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NANOPLASTICS.

A Systematic Risk Analysis for Human Health, Ecosystems, and the Environment



HUMAN HEALTH

ECOSYSTEMS

ENVIRONMENT

ELECTROSTATIC INTERACTIONS

2026

AUTHORS

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The ALLATRA Global Research Center (ALLATRA GRC) carries out interdisciplinary research on global environmental, climate, and societal processes, with a focus on systematic analysis, risk assessment, and development of evidence-based recommendations for policy, strategic planning, and practical implementation of relevant measures.

The Center's research activities are undertaken by an international network of scientists and experts from diverse disciplines. The Center provides a platform for scientific collaboration and international dialogue on issues affecting human societies and the natural environment.

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TABLE OF CONTENTS

Part 1. Introduction. Plastic Is Everywhere 21

Part 2. Properties of Micro- and Nanoplastics as a Factor of Their Activity 24
- Mechanisms of Plastic Degradation 25
- Increase in Specific Surface Area and the Role of Surface Processes 27
- Formation of Surface Charge and Behavior in Aquatic Environment 28
- Electrostatic Mechanisms of Microplastics' Interaction with Soil Systems 31
- Linking Electrokinetic Properties to Biological Interactions 32
- Reaccumulation of Electrostatic Charge by Micro- and Nanoplastics 36

Part 3. The Impact of Micro- and Nanoplastics on the Universal Mechanisms of Natural Systems 38
- Forests 39
- Animals 47
- Insects 51
- Birds 56
- Marine Life 60

Part 4. Micro- and Nanoplastics as a Factor of Cellular Dysfunction: Mechanisms and Biological Consequences 62
- Electrophysiological Foundation of the Human Body and Its Vulnerability to Micro- and Nanoplastics 63
- Mechanisms of MNP Internalization and the Role of Surface Charge 65
- Disruption of Electrical Stability and Neuronal Membrane Potential 68
- Impact on Ion Channels 71
- Disruption of Neurotransmitter Transmission 72
- Induction of Oxidative Stress and Inflammation 74
- Mitochondrial Dysfunction and Energy Deficit 76
- Structural Protein Degradation Induced by Nanoplastics 82

Part 5. Micro- and Nanoplastics as a Potential Neurobiological Risk Factor 90
- Bioaccumulation and Penetration into the CNS 91
- Neuroinflammation and Neurodegeneration 96
- Potential Clinical and Epidemiological Implications 100

Part 6. Systemic Impact of Micro- and Nanoplastics on the Human Body Through the Destabilization of the Gut–Brain Axis and Disruption of Intestinal Barrier Functions 102
Impairment of Physical Integrity and Selective Permeability of the Intestinal Barrier .. 103
Role of Surface Charge and Formation of the "Protein Corona" 104
Immune Activation and the Chronicity of Inflammation 105
Clinical Evidence from Inflammatory Bowel Disease 106
Destabilization of Microbiota as a Key Regulator 106
Systemic Effects of Dysbiosis: Impact on the Central Nervous System 108

Part 7. Micro- and Nanoplastics as a Factor in Systemic Destabilization of Immune Defense and Amplification of Infectious Risks 110
Immunotoxicity of Micro- and Nanoplastics 111
MNPs and Damage to Lymphoid Organs 113
Electrostatic Properties of MNPs and Increased Pathogenicity of Microorganisms .. 115
The Role of Microplastics in the Spread of Viral and Bacterial Pathogens 116
Protein Corona and Immune Recognition of MNPs 117
Microplastics as a Factor of Increasing Antibiotic Resistance 118
Sorption properties of microplastics and accumulation of pollutants 119
External electron transfer as a mechanism of bacterial metabolic activation 119

Part 8. Inhalation Exposure to Micro- and Nanoplastics: Risks to the Respiratory System 121
Scale of Impact and Physical Penetration Capability 123
Accumulation in Tissues and the Mechanism of Cell Damage 123
Cellular and Intracellular Toxicity 126
Disruption of Local Homeostasis and Respiratory Tract Dysbiosis 128
Experimental and Clinical Evidence Base 129
Carcinogenic Potential of Micro- and Nanoplastics 132

Part 9. The Systemic Impact of Micro- and Nanoplastics on the Musculoskeletal System: From Cellular Toxicity to the Global Disease Burden 134
Identification of MNPs in Human Bone and Skeletal Muscle 136
Mechanisms of Spinal Degeneration 138
A New Paradigm for Bone Loss: Osteocytic Osteolysis 139
The Impact of MNPs on Bone Metabolism and Structure 139
The Specifics of Nanotoxicity 141
Skeletal Muscle Degradation and Mitochondrial Dysfunction 142
Clinical Picture: Osteoarthritis and Epidemiological Evidence 142
Degenerative Bone Diseases: Challenges for Society and Healthcare 143

Part 10. The Impact of Micro- and Nanoplastics on Human Reproductive Health 145
Damage to Male Reproductive Cells and Impairment of Spermatogenesis 146
Hormonal Imbalance and Its Consequences 151
Neurogenic Mechanisms of Erectile Dysfunction 151
Epidemiology of Erectile Dysfunction: Current Trends 152
Impact of MNPs on the Female Reproductive System 152

Part 11. The Impact of Micro- and Nanoplastics on Prenatal and Postnatal Development 156
Penetration of Micro- and Nanoplastics Through the Placental Barrier 157
Mechanisms of Placental Toxicity: Cellular and Molecular Effects 159
Preterm Birth and the Global Burden of Perinatal Pathology 163
Impact on Embryonic Tissues and Organogenesis 163
Rising Prevalence of Congenital Anomalies 170
Postnatal Exposure to MNPs 175
Endocrine, Immune, and Epigenetic Effects 176
Vulnerability of the Developing Brain 177
Sudden Infant Death Syndrome 178

Part 12. Micro- and Nanoplastics as a Potential Risk Factor for Cardiovascular Diseases 182
Mitochondrial Dysfunction as a Mechanism of Cardiac Damage 183
Microplastics in Atherosclerotic Plaques and the Risk of Vascular Events 185
Disruption of Vascular Homeostasis and Endothelial Dysfunction 185
Impact on Blood Cells and Thrombogenesis Processes 186
Micro- and Nanoplastics as a Potential Risk Factor for Sudden Cardiac Death 189

Part 13. Micro- and Nanoplastics as a Risk Factor for Oncogenesis: From Cellular Mechanisms to Global Epidemiology 195
Global Dynamics and Prognosis of Oncological Morbidity 196
Epidemiological Shift: The Phenomenon of Early-Onset Oncogenesis 197
Cellular Mechanisms: Oxidative Stress and DNA Damage 200
Mitochondrial Dysfunction as a Mechanism of the Oncogenic Impact of Nanoplastics 204
Micro- and Nanoplastics as a Potential Factor in Tumor Metastasis 206
The Problem of Resistance to Therapy 207

Part 14. Micro- and Nanoplastics as a Factor in Accelerated Biological Aging 209
Population Manifestations of Accelerated Biological Aging 210
Biological Aging: What Occurs at the Cellular Level 211
Activation of Key Biological Aging Mechanisms by Micro- and Nanoplastics 213

Part 15. The Influence of MNPs on Climate Processes 214
The Role of Oceans in the Climate System 215
The Impact of Micro- and Nanoplastics on Oceanic Processes 215
Mechanisms of MNPs on the Aqueous Environment 218
The Mediterranean Sea as a Model of MNP Concentration Influence on the Marine Thermal Regime 220
Global Distribution of MNPs 221
The Influence of Micro- and Nanoplastics on Ocean Acidification 223
Influence of Micro- and Nanoplastics on the Atmosphere 225
Energy Balance and the Climate Crisis 226

Part 16. The Illusory Nature of Existing Technological Solutions to Global Plastic Pollution 228
Limitations of Mechanical Cleanup: Scale and Time 229
Ecological Risks of Ecosystem Intervention 230
Risks of Traditional Landfilling and Waste Migration 231
The Disposal Problem and Pollution Transfer to the Atmosphere 231
Thermal Degradation as a Source of Micropollution 234
The Recycling Paradox 235
Micro- and Nanoplastics: Irreversibility of Pollution and Biological Integration 237

Part 17. From the Illusion of "Purification" to Strategies for MNP Neutralization 240

Part 18. Discussion on the Risks of Micro- and Nanoplastics to Living Organisms 245

Part 19. Research Perspective: Can Subsurface Electrical Organization of Nanoplastics Contribute to Their Behavior? 253

Part 20. Conclusion 260

References 262

ABBREVIATIONS AND ACRONYMS

Polymer Types and Modifications

Abbreviation	Full term
HDPE	high-density polyethylene
LDPE	low-density polyethylene
PAN	polyacrylonitrile
PBAT	polybutylene adipate-co-terephthalate
PC	polycarbonate
PE	polyethylene
PET	polyethylene terephthalate
PP	polypropylene
PPO	polyphenylene oxide
PS	polystyrene
PS-COOH	carboxylated polystyrene
PS-NH2	aminated polystyrene
PU (PUR)	polyurethane
PVC	polyvinyl chloride

Plastic Particle Terminology

Abbreviation	Full term
MNP (MNPs, M/NP, M/NPs, MPs/NPs)	micro- and nanoplastics
MP (MPs)	microplastics
NP (NPs)	nanoplastics
PS-MP (PS-MPs)	polystyrene microplastics
PS-NP (PS-NPs)	polystyrene nanoparticles

Environmental Contaminants

Abbreviation	Full term
EPFR	environmentally persistent free radicals
MPW	mixed plastic waste
OPs	organic pollutants
PAH	polycyclic aromatic hydrocarbons
PCB	polychlorinated biphenyls
PCDD	polychlorinated dibenzo-p-dioxins
PCDD/F	polychlorinated dibenzo-p-dioxins/furans
PCDF	polychlorinated dibenzofurans
PFAS	per- and polyfluoroalkyl substances
PM	particulate matter
POP	persistent organic pollutants

Cell Lines and Experimental Biological Models

Abbreviation	Full term
A549 cells	human lung adenocarcinoma cell line A549
bEnd.3	mouse brain endothelial cell line bEnd.3
HEK	human embryonic kidney
HNEC	human nasal epithelial cells
SH-SY5Y	human neuroblastoma cell line SH-SY5Y

Chemical Compounds, Materials, Biomolecules, Genes, Proteins, Receptors, and Reagents

Abbreviation	Full term
5-HT	5-hydroxytryptamine (serotonin)
5-HT3	5-hydroxytryptamine receptor 3
AMPAR	α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor
ATP	adenosine triphosphate
BCL2L15	BCL2 like 15
BDNF	brain-derived neurotrophic factor
BMP	bone morphogenetic protein
CI	Complex I

CI-NDUFB8	complex I, NADH:ubiquinone oxidoreductase subunit B8
CII-SDHB	complex II, succinate dehydrogenase subunit B
CIII-UQCRC2	complex III, ubiquinol-cytochrome c reductase core protein 2
CINC-1	cytokine-induced neutrophil chemoattractant-1
CINC-2	cytokine-induced neutrophil chemoattractant-2
CIV-MTCO2	complex IV, mitochondrially encoded cytochrome c oxidase II
CL	clathrin-coated vesicles
CPZ	chlorpromazine
CV-ATP5A	complex V, ATP synthase F1 subunit alpha
DAMP	damage-associated molecular patterns
DNA	deoxyribonucleic acid
DOPC	1,2-dioleoyl-sn-glycero-3-phosphocholine
EEA1	early endosome antigen-1
EPS	extracellular polymeric substances
F10T	flavin-tryptophan dyad
FAD	flavin adenine dinucleotide
FAs	fulvic acids
FIB	fibrinogen
FITC	fluorescein isothiocyanate
FM 4-64	N-(3-triethylammoniumpropyl)-4-(6-(4-(diethylamino) phenyl) hexatrienyl) pyridinium dibromide
FOXD3	forkhead box D3
GABA	gamma-aminobutyric acid
GPLD1	glycosylphosphatidylinositol-specific phospholipase D1
GPX3	glutathione peroxidase 3
HAs	humic acids
HOCl	hypochlorous acid
IFN-γ	interferon gamma
IL-17	interleukin-17
IL-19	interleukin-19
IL-1β	interleukin-1 beta
IL-6	interleukin-6
IL-8	interleukin-8
LAMP2	lysosome-associated membrane protein 2
LDH	lactate dehydrogenase
MPO	myeloperoxidase
mROS	mitochondrial reactive oxygen species
mtDNA	mitochondrial DNA
mtDNAcn	mitochondrial DNA content
NAC	non-amyloid component

NLRP3	NOD-like receptor family, pyrin domain containing 3
NMs	nanomaterials
NOx	nitrogen oxides
p53	tumor protein p53
PGA5	pepsinogen A5
pS129	phosphorylated at serine 129
RCS	reactive chlorine species
ROS	reactive oxygen species
SNAI2	Snail family transcriptional repressor 2
TGF-□	transforming growth factor beta
Th2	T helper 2 cells
TLR4	Toll-like receptor 4
TNF-□	tumor necrosis factor alpha
WNT1	Wnt family member 1
ZO	zonula occludens

Signaling Pathways and Cellular Processes

Abbreviation	Full term
AMPK	AMP-activated protein kinase
EET	extracellular electron transfer
EMT	epithelial–mesenchymal transition
ERK1/2	extracellular signal-regulated kinases 1/2
ETC	electron transport chain
FGP	fibroblast growth factor pathway
HR	homologous recombination
JNK	c-Jun N-terminal kinase
MAPK	mitogen-activated protein kinase
NETosis	neutrophil extracellular trap formation
NF-κB	nuclear factor kappa B
Nrf2	nuclear factor erythroid 2–related factor 2
OXPHOS	oxidative phosphorylation
p38	p38 mitogen-activated protein kinase
PI3K/Akt	phosphoinositide 3-kinase / protein kinase B (Akt)
PKA	protein kinase A
SASP	senescence-associated secretory phenotype
STAT	signal transducer and activator of transcription
TCA	tricarboxylic acid
Wnt/β	Wnt/β-catenin signaling pathway

Cells, Tissues, Anatomy, and Physiological Systems

Abbreviation	Full term
ANS	autonomic nervous system
BBB	blood-brain barrier
CNS	central nervous system
CTB	cytotrophoblast
ENS	enteric nervous system
ER	endoplasmic reticulum
GALT	gut-associated lymphoid tissue
GI	gastrointestinal
GPe	globus pallidus externa
HPA	hypothalamic-pituitary-adrenal
MGB	microbiota-gut-brain
MMP	mitochondrial membrane potential
NK cell	natural killer cell
PLT	platelet
RBC (RBCs)	red blood cells
SNpc	substantia nigra pars compacta
STB	syncytiotrophoblast
TJ	tight junction

Methods, Assays, Stains, Microscopy, and Analytical Techniques

Abbreviation	Full term
AB-PAS	Alcian blue–periodic acid–Schiff stain
CCK-8	Cell Counting Kit-8
CT	computed tomography
DAPI	4',6-diamidino-2-phenylindole
DHE	dihydroethidium
EDX	energy-dispersive X-ray spectroscopy
fs-ESHS	femtosecond elastic second harmonic scattering
H&E	hematoxylin and eosin
iDISCO	immunolabeling-enabled three-dimensional imaging of solvent-cleared organs
LD-IR	laser direct infrared spectroscopy
MTT	thiazolyl blue tetrazolium (3-(4,5-dimethylthiazol-2-yl)-2,5-di-phenyltetrazolium) bromide

NMR	nuclear magnetic resonance
photo-CIDNP	photo-chemically induced dynamic nuclear polarization
Py-GC/MS	pyrolysis gas chromatography-mass spectrometry
SD-FGD	semi-dry flue gas deacidification
SEM	scanning electron microscopy
TEER	trans-endothelial electrical resistance
TEM	transmission electron microscopy
TPFM	two-photon fluorescence microscopy
TUNEL	terminal deoxynucleotidyl transferase dUTP nick end labeling
UV	ultraviolet
YG	nonfunctionalized fluoresbrite yellow green
µFTIR	micro-Fourier transform infrared

Diseases and Health Outcomes

Abbreviation	Full term
ALS	amyotrophic lateral sclerosis
AMR	antimicrobial resistance
ASD	autism spectrum disorder
BD	birth defect
CHD	congenital heart disease
COPD	chronic obstructive pulmonary disease
CVD (CVDs)	cardiovascular diseases
ED	erectile dysfunction
IBD	inflammatory bowel disease
MS	multiple sclerosis
NCD	non-communicable diseases
OA	osteoarthritis
SCD	sudden cardiac death
SIDS	sudden infant death syndrome
TOPFA	terminations of pregnancy for fetal anomaly

Organizations, Programs, and Datasets

Abbreviation	Full term
ADDM	Autism and Developmental Disabilities Monitoring
CDC	Centers for Disease Control and Prevention
EU	European Union
EUROCAT	European Surveillance of Congenital Anomalies
IARC	International Agency for Research on Cancer
NOAA	National Oceanic and Atmospheric Administration
OECD	Organisation for Economic Co-operation and Development
UN SDG	United Nations Sustainable Development Goals
US	United States
UTS	University of Technology Sydney
WHO	World Health Organization
WWF	World Wildlife Fund

Statistics, Metrics, and Modelling Terms

Abbreviation	Full term
ANOVA	analysis of variance
CIs	confidence intervals
DALY	disability-adjusted life years
GAM	generalized additive models
GFN2-xTB	geometry, frequency, noncovalent, extended tight-binding
MD	(version 2)
OHC	molecular dynamics
SD	ocean heat content
	standard deviation

EXECUTIVE SUMMARY

Plastic has become a cornerstone of the modern economy. However, its high resistance to degradation leads to environmental accumulation and gradual fragmentation into micro- and nanoparticles. According to estimates, over 9 billion tons of plastic have been produced since the mid-20th century, with more than 400 million tons of waste generated annually, yet less than 10% of it is recycled. A significant share ends up in the oceans, soil, and air.

A key change is observed as plastic transitions to the micro- and especially the nanoscale: the material begins to behave as a colloidal and surface-active particle rather than a relatively inert macro fragment. Its specific surface area, adsorption capacity, and the role of its electrostatic properties increase sharply. This means that even with the same mass of pollution, its biological and ecological activity may increase substantially.

Simultaneously, the practical problem of risk management becomes more acute: the smallest fractions are the most difficult to detect and measure comparably, while unified standards for sampling, sample preparation, and measurement — especially for nanoplastics — are still being established. Consequently, management decisions in the fields of regulation, investment, and control are often made under conditions of limited measurability, despite existing data indicating the widespread nature of the impact.

Key Findings of the Report

The impact of micro- and nanoplastics is pervasive, chronic, and multi-channel. Micro- and nanoplastics (MNPs) are found in the air, water, soil, and food products. For humans, this means continuous exposure through inhalation, food, and water intake, as well as through specific sources (including medical infusion systems).

At the micro- and nanoscale, the surface and electrical properties of plastic particles are critical. Particles acquire and modify their charge in different environments (triboelectric in the air; charge/ζ-potential and electrical double layer in water; and as the "effective charge" of a "particle–biomolecular corona" complex in biological media).

This increases the likelihood of:

- Adhesion to cell membranes and intracellular uptake (endocytosis and other pathways);
- Protein binding with the formation of a bio/eco-corona, which defines the particles' "biological identity";
- The transport of adsorbed pollutants and microorganisms (the "Trojan horse" effect).

The report systematizes the convergent mechanisms that arise from exposure of living organisms to MNPs, which recur across different models:

- Oxidative stress and inflammatory responses;
- Mitochondrial dysfunction (decreased membrane potential, reduced ATP synthesis, increased reactive oxygen species (ROS) formation, and mitochondrial DNA damage);

- Disruption of cellular electrophysiology (impact on membrane potential, ion channels, and synaptic transmission);
- Conformational changes in proteins and the risk of proteotoxic stress;
- Disruption of barrier functions (intestinal epithelium, blood-brain barrier, placental barrier) as a potential pathway for systemic effects.

There is growing evidence of the actual accumulation of micro- and nanoplastics in human tissues, including highly sensitive organs. The report presents data on the detection of particles in lung tissue, the placenta, bone marrow, and other tissues. Some individual studies have documented the presence of fragments <200 nm in brain tissue, as well as higher levels of MNPs in the brain compared to the liver and kidneys. These observations are not equivalent to proven clinical harm; however, they increase the significance of the topic for systemic risk assessment and indicate the need for a detailed and comprehensive study of the impact of MNPs on human health.

For a number of outcomes, clinically significant associations have already been identified and require further verification of a causal link. The report notes, in particular, data on the presence of micro- and nanoplastics in atherosclerotic plaques and a statistical correlation with an increased risk of major cardiovascular events. Such results should be considered a serious basis for in-depth research and monitoring.

Ecosystem-level consequences may manifest through "universal mechanisms" of communication and resilience. The report discusses how electrostatically active plastic particles are potentially capable of disrupting subtle signaling processes (including bioelectric ones) in plants, pollinators, and marine organisms, as well as influencing soil interactions and biocenoses (biological communities). Even with a cautious interpretation, this indicates a risk of reduced ecosystem resilience under long-term exposure.

Risks

Main groups of potential risks:

- **Regulatory and Compliance Risks**: Likely tightening of requirements for monitoring micro- and nanoparticles, emissions/effluents, as well as materials that come into contact with food and medical solutions.
- **Operational Risks**: Potential indoor air contamination; emissions of micro-fractions during recycling and incineration stages; passage through wastewater treatment plants; and the secondary formation of nanofractions due to mechanical stress.
- **Reputational Risks**: Growing public sensitivity toward microparticles and "invisible" pollution.
- **Long-term Socio-economic Risks**: Potential contribution to the burden of chronic diseases and the degradation of ecosystem services (pollination, bioproductivity, water and soil quality).

Proposed Directions for Further Action and Research

The report notes that polymers remain a vital foundation for technological development, including in medicine, transportation, energy, electronics, and construction. Consequently, a realistic strategy is not to “ban the material,” but to manage the risks associated with micro- and nanofractions and to reduce key scientific uncertainties.

Priority 1: Unified Monitoring Standards and Comparable Metrics

- Introduce and harmonize standards for sampling, sample preparation, polymer identification, and quantification.
- Specifically establish a control boundary for particles <10 μm as a practical threshold. The report emphasizes that at these sizes, the likelihood of cellular internalization and systemic effects increases.
- Approve minimum reporting requirements: size distribution, shape, polymer type, indicators of surface "aging" (weathering), and charge/ζ-potential parameters (where applicable).

Priority 2: Targeted Scientific Program on the "Charge Architecture" of Nanoplastics

The report highlights a critical hypothesis: ζ-potential may not fully capture the electrical nature of nanoplastics, and their behavior may depend on internal (subsurface) charge organization (e.g., "soft particles," volume charges, charge traps, electret-like states). Without understanding this architecture, it is difficult to:

- Predict particle migration in water and air, as well as their aggregation;
- Assess interactions with proteins (bio-corona formation), membranes, and biofilms;
- Design methods for the physical or biophysical "deactivation" of particles.

Recommended Format: An interdisciplinary "mission-oriented R&D" program combining materials science, colloid chemistry, toxicology, biophysics, and clinical sciences.

The fact of exposure and biological interactions is well-supported, whereas precise quantitative risk estimates for humans and causal relationships for a number of clinical outcomes remain to be clarified.

Micro- and nanoplastics should be regarded as a new class of anthropogenic particles with potentially significant biophysical activity, where size, surface, and electrical properties play a decisive role.

with potentially significant biophysical activity, where size, surface, and electrical properties play a decisive role.

A rational course of action is to standardize monitoring (with <10 μm as a critical range), invest in researching hidden charge architecture, and develop biophysical strategies for neutralizing electrostatic activity, while simultaneously reducing microfraction emissions at key points in the product life cycle.

PART 1.

INTRODUCTION.
PLASTIC IS EVERYWHERE

Plastics have become an integral part of modern civilization; however, their exceptional resistance to degradation has provoked an unprecedented global ecological crisis.[1] Since the mid-20th century, humanity has produced over 9 billion tons of plastic. Annually, more than 400 million tons of plastic waste are generated worldwide. According to global estimates, less than 10% of manufactured plastic is recycled, whereas over 90% accumulates in the environment, is disposed of via landfills and incineration, or enters uncontrolled waste streams.[2]

Consequently, a significant portion of plastic materials eventually ends up in natural ecosystems. To date, the World Ocean remains one of the largest sinks for plastic waste: researchers estimate that it already harbors approximately 200 million tons of plastic debris, and this mass continues to increase.[2]

Upon entering the environment, plastic does not disappear but undergoes gradual fragmentation driven by solar radiation, mechanical breakdown, and chemical processes.[1,3] This results in the formation of a broad spectrum of fine particles. In this report microplastics are defined as particles ranging in size from 1 µm to 5 mm, and nanoplastics as particles smaller than 1 µm; however, it is important to note that these boundaries are not yet fully standardized in the scientific literature.[3,4]

The delineation of these classes holds fundamental biophysical significance, as the transition to the nanoscale radically alters the behavior of materials. While a single plastic fragment with a diameter of 1 mm is equivalent in mass to a trillion nanoparticles of 100 nm in size, the cumulative surface area of these nanoparticles is nearly 10,000 times greater.[4] As fragmentation progresses to micro- and nanoscales, the behavior of the particles becomes increasingly governed by the laws of colloid chemistry and surface phenomena. The colossal increase in specific surface area, combined with inherent hydrophobicity, transforms a relatively inert polymer into a highly active physicochemical entity.[5] In aqueous media, nanoplastics are prone to aggregation[4] and possess the capacity to adsorb and concentrate a diverse array of hydrophobic pollutants.[5] The toxicological potential of the particles is further amplified by chemical additives (plasticizers, flame retardants, dyes) that leach from the polymer matrix during its degradation.[6]

The investigation of these particles is additionally complicated by the fact that the finest fractions are the most difficult to detect and reliably quantify. Analytical methods are evolving rapidly; however, unified standards for sampling, sample preparation, identification, and measurement of microplastics, and particularly nanoplastics, are still in the formative stages. Consequently, the results of different studies are not always fully comparable, and the actual concentrations of nanoplastics in a number of objects may be underestimated.[4]

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[5] *Ašmonaitė, G., Tivefälth, M., Westberg, E., et al. Microplastics as a Vector for Exposure to Hydrophobic Organic Chemicals in Fish: A Comparison of Two Polymers and Silica Particles Spiked With Three Model Compounds. Front. Environ. Sci. 8, 87 (2020). https://doi.org/10.3389/fenvs.2020.00087*

[6] *Campanale, C., Massarelli, C., Savino, I., et al. A Detailed Review Study on Potential Effects of Microplastics and Additives of Concern on Human Health. IJERPH 17, 1212 (2020). https://doi.org/10.3390/ijerph17041212*

Nevertheless, it is already evident that micro- and nanoplastics have long transcended the confines of landfills and oceanic waters. They are detected in the air, soils, agricultural produce, drinking water, and food products.[1,3,7] For humans, this entails continuous exposure to such particles, including through food, water, and inhaled air. Concurrently, accumulating evidence indicates that small-sized plastic particles are capable of interacting with epithelial barriers, cellular membranes, the immune system, and intracellular structures.[4,8] Due to their ultra-small size, nanoplastics can bypass innate defense systems and cross the most complex biological barriers: the intestinal, blood-brain, and placental barriers.[4,7] The ability of polymers to migrate through food chains leads to bioaccumulation and biomagnification, which are already inducing developmental and reproductive disruptions in aquatic organisms.[1] The presence of fragments smaller than 200 nm in human brain tissues corroborates the invasiveness of these pollutants.[4]

The intracellular presence of nanoplastics can trigger oxidative stress, elicit an inflammatory response, and induce dysfunction of vital organelles, primarily mitochondria, initiating apoptosis.[4,8]

Furthermore, possessing a high surface charge density, plastic is capable of altering the spatial conformation of proteins, disrupting the electrophysiological signaling of membranes, and acting as a “Trojan horse” by transporting heavy metals, antibiotics, and pathogenic microorganisms on its surface into biological systems.[8]

In this context, it is crucial to distinguish established facts from open questions: the existence of exposure and biological interactions is already well-documented, whereas the precise degree of risk to humans continues to be explored.

This is precisely why the key question is not merely how much plastic has accumulated in the environment, but also what happens to its properties upon transitioning to micro- and nanoscales. At this scale, plastic ceases to be merely a passive fragment of waste and begins to behave as an active dispersed entity, capable of interacting with living and non-living systems in novel ways. This renders the issue of micro- and nanoplastics simultaneously an ecological, toxicological, and biophysical problem.

This report examines how the physicochemical properties of microplastics, and particularly nanoplastics, dictate their behavior in the environment and in biological systems, encompassing surface interactions, cellular responses, and effects at the tissue and systemic levels, including potential implications for human health. Special attention is dedicated to those mechanisms already corroborated by modern data, as well as to questions that currently remain subjects of scientific debate. Such an approach allows micro- and nanoplastics to be viewed not just as a symbol of global pollution, but as a distinct class of anthropogenic particles that demands rigorous, interdisciplinary scientific analysis.

[7] *Hu, T., Zhang, C., Zhu, Y., et al. Abundance of microplastics and nanoplastics in urban atmosphere. Science Advances 12, eadz7779 (2026).* https://doi.org/10.1126/sciadv.adz7779

[8] *Yee, M. S.-L., Hii, L.-W., Looi, C. K., et al. Impact of Microplastics and Nanoplastics on Human Health. Nanomaterials 11, 496 (2021).* https://doi.org/10.3390/nano11020496

PART 2.

PROPERTIES OF **MICRO-** AND **NANOPLASTICS** AS A FACTOR OF THEIR ACTIVITY

The environmental threat posed by plastics extends beyond their chemical stability and accumulation in the environment.

As plastic breaks down into micro- and nanoparticles, its behavior at this scale becomes governed by the principles of colloid chemistry. Within this size range, the surface and electrostatic properties of the particles play a critical role, fundamentally altering how they interact with water, living organisms, and other environmental components. Combined with their high resistance to degradation, this transforms micro- and nanoplastic particles into long-lasting, physicochemically active entities. They readily engage in interfacial processes like adsorbing substances, binding ions, and interacting with biological surfaces.

Plastic is a human-made synthetic material with no natural counterparts. The word "plastic" comes from the Greek *plastikos*, meaning "capable of being moulded or shaped." This material has been developed through the chemical processing of monomers, such as ethylene and propylene. Unlike natural polymers like cellulose, chitin, or natural proteins, synthetic plastics have no evolutionary history in the biosphere and did not exist in nature until the 20th century.[9]

MECHANISMS OF PLASTIC DEGRADATION

Long hydrocarbon chains containing strong covalent C–C and C–H bonds make up most synthetic plastics (Fig. 2.1).

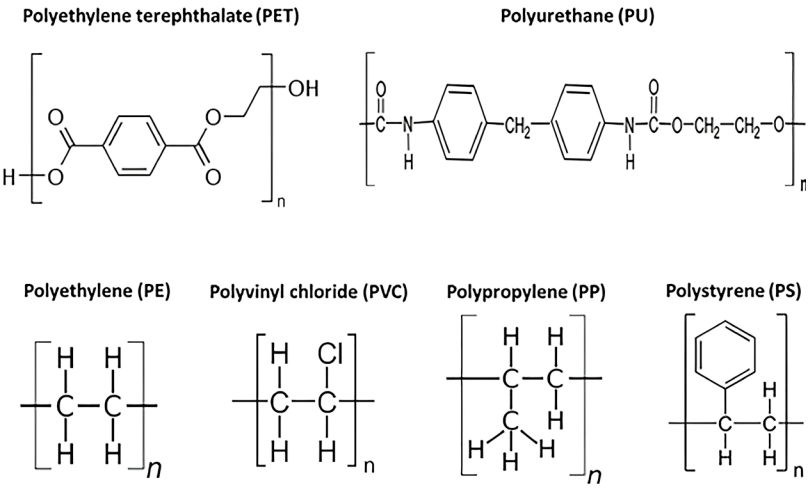


Figure 2.1.

Structures of major commercially available synthetic polymers.

Source: Mohanan, N., Montazer, Z., Sharma, P. K., et al. Microbial and Enzymatic Degradation of Synthetic Plastics. Front. Microbiol. 11, 580709 (2020).

https://doi.org/10.3389/fmicb.2020.580709

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[9] *PlasticsEurope. History of Plastics. https://plasticseurope.org/plastics-explained/history-of-plastics/ (Accessed 20 March 2026).*

These bonds are highly stable and resistant to water, hydrolysis under typical environmental conditions and most natural chemical processes.[10]

Additionally, the biosphere lacks effective biological mechanisms to break these bonds down. Living organisms decompose substances using enzymes that evolved over time to interact with natural compounds. Since synthetic polymers lack a long evolutionary history in nature, widespread enzyme systems capable of effectively breaking down their structure do not exist.[11]

Polymer	Density (g/L)	Crystallinity (%)	Life span (years)
PET	1.35	0–50	450
LDPE	0.91–0.93	50	10–600
HDPE	0.94–0.97	70	>600
PS	1.03–1.09	0	50–80
PP	0.90–0.91	50	10–600
PVC	1.35–1.45	0	50–150

Figure 2.2.

Selected properties of major synthetic thermoplastic polymers. PET, Polyethylene terephthalate; LDPE, Low density polyethylene; HDPE, High density polyethylene; PS, Polystyrene; PP, Polypropylene; PVC, Polyvinyl chloride.

Source: Mohanan, N., Montazer, Z., Sharma, P. K., et al. Microbial and Enzymatic Degradation of Synthetic Plastics. Front. Microbiol. 11, 580709 (2020).

https://doi.org/10.3389/fmicb.2020.580709

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High molecular weight and significant polymer chain length also hinder biodegradation (Fig. 2.2). Enzymes struggle to interact with such large hydrophobic molecules, and even during oxidation, the chain scission proceeds slowly. In the early stages, the material primarily fragments, while complete decomposition into mineral components almost never occurs.

In nature, plastics do not disappear; instead, they gradually age under ultraviolet radiation, oxidize, lose mechanical strength, crumble, and break into increasingly smaller particles.[11] Ultimately, macroplastics transform into micro- and nanoplastics (MNPs). Rather than eliminating pollution, this process shifts them into a more dispersed and bioavailable form.

[10] *Bacha, A.-U.-R., Nabi, I., Zaheer, M., et al. Biodegradation of macro- and micro-plastics in environment: A review on mechanism, toxicity, and future perspectives. Science of The Total Environment 858, 160108 (2023). https://doi.org/10.1016/j.scitotenv.2022.160108*

[11] *Mohanan, N., Montazer, Z., Sharma, P. K., et al. Microbial and Enzymatic Degradation of Synthetic Plastics. Front. Microbiol. 11, 580709 (2020). https://doi.org/10.3389/fmicb.2020.580709*

INCREASE IN SPECIFIC SURFACE AREA AND THE ROLE OF SURFACE PROCESSES

As particle size decreases, the surface area-to-volume ratio spikes, that leading to surface processes to take on a dominant role.[12]

Plastic fragmentation radically changes both its quantitative and surface characteristics. If a particle roughly 5 mm in diameter breaks down into particles approximately 1 μm in size, it can produce about 10^{11}–10^{12} smaller particles. At the same time, the total surface area increases by several orders of magnitude and can exceed the original area by thousands of times.[13]

This means that as particles become smaller, the proportion of functional groups available for interaction increases, as does the number of adsorption sites.[14] This promotes the binding of proteins, metal ions, lipids, and other molecules from the environment (Fig. 2.3, 2.4). The surface of nanoplastics thus becomes a zone of intense intermolecular interaction and a site for the formation of adsorption complexes.[15]

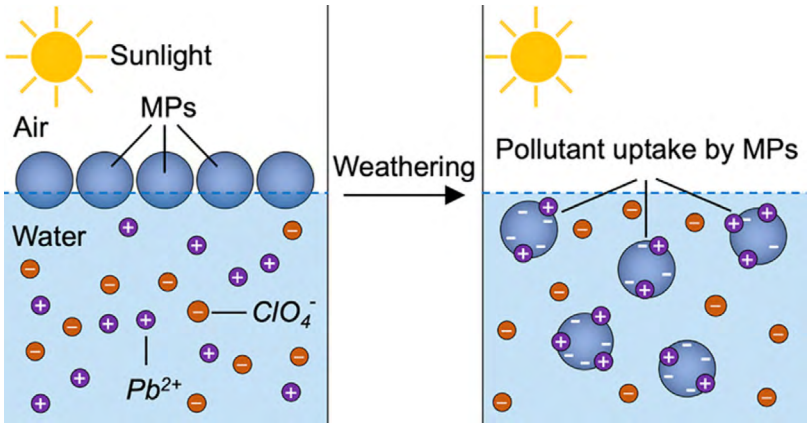


Figure 2.3.

Within 10 days of accelerated weathering, microplastics become dramatically more dispersible in the water column and can more than double the surface uptake of common chemical pollutants, such as malachite green and lead ions.

Source: Al Harraq, A., Brahana, P. J., Arcemont, O., et al. Effects of Weathering on Microplastic Dispersibility and Pollutant Uptake Capacity. ACS Environ. Au 2, 549–555 (2022). https://doi.org/10.1021acsenvironau.2c00036

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As particle size decreases, surface and electrostatic characteristics begin to define their behavior, strengthening colloidal and biological interactions.

[12] *Rahman, A. M. N. A. A., Rusli, A., Abdullah, M. K., et al. A review of microplastic surface interactions in water and potential capturing methods. Water Science and Engineering 17, 361–370 (2024). https://doi.org/10.1016/j.wse.2023.11.008*

[13] *The estimate is based on the idealized case of spherical particles and total mass conservation during fragmentation.*

[14] *Yang, H., Chen, Z., Kong, L., et al. A Review of Eco-Corona Formation on Micro/Nanoplastics and Its Effects on Stability, Bioavailability, and Toxicity. Water 17, 1124 (2025). https://doi.org/10.3390/w17081124*

[15] *Lundqvist, M., Stigler, J., Elia, G., et al. Nanoparticle size and surface properties determine the protein corona with possible implications for biological impacts. Proc. Natl. Acad. Sci. U.S.A. 105, 14265–14270 (2008). https://doi.org/10.1073/pnas.0805135105*

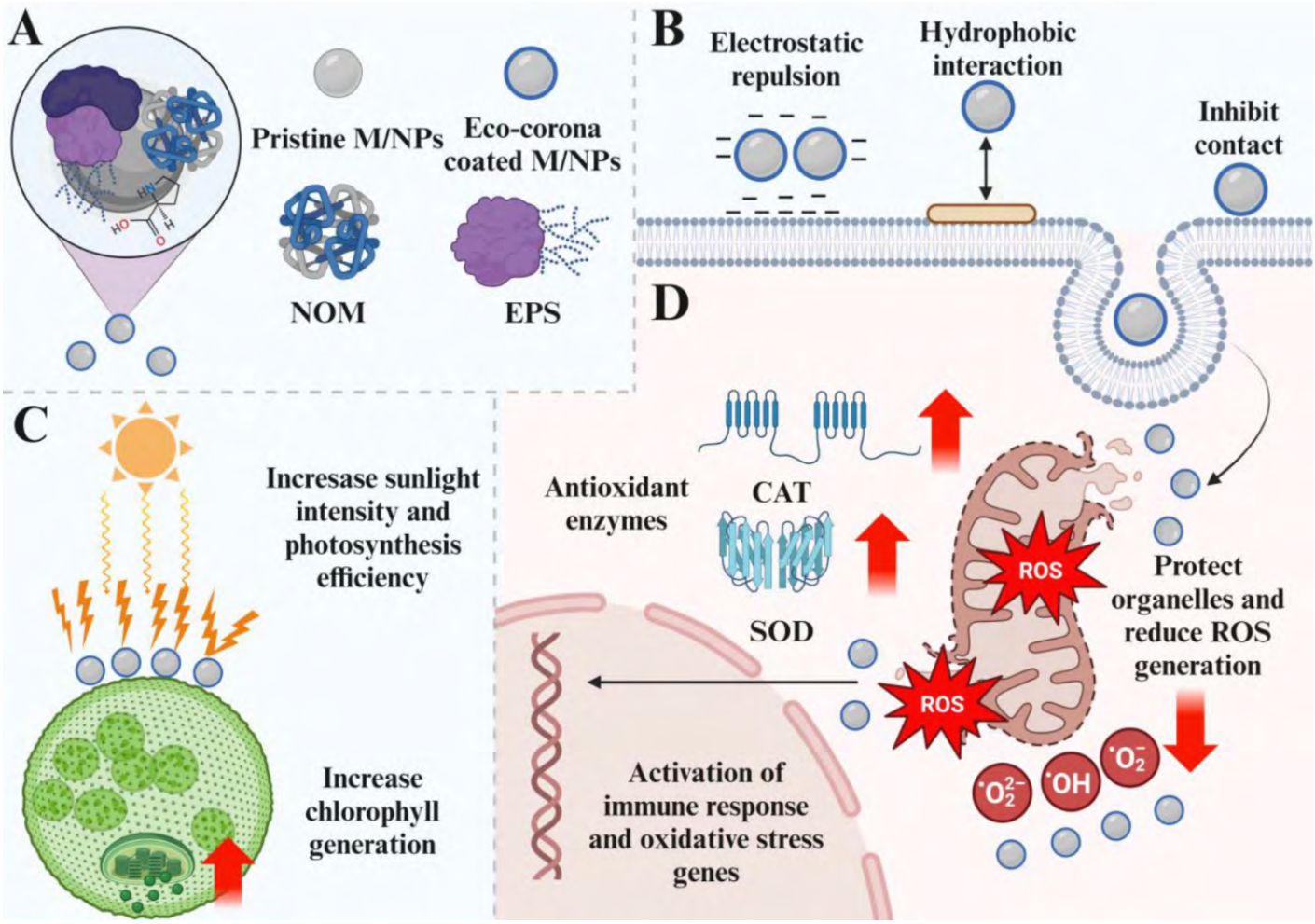


Figure 2.4.

Formation of eco-corona and the toxicity of M/NPs coated with eco-corona to aquatic organisms. (A) Formation of eco-corona; (B) Effects the adhesion of M/NPs to the cell surface; (C) Protecting algae from damage by M/NPs; (D) Effects on M/NPs-induced cytotoxicity and gene expression.

Source: Yang, H., Chen, Z., Kong, L., et al. A Review of Eco-Corona Formation on Micro/Nanoplastics and Its Effects on Stability, Bioavailability, and Toxicity. Water 17, 1124 (2025). https://doi.org/10.3390/w17081124
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FORMATION OF SURFACE CHARGE AND BEHAVIOR IN AQUATIC ENVIRONMENTS

Large plastic fragments in aquatic environments are less influenced by surface phenomena, as their behavior is governed primarily by the bulk properties of the material. However, when fragmented into micro- and nanoscale particles, the contribution of surface processes increases sharply,[12] because surface electrostatic charge begins to affect how the particles behave (Fig. 2.5).

"When exposed to erratic water environments with the presence of organic and synthetic impurities, pH, temperature, and salt, microplastic surfaces may be potentially active and generate charges in water."[12]

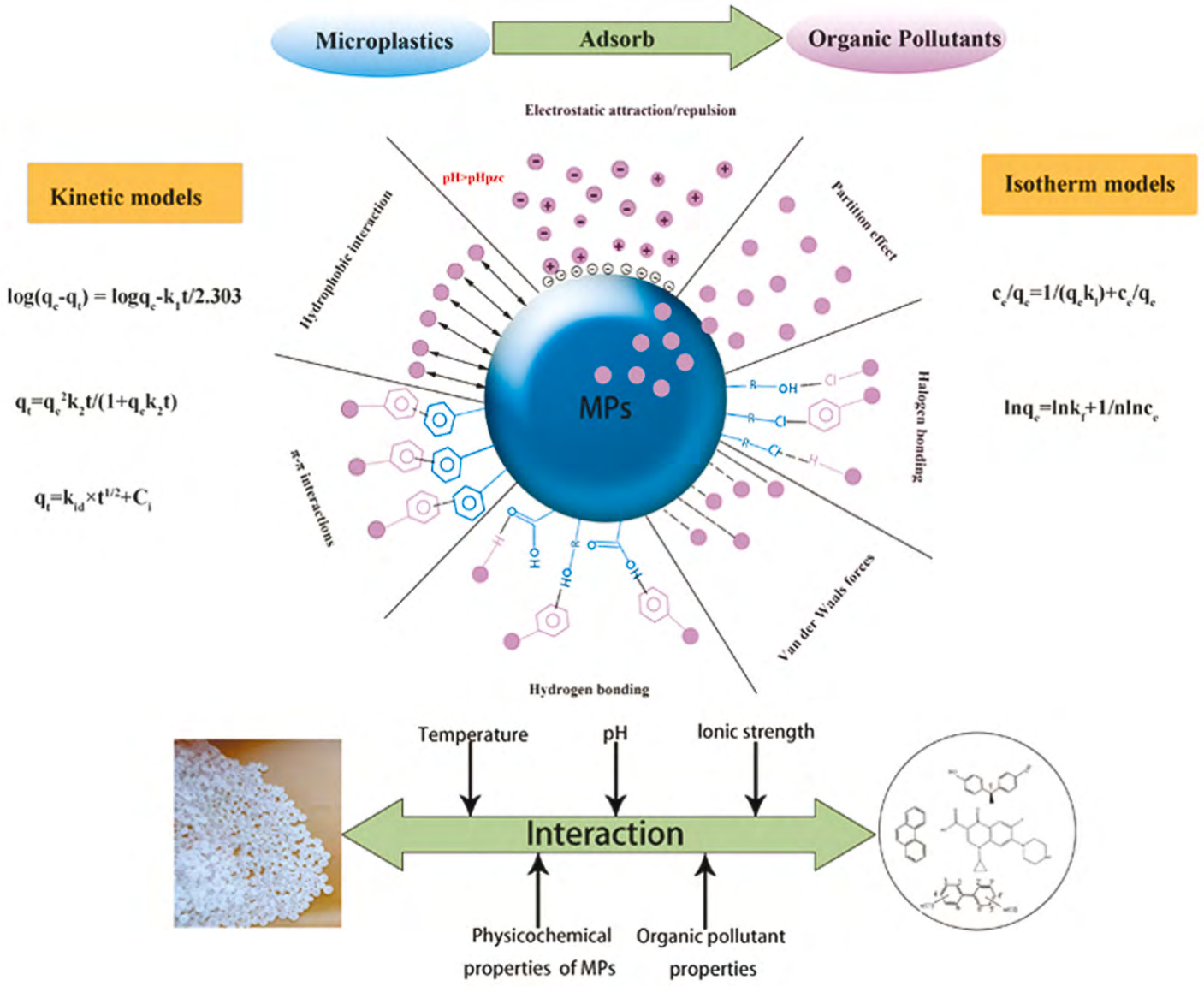


Figure 2.5.

Interaction between microplastics and organic pollutants.

Source: Fu, L., Li, J., Wang, G., et al. Adsorption behavior of organic pollutants on microplastics. Ecotoxicology and Environmental Safety 217, 112207 (2021).

https://doi.org/10.1016/j.ecoenv.2021.112207 License: CC BY-NC-ND 4.0.

In aquatic environments, micro- and nanoplastic particles acquire or change their surface charge and engage in electrostatic interactions with surrounding ions, molecules, and particles. This surface charge forms through the ionization of surface functional groups, adsorption of ions from solution, and interactions with environmental components.[12] This charge also determines the particles' zeta potential (ζ-potential), a key parameter for their colloidal stability and inter-particle interactions.

Zeta potential (ζ-potential) is the electrostatic potential at the hydrodynamic shear plane of a particle (boundary between a moving particle and the surrounding liquid), reflecting the effective surface charge and governing colloidal stability and inter-particle interactions.

This mechanism is particularly pronounced in liquid media,[12] as water provides high ion mobility and facilitates the formation of an electrical double layer at the particle-solution interface. This layer largely determines the forces of attraction and repulsion between particles, influencing aggregation, stable dispersion, and the adsorption of charged particles.

In these dynamic and chemically complex systems, the surfaces of micro- and nanoplastic particles can act as active centers for interfacial processes.

The environment's acid-base balance is a key factor regulating these processes. pH levels significantly influence the surface properties of microplastic particles and the nature of their interactions in aquatic environments (Fig. 2.6).[12]

Analysis of microparticles in water reveals a characteristic link between the pH and the sign of the surface charge: lower pH values often lead to positive charges, while higher pH values are associated with positive charge, while higher values result in negative charge. Near the isoelectric point, the net charge approaches zero.

Even minor changes in acidity can affect the surface charge of MNP particles. This, in turn, determines whether the particles will attract and stick together or remain in a stable, dispersed state. Increased salinity also weakens the electrical repulsion between particles and can promote their aggregation.

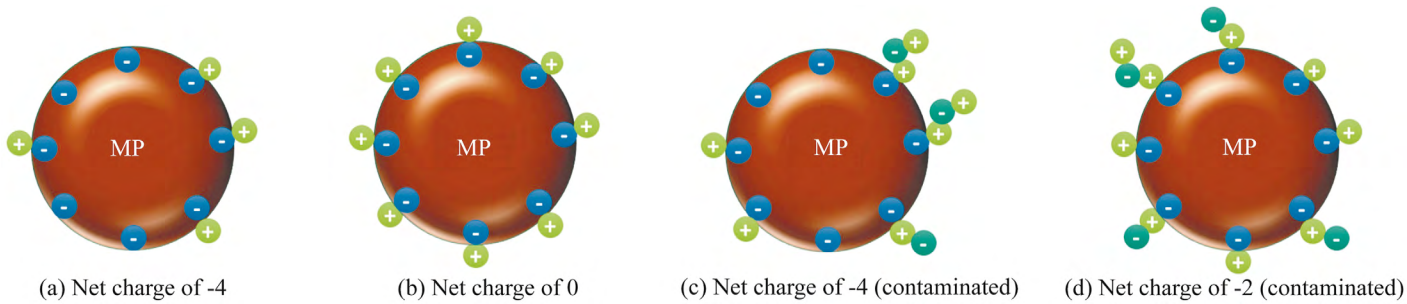


Figure 2.6.

Illustration of how cations and anions of water contaminants affect net surface charge of microplastics (MPs).

Source: Rahman, A. M. N. A. A., Rusli, A., Abdullah, M. K., et al. A review of microplastic surface interactions in water and potential capturing methods. Water Science and Engineering 17, 361–370 (2024).

https://doi.org/10.1016/j.wse.2023.11.008 License: CC BY-NC-ND 4.0.

In turn, due to their physicochemical properties, micro- and nanoplastics can alter the thermodynamic and hydrochemical parameters of aquatic environments. The potential consequences of these changes for the biosphere are examined in Part 15.

ELECTROSTATIC MECHANISMS OF MNPs INTERACTION WITH SOIL SYSTEMS

The surface charge of microplastics plays a role not only in aquatic environments, but also in their interaction with soils. Experimental data show that electrokinetic characteristics of microplastic particles largely determine how the particles attach to soil components.

A study on microplastic adhesion to soils found that microplastics' attachment ability significantly correlated with the soil's zeta potential. This indicates that electrostatic interactions play a key role in this process. The dominant mechanisms for microplastic attachment to soils are electrostatic attraction and physical entrapment within the soil's porous structure. The surface charge of micro- and nanoplastics allows them to interact more effectively with soil particles and makes them more likely to stay and accumulate in the soils compared to electrically neutral particles.[16]

This increases the likelihood of long-term contact between micro- and nanoplastics and root systems of plants as well as soil microbiota, which can lead to the disruption of biological processes[17] and changes in the physicochemical properties of the soil.[18]

[16] *Wang, Y., Wang, F., Xiang, L., et al. Attachment of positively and negatively charged submicron polystyrene plastics on nine typical soils. Journal of Hazardous Materials 431, 128566 (2022). https://doi.org/10.1016/j.jhazmat.2022.128566*

[17] *Chen, Z., Carter, L. J., Banwart, S. A., et al. Microplastics in Soil–Plant Systems: Current Knowledge, Research Gaps, and Future Directions for Agricultural Sustainability. Agronomy 15, 1519 (2025). https://doi.org/10.3390/agronomy15071519*

[18] *Mondol, M., Angon, P. B. & Roy, A. Effects of microplastics on soil physical, chemical and biological properties. Natural Hazards Research 5, 14–20 (2025). https://doi.org/10.1016/j.nhres.2024.02.002*

LINKING ELECTROKINETIC PROPERTIES TO BIOLOGICAL INTERACTIONS

A link between the surface charge of microplastics and their biological interactions is also confirmed at the cellular level. Experimental studies show that the particles' zeta (ζ) potential, which reflects their electrokinetic properties, significantly influences their interaction with cellular surfaces.

A study investigating differences in how microplastic models interact with cells has found:

"(...) the ζ-potential determines particle-cell adhesion strength. Furthermore, we find that due to this effect, the ζ-potential also strongly determines the internalization of the microplastic particles into cells. Therefore, the ζ-potential can act as a proxy of microplastic-cell interactions and may govern adverse effects reported in various organisms exposed to microplastics."[19]

When micro- and nanoplastics enter biological fluids (such as blood plasma, bronchoalveolar fluid, or digestive fluids), a protein corona — a layer of adsorbed proteins — rapidly forms on their surface.[20] This corona largely dictates the particle's biological identity, how it is recognized by cells, and its uptake pathways.[21]

The formation of this corona is a thermodynamically favorable process driven by a reduction in free energy at the phase boundary.[22] It occurs through hydrophobic, van der Waals, and electrostatic interactions. The surface charge of the particles, characterized by the zeta potential, plays a critical role during the initial stage of adsorption: it attracts oppositely charged proteins, thereby influencing the rate of formation, composition, and stability of the corona.[23]

Various types of coronas can form on the surface of micro- and nanoplastics,[24] including hard and soft protein coronas, as well as eco-coronas and bio-coronas (Fig. 2.7).

[19] *Wieland, S., Ramsperger, A. F. R. M., Gross, W., et al. Nominally identical microplastic models differ greatly in their particle-cell interactions. Nat Commun 15, 922 (2024). https://doi.org/10.1038/s41467-024-45281-4*

[20] *Pino, P. del, Pelaz, B., Zhang, Q., et al. Protein corona formation around nanoparticles – from the past to the future. Mater. Horiz. 1, 301–313 (2014). https://doi.org/10.1039/C3MH00106G*

[21] *Cai, R., Baimanov, D., Yuan, H., et al. Protein Corona-Directed Cellular Recognition and Uptake of Polyethylene Nanoplastics by Macrophages. Environ. Sci. Technol. 58, 14158–14168 (2024). https://doi.org/10.1021/acs.est.4c05215*

[22] *Walkey, C. D. & Chan, W. C. W. Understanding and controlling the interaction of nanomaterials with proteins in a physiological environment. Chem. Soc. Rev. 41, 2780–2799 (2012). https://doi.org/10.1039/C1CS15233E*

[23] *Du, T., Yu, X., Shao, S., et al. Aging of Nanoplastics Significantly Affects Protein Corona Composition Thus Enhancing Macrophage Uptake. Environ. Sci. Technol. 57, 3206–3217 (2023). https://doi.org/10.1021/acs.est.2c05772*

[24] *Cao, J., Yang, Q., Jiang, J., et al. Coronas of micro/nano plastics: a key determinant in their risk assessments. Part Fibre Toxicol 19, 55 (2022). https://doi.org/10.1186/s12989-022-00492-9*

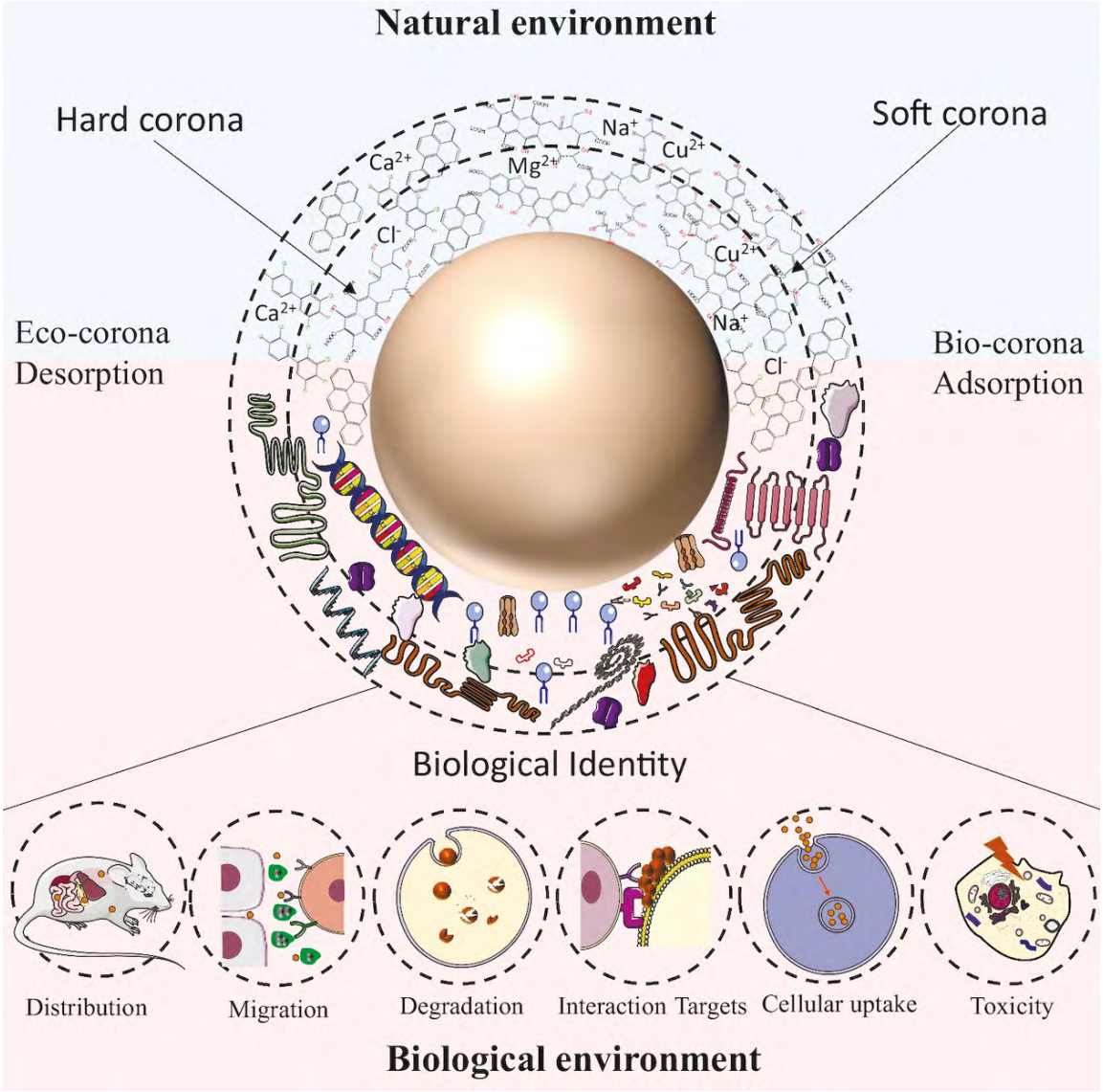


Figure 2.7.

Eco-coronas (top) and bio-coronas (bottom) on M/NP. The natural components in the environment can be adsorbed on the surface of M/NP and form eco-coronas, which consist of metal ions (Cu^{2+}, Mg^{2+}, Pd^{2+} etc.), inorganic salts (Na^{+}, Cl^{-}, etc.), natural organic matters (HAs, FAs, etc.), and persistent organic pollutants (PCBs, PAHs, etc.). When this M/NP encounters biological systems, it may interact with abundant biomolecules, including proteins, lipids, and nucleic acids to replace some of the constituents in eco-coronas and form bio-coronas on surface. The corona structures offer new environmental or biological identities for M/NP and impact its distribution, migration, degradation, interaction targets, cellular internalization and toxicity in organisms.

Source: Cao, J., Yang, Q., Jiang, J., et al. Coronas of micro/nano plastics: a key determinant in their risk assessments. Part Fibre Toxicol 19, 55 (2022).
https://doi.org/10.1186/s12989-022-00492-9
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Unlike certain inorganic nanomaterials[25] — where the protein corona often partially passivates the original surface and dampens its direct biological impact[26] — the corona on nanoplastics typically acts not merely as a shielding layer, but as a dynamic envelope whose properties are largely dictated by the underlying polymer surface itself.[27,28] Due to their hydrophobic nature, surface roughness, and the presence of oxidation products and manufacturing additives, plastic micro- and nanoparticles can alter the spatial structure (conformation) of adsorbed proteins (Fig. 2.8, 2.9),[29,30] thereby fostering a highly specific "biological identity" for the particle.[22,31]

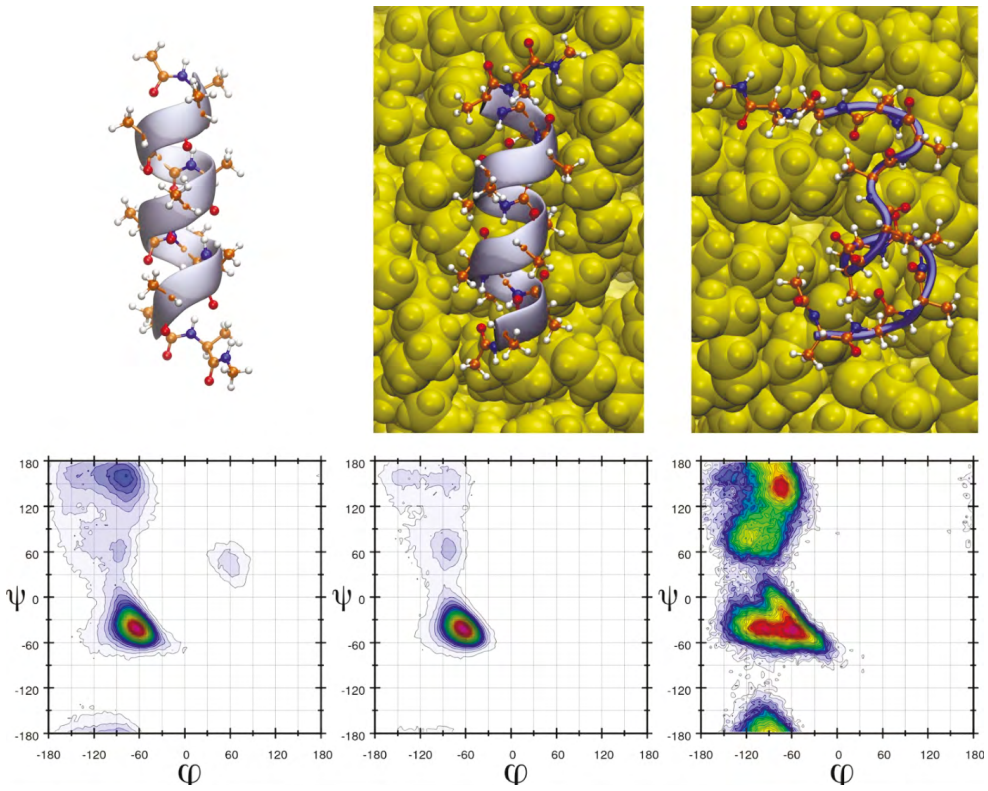


Figure 2.8.

Three dimensional structures (above) of the α-helical peptide composed of 12 alanine amino acids in the absence of plastics (left), and in the presence of a polyethylene (middle) and nylon-6,6 (right) nanoplastic particle. Ramachandran plots (below) show the distribution of the peptide backbone dihedral angles □ and □, revealing that the □-helical structure is enhanced in the presence of polyethylene, whereas nylon-6,6 unfolds the helix and changes it into a more □-sheet-like structure.

Source: Hollóczki, O. & Gehrke, S. Nanoplastics can change the secondary structure of proteins. Sci Rep 9, 16013 (2019). https://doi.org/10.1038/s41598-019-52495-w

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[25] Corbo, C., Molinaro, R., Parodi, A., et al. The Impact of Nanoparticle Protein Corona on Cytotoxicity, Immunotoxicity and Target Drug Delivery. Nanomedicine (Lond.) 11, 81–100 (2016). https://doi.org/10.2217/nnm.15.188

[26] Khang, D., Lee, Y. K., Choi, E.-J., et al. Effect of the protein corona on nanoparticles for modulating cytotoxicity and immunotoxicity. IJN 10, 97–113 (2014). https://doi.org/10.2147/IJN.S72998

[27] Wang, J., Cong, J., Wu, J., et al. Nanoplastic-protein corona interactions and their biological effects: A review of recent advances and trends. TrAC Trends in Analytical Chemistry 166, 117206 (2023). https://doi.org/10.1016/j.trac.2023.117206

[28] Luo, H., Du, Q., Zhong, Z., et al. Protein-coated microplastics corona complex: An underestimated risk of microplastics. Science of The Total Environment 851, 157948 (2022). https://doi.org/10.1016/j.scitotenv.2022.157948

[29] Hollóczki, O. & Gehrke, S. Nanoplastics can change the secondary structure of proteins. Sci Rep 9, 16013 (2019). https://doi.org/10.1038/s41598-019-52495-w

[30] Windheim, J., Colombo, L., Battajni, N. C., et al. Micro- and Nanoplastics' Effects on Protein Folding and Amyloidosis. International Journal of Molecular Sciences 23, 10329 (2022). https://doi.org/10.3390/ijms231810329

[31] Ducoli, S., Federici, S., Nicsanu, R., et al. A different protein corona cloaks "true-to-life" nanoplastics with respect to synthetic polystyrene nanobeads. Environ. Sci.: Nano 9, 1414–1426 (2022). https://doi.org/10.1039/D1EN01016F

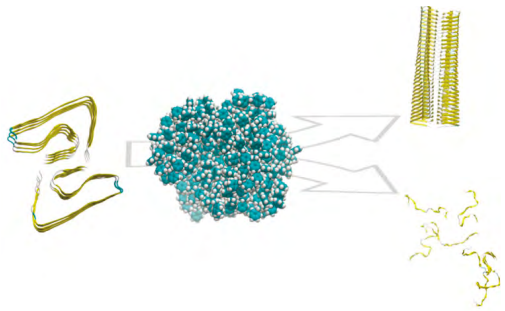


Figure 2.9.

Schematic of the role of polystyrene NP on amyloid aggregation. Depending on the features of the NP and the protein/NP ratio, the aggregation is promoted or inhibited.

Source: Windheim, J., Colombo, L., Battajni, N. C., et al. Micro- and Nanoplastics' Effects on Protein Folding and Amyloidosis. International Journal of Molecular Sciences 23, 10329 (2022). https://doi.org/10.3390/ijms231810329 License: CC BY 4.0.

As a result, a functionally modified protein corona is formed, which can alter how the particles interact with cells and modify their subsequent biological effects.[32] Following the formation of the protein corona, there is typically a partial attenuation[33] and shift in the particle's zeta potential.[34] In other words, while the corona generally shields the original surface charge, it does not necessarily neutralize it completely. This outcome depends on the protein composition, the initial surface characteristics, the pH, and the incubation time.

Owing to their small size, surface charge, and the formation of a protein corona, nanoplastics can interact highly effectively with cellular structures and affect molecular mechanisms (Fig. 2.10).[27, 35] This can disrupt intracellular processes and potentially compromise overall bodily functions.

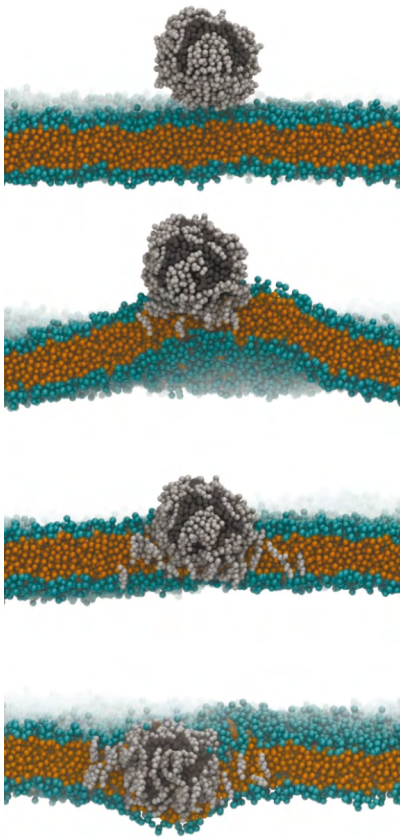


Figure 2.10.

Snapshots of the simulations showing the entry of the plastic particle with a corona of 150 cholesterol molecules into the DOPC bilayer, a model blood–brain barrier (orange: hydrophobic moieties of the DOPC molecules; green: hydrophilic groups of the DOPC; dark grey: polystyrene; light grey: cholesterol). The nanoparticle approaches the membrane in the beginning of the simulation (above), and then spontaneously diffuses into its hydrophobic core, followed by the dissociation of its corona, and the disentanglement of the polymer chains by the end of the 1 μs run (below).

Source: Kopatz, V., Wen, K., Kovács, T., et al. Micro- and Nanoplastics Breach the Blood–Brain Barrier (BBB): Biomolecular Corona's Role Revealed. Nanomaterials 13, 1404 (2023).

https://doi.org/10.3390/nano13081404
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[32] *Ramsperger, A. F. R. M., Narayana, V. K. B., Gross, W., et al. Environmental exposure enhances the internalization of microplastic particles into cells. Sci. Adv. 6, eabd1211 (2020). https://doi.org/10.1126/sciadv.abd1211*
[33] *Giulimondi, F., Digiacomo, L., Pozzi, D., et al. Interplay of protein corona and immune cells controls blood residency of liposomes. Nat Commun 10, 3686 (2019). https://doi.org/10.1038/s41467-019-11642-7*
[34] *Wang, L., Hartel, N., Ren, K., et al. Effect of protein corona on nanoparticle–plasma membrane and nanoparticle–biomimetic membrane interactions. Environ. Sci.: Nano 7, 963–974 (2020). https://doi.org/10.1039/D0EN00035C*
[35] *Kopatz, V., Wen, K., Kovács, T., et al. Micro- and Nanoplastics Breach the Blood–Brain Barrier (BBB): Biomolecular Corona's Role Revealed. Nanomaterials 13, 1404 (2023). https://doi.org/10.3390/nano13081404*

The mechanisms underlying the impacts of MNPs and and their consequences for animals and humans are also examined in this report.

REACCUMULATION OF ELECTROSTATIC CHARGE BY MICRO- AND NANOPLASTICS

As dielectric materials, micro- and nanoplastics are capable of accumulating static charge. In this sense, they may be compared to electrets — materials that can accumulate static electricity and retain it for extended periods of time.

Importantly, plastic particles are capable of both retaining a surface electric charge and reacquiring it through interactions with their surrounding environment. In aerosol or dry conditions, charges can form as a result of the triboelectric effect[36] (contact and friction with various surfaces) (Fig. 2.11). In liquid environments, a charge is determined by the ionization of functional groups, ion adsorption, and formation of an electrical double layer. As particles move between different environments, their surface charge may be repeatedly altered and re-established.

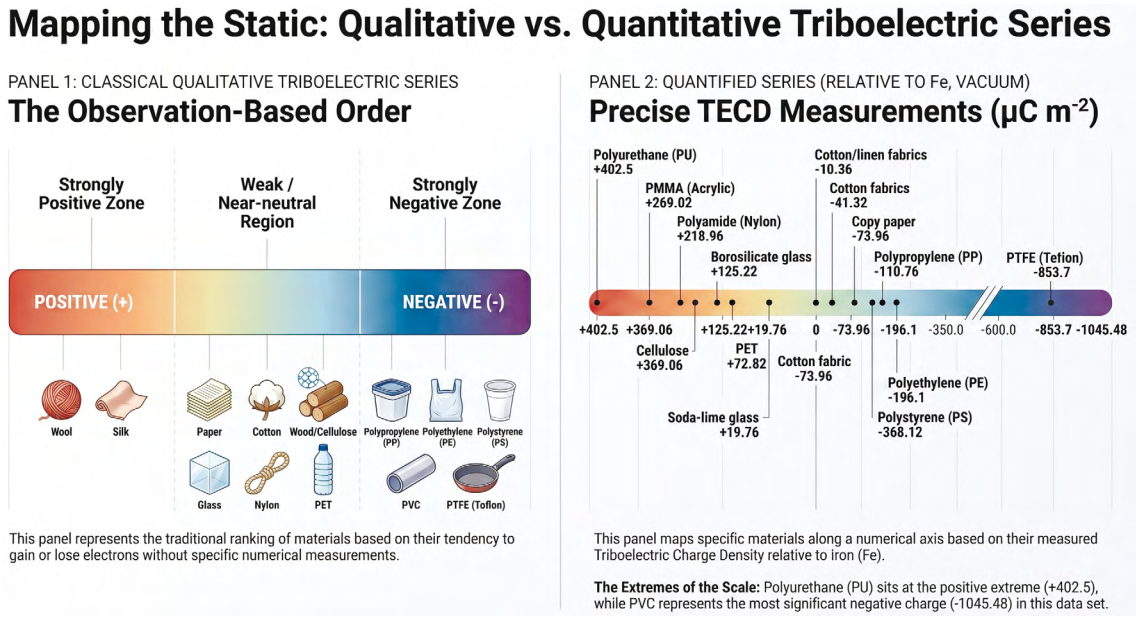


Figure 2.11.

Triboelectric series characterizing the relative ability of materials to acquire a positive or negative electric charge upon contact interaction (triboelectric effect).

Data source: Liu, D., Zhou, L., Cui, S., et al. Standardized measurement of dielectric materials' intrinsic triboelectric charge density through the suppression of air breakdown. Nat Commun 13, 6019 (2022). https://doi.org/10.1038/s41467-022-33766-z

[36] *Németh, E., Albrecht, V., Schubert, G., et al. Polymer triboelectric charging: dependence on thermodynamic surface properties and relative humidity. Journal of Electrostatics 58, 3–16 (2003). https://doi.org/10.1016/S0304-3886(02)00137-7*

Consequently, the electrical charge of plastic is not a static property of the material; rather, it is dictated by the environment in which the particle is located. In air, this manifests as a static or triboelectric charge, reflecting the accumulation of free charges through contact and friction. In aqueous environments, the focus shifts to surface charge and zeta potential — parameters of colloidal electrochemistry that govern stability, aggregation, and interfacial interactions. In biological systems, the material's initial properties undergo further transformation: once a bio-corona forms, what is actually measured is the effective charge of the "plastic + adsorbed biomolecules" complex, which ultimately dictates its cellular and immune interactions. In other words, it is not merely the terminology that changes, but the fundamental physical state in which the particle exists. Recognizing this is absolutely critical for understanding the ecological and biological activity of micro- and nanoplastics.

Millions of tons of plastics are already present in the biosphere, continuously fragmenting into micro- and nanoparticles under the influence of physical and chemical factors. As the size of plastics decreases, the nature of their behavior changes dramatically. The specific surface area increases sharply, and the role of surface electric charge and interfacial interactions becomes more pronounced. With a reduction in size, plastics transition from a state of being a relatively inert macro-material into a dispersed form with pronounced physicochemical activity.

Microplastics and especially nanoplastics, possessing surface electrostatic charge, become capable of binding and transporting ions, organic molecules, and proteins, participating in the transport of substances, and actively interacting with natural phase boundaries such as water, soil colloids, and aerosol particles. Charged micro- and nanoparticles interact more easily with cell membranes, protein structures, and the extracellular matrix, which increases the likelihood of their cellular uptake and intracellular accumulation. As a result, they can interfere with intracellular processes, alter signaling pathways, enhance the formation of reactive oxygen species, and provoke inflammatory reactions. Thus, their impact is not limited to the local level and may lead to systemic disruptions.

Therefore, the resistance of plastic to degradation, combined with its particles' capacity to carry an electrical charge, renders it a potentially hazardous agent capable of exerting long-term impacts on natural systems.

PART 3.

THE IMPACT OF MICRO- AND NANOPLASTICS **ON THE UNIVERSAL MECHANISMS OF NATURAL SYSTEMS**

Natural ecosystems function through a complex network of communication and interaction between their components, which ensures their resilience and ability to adapt to environmental changes. However, exposure to micro- and nanoplastics can disrupt this balance, leading to significant damage to ecosystems. Given current data on the systemic accumulation of MNPs in forests and their impact on plants and animals, it is imperative to recognize that plastic pollution is not merely an ecological problem, but a serious threat to the biosphere as a whole.

FORESTS

Communication mechanisms in natural ecosystems often remain invisible; however, it is these mechanisms that form the resilience and functional integrity of living systems.[37]

One of the most illustrative examples is forest communities, where trees and other plants can be interconnected via mycorrhizal networks. Underground mycorrhizal structures form an interconnected system for the exchange of water, nutrients, and signaling compounds (Fig. 3.1). In scientific and popular literature, this phenomenon is often referred to as the "Wood Wide Web."[38]



Figure 3.1.

Illustration of interconnection between trees and plans, known as the "Wood Wide Web".

[37] *Gorzelak, M. A., Asay, A. K., Pickles, B. J., et al. Inter-plant communication through mycorrhizal networks mediates complex adaptive behaviour in plant communities. AoB PLANTS 7, plv050 (2015). https://doi.org/10.1093/aobpla/plv050*

[38] *GFBI consortium, Steidinger, B. S., Crowther, T. W., et al. Climatic controls of decomposition drive the global biogeography of forest-tree symbioses. Nature 569, 404–408 (2019). https://doi.org/10.1038/s41586-019-1128-0*

Such connections are regarded as a factor allowing plants to not only redistribute resources, but also coordinate responses to external stimuli.

Plant communication is implemented through several mechanisms. Alongside chemical signaling[39,40] — via the secretion of specific molecules — plants exhibit bioelectrical processes that ensure rapid systemic reactions.

> *"Ordered flow of electrical currents between cells and organs allows a given organism for universal, rapid, and efficient communication of the external changes."* [41]

Changes in electric potential within tissues can function as signals that propagate through the plant, triggering physiological responses to stress.[42]

A number of studies use the term "electrome" to designate the total electrical activity of living tissues — the aggregate of bioelectrical signaling and ionic currents occurring at the cellular and tissue levels.[43]

It has been established that the bioelectric activity of plants is not chaotic. Research shows that these signals are highly structured: they demonstrate specific patterns, stability, and temporal continuity.[44] These characteristics suggest that electrical processes may be involved in long-distance signal transmission within the plant.

Electrical signals are involved in the regulation of physiological responses.[45] For instance, when a plant's leaves are damaged by insects, these signals trigger the plant's defensive mechanisms.

The coordination of bioelectrical activity is also recorded in natural conditions, including those associated with external astronomical events. A study published in Royal Society Open Science, led by Alessandro Chiolerio, investigated the electrical signals of spruce trees (Picea abies) (Fig. 3.2) to characterize their bioelectrical activity during a partial solar eclipse that occurred in a forest located in the Dolomites mountain region, northeastern Italy.[46]

[39] *Mahanta, D. K., Komal, J., Samal, I., et al. Plant Defense Responses to Insect Herbivores Through Molecular Signaling, Secondary Metabolites, and Associated Epigenetic Regulation. Plant Enviro Interactions 6, e70035 (2025). https://doi.org/10.1002/pei3.70035*

[40] *Meents, A. K. & Mithöfer, A. Plant–Plant Communication: Is There a Role for Volatile Damage-Associated Molecular Patterns? Front. Plant Sci. 11, 583275 (2020). https://doi.org/10.3389/fpls.2020.583275*

[41] *Szechyńska-Hebda, M., Lewandowska, M. & Karpiński, S. Electrical Signaling, Photosynthesis and Systemic Acquired Acclimation. Front. Physiol. 8, 684 (2017). https://doi.org/10.3389/fphys.2017.00684*

[42] *Li, R., Yang, Y., Lou, H., et al. Electrical and calcium signaling in plant systemic defense: from local wounds to global responses. New Phytologist 247, 1633–1642 (2025). https://doi.org/10.1111/nph.70301*

[43] *Souza, G. M., Ferreira, A. S., Saraiva, G. F. R., et al. Plant "electrome" can be pushed toward a self-organized critical state by external cues: Evidences from a study with soybean seedlings subject to different environmental conditions. Plant Signaling & Behavior 12, e1290040 (2017). https://doi.org/10.1080/15592324.2017.1290040*

[44] *Gloor, P. A. Plant Bioelectrical Signals for Environmental and Emotional State Classification. Biosensors 15, 744 (2025). https://doi.org/10.3390/bios15110744*

[45] *Kloth, K. J. & Dicke, M. Rapid systemic responses to herbivory. Current Opinion in Plant Biology 68, 102242 (2022). https://doi.org/10.1016/j.pbi.2022.102242*

[46] *Chiolerio, A., Gagliano, M., Pilia, S., et al. Bioelectrical synchronization of Picea abies during a solar eclipse. Royal Society Open Science 12, 241786 (2025). https://doi.org/10.1098/rsos.241786*

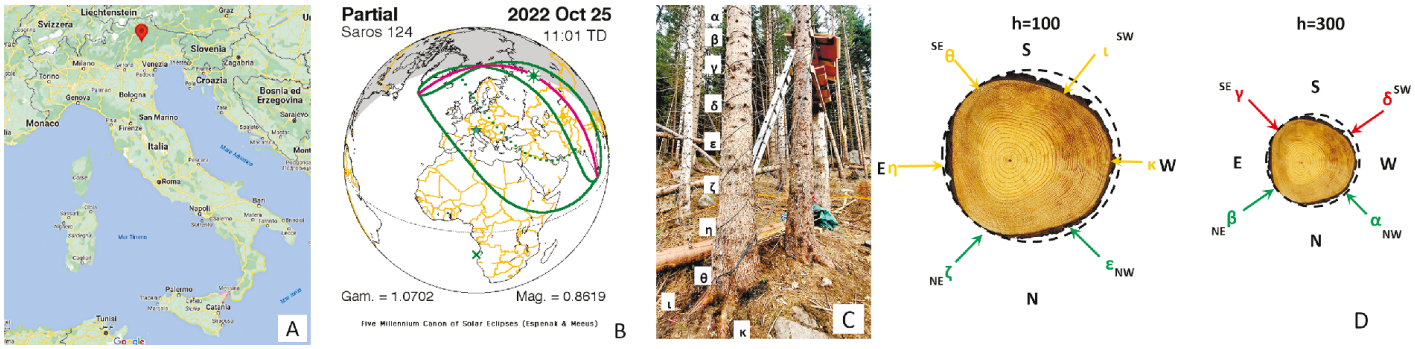


Figure 3.2.

Experimental set-up to simultaneously monitor the electrome of multiple trees during solar eclipse. (A) Location of the experimental site at the Costa Bocche forest near Paneveggio in the Dolomites area, Italy; (B) Diagram of the Saros 124 event, the solar eclipse that occurred on October 25, 2022; (C) In situ installation of 10 xylematic electrodes (labelled with Greek letters) on a spruce tree; (D) Horizontal view of the phloematic configuration with electrodes in contact with the superficial layer of the tree and radially arranged in two arrays.

Source: Chiolerio, A., Gagliano, M., Pilia, S., et al. Bioelectrical synchronization of Picea abies during a solar eclipse. Royal Society Open Science 12, 241786 (2025). https://doi.org/10.1098/rsos.241786 License: CC BY 4.0.

It was revealed that during the solar eclipse, researchers observed a synchronization in the bioelectric signals of trees, forming a cohesive, collective pattern. Furthermore, trees of different age groups often reacted differently: more mature trees tended to respond earlier or with greater intensity. Such observations are interpreted as manifestations of age-related differences in physiological sensitivity and the adaptive dynamics of the ecosystem.

Consequently, bioelectrical processes are viewed as one of the mechanisms ensuring growth coordination, external threat signaling, and plant adaptation to stressors.

In light of this, the issue of the impact of external anthropogenic pollutants becomes increasingly critical. Micro- and nanoplastics occupy an important place among those pollutants; these particles are capable of accumulating and retaining a surface electrostatic charge, which enhances their interactions with biological structures and may disrupt the natural regulatory mechanisms of ecosystems.

In recent years, forest ecosystems are increasingly viewed as zones of significant microplastic accumulation. Research findings indicate that forests are capable of capturing microplastics from the atmosphere: air currents carry particles that are trapped by tree canopies and leaves, and subsequently enter the forest environment via precipitation.[47]

Nature intended for this process to facilitate the cleansing of air from dust and aerosol pollutants. However, under modern conditions, this mechanism facilitates the accumulation of persistent MNP particles in foliage and soils. Unlike organic components, plastics are not integrated into natural decomposition cycles and can persist in the ecosystem for long periods.

According to quantitative estimates, dozens of microplastic particles can settle per unit area of the tree canopy every single day.[47]

[47] *Weber, C. J. & Bigalke, M. Forest soils accumulate microplastics through atmospheric deposition. Commun Earth Environ 6, 702 (2025). https://doi.org/10.1038/s43247-025-02712-4*

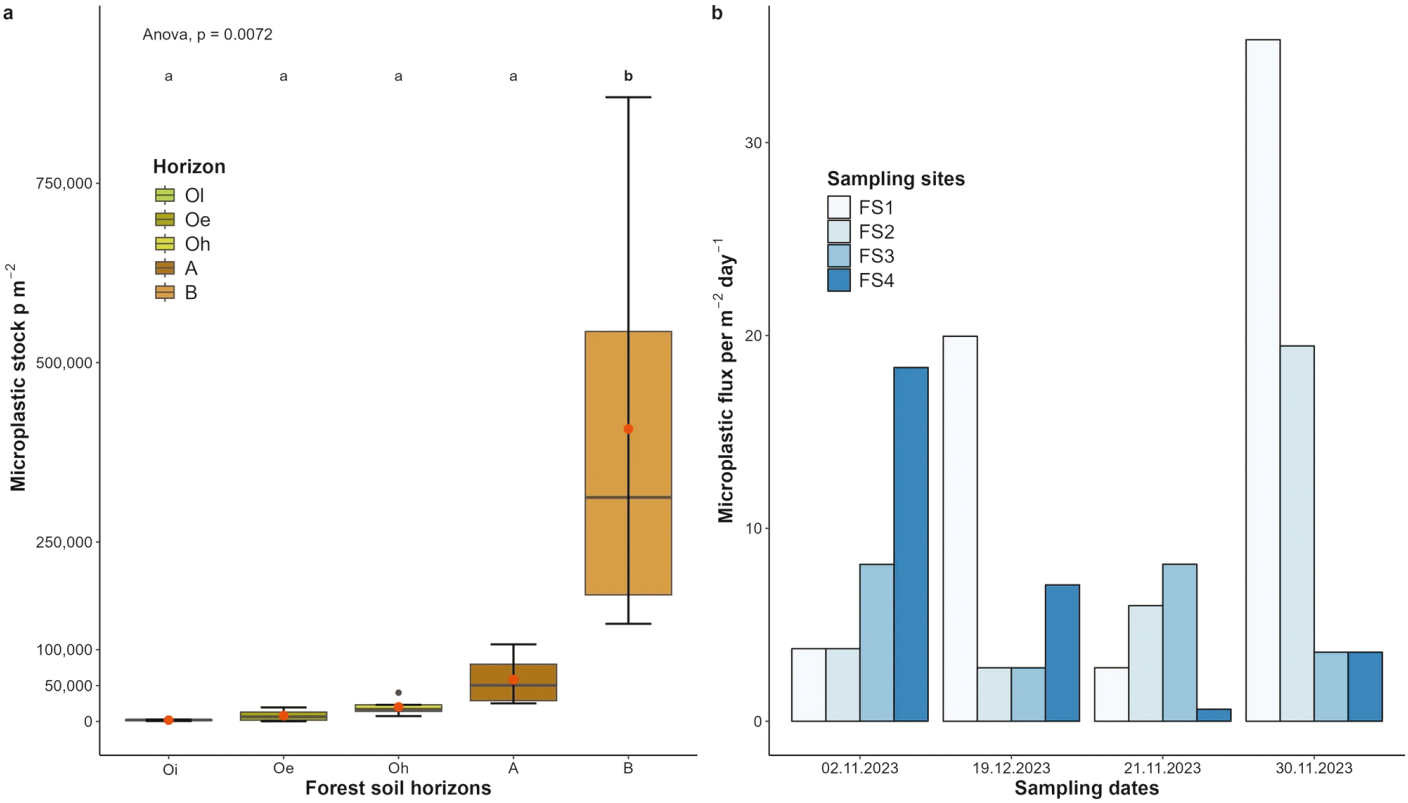


Figure 3.3.

Microplastic stocks in forest soil and microplastic flux with throughfall deposition.

a) Microplastic stock (particles per square metre soil, p m^{-2}) in different forest soil organic (Oi, Oe, Oh) and mineral (A, B) horizons (n = 4).

b) Microplastic flux with throughfall deposition (particles per m^2 per day, p m^2 day^{-1}) on sampling dates at each of the four forest sampling sites.

Source: Weber, C. J. & Bigalke, M. Forest soils accumulate microplastics through atmospheric deposition. Commun Earth Environ 6, 702 (2025). https://doi.org/10.1038/s43247-025-02712-4 License: CC BY 4.0.

The annual rates of microplastic accumulation in forest soils (Fig. 3.3) and their correlation with plastic production scales in Europe have also been evaluated (Fig. 3.4). When considering atmospheric deposition as one of the primary sources of influx, it has been shown that over a period of approximately 70 years, forest soils can accumulate about 400,000 microplastic particles per square meter, which underscores the scale and long-term nature and pervasive character of this process.[47]

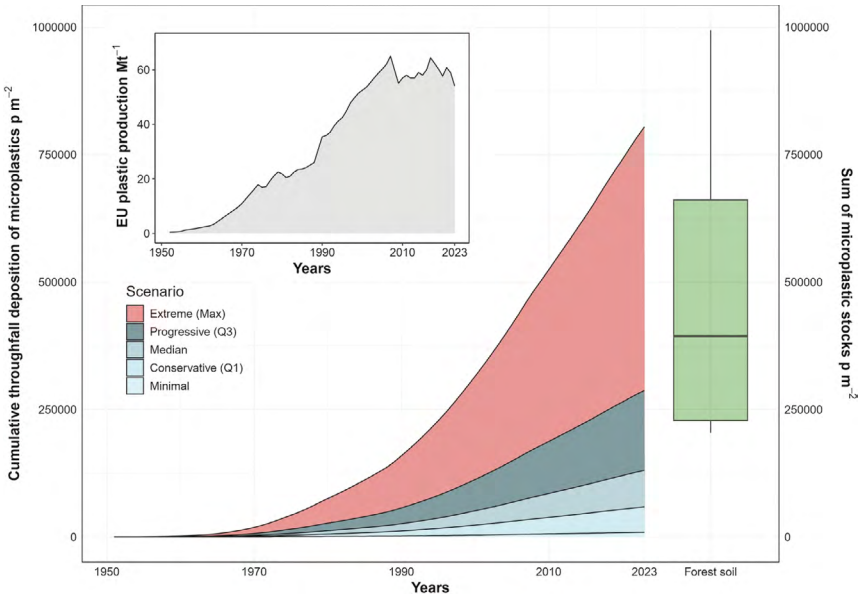


Figure 3.4.

Estimated cumulative throughfall deposition of microplastics (p m^{-2}) under different scenarios ranging from minimal to extreme combined with European Union (EU) plastic production (Mt^{-1}) according Plastic Europe (2014, 2023) and fit to total microplastic stocks (p m^{-2}) in forest soils.

Source: Weber, C. J. & Bigalke, M. Forest soils accumulate microplastics through atmospheric deposition. Commun Earth Environ 6, 702 (2025). https://doi.org/10.1038/s43247-025-02712-4

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Special attention is being paid to the role of the electrostatic properties of micro- and nanoplastic particles.

It is noted that the surface charge of nanoparticles affects their interaction with the plant root systems and the efficiency of tissue penetration. Research shows that negatively charged polymer nanoparticles can penetrate root tissues and are gradually transported within the plant, whereas positively charged particles tend to accumulate on the root surface and show minimal uptake. (Fig. 3.5, 3.6).[48]

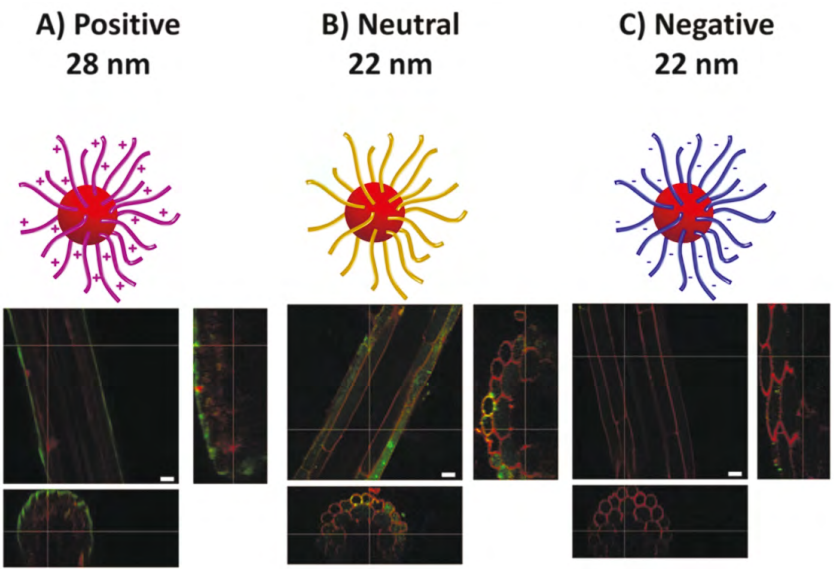


Figure 3.5.

Root cross-sections illustrate nanoparticle accumulations. Cross-sections of root hair zones after uptake of small nanoparticles with (a) positive, (b) neutral, and (c) negative surface charges, respectively. Propidium iodide staining was used to outline the cellular structure (red) while the nanoparticles are in green, and areas of co-localisation appear yellow. Scale bar = 20 µm.

Source: Parkinson, S. J., Tungsirisurp, S., Joshi, C., et al. Polymer nanoparticles pass the plant interface. Nat Commun 13, 7385 (2022). https://doi.org/10.1038/s41467-022-35066-y License: CC BY 4.0.

[48] *Parkinson, S. J., Tungsirisurp, S., Joshi, C., et al. Polymer nanoparticles pass the plant interface. Nat Commun 13, 7385 (2022). https://doi.org/10.1038/s41467-022-35066-y*

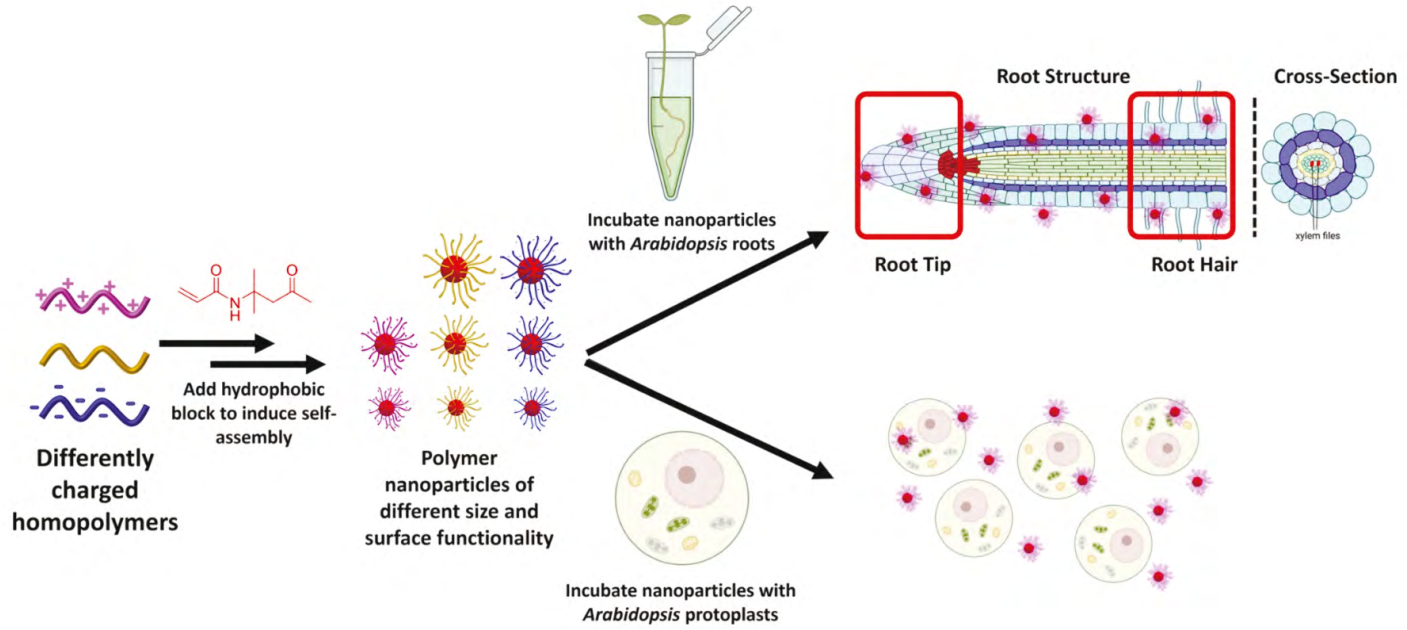


Figure 3.6.

Synthesis of polymeric nanoparticles and subsequent uptake pathways explored. The charges associated with each class of nanoparticle: magenta = positively charged; gold = neutral; blue = negatively charged.

Source: Parkinson, S. J., Tungsirisurp, S., Joshi, C., et al. Polymer nanoparticles pass the plant interface. Nat Commun 13, 7385 (2022). https://doi.org/10.1038/s41467-022-35066-y License: CC BY 4.0.

The accumulation and penetration of nanoparticles (Fig. 3.7) may be accompanied by disturbances in physiological processes, including nutrient uptake, which manifests as growth inhibition and alterations in the morphological traits of the root system.[48,49]

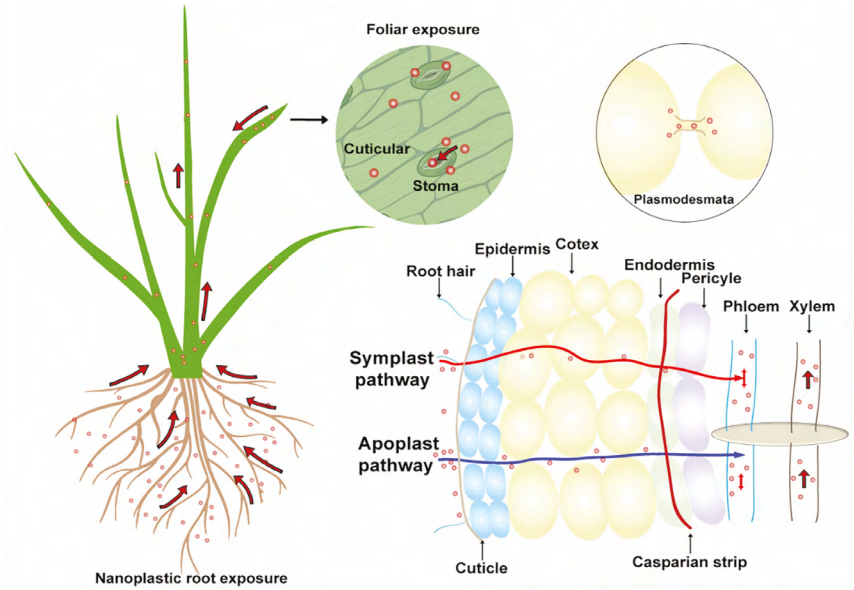


Figure 3.7.

Schematic diagram of the absorption and translocation pathways of NPs in plants.

Source: Zhou, P., Wang, L., Gao, J., et al. Nanoplastic–plant interaction and implications for soil health. Soil Use and Management 39, 13–42 (2023).

https://doi.org/10.1111/sum.12868

License: CC BY-NC 4.0.

[49] *Hussain, K., Fox, J.-P., Ma, X., et al. Impact of polystyrene nanoplastics on physiology, nutrient uptake, and root system architecture of aeroponically grown citrus plants. NanoImpact 37, 100536 (2025). https://doi.org/10.1016/j.impact.2024.100536*

In addition, electrostatically charged micro- and nanoplastic particles are capable of adhering to the leaf surface and penetrating into plant cells.[50,51,52]

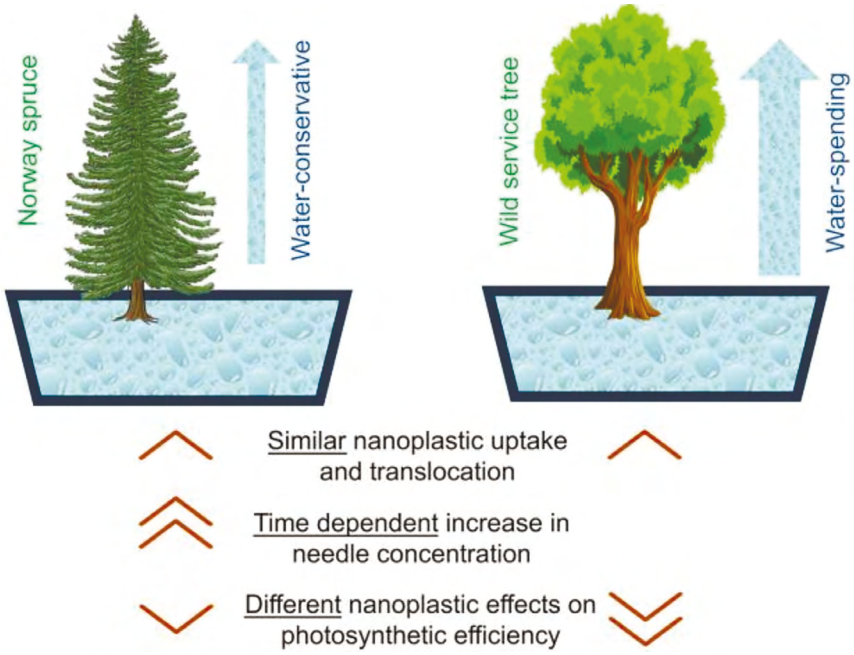


Figure 3.8.

Nanoplastic particles negatively affected photosynthesis in a coniferous species, the Norway spruce (Picea abies), and a deciduous species, the wild service tree (Sorbus torminalis), due to their uptake and translocation into the aboveground organs of the trees.

Source: Murazzi, M. E., Pradel, A., Schefer, R. B., et al. Uptake and physiological impacts of nanoplastics in trees with divergent water use strategies. Environ. Sci.: Nano 11, 3574–3584 (2024).

https://doi.org/10.1039/D4EN00286E

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> *"...uptake and transport of micro- and nanoplastics are complex processes influenced by multiple factors, including particle size, surface charge, mechanical properties, and physiological characteristics of plants, as well as external environmental conditions."*[50]

With prolonged exposure to micro- and nanoplastics, plants exhibit a decline in photosynthetic efficiency (Fig. 3.8),[53,54] potentially leading to reduced oxygen production, in part due to damage to chloroplasts [55] — the energy centers of plant cells (Fig. 3.9).

[50] *Yu, Z., Xu, X., Guo, L., et al. Uptake and transport of micro/nanoplastics in terrestrial plants: Detection, mechanisms, and influencing factors. Science of The Total Environment 907, 168155 (2024). https://doi.org/10.1016/j.scitotenv.2023.168155*

[51] *Wang, K., Dong, X., Wang, S., et al. The Behavior of Electrostatic Droplets After Impacting Pepper Leaves. Horticulturae 11, 608 (2025). https://doi.org/10.3390/horticulturae11060608*

[52] *Leitão, I. A., Van Schaik, L., Iwasaki, S., et al. Accumulation of airborne microplastics on leaves of different tree species in the urban environment. Science of The Total Environment 948, 174907 (2024). https://doi.org/10.1016/j.scitotenv.2024.174907*

[53] *Murazzi, M. E., Pradel, A., Schefer, R. B., et al. Uptake and physiological impacts of nanoplastics in trees with divergent water use strategies. Environ. Sci.: Nano 11, 3574–3584 (2024). https://doi.org/10.1039/D4EN00286E*

[54] *Li, J., Zheng, X., Liu, X., et al. Effect and mechanism of microplastics exposure against microalgae: Photosynthesis and oxidative stress. Science of The Total Environment 905, 167017 (2023). https://doi.org/10.1016/j.scitotenv.2023.167017*

[55] *Okeke, E. S., Chukwudozie, K. I., Addey, C. I., et al. Micro and nanoplastics ravaging our agroecosystem: A review of occurrence, fate, ecological impacts, detection, remediation, and prospects. Heliyon 9, e13296 (2023). https://doi.org/10.1016/j.heliyon.2023.e13296*

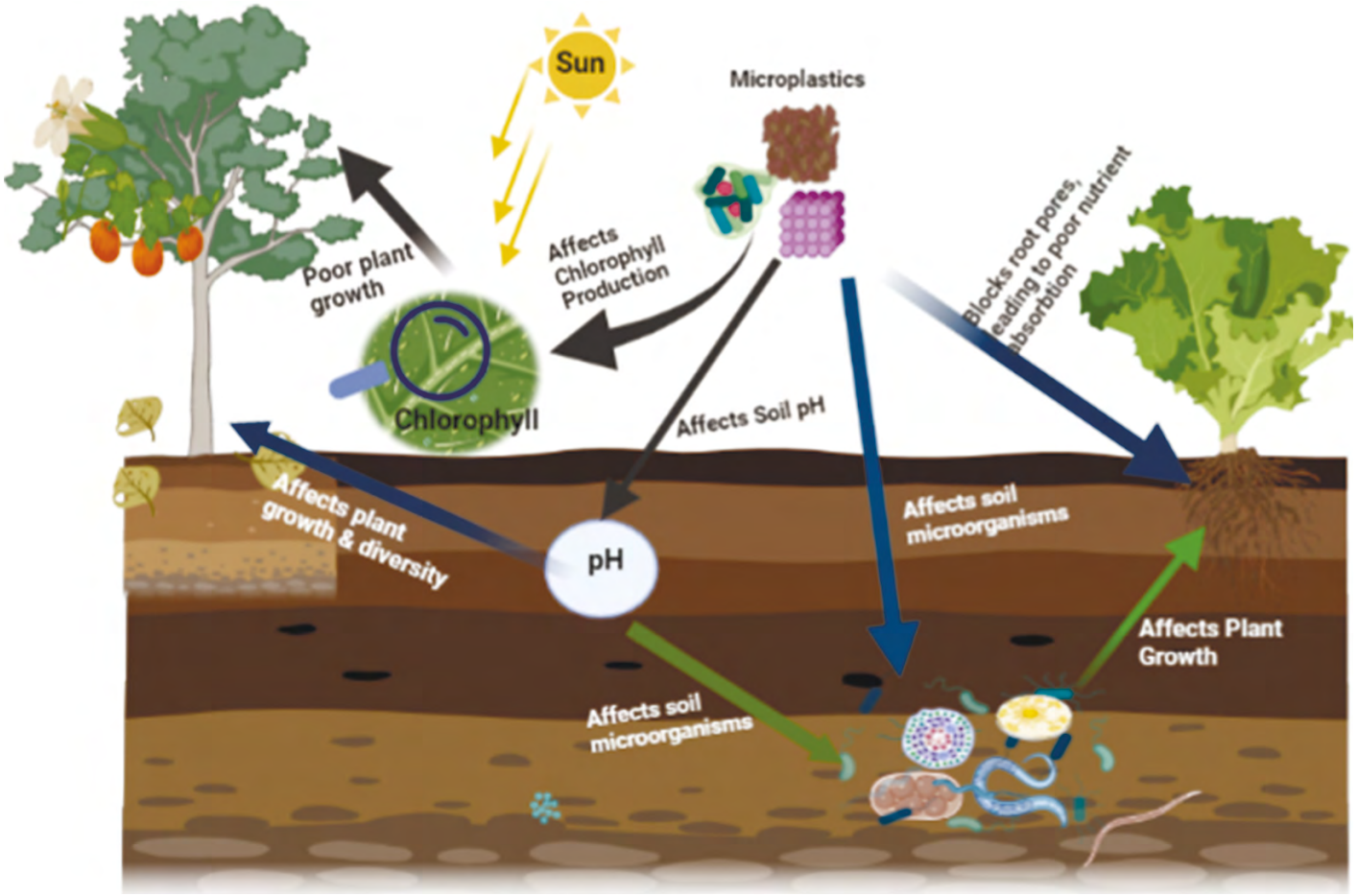


Figure 3.9.

Occurrence, transport, and the fate of MNPs in agroecosystems. MNPs are distributed in agroecosystems horizontally and vertically, affecting plant diversity, soil biota, and nutrient cycling. This results in severe ecological risks.

Source: Okeke, E. S., Chukwudozie, K. I., Addey, C. I., et al. Micro and nanoplastics ravaging our agroecosystem: A review of occurrence, fate, ecological impacts, detection, remediation, and prospects. Heliyon 9, e13296 (2023).

https://doi.org/10.1016/j.heliyon.2023.e13296

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Increasing evidence indicates that micro- and nanoplastics (MNPs) may impair ecosystem functioning by disrupting soil processes, plant root systems, and microbial communities. These effects may have implications for key biogeochemical cycles, including carbon dynamics, although direct evidence is still lacking.

This concern arises in the context of already observed alterations in forest carbon balance: certain regions of the Amazon rainforest, particularly in its eastern areas, have been reported to exhibit a shift in which carbon emissions exceed carbon sequestration.[56]

[56] *Maia, V. A., Santos, A. B. M., De Aguiar-Campos, N., et al. The carbon sink of tropical seasonal forests in southeastern Brazil can be under threat. Sci. Adv. 6, eabd4548 (2020). https://doi.org/10.1126/sciadv.abd4548*

ANIMALS

In the context of the described mechanisms of micro- and nanoplastics impacts on forest ecosystems and habitats, it is important to consider the general trend of declining global wildlife.[57]

> *"Despite specific differences, the network of electrical signaling is present at almost each level of complexity, from unicellular bacteria and fungi to multi-cellular organisms like plants and animals."*[58]

Plastic pollution constitutes a major global challenge for most ecosystems, affecting both terrestrial and aquatic environments.

Concurrently, Earth's global ecosystems are already in a state of deep crisis. According to the World Wildlife Fund, wildlife populations have declined by more than 70% over the last 50 years due to habitat loss, climate change, and ecosystem degradation.[57]

In this context, plastic pollution becomes an exacerbating factor of pressure on the living organisms.

Plastic waste exerts a direct mechanical impact on animals. Around 100,000 of species, including marine mammals and birds, die every year from plastic ingestion or entanglement in it.[59] At the same time, micro- and nanoplastics pose a particular danger. Small-sized electrostatically charged plastic particles are capable of penetrating organisms and affecting their physiological functions.

Micro- and nanoplastics can even reach the central nervous system of animals, including brain tissue (Fig. 3.10).[60] These particles can damage brain cells and disrupt the transmission of nerve impulses (Fig. 3.11).[61]

[57] *WWF. 2024 Living Planet Report. A System in Peril. (WWF, Gland, Switzerland, 2024). https://livingplanet.panda.org/ (Accessed 20 March 2026).*

[58] *Szechyńska-Hebda, M., Lewandowska, M. & Karpiński, S. Electrical Signaling, Photosynthesis and Systemic Acquired Acclimation. Front. Physiol. 8, 684 (2017). https://doi.org/10.3389/fphys.2017.00684*

[59] *WWF-Australia. Plastic in Our Oceans Is Killing Marine Mammals. (2025) https://wwf.org.au/blogs/plastic-in-our-oceans-is-killing-marine-mammals/ (Accessed 20 March 2026).*

[60] *Su, M., Gan, S., Gao, R., et al. Toxicity Mechanisms of Microplastic and Its Effects on Ruminant Production: A Review. Biomolecules 15, 462 (2025). https://doi.org/10.3390/biom15040462*

[61] *Vojnits, K., De León, A., Gibon, J., et al. A systematic review of the potential neurotoxicity of micro-and nanoplastics: the known and unknown. Part Fibre Toxicol 22, 29 (2025). https://doi.org/10.1186/s12989-025-00647-4*

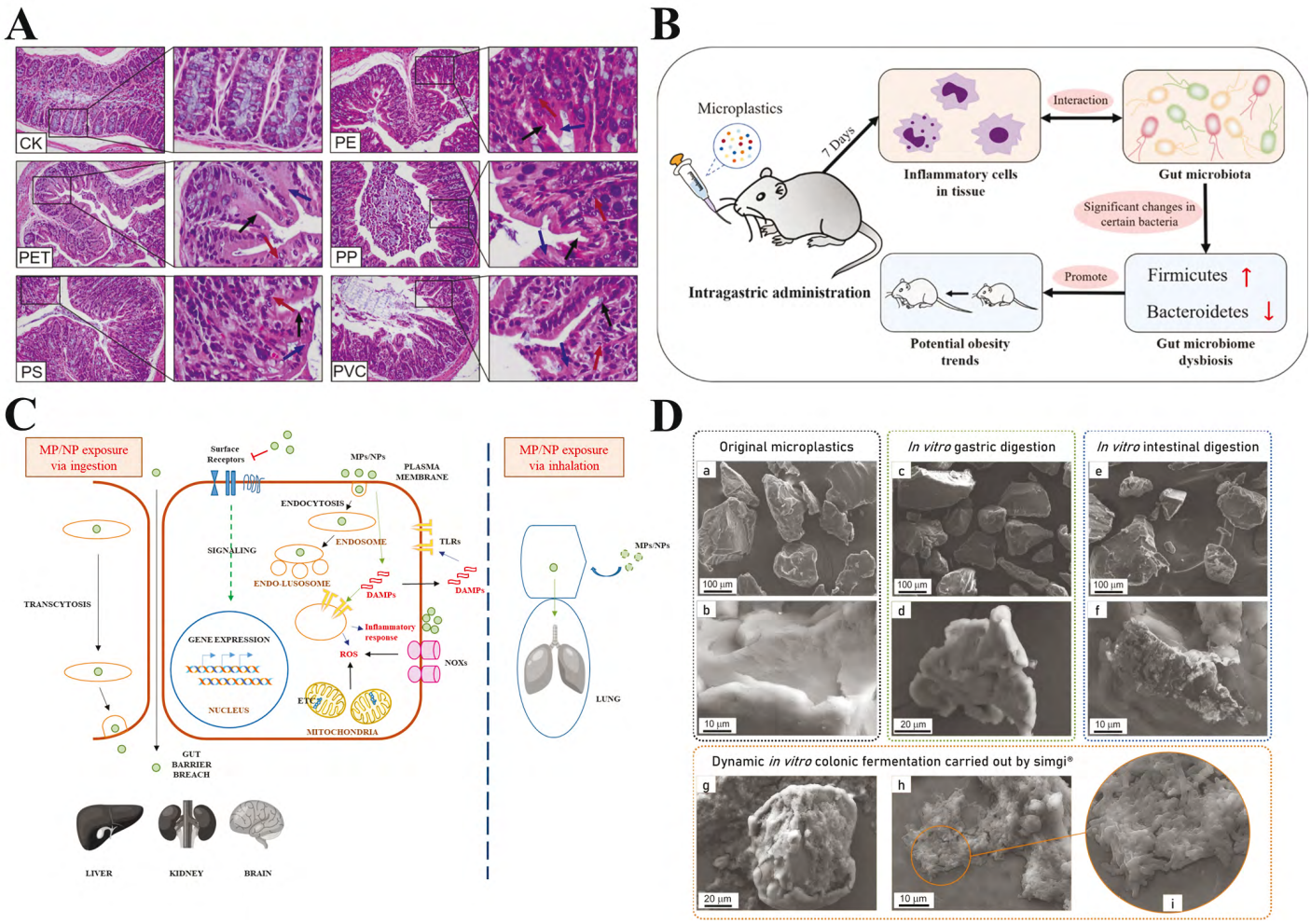


Figure 3.10.

Effects of MP exposure on mice, potential cytotoxic mechanisms of MPs/NPs, and scanning electron microscopy of PET MPs.

Source: Su, M., Gan, S., Gao, R., et al. Toxicity Mechanisms of Microplastic and Its Effects on Ruminant Production: A Review. Biomolecules 15, 462 (2025). https://doi.org/10.3390/biom15040462

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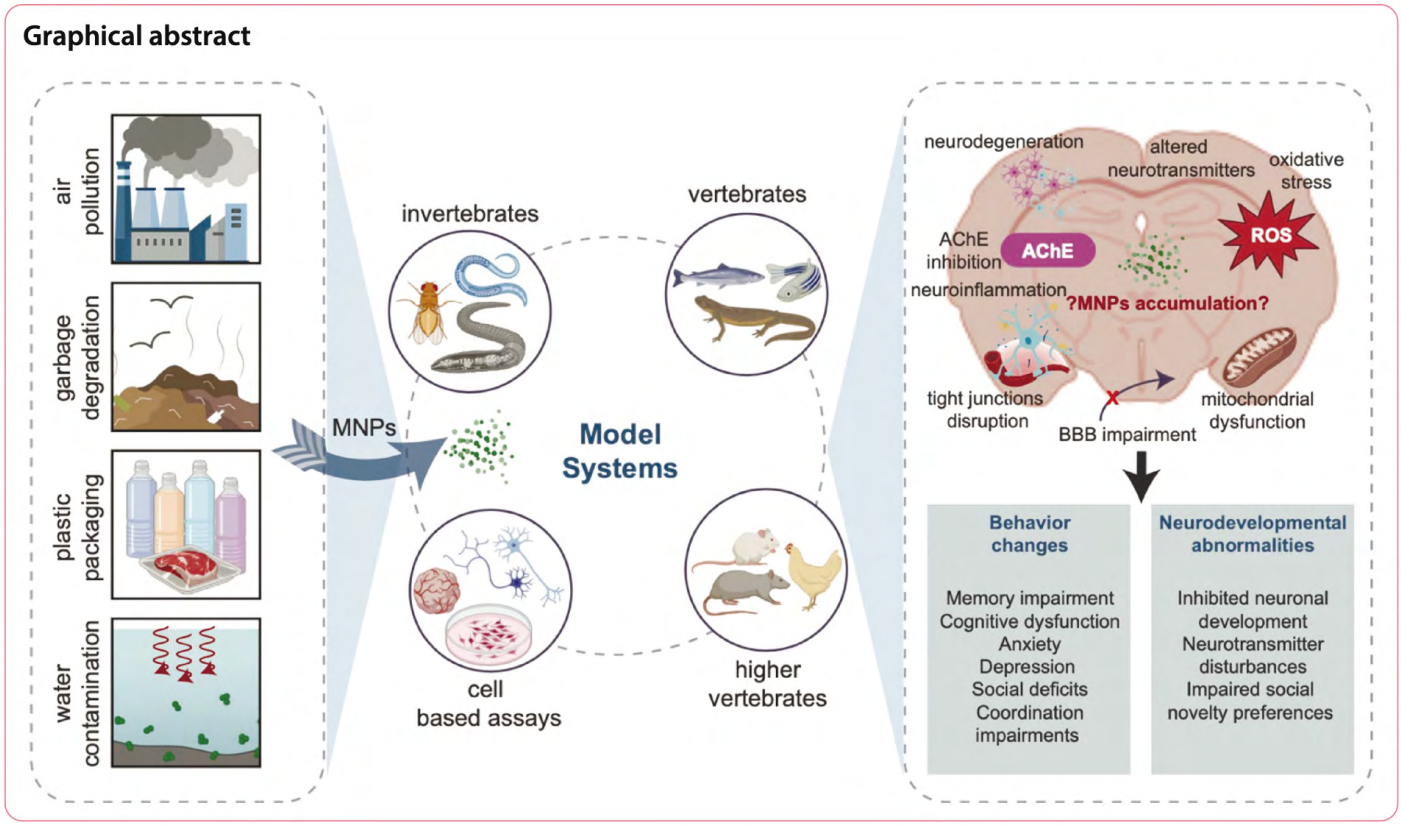


Figure 3.11.

Schematic illustration of neurotoxic micro- and nanoplastics outcomes, such as oxidative stress, cholinergic dysfunction, neurotransmitter imbalances, and neuronal apoptosis. They vary widely depending on particle size, shape, polymer type, exposure concentration, and host species.

Source: Vojnits, K., De León, A., Gibon, J., et al. A systematic review of the potential neurotoxicity of micro-and nanoplastics: the known and unknown. Part Fibre Toxicol 22, 29 (2025). https://doi.org/10.1186/s12989-025-00647-4 License: CC BY 4.0.

These conditions are associated with changes in behavior and motor function, indicating a potential risk of neurological impairment.

Neurological changes induced by exposure to MNPs reduce the ability of animals to adapt to their natural environments. They face increasing difficulty navigating, foraging effectively, avoiding predators, and interacting with others of their species.

The cumulative exposure to MNPs may reduce the probability of survival and reproductive performance in animals (Fig. 3.12).[62] This allows the potential neurotoxic effects of MNPs to be viewed as a significant environmental risk factor capable of influencing population resilience and ecosystem functioning.

[62] *Dindo, S., Tovar-Pascual, L., Baldassarro, V. A., et al. Nanoplastics internalization impairs mitochondrial activity in equine sperm. Theriogenology 250, 117711 (2026). https://doi.org/10.1016/j.theriogenology.2025.117711*

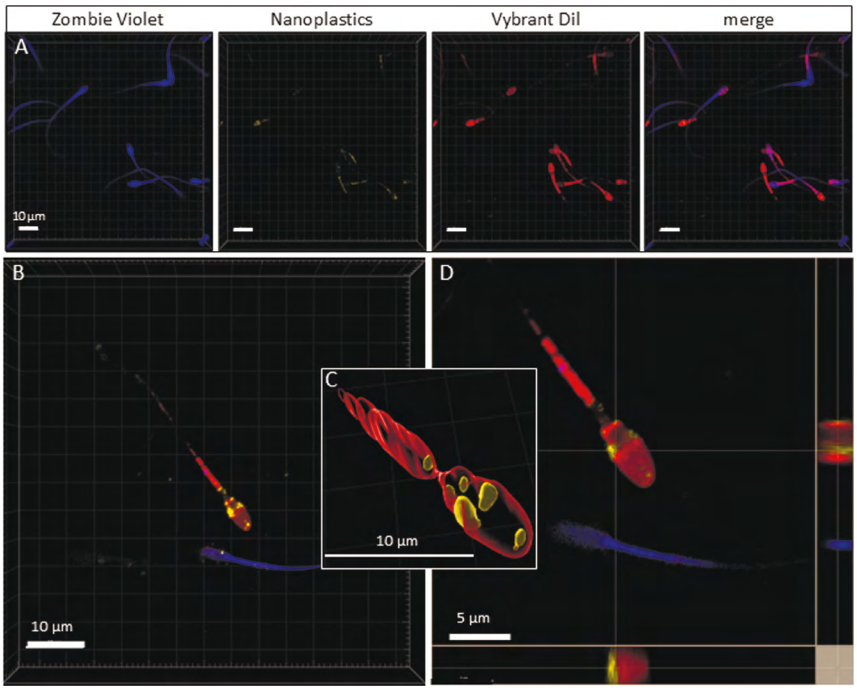


Figure 3.12.

Representative images of internalization and localization of nanoplastics (NP) in live spermatozoa. Sperm were incubated for 1 h at 38 °C with 100 µg/mL fluorescent NP (yellow), and stained with Zombie Violet (blue, dead sperm) and Vybrant Dil (red, membrane marker).

Source: Dindo, S., Tovar-Pascual, L., Baldassarro, V. A., et al. Nanoplastics internalization impairs mitochondrial activity in equine sperm. Theriogenology 250, 117711 (2026). https://doi.org/10.1016/j.theriogenology.2025.117711

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The impact of micro- and nanoplastics is not limited to wildlife. Signs of contamination are also being recorded in domestic animals as well. Notably, polyethylene terephthalate (PET), a plastic widely used in the production of bottles, has been detected in pet food (Fig. 3.13).[63]

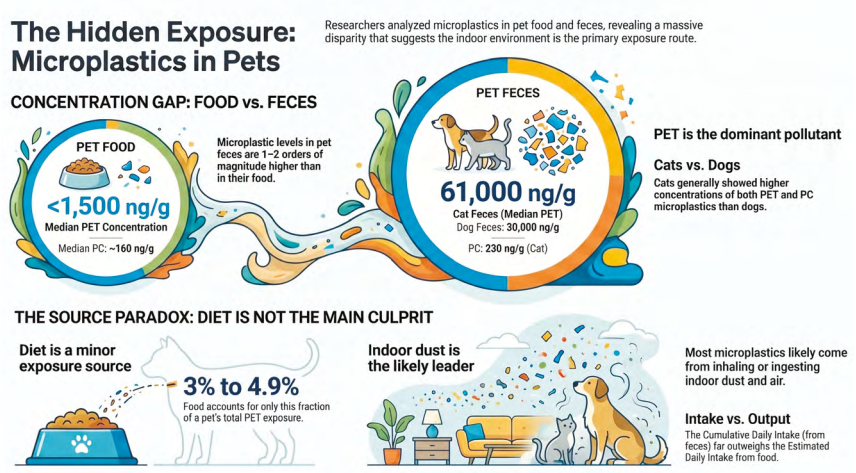


Figure 3.13.

PET was detected at concentrations in the range of <1,500 ng/g to 12,000 ng/g (median: <1,500 ng/g) and <1,500 to 4,600 ng/g (median: <1,500 ng/g) in cat and dog foods, respectively.

Data source: Zhang, J., Wang, L. & Kannan, K. Polyethylene Terephthalate and Polycarbonate Microplastics in Pet Food and Feces from the United States. Environ. Sci. Technol. 53, 12035–12042 (2019).

https://doi.org/10.1021/acs.est.9b03912

Furthermore, microplastic particles have been found in the tissues of cats and dogs. One study analyzed tissue samples from 49 animals (24 cats and 25 dogs) that died of natural causes. It revealed that in more than 70% of the animals, microplastics were detected in multiple organs simultaneously, pointing to the systemic nature of this accumulation.[64]

[63] *Zhang, J., Wang, L. & Kannan, K. Polyethylene Terephthalate and Polycarbonate Microplastics in Pet Food and Feces from the United States. Environ. Sci. Technol. 53, 12035–12042 (2019). https://doi.org/10.1021/acs.est.9b03912*

[64] *Prata, J. C., Silva, A. L. P., da Costa, J. P., et al. Microplastics in Internal Tissues of Companion Animals from Urban Environments. Animals 12, 1979 (2022). https://doi.org/10.3390/ani12151979*

INSECTS

The extinction rate of insects is 8 times higher than that of mammals, birds, and reptiles.[65]

More than 75 % of the world's leading food crops—including many fruits, vegetables, nuts, and oilseeds—depend, to varying degrees, on animal pollinators, particularly bees.[66] As such, pollinators represent a highly vulnerable group of considerable ecological and socioeconomic importance.

Because of this, the steady decline in bee populations observed across many regions is seen as a key indicator of ecosystem degradation. There is compelling evidence of a significant reduction in both wild and domestic honeybee populations.[67,68] If current trends continue, there will be further aggravation of the situation, potentially leading to substantial population losses in the coming years. Bee die-off signifies a disruption in the functional link connecting plant communities and food chains, natural processes, and the stability of human life support systems.

In recent years, growing evidence has suggested a potential link between microplastic pollution in the environment and the decline of insect populations.[69,70]

The vulnerability of bees to such impacts stems not only from their constant contact with the environment, but also from the specifics of their sensory and behavioral organization. The way pollinators interact with the world is largely based on subtle signaling mechanisms, including the perception of electrical stimuli.[71] Bees navigate not only by a flower's scent, color, and shape, but are also capable of sensing floral electric fields (Fig. 3.14).[72]

When a bee approaches a flower, an electric interaction occurs. During flight (Fig. 3.15), the bee's body becomes electrified due to air friction and acquires a positive charge, whereas flowers generally remain grounded and hold a negative charge.[73,74]

[65] *United Nations Environment Programme (UNEP). Insect Declines Are a Stark Warning to Humanity. (2019) https://www.unep.org/news-and-stories/story/insect-declines-are-stark-warning-humanity (Accessed 20 March 2026).*

[66] *Potts, S. G., Biesmeijer, J. C., Kremen, C., et al. Global pollinator declines: trends, impacts and drivers. Trends in Ecology & Evolution 25, 345–353 (2010). https://doi.org/10.1016/j.tree.2010.01.007*

[67] *Kohl, P. L. & Rutschmann, B. European wild honeybee populations are endangered. EcoEvoRxiv preprint (2025). https://doi.org/10.32942/X2SW5M*

[68] *Zattara, E. E. & Aizen, M. A. Worldwide occurrence records suggest a global decline in bee species richness. One Earth 4, 114–123 (2021). https://doi.org/10.1016/j.oneear.2020.12.005*

[69] *Reddy, N. K. S., Kamal, A. P., Sathwika, N. & Sadafale, G. V. R. Microplastic pollution and its effects on insects: a hidden crisis in our ecosystems. Indian Farmer 12, 116–120 (2025).*

[70] *Abbas, M., Jafir, M., Nazir, T., et al. A global meta-analysis reveals the toxicity of plastics on insect health. Ecotoxicology and Environmental Safety 302, 118611 (2025). https://doi.org/10.1016/j.ecoenv.2025.118611*

[71] *Clarke, D., Whitney, H., Sutton, G., et al. Detection and Learning of Floral Electric Fields by Bumblebees. Science 340, 66–69 (2013). https://doi.org/10.1126/science.1230883*

[72] *Clarke, D., Morley, E. & Robert, D. The bee, the flower, and the electric field: electric ecology and aerial electroreception. J Comp Physiol A 203, 737–748 (2017). https://doi.org/10.1007/s00359-017-1176-6*

[73] *Montgomery, C., Koh, K. & Robert, D. Measurement of electric charges on foraging bumblebees (Bombus terrestris). J. Phys.: Conf. Ser. 1322, 012002 (2019). https://doi.org/10.1088/1742-6596/1322/1/012002*

[74] *AskNature (Biomimicry Institute). Bees and Flowers Harness Static Electricity to Spread Pollen. (2024) https://asknature.org/strategy/bees-and-flowers-harness-static-electricity-to-spread-pollen/ (Accessed 20 March 2026).*

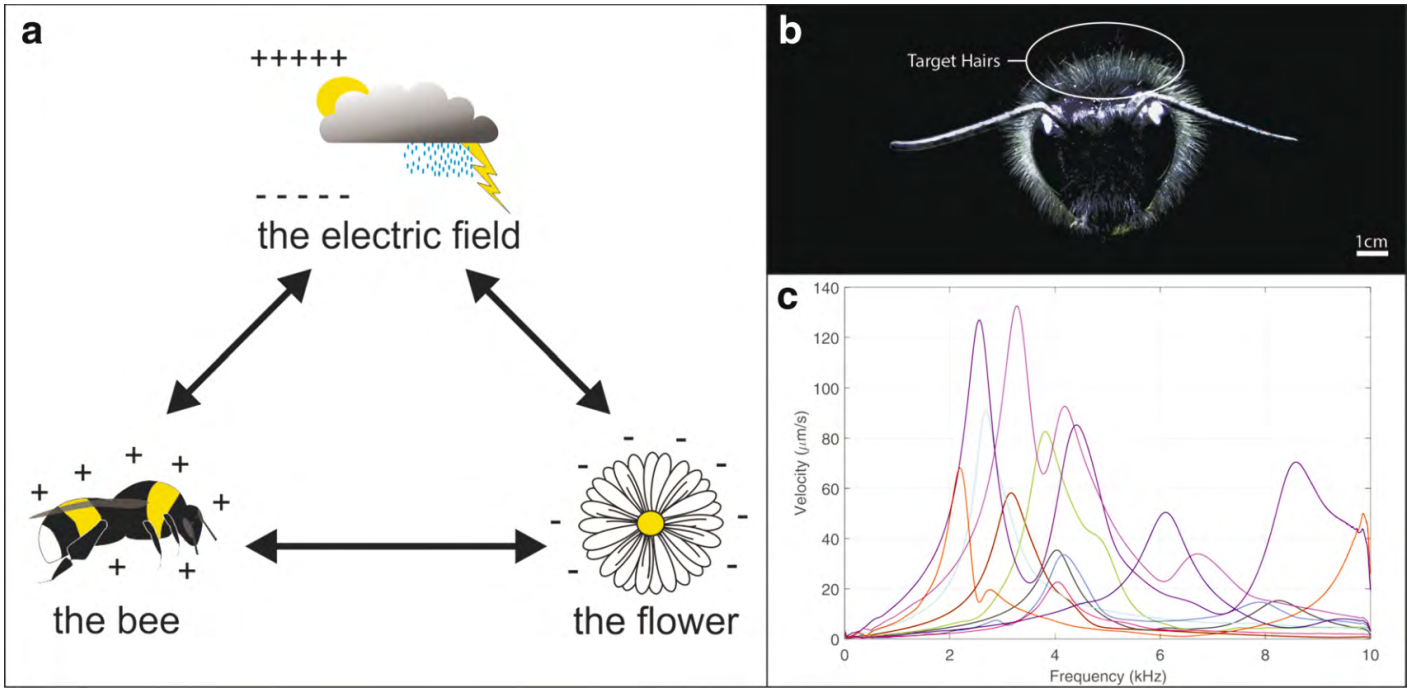


Figure 3.14.

Electromechanical reception in bumblebees and electrical ecology of pollination. a Interactions between bee, flower, and atmospheric electric field cannot be separated, as each of them influence the other. b Frontal confocal photograph of a bumble bee illustrating target hairs used to establish the site of electromechanical detection of electric fields in bumble bees. c Mechanical response of diverse bumble bee hairs to an electric field stimulus.

Source: Clarke, D., Morley, E. & Robert, D. The bee, the flower, and the electric field: electric ecology and aerial electroreception. J Comp Physiol A 203, 737–748 (2017). https://doi.org/10.1007/s00359-017-1176-6 License: CC BY 4.0.

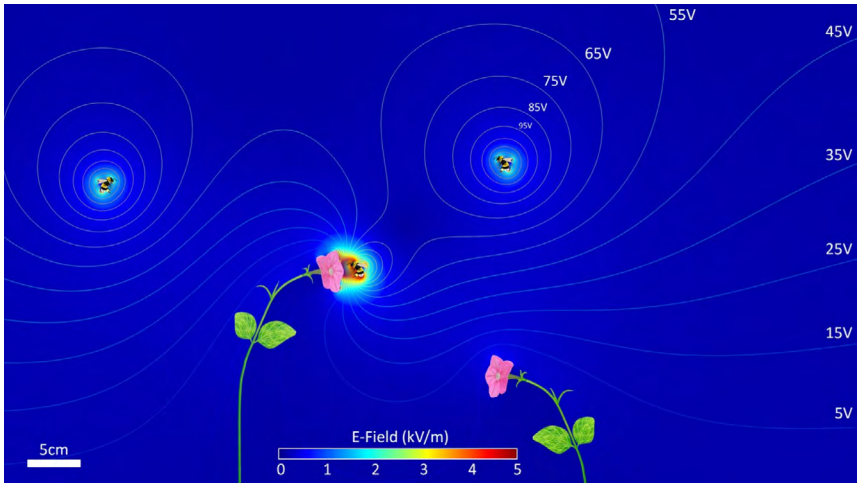


Figure 3.15.

Visualising electric ecology. Finite-element model of electric interactions between positively charged bumble bees and grounded petunias (Petunia sp.) against the background of the atmospheric potential gradient.

Source: Clarke, D., Morley, E. & Robert, D. The bee, the flower, and the electric field: electric ecology and aerial electroreception. J Comp Physiol A 203, 737–748 (2017). https://doi.org/10.1007/s00359-017-1176-6 License: CC BY 4.0.

The resulting electric field facilitates the transfer of pollen to the bee even before physical contact with the flower is made (Fig. 3.16).[72]

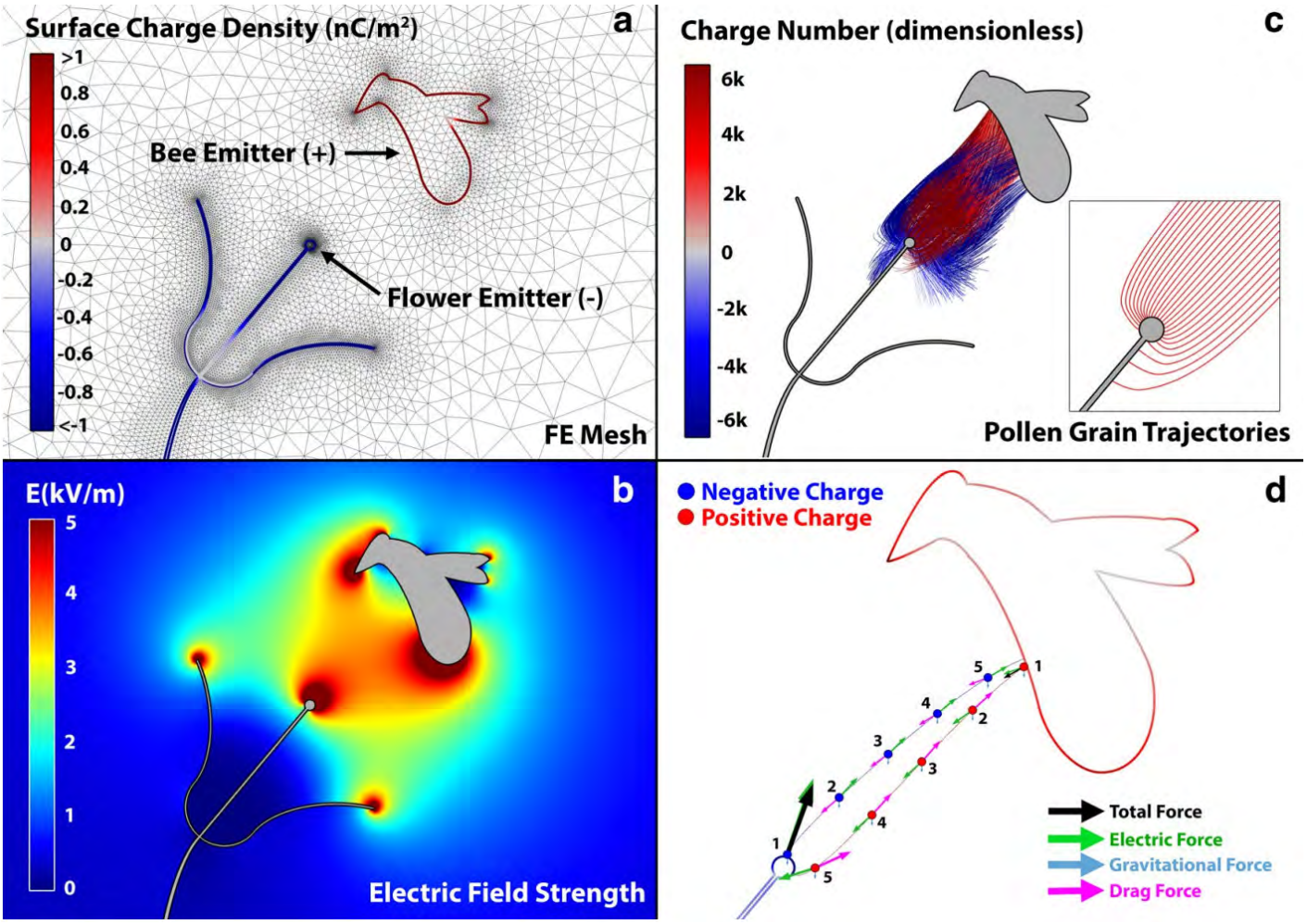


Figure 3.16.

Finite-element modelling and visualisation of bee/flower electrostatic interaction in pollen transfer.

a) Model construction, showing the finite-element optimised mesh, and surface charge density (scale bar, nC/m2).

b) Model solution, showing the electric field (kV/m) arising between bee and flower, resulting in electric field hot spots, where electrical forces on charges are higher.

c) Simulation of bidirectional pollen grain transfer between flower and bee, taking into account gravitational, electrical, and viscous drag forces.

d) Close-up finite-element visualisation of two pollen grains during their opposite journey from bee to flower (blue) and flower to bee (red). Pollen grain colour indicates its charge (red positive, blue negative).

Source: Clarke, D., Morley, E. & Robert, D. The bee, the flower, and the electric field: electric ecology and aerial electroreception. J Comp Physiol A 203, 737–748 (2017). https://doi.org/10.1007/s00359-017-1176-6 License: CC BY 4.0.

Clarke et al. further shows that bees are able to distinguish flowers by the parameters of their electric fields (Fig. 3.17), associate specific electrical signals with the presence of nectar, and evaluate the “freshness” of a flower based on the level of its residual charge.[72]

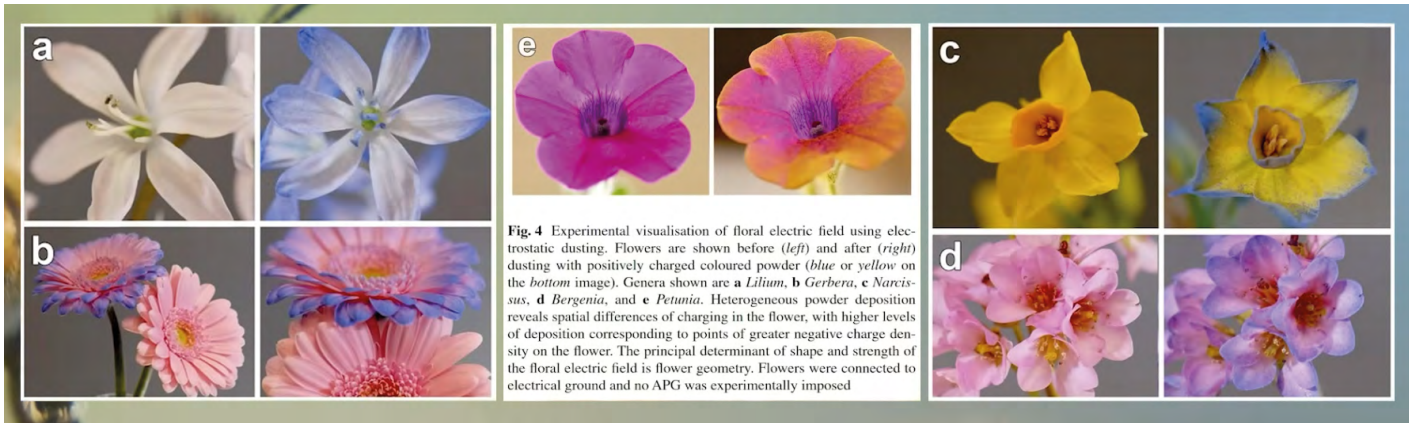


Figure 3.17.

Experimental visualisation of floral electric field using electrostatic dusting. Flowers are shown before (left) and after (right) dusting with positively charged coloured powder (blue or yellow on the bottom image). Genera shown are a Lilium, b Gerbera, c Narcissus, d Bergenia, and e Petunia. Heterogeneous powder deposition reveals spatial differences of charging in the flower, with higher levels of deposition corresponding to points of greater negative charge density on the flower.

Source: Clarke, D., Morley, E. & Robert, D. The bee, the flower, and the electric field: electric ecology and aerial electroreception. J Comp Physiol A 203, 737–748 (2017). https://doi.org/10.1007/s00359-017-1176-6 License: CC BY 4.0.

After a bee visits a flower, its electric field weakens, which other bees can perceive as an indirect sign of lower nectar availability.[71,75]

Moreover, a bee's electric charge can stimulate a plant[71] to increase its scent emission, which in turn enhances the flower's attractiveness to other pollinators. Thus, the interaction between bees and flowering plants involves not only chemical and visual signals, but also electrical signaling mechanisms.

Electrostatically charged micro- and nanoplastic particles can be viewed as a factor that potentially disrupts these finely tuned sensory systems of bees.

MNPs can distort electrical signals, affecting the bee's nervous system and cognitive functions. As a result, bees struggle to recognize flowers and nectar sources, lose the ability to navigate effectively (in particular, returning to the hive), and exhibit impairments in social recognition. Although the severity of effects increases with concentration, even low or environmentally relevant concentrations may affect insect fitness.[76]

[75] *Montgomery, C., Vuts, J., Woodcock, C. M., et al. Bumblebee electric charge stimulates floral volatile emissions in Petunia integrifolia but not in Antirrhinum majus. Sci Nat 108, 44 (2021). https://doi.org/10.1007/s00114-021-01740-2*

[76] *Pasquini, E., Ferrante, F., Passaponti, L., et al. Microplastics reach the brain and interfere with honey bee cognition. Science of The Total Environment 912, 169362 (2024). https://doi.org/10.1016/j.scitotenv.2023.169362*

The physiological vulnerability of bees is aggravated by their specific morphology and lifestyle: a bee's body is covered in sensitive hairs, and it is constantly in contact with plant surfaces, the air, and aerosol particles. Under such conditions, microplastics can settle on the insects' bodies and, as hypothesized, enter their organisms relatively easily.

According to research by Professor David Baracchi (University of Florence), microplastics can penetrate a bee's body through the cuticle or by ingestion, and reach the brain within 72 hours (Fig. 3.18). This is followed by synaptic destruction, neuroinflammation, cell death, and neurotransmitter disruption. The bee loses mass, its metabolism slows down, and its immunity decreases.[76]

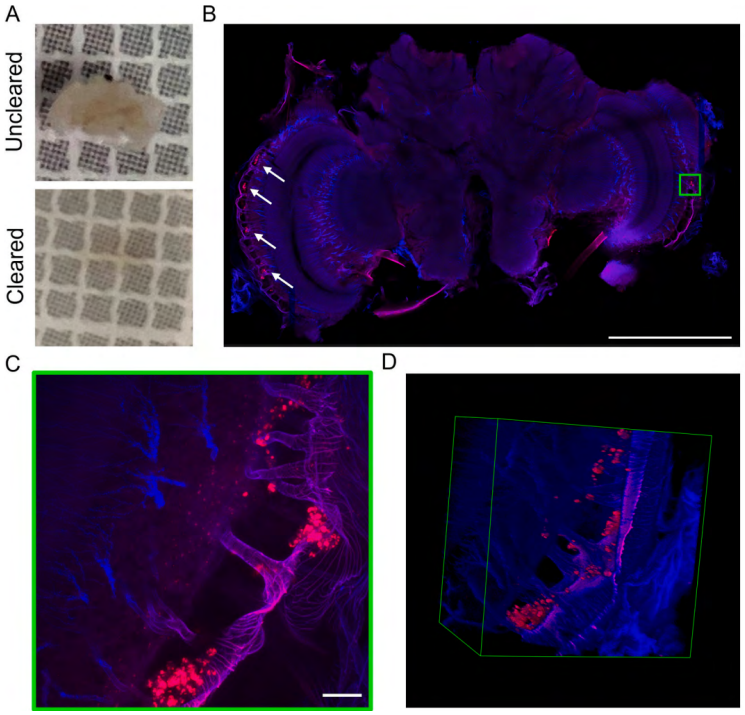


Figure 3.18.
Detection of significant amounts of microplastics in honeybee brain.
A) Photos of a dissected brain before and after the iDISCO clearing.
B) Single plane (depth ~ 200 µm) of a 3D reconstruction obtained with Two-Photon Fluorescence Microscopy (TPFM) of the whole brain with a 10× objective, resolution of 0.51 × 0.51 × 2 µm³. In blue = autofluorescence signal of the tissue; in red = red fluorescence MPs (pointed with white arrows). Scale bar = 1000 µm.
C) High-resolution inset of the green region of interest in B acquired with a 63× objective. The image is the maximum intensity projection of a 150 µm depth stack quired with a resolution of 0.17 × 0.17 × 1 µm³. Scale bar = 20 µm.
D) 3D rendering of the stack in C. Dimensions = 170 × 170 × 150 µm³.

Source: Pasquini, E., Ferrante, F., Passaponti, L., et al. Microplastics reach the brain and interfere with honey bee cognition. Science of The Total Environment 912, 169362 (2024). https://doi.org/10.1016/j.scitotenv.2023.169362 License: CC BY 4.0.

Moreover, bees exhibit behavioral changes, including impairments in learning and memory.[76] It is indicated that under experimental conditions, polystyrene at concentrations that may occur in the environment can impair memory formation and retrieval in honeybees. This is assessed as particularly alarming, since learning and memory skills are critical for bees to successfully forage and navigate.

These effects add to the overall picture of the pollinator ecological crisis, highlighting the need to view micro- and nanoplastics as a potentially significant anthropogenic factor in the decline of insects and their associated ecosystem functions.

BIRDS

A steady and statistically confirmed trend of declining wild bird populations is being documented worldwide. According to observational data, North America alone has lost approximately 3 billion birds over the past 50 years, roughly one in every four individuals.[77]

This underscores the systemic nature of the ongoing changes and points to a serious deterioration in the state of natural ecosystems.

The biological functioning of the organism of birds is directly linked to the transmission of electrical signals, ensuring the operation of their nervous system and the coordination of vital processes. Nerve impulses traveling from the brain to organs and muscles control the movement of wings, tails, and bodies during flight. These impulses also ensure vision and the coordination of motor responses, participate in cognitive processes (including threat assessment and predator avoidance), and regulate vocalization, i.e. singing and other acoustic signals. Thus, electrical mechanisms are one of the fundamental pillars of bird behavior and survival.

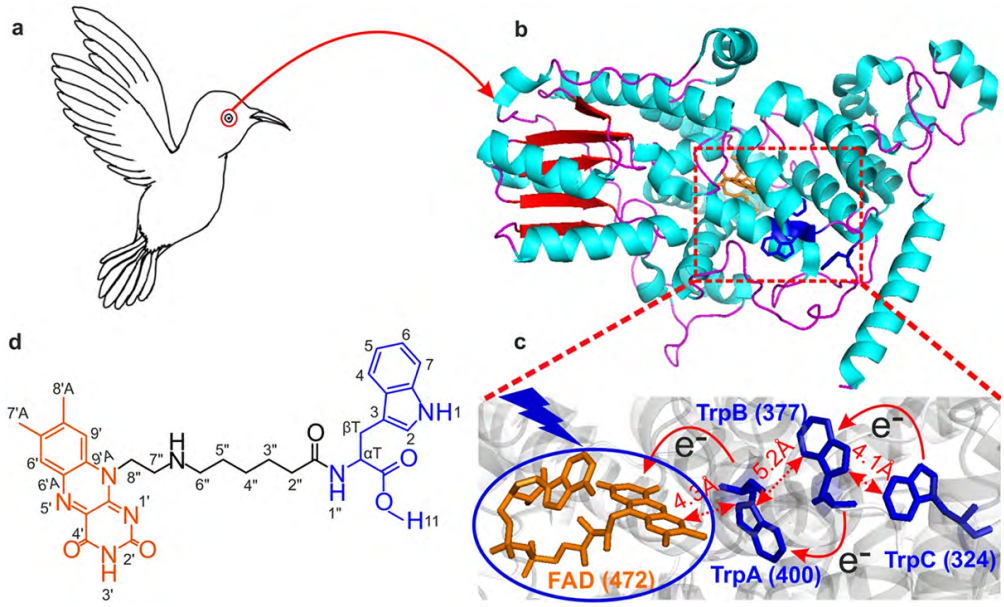


Figure 3.19.

Structural representation of natural cryptochrome and artificial dyad F10T. **(a,b)** Migratory bird's magnetic sense is based on the protein cryptochrome [PDB entry 1U3D], found in the retina. **(c)** Relative positions of the FAD cofactor and the Trp-triad (TrpA, TrpB and TrpC) of Arabidopsis thaliana cryptochrome. The FAD cofactor is colored in orange and the tryptophans are shown in blue. **(d)** Flavin-tryptophan dyad F10T consisting of a flavin residue (orange) and a tryptophan (blue) connected by an aliphatic linker (black).

Source: Paul, S., Kiryutin, A. S., Guo, J., et al. Magnetic field effect in natural cryptochrome explored with model compound. Sci Rep 7, 11892 (2017). https://doi.org/10.1038/s41598-017-10356-4 License: CC BY 4.0.

[77] *Rosenberg, K. V., Dokter, A. M., Blancher, P. J., et al. Decline of the North American avifauna. Science 366, 120–124 (2019). https://doi.org/10.1126/science.aaw1313*

A unique aspect of bird biology is their capacity for long-distance migration and high-precision navigation. Many migratory species of birds travel thousands of miles and return to the same locations year after year.

According to scientific studies, the Earth's magnetic field serves as one of the key points of reference during migration. It has been proposed that the primary detector is a specialized molecular mechanism in the birds' visual systems (Fig. 3.19) that allows them to perceive magnetic field parameters (Fig. 3.20) and convert them into electrical signals.[78,79] As a result, magnetic information is integrated in the brain with visual and other sensory inputs, potentially providing an additional directional reference that supports stable orientation during long-distance migration. This process can be metaphorically described as if the visual scene were subtly overlaid with directional cues or faint "patterns" linked to the Earth's magnetic field, although the exact nature of this perception remains unclear.

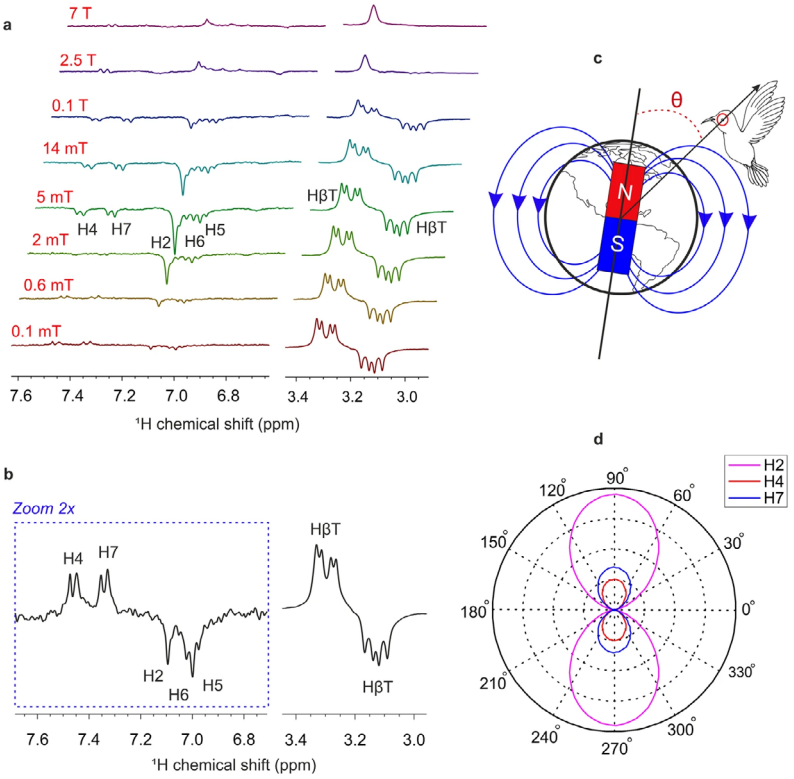


Figure 3.20.

Magnetic field effect on ^{1}H photo-CIDNP NMR spectra of F10T. **(a)** ^{1}H photo-CIDNP NMR spectra of F10T (in methanol-d_4) obtained at different polarizing magnetic field (B_P) strengths: (i) 0.1 mT, (ii) 0.6 mT, (iii) 2 mT, (iv) 5 mT, (v) 14 mT, (vi) 0.1 T, (vii) 2.5 T, (viii) 7 T. **(b)** ^{1}H photo-CIDNP NMR spectra of F10T obtained with the Earth's magnetic field as the polarizing magnetic field (B_P). **(c)** Magnetic force lines of Earth's magnetic field. **(d)** Proposed variation of ^{1}H photo-CIDNP intensities with the change of direction in fully freezed and perfectly aligned F10T dyad.

Source: Paul, S., Kiryutin, A. S., Guo, J., et al. Magnetic field effect in natural cryptochrome explored with model compound. Sci Rep 7, 11892 (2017). https://doi.org/10.1038/s41598-017-10356-4 License: CC BY 4.0.

In this regard, the potential impact of micro- and nanoplastics on the neurophysiological processes in birds is of serious concern. Upon entering the body of birds, electrostatically charged MNPs can potentially distort the transmission of electrical impulses and disrupt nervous system function. These disruptions are linked to impaired motor coordination, weakening of memory and learning ability, changes in vocal activity, and reduced accuracy of spatial orientation. At the behavioral level, such changes may manifest as more chaotic flight patterns, unstable reactions, and weakened vocalization.

[78] *Rodgers, C. T. & Hore, P. J. Chemical magnetoreception in birds: The radical pair mechanism. Proc. Natl. Acad. Sci. U.S.A. 106, 353–360 (2009). https://doi.org/10.1073/pnas.0711968106*

[79] *Paul, S., Kiryutin, A. S., Guo, J., et al. Magnetic field effect in natural cryptochrome explored with model compound. Sci Rep 7, 11892 (2017). https://doi.org/10.1038/s41598-017-10356-4*

Beyond neurophysiological effects, plastic affects internal organs (Fig. 3.21), including the liver, kidneys, stomach, and brain.Researchers have described symptoms similar to neurodegenerative changes, as well as a decline in the behaviors needed for reproductive performance. Even outwardly healthy birds may lose the ability to fully care for their mates and offspring, leading to reduced reproductive performance and, consequently, exacerbating negative population dynamics.[80,81]

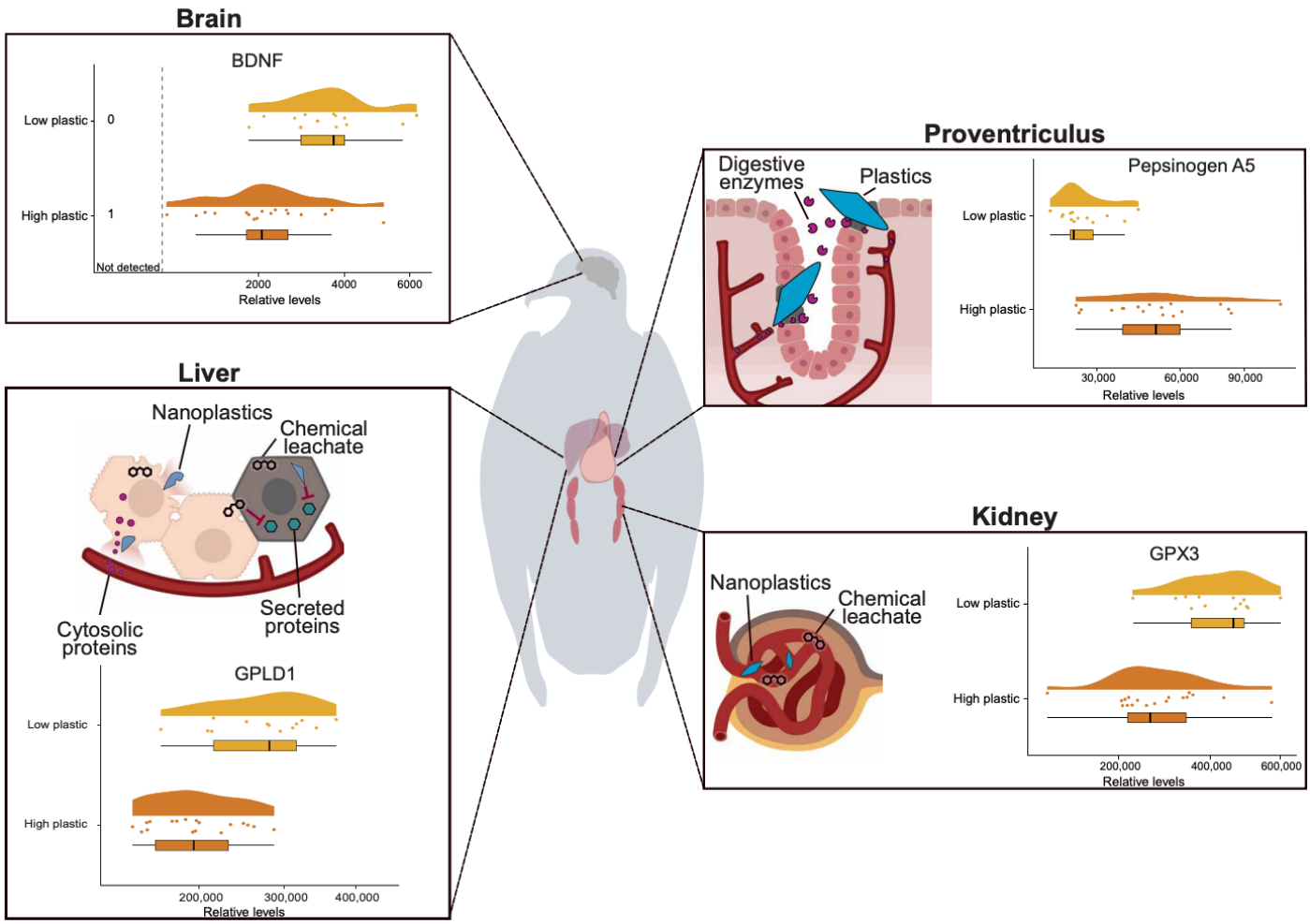


Figure 3.21.

Plastic ingestion induces proteomic signatures distal to the site of exposure.

Anatomical diagram of sable shearwater (A. carneipes) exposed to low (n = 13) and high (n = 18) quantities of ingested plastics. Proteomics on the plasma of chicks exposed to high quantities of ingested plastic revealed loss of brain, liver, and kidney function and stomach integrity disruption. Proteins of interest include BDNF (brain-derived neurotrophic factor), GPLD1 (glycosylphosphatidylinositol-specific phospholipase D1), GPX3 (glutathione peroxidase 3), and PGA5 (pepsinogen A5).

Source: De Jersey, A. M., Lavers, J. L., Bond, A. L., et al. Seabirds in crisis: Plastic ingestion induces proteomic signatures of multiorgan failure and neurodegeneration. Sci. Adv. 11, eads0834 (2025). https://doi.org/10.1126/sciadv.ads0834 License: CC BY 4.0.

[80] *De Jersey, A. M., Lavers, J. L., Bond, A. L., et al. Seabirds in crisis: Plastic ingestion induces proteomic signatures of multiorgan failure and neurodegeneration. Sci. Adv. 11, eads0834 (2025). https://doi.org/10.1126/sciadv.ads0834*

[81] *Carrasco, L., Jiménez-Mora, E., Utrilla, M. J., et al. Birds as Bioindicators: Revealing the Widespread Impact of Microplastics. Birds 6, 10 (2025). https://doi.org/10.3390/birds6010010*

The most vulnerable group appear to be fledglings (Fig. 3.22), as their nervous systems are in a stage of active formation.[80]

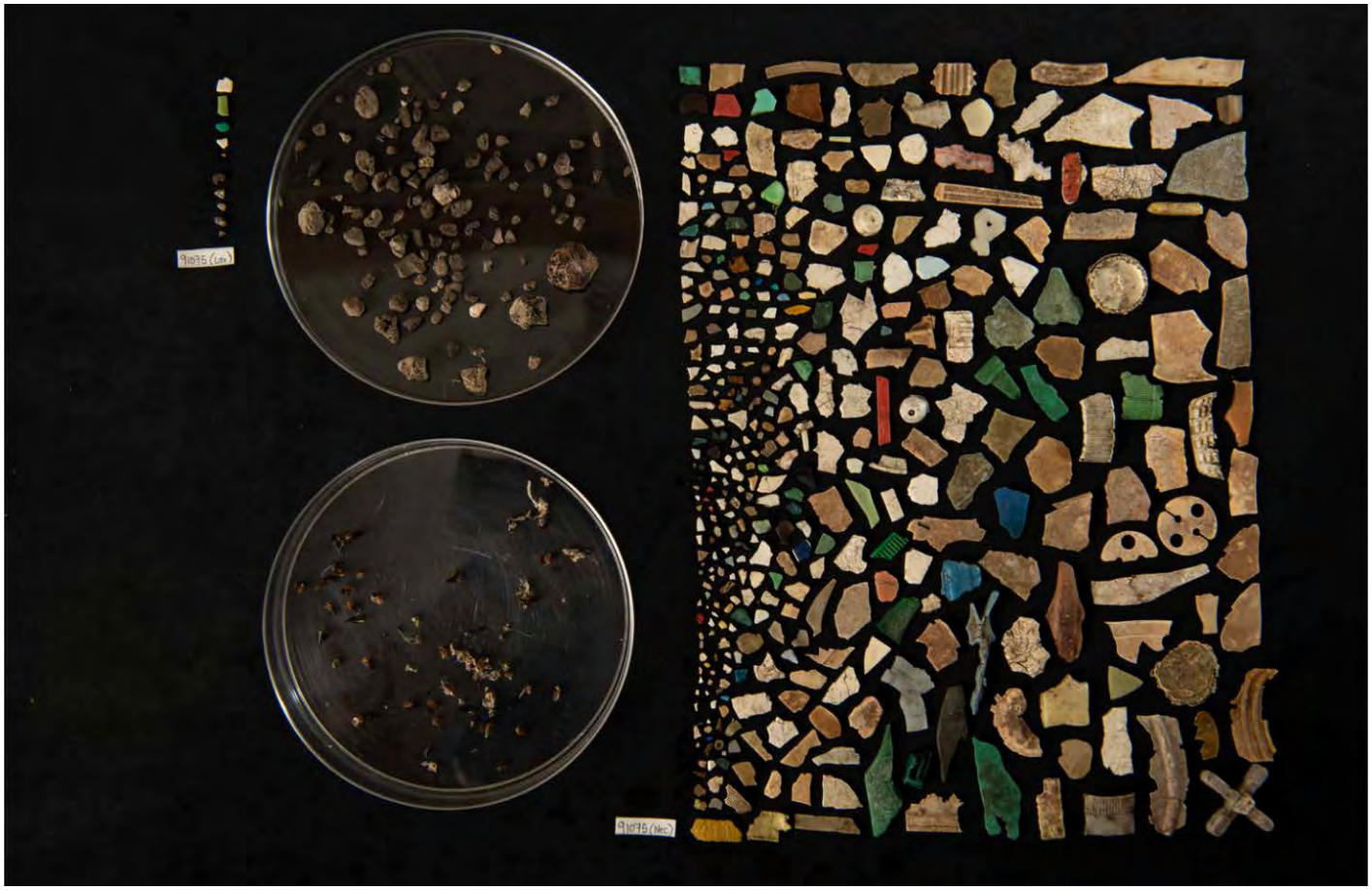


Figure 3.22.

Four hundred three pieces of plastic removed from sable shearwater A. carneipes 90-day-old chick. On the left are five plastic items (0.073 g) removed during lavage, and on the right are the 398 items (41.812 g) that remained inside the stomach after lavage. The 398 items could not be removed during lavage as the large volume of plastic created a blockage. Instead, these items were recorded during necropsy after the bird was deceased. Pumice (128 pieces, 6.819 g) and squid beaks (not counted) also ingested by the bird are displayed in the petri dishes. The image was captured by J. Gilligan. This is the largest count record of plastic ingestion published for this species.

Source: De Jersey, A. M., Lavers, J. L., Bond, A. L., et al. Seabirds in crisis: Plastic ingestion induces proteomic signatures of multiorgan failure and neurodegeneration. Sci. Adv. 11, eads0834 (2025). https://doi.org/10.1126/sciadv.ads0834
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MARINE LIFE

The scale of ecosystem transformation is especially evident in the dynamics of biological indices. Over the last 50 years, marine vertebrate populations have declined by 56%. The most pronounced decline is noted in freshwater ecosystems, where the figure reaches 85%.[82]

In underwater environments, the conditions for perception differ fundamentally from those on land. Vision often loses its precision, and sound signals are distorted by the physical properties of water. Due to that, many marine organisms use alternative sensory channels including electroreception — the ability to perceive and navigate by weak electric fields.

Electroreception is of key importance for the behavior and survival of many species. For instance, predators, including sharks, are able to detect extremely weak electrical impulses generated by the muscle and heart activity of potential prey.[83]

Even a stationary fish can be detected by bioelectric signals generated during its life processes. Since these fields are created by the constant flow of ions through the body's surfaces, bioelectric signals are a physiologically unavoidable component of living systems in aquatic environments. Their parameters can change as the organism moves or as other living objects appear nearby, providing additional sensory information.

In the given context, electrostatically charged micro- and nanoplastic particles can be considered a factor capable of distorting or blocking bioelectric signaling. Such interference with sensory communication and navigation potentially increases the vulnerability of aquatic organisms. Disruption of electroreceptor perception accuracy can lead to a loss of effectiveness in key behavioral strategies such as searching for prey, avoiding predators, and navigating during migrations. As a result, individual survival chances decrease and the risks of adverse consequences at the population level increase.

In addition to sensory disruptions, fish exposed to MNPs exhibit a wide range of toxic effects (Fig. 3.23), including immune system dysfunction, changes in blood parameters, signs of neurotoxicity, growth stunting, and a decline in reproductive function.[84]

[82] *WWF. 2024 Living Planet Report. A System in Peril. (WWF, Gland, Switzerland, 2024). https://livingplanet.panda.org/ (Accessed 20 March 2026).*

[83] *Kalmijn, A. J. The Electric Sense of Sharks and Rays. J Exp Biol 55, 371–383 (1971). https://doi.org/10.1242/jeb.55.2.371*

[84] *Ghosh, T. Microplastics bioaccumulation in fish: Its potential toxic effects on hematology, immune response, neurotoxicity, oxidative stress, growth, and reproductive dysfunction. Toxicology Reports 14, 101854 (2025). https://doi.org/10.1016/j.toxrep.2024.101854*

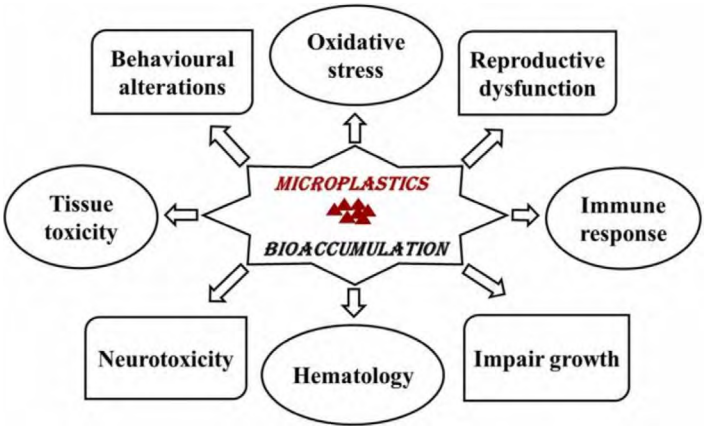


Figure 3.23.

Schematic representation of the potential toxic effects of microplastic accumulation in fish.

Source: Ghosh, T. Microplastics bioaccumulation in fish: Its potential toxic effects on hematology, immune response, neurotoxicity, oxidative stress, growth, and reproductive dysfunction. Toxicology Reports 14, 101854 (2025). https://doi.org/10.1016/j.toxrep.2024.101854 License: CC BY 4.0.

Thus, the impact of MNPs is evident not only in behavior and navigation, but also at the physiological level, affecting the mechanisms of resilience, development, and reproduction.

On a broader systemic level, it is important to recognize that processes within the biosphere are based on a complex network of interactions, a significant portion of which is mediated by subtle electrical and electrochemical signals. Electrostatically charged MNPs, which are widespread throughout the environment and integrated into various ecosystems, have the potential to disrupt such signaling mechanisms.

The examples provided above demonstrate only specific instances of a much larger problem: when signal transmission is impaired, biological connections weaken, organism functions destabilize, and ecosystem resilience declines.

Given the ubiquitous presence of MNPs and the trend of a rapid increase in the amount of plastic particles, there is a risk of systemic failures affecting various levels of biological organization, from the physiology of individual organisms to the stability of the biosphere as a whole. Since humans depend on the functioning of natural systems, air, water, and biological communities, biosphere degradation inevitably acquires the character of not only an ecological, but also an anthropogenic threat.

PART 4.

MICRO- AND NANOPLASTICS **AS A FACTOR OF CELLULAR DYSFUNCTION**: MECHANISMS AND BIOLOGICAL CONSEQUENCES

ELECTROPHYSIOLOGICAL FOUNDATION OF THE HUMAN BODY AND ITS VULNERABILITY TO MICRO- AND NANOPLASTICS

The human body can be viewed as a coherent biological system in which electrical processes play a key role in the coordination of cells, tissues, and organs.[85,86] Each cell maintains a membrane potential—an electrical charge difference between the inner and outer sides of the cell membrane (Fig. 4.1). This arises due to the unequal distribution of ions (sodium, potassium, calcium, chloride) and the selective permeability of the membrane. Ion channels and pumps (Fig. 4.2) constantly transport ions across the membrane, maintaining electrochemical gradients and ensuring the stability of the membrane potential. These mechanisms allow excitable cells (neurons, cardiomyocytes, muscle cells) to generate and conduct action potentials, which underlie neural transmission, the electrical activity of the heart, and muscle contraction. Maintaining ion balance and the electrical properties of cells is a prerequisite for the normal functioning of tissues and organs.[87]

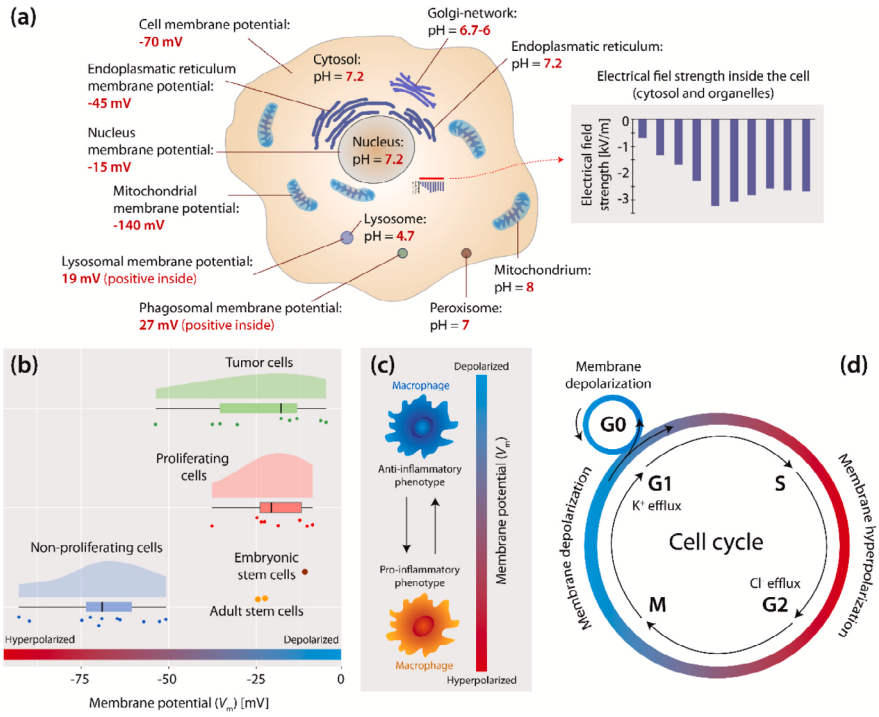


Figure 4.1.

Visualizations of the importance of the membrane potential (Vm) for cellular function. **(a)** Membrane potentials and pH values of subcellular structures of a cell. The values represent typical values measured. The insert shows measured electric field strength values (with a fluorescence probe) from a section inside the cell (covering cytosol and cell organelles). **(b)** Cell types can be grouped according to their Vm. **(c)** The phenotype of macrophages depends on Vm. **(d)** Vm during the cell cycle with hyperpolarization during the S and G2 phase and depolarization during the M G1 phase as well as at G0.

Source: Funk, R. H. W. & Scholkmann, F. The significance of bioelectricity on all levels of organization of an organism. Part 1: From the subcellular level to cells. Progress in Biophysics and Molecular Biology 177, 185–201 (2023). https://doi.org/10.1016/j.pbiomolbio.2022.12.002 License: CC BY 4.0.

[85] *Hall, J. E. & Hall, M. E. Guyton and Hall Textbook of Medical Physiology. (Elsevier, Philadelphia, PA, 2021).*

[86] *Funk, R. H. W. & Scholkmann, F. The significance of bioelectricity on all levels of organization of an organism. Part 1: From the subcellular level to cells. Progress in Biophysics and Molecular Biology 177, 185–201 (2023). https://doi.org/10.1016/j.pbiomolbio.2022.12.002*

[87] *Herrmann, T., Leavitt, L. & Sharma, S. Physiology, Membrane. in StatPearls [Internet] (StatPearls Publishing, 2023). https://www.ncbi.nlm.nih.gov/sites/books/NBK538211/ (Accessed 20 March 2026).*

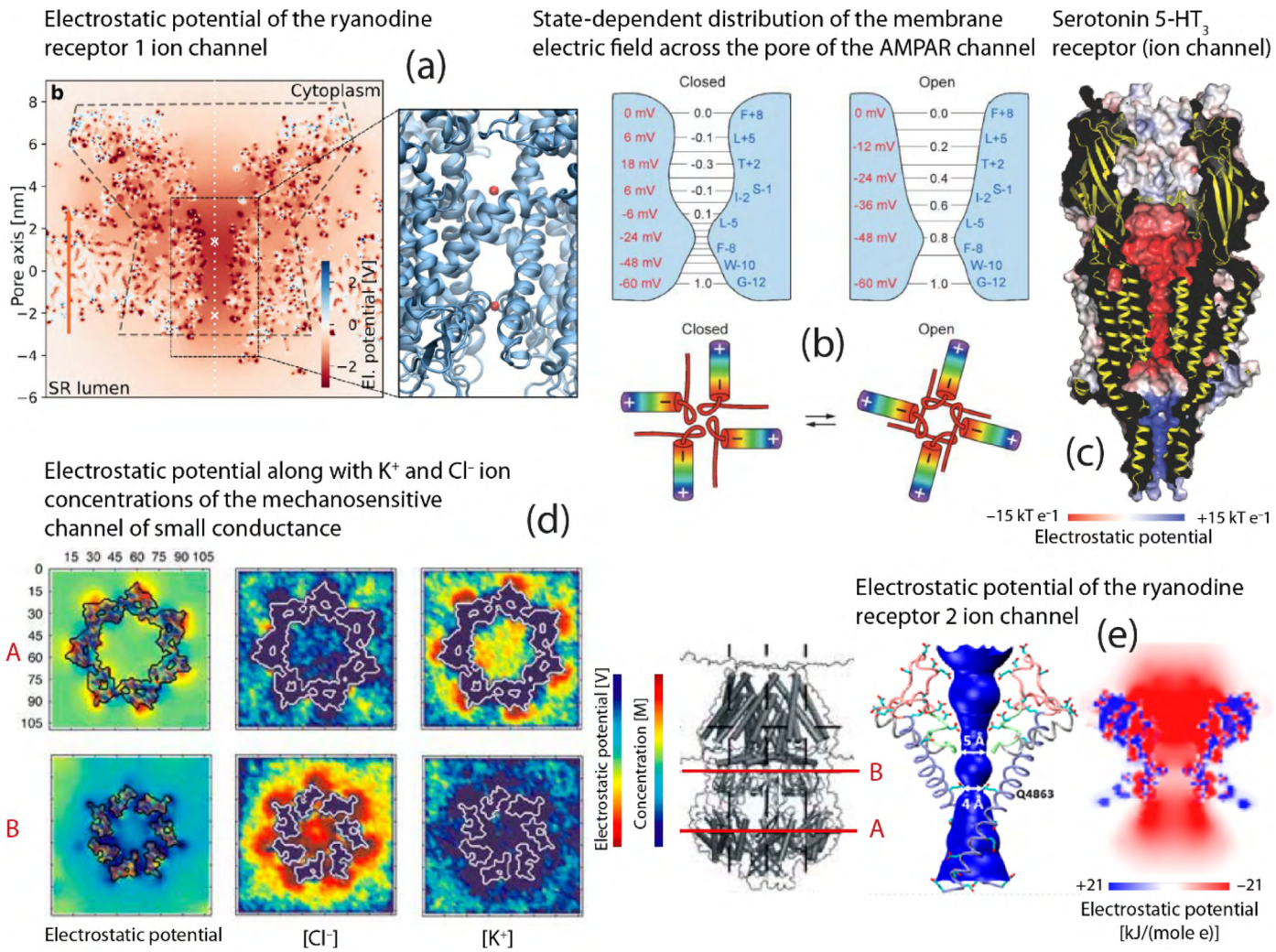


Figure 4.2.

Electrostatic properties of ion channels. **(a)** Electrostatic potential on a slice of the ryanodine receptor 1 (RnR1) $Ca2^+$ ion channel (found in mammalian skeletal muscle). **(b)** State-dependent distribution of the membrane electric field across the α-amino-3-hydroxy-5-methylisoxazole-4-proprionic acid receptor (AMPAR) channel pore. **(c)** Electrostatic potential of the mouse 5-hydroxytryptamin 3 (5-HT3) receptor (ion channel) **(d)** 2D sliced through a mechanosensitive channel of small conductance (MscS) showing the electrostatic potential along with the K^+ and Cl^- ion concentrations. **(e)** Electrostatic potential on a slice of the ryanodine receptor 1 (RnR1) $Ca2^+$ ion channel (found primarily in cardiac muscle).

Source: Funk, R. H. W. & Scholkmann, F. The significance of bioelectricity on all levels of organization of an organism. Part 1: From the subcellular level to cells. Progress in Biophysics and Molecular Biology 177, 185–201 (2023).

https://doi.org/10.1016/j.pbiomolbio.2022.12.002 License: CC BY 4.0.

In an anthropogenic environment, the significance of factors capable of altering the electrical properties of cells is increasing. Due to their small size, high specific surface area, and capacity to carry a surface charge, nanoplastics are considered potentially active physicochemical agents capable of interacting with cell membranes. Such interactions may affect the membrane potential, ion fluxes, and related metabolic processes, including mitochondrial function and cellular energy metabolism. The proposed mechanisms underlying this effect will be examined and substantiated in this section.

MECHANISMS OF MNP INTERNALIZATION AND THE ROLE OF SURFACE CHARGE

The internalization (cellular entry) of micro- and nanoplastic particles (Fig. 4.3) depends on their size, shape, and surface properties.[88] For most nanoparticles, the primary mechanism of cellular entry is endocytosis—an active engulfment process resulting in the formation of intracellular vesicles.[89] This involves various endocytotic subtypes, including clathrin-mediated endocytosis, caveolin-mediated endocytosis, and macropinocytosis; their respective contributions are determined by particle size and cell type. Generally, particles approximately 60–200 nm in size are predominantly internalized via clathrin-mediated endocytosis, whereas the internalization of particles ranging from about 200 nm to <1 μm may occur via the caveolin-mediated pathway.[90] Particles larger than 150 nm can also be engulfed by cells through macropinocytosis. Micrometer-scale particles up to ~3 μm can be taken up by immune system cells exhibiting pronounced phagocytic activity (professional phagocytes). Ultrafine nanoparticles smaller than ~10 nm can interact with the lipid bilayer and, under certain conditions, induce local membrane alterations, potentially facilitating their transmembrane transport.[91,92] Furthermore, paracellular transport through intercellular spaces is possible upon the disruption of epithelial barrier function (e.g., in the intestines or respiratory tract).[93] Large microplastic particles ~10 μm in size predominantly interact with the epithelial surface and can cause cellular damage via mechanical stress, as well as induce alterations in cellular metabolism. [94]

A key role is played by the surface charge of the particles The cell membrane normally possesses a negative electrical potential, creating conditions for electrostatic interactions with charged nanoparticles (Fig. 4.4). If particles carry a positive or partially positive charge, they are more strongly attracted to the membrane, which enhances their adhesion and increases the probability of cellular uptake via endocytosis. In certain cases, close contact of charged particles with the membrane can also lead to local structural disruptions—for instance, temporary impairment of barrier function or membrane deformation.[95,96,97,98]

[88] *Yee, M. S.-L., Hii, L.-W., Looi, C. K., et al. Impact of Microplastics and Nanoplastics on Human Health. Nanomaterials 11, 496 (2021). https://doi.org/10.3390/nano11020496*

[89] *Liu, Y.-Y., Liu, J., Wu, H., et al. Endocytosis, Distribution, and Exocytosis of Polystyrene Nanoparticles in Human Lung Cells. Nanomaterials 13, 84 (2022). https://doi.org/10.3390/nano13010084*

[90] *Popa, R. P. & Tabaran, A. F. A Systematic Review of the Toxicokinetics of Micro- and Nanoplastics in Mammals Following Digestive Exposure. Applied Sciences 15, 6135 (2025). https://doi.org/10.3390/app15116135*

[91] *Guo, Y., Terazzi, E., Seemann, R., et al. Direct proof of spontaneous translocation of lipid-covered hydrophobic nanoparticles through a phospholipid bilayer. Science Advances 2, e1600261 (2016). https://doi.org/10.1126/sciadv.1600261*

[92] *Łazarski, G., Rajtar, N., Romek, M., et al. Interaction of Polystyrene Nanoplastic with Lipid Membranes. J. Phys. Chem. B 129, 4110–4122 (2025). https://doi.org/10.1021/acs.jpcb.5c00738*

[93] *Donkers, J. M., Höppener, E. M., Grigoriev, I., et al. Advanced epithelial lung and gut barrier models demonstrate passage of microplastic particles. Micropl.&Nanopl. 2, 6 (2022). https://doi.org/10.1186/s43591-021-00024-w*

[94] *Cheng, Y., Chen, J., Fu, R., et al. Molecular mechanism differences between nanoplastics and microplastics in colon toxicity: nanoplastics induce ferroptosis-mediated immunogenic cell death, while microplastics cause cell metabolic reprogramming. J Nanobiotechnol 23, 505 (2025). https://doi.org/10.1186/s12951-025-03545-1*

[95] *Jeon, S., Clavadetscher, J., Lee, D.-K., et al. Surface Charge-Dependent Cellular Uptake of Polystyrene Nanoparticles. Nanomaterials 8, 1028 (2018). https://doi.org/10.3390/nano8121028*

[96] *Wieland, S., Ramsperger, A. F. R. M., Gross, W., et al. Nominally identical microplastic models differ greatly in their particle-cell interactions. Nat Commun 15, 922 (2024). https://doi.org/10.1038/s41467-024-45281-4*

[97] *Dai, S., Ye, R., Huang, J., et al. Distinct lipid membrane interaction and uptake of differentially charged nanoplastics in bacteria. J Nanobiotechnol 20, 191 (2022). https://doi.org/10.1186/s12951-022-01321-z*

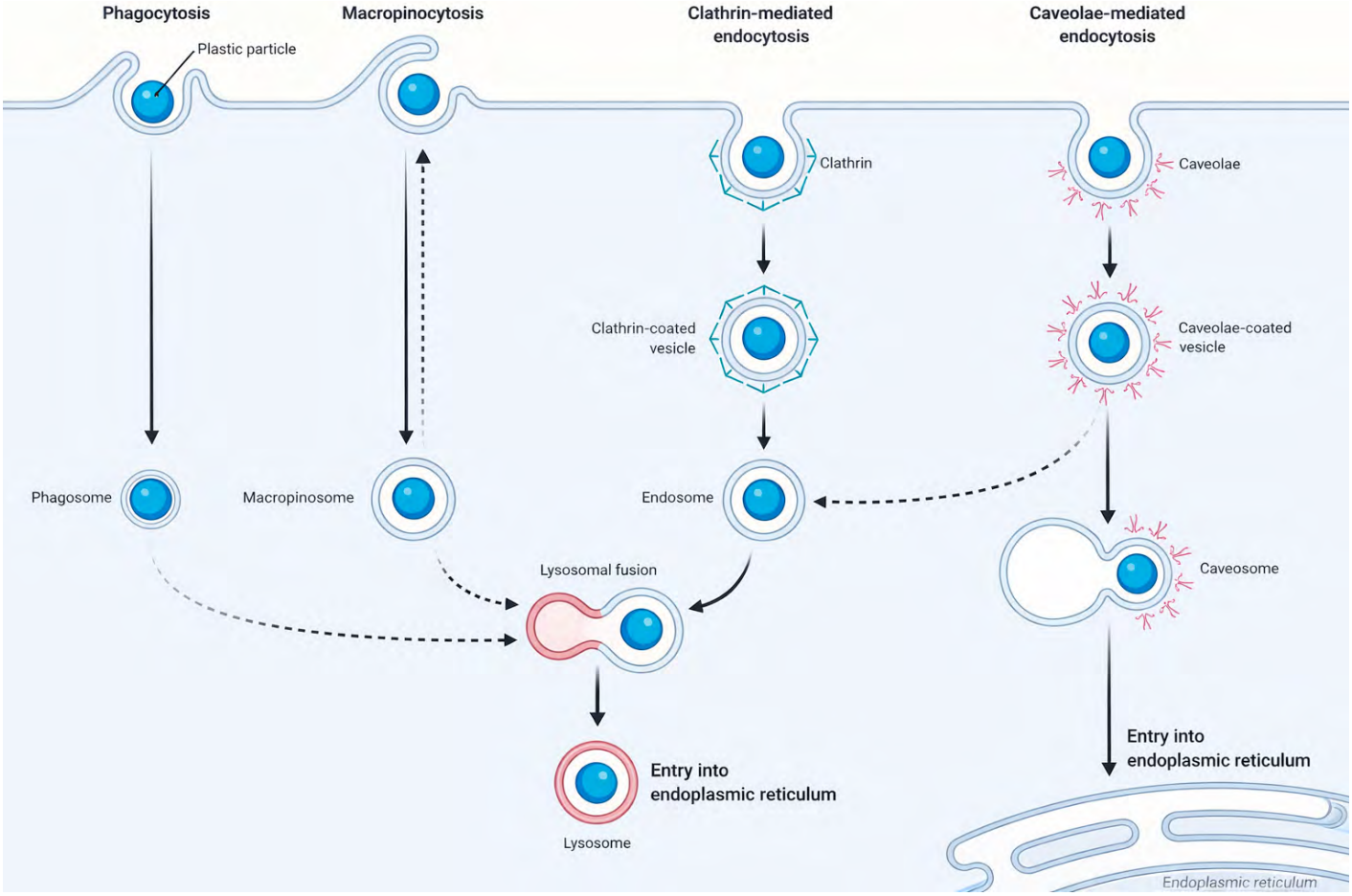


Figure 4.3.

Routes of cellular uptake of plastic particles. Phagocytosis, macropinocytosis, clathrin- and caveolae-mediated endocytosis are the common endocytotic pathways that have been identified for cellular uptake of plastic particles. Micro- and nanoplastics can be absorbed by cells through different routes, of which endocytotic nanoparticle uptake is the primary route where adhesive interaction of nanoparticles (or inactive permeation of the cell membrane) with channel- or transport-protein occurs.

Source: Yee, M. S.-L., Hii, L.-W., Looi, C. K., et al. Impact of Microplastics and Nanoplastics on Human Health. Nanomaterials 11, 496 (2021). https://doi.org/10.3390/nano11020496 License: CC BY 4.0.

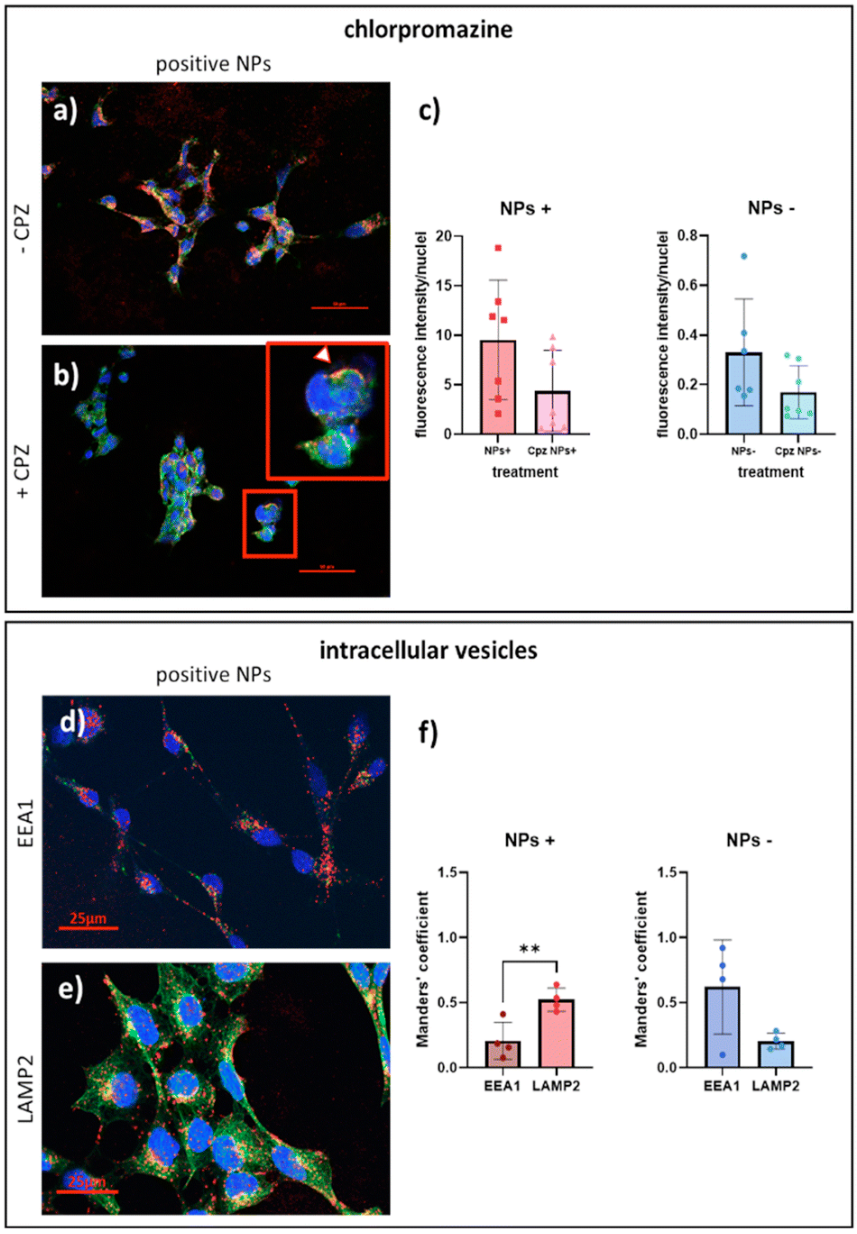


Figure 4.4.

Clathrin-mediated internalization pathway and intracellular fate of PS-NPs. Upper panel) Effect of chlorpromazine (CPZ). Positive PS-NPs were administered for 2 hours to HEK 293 cells, a) not pre-treated (−CPZ) or b) pre-treated (+CPZ) for 30 minutes with chlorpromazine (CPZ) to investigate the clathrin-mediated internalization pathway. In the red square in b, a higher magnification of a cell showing the NPs sticking to the plasma membrane was shown. Blue: nuclei; red: PS-NPs; green: cell membranes. Scale bar: 50 µm. c) Quantification of NPs uptake in cells pre-treated or not with chlorpromazine before being exposed for 2 hours to positive or negative PS-NPs. Data are the mean ± SD (standard deviation). Lower panel) Vesicle-mediated endocytosis. Representative confocal images of cells treated with positive PS-NPs and stained with d) early endosome antigen-1 (EEA1) or e) lysosome-associated membrane protein-2 (LAMP2). Blue: nuclei; red: PS-NPs; green: intracellular vesicles. Scale bar: 25 µm. f) Pearson's coefficient of positive and negative PS-NPs with the vesicles. Data are the mean ± SD. **p = 0.0877, unpaired t-test.

Source: Moscatiello, G. Y., Natale, C., Inserra, M., et al. The surface charge both influences the penetration and safety of polystyrene nanoparticles despite the protein corona formation. Environ. Sci.: Nano 12, 2857–2870 (2025). https://doi.org/10.1039/D4EN00962B License: CC BY-NC 3.0.

"An accumulation of the red signal to the periphery of cells was probably due to the electrochemical attraction between the positive surface of PS-NPs and the net anionic charge of the cell membrane."[98]

"The effect of positively charged PS-NPs in cells and worms is likely due to the z-potential rather than the amount of their accumulation."[98]

[98] *Moscatiello, G. Y., Natale, C., Inserra, M., et al. The surface charge both influences the penetration and safety of polystyrene nanoparticles despite the protein corona formation. Environ. Sci.: Nano 12, 2857–2870 (2025). https://doi.org/10.1039/D4EN00962B*

In general, experimental data show that micro- and nanoplastics can impact cells through several mechanistic pathways: they can disrupt signaling between cellular structures, impair the function of mitochondria (the cell's powerhouse), and exacerbate oxidative stress. Such impacts affect fundamental mechanisms of cellular regulation and structural integrity, initiating damage cascades that can lead to cellular dysfunction and apoptosis.[88] Neural tissue is considered particularly vulnerable, as neurons are highly sensitive to disturbances in energy metabolism and signaling; the severity and outcome of these effects depend on particle size, charge, and concentration.

DISRUPTION OF ELECTRICAL STABILITY AND NEURONAL MEMBRANE POTENTIAL

The neuron function relies on the generation and transmission of electrical signals, which requires strict maintenance of the potential difference between the inner and outer sides of the cell membrane (Fig. 4.5). This electrochemical gradient ensures cellular excitability and readiness for impulse transmission. The presence of nanoplastic particles carrying an intrinsic electrostatic charge can disrupt this equilibrium (Fig. 4.6, 4.7).

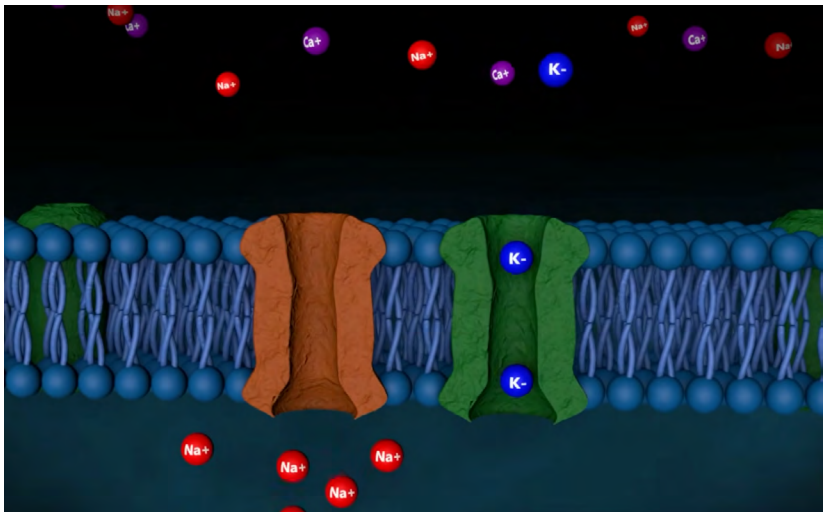


Figure 4.5.

Schematic representation of ion distribution and membrane potential formation in a neuron. The uneven distribution of Na^+, K^+, Ca^{2+} и Cl^- ions across the lipid membrane creates an electrochemical gradient. The opening of ion channels leads to local changes in the membrane potential. Upon reaching the threshold level, an action potential—an electrical impulse that propagates along the neuron—is generated.

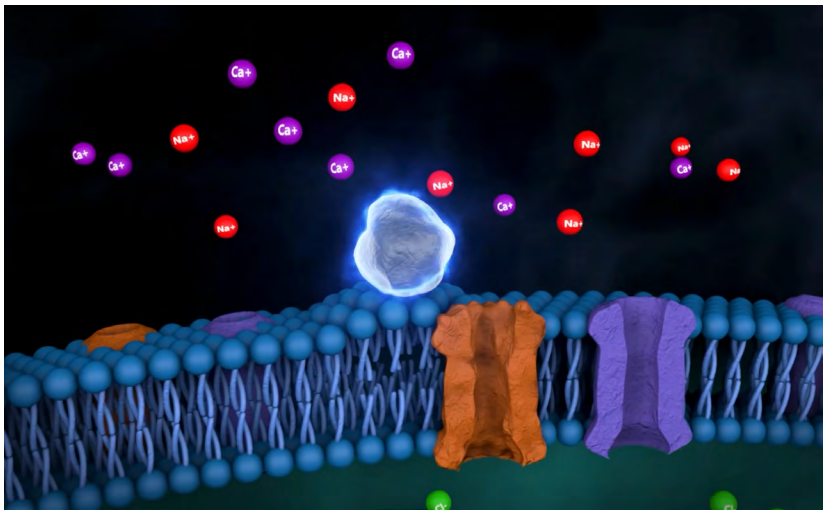


Figure 4.6.

Schematic representation of the effect of a charged nanoplastic particle on a lipid membrane. The electrostatic field of the particle can alter the distribution of ions near the membrane, potentially disrupting the electrochemical equilibrium necessary for the normal electrical activity of a neuron.

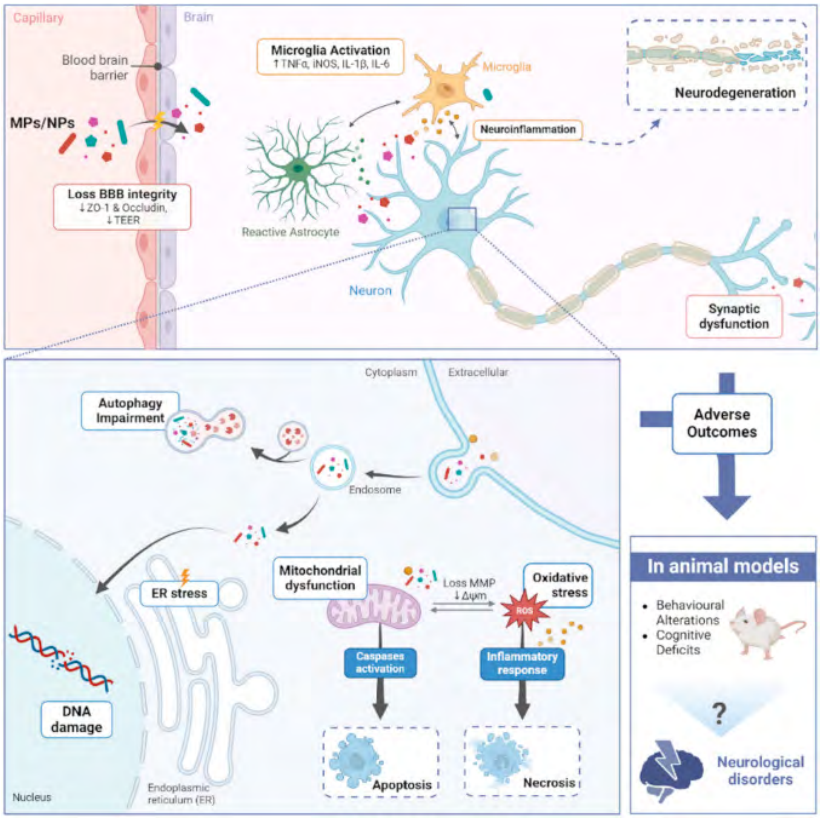


Figure 4.7.

Mechanisms of micro- and nanoplastics (MPs/NPs)-induced neurotoxicity. The scheme illustrates the multifactorial processes through which MPs/NPs disrupt brain function and lead to neurotoxicity.

Source: Araújo, A. M., Mota, C., Ramos, H., et al. The neurotoxic threat of micro- and nanoplastics: evidence from In Vitro and In Vivo models. Arch Toxicol 99, 3505–3525 (2025). https://doi.org/10.1007/s00204-025-04091-3

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This is corroborated by experimental data on the interaction of polystyrene nanoparticles with phospholipid membranes (Fig. 4.8).

"We have shown that PS-NH2 has the most potent capacity of membrane disruption, pointing all our findings to membrane depolarization by different mechanisms. First, hypothetical internalization of positively charged PS-NH_2 would decrease the negative membrane potential. Second, selective pores induced by PS-NH_2 are cation-selective for the inward flow but anion-selective for the outward one, suggesting a highly asymmetric membrane charge distribution produced either from NP adhesion or from major lipid reorganizations. Note that both the entrance of positive charges and the exit of negative ones decrease the membrane potential. Interestingly, negatively charged PS and PS-COOH follow the opposite pattern: either by NP internalization or by the charge transferred by their induced selective pores, the membrane potential increases leading to hyperpolarization."[99]

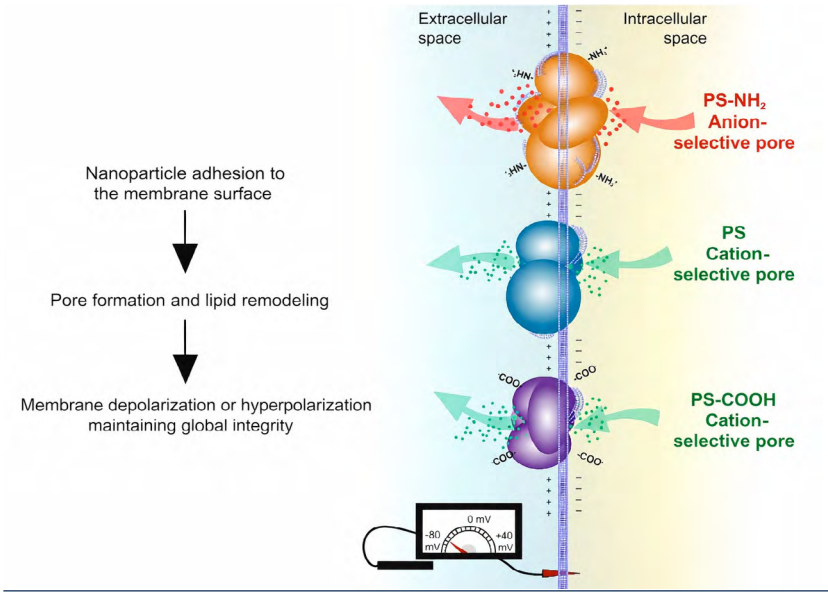


Figure 4.8.

Schematic representation of the complex modulation of the cell transmembrane electric potential exerted by PS NPs.

Source: Perini, D. A., Parra-Ortiz, E., Varó, I., et al. Surface-Functionalized Polystyrene Nanoparticles Alter the Transmembrane Potential via Ion-Selective Pores Maintaining Global Bilayer Integrity. Langmuir 38, 14837–14849 (2022). https://doi.org/10.1021/acs.langmuir.2c02487

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The alteration of ion gradients around cells under the influence of polystyrene nanoparticles (PS-NPs) is linked to various molecular mechanisms, such as nanoparticle internalization, local disruption of membrane integrity, the creation of hydrophilic pores, and potentially the physical blockade of ion channels, such as K^+ or Na^+ channels.[99] These mechanisms are likely not mutually exclusive but may act concertedly or even synergistically.

Alterations in membrane potential parameters can lead to atypical neuronal activity: either premature activation or, conversely, a reduced capacity to generate an action potential. Such disruptions at the single-cell level destabilize the functioning of neural networks and reduce the precision of signal transmission in the nervous system.

[99] *Perini, D. A., Parra-Ortiz, E., Varó, I., et al. Surface-Functionalized Polystyrene Nanoparticles Alter the Transmembrane Potential via Ion-Selective Pores Maintaining Global Bilayer Integrity. Langmuir 38, 14837–14849 (2022). https://doi.org/10.1021/acs.langmuir.2c02487*

IMPACT ON ION CHANNELS

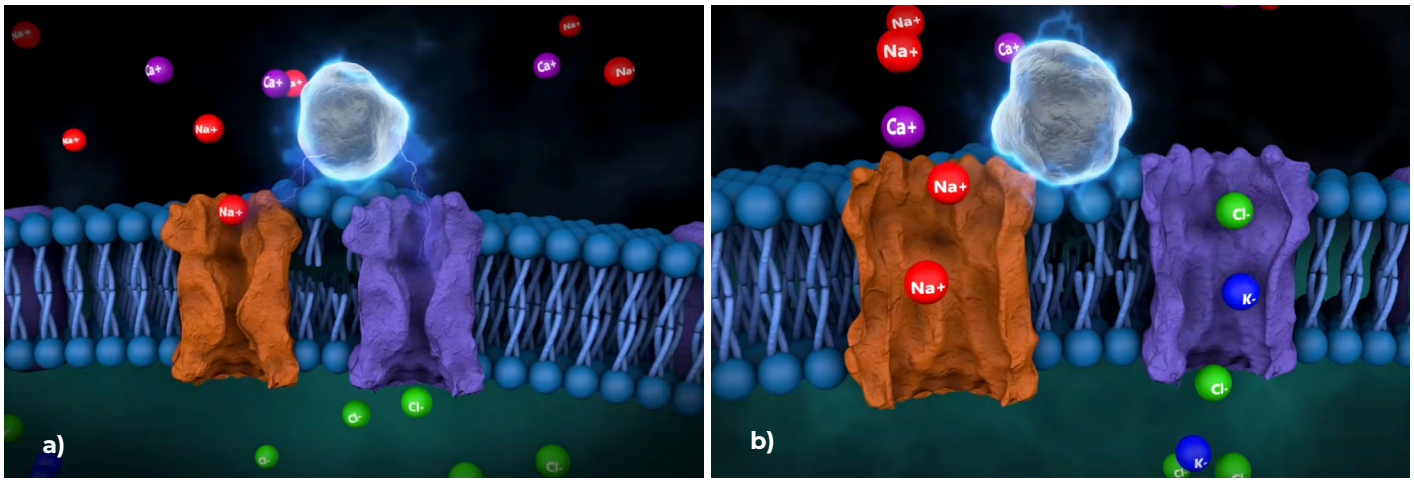


Figure 4.9.

Schematic representation of the potential effects of a charged nanoplastic particle on the conformation (spatial structure) of membrane ion channels.

a) Alteration of the channel structure may hinder its normal opening.

b) Conformational disruption may lead to excessive or prolonged channel opening, resulting in impaired ion transport.

Nanoplastics can directly interact with ion channels—membrane protein "gates" that regulate the transport of calcium, sodium, and potassium into and out of the cell.[100,101] Particles possessing a surface electrostatic charge can alter the spatial conformation of these channels, causing them either to fail to open in response to physiological signals or to remain open longer than required (Fig. 4.9). The disruption of the fine-tuned regulation of ion fluxes leads to the destabilization of cellular electrical activity and impaired signal transmission, particularly in the nervous system. Because ion currents are the foundation of the membrane potential and cellular signaling, their dysregulation impacts intracellular ion balance, signaling pathways, and the regulation of cellular processes. Under chronic exposure, this can trigger stress responses and other pathological mechanisms that promote cellular damage.

[100] *Kustra, A., Maliszewska-Olejniczak, K., Sekrecka-Belniak, A., et al. Polystyrene Nanoplastics in Human Gastrointestinal Models—Cellular and Molecular Mechanisms of Toxicity. IJMS 26, 11738 (2025). https://doi.org/10.3390/ijms262311738*

[101] *Yin, S., Liu, J., Kang, Y., et al. Interactions of nanomaterials with ion channels and related mechanisms. British J Pharmacology 176, 3754–3774 (2019). https://doi.org/10.1111/bph.14792*

DISRUPTION OF NEUROTRANSMITTER TRANSMISSION

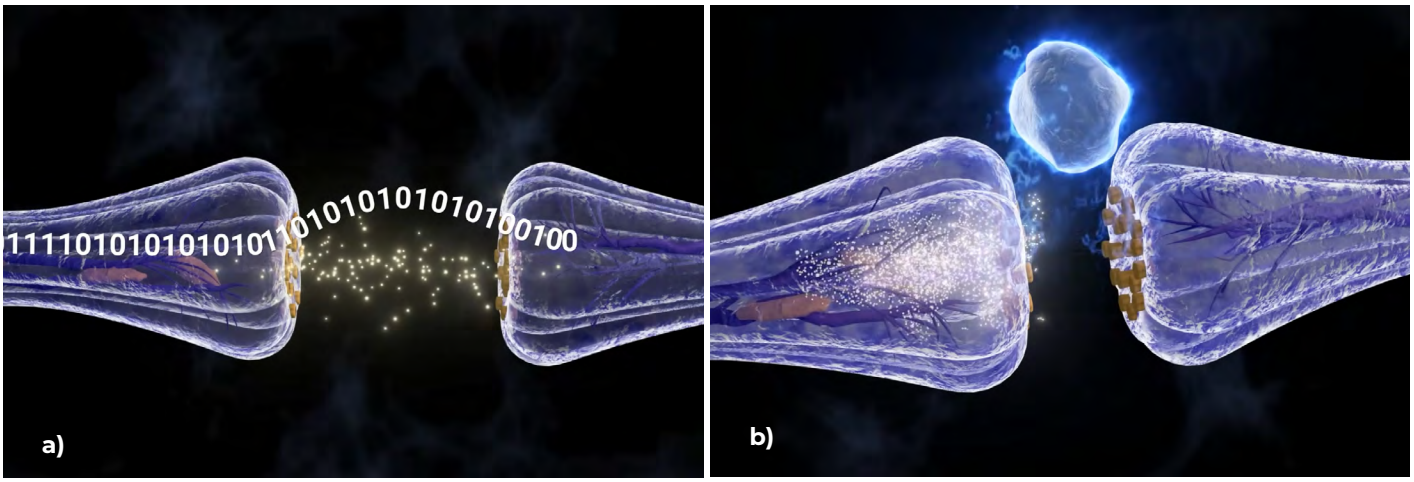


Figure 4.10.

Schematic representation of signal transmission between neurons at the synapse.

a) Normal synaptic transmission — the release of neurotransmitters from the presynaptic terminal and their diffusion across the synaptic cleft ensure signal transmission to the postsynaptic membrane.

b) The presence of a nanoplastic particle near the synapse can potentially disrupt the electrical state of the membrane and neurotransmitter release processes, which may affect the efficiency of interneuronal communication.

Signal transmission between neurons is an electrochemical process fundamentally based on synaptic communication. Neurotransmitters—specialized molecules mediating information transfer from one neuron to another—play a key role in this mechanism (Fig. 4.10a). They are released at precise moments and in strictly defined quantities. This process depends on the electrical state of the neuronal membrane, the function of ion channels, and the influx of calcium into the cell.

Electrostatically charged nanoplastics can disrupt the influx of calcium ions into the cell (Fig. 4.10b).[101,102] Because it is specifically the elevation of intracellular Ca^{2+} concentration that triggers neurotransmitter release into the synaptic cleft, any perturbations in this process result in weakened, delayed, or aberrant neurotransmitter release (Fig. 4.11, 4.12). Consequently, the precision of signal transmission between neurons is diminished, and the coordinated operation of neural networks is impaired.

[102] *Ho, K. H., Yeo, X. Y., Park, C., et al. Exposure to Nanoplastics Disrupts Neurotransmitter Release in Rat Hippocampal Neurons. Environ. Health envhealth. XXXX, XXX-XXX (2025).* https://doi.org/10.1021/envhealth.5c00477

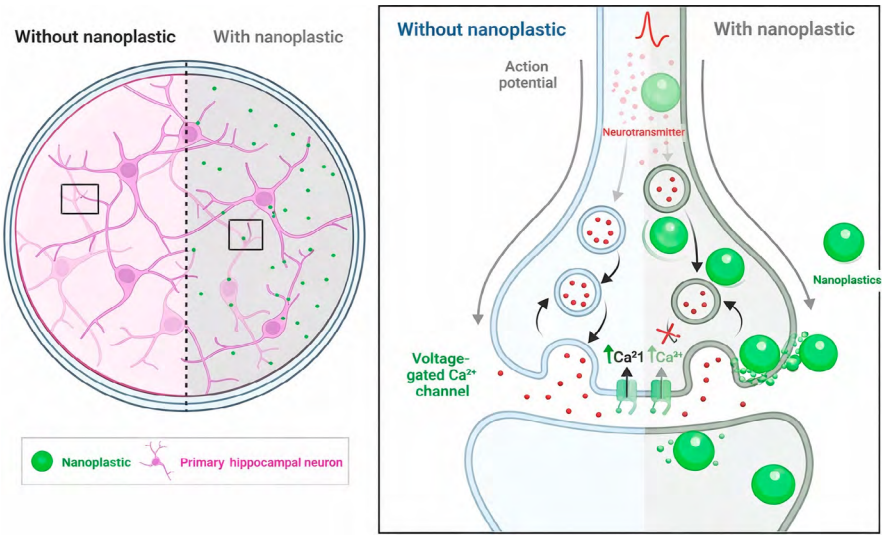


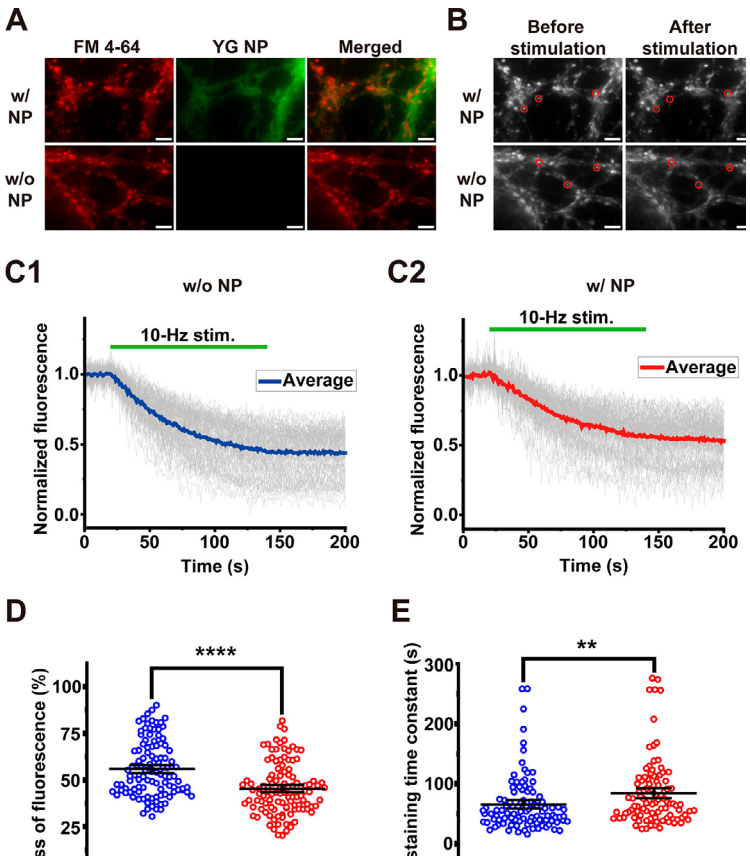
Figure 4.11.

Schematic representation of the impact of nanoplastics on hippocampal neuron function. The accumulation of nanoplastic particles in the presynaptic terminal reduces the Ca^{2+}-dependent release of neurotransmitters from synaptic vesicles, leading to impaired synaptic transmission between neurons.

Source: Ho, K. H., Yeo, X. Y., Park, C., et al. Exposure to Nanoplastics Disrupts Neurotransmitter Release in Rat Hippocampal Neurons. Environ. Health envhealth. XXXX, XXX-XXX (2025). https://doi.org/10.1021/envhealth.5c00477 License: CC BY-NC-ND 4.0.

Figure 4.12.

Exposure to nanoplastics decreases neurotransmitter release in hippocampal neurons. (A) Representative images of FM 4-64-loaded synaptic vesicles (red) in the presynaptic terminals of hippocampal neurons with (w/) 1 μg/mL YG nanoplastics (YG NP, green) and without (w/o) NP. FM 4-64 puncta represent individual presynaptic terminals containing FM 4-64-loaded synaptic vesicles. Scale bar: 5 μm. (B) Representative images of FM 4-64 puncta before and after 1200 electric field stimuli with (w/) and without (w/o) 1 μg/mL NP. Red circles mark ROIs representing FM 4-64 puncta that showed release and were used for analyses. Scale bar: 5 μm. (C) Normalized fluorescence intensity traces of FM 4-64 during 1200 electrical stimuli without (w/o) NP (n = 109 boutons, N = 5 experiments) (C1) and with (w/) NP (n = 112, N = 7) (C2). The blue and red lines represent the average normalized fluorescence traces of FM 4-64 without (w/o) and with (w/) NP, respectively. (D) Percentage of normalized fluorescence loss of FM 4-64 in hippocampal neurons without (w/o) NP (n = 109, N = 5) and with (w/) NP (n = 112, N = 7). ****p < 0.0001 (independent two-tailed Student's t-test). (E) Destaining time constant of FM 4-64 during stimulation without (w/o) NP (n = 100, N = 5) and with (w/) NP (n = 97, N = 7). **p < 0.01 (Mann–Whitney U test).



Source: Ho, K. H., Yeo, X. Y., Park, C., et al. Exposure to Nanoplastics Disrupts Neurotransmitter Release in Rat Hippocampal Neurons. Environ. Health envhealth. XXXX, XXX-XXX (2025). https://doi.org/10.1021/envhealth.5c00477 License: CC BY-NC-ND 4.0.

Clinically, such disruptions can manifest as memory impairment, reduced attention span, and other cognitive disorders.[103]

INDUCTION OF OXIDATIVE STRESS AND INFLAMMATION

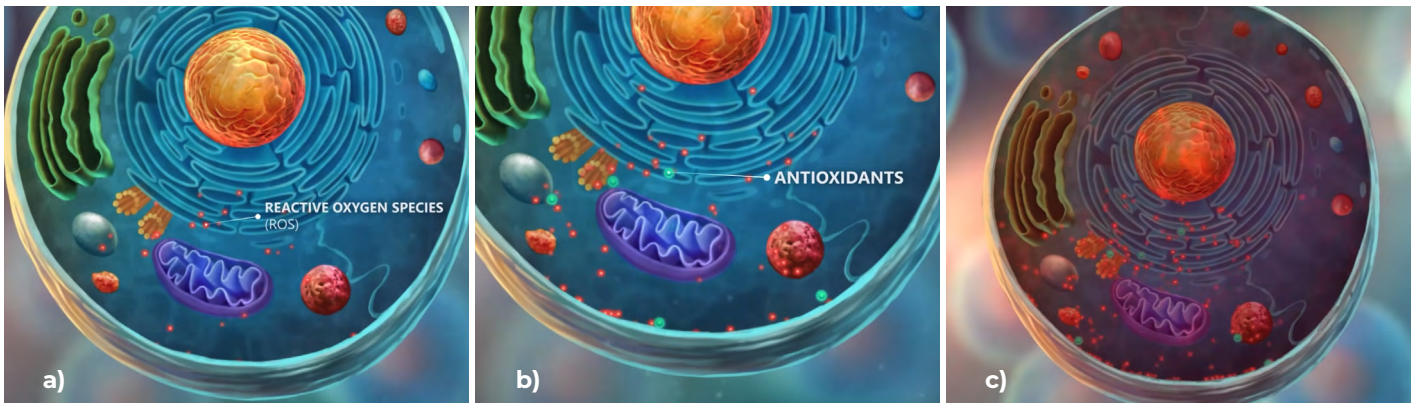


Figure 4.13.

Schematic representation of oxidative stress formation in the cell. Under normal conditions **(a)**, reactive oxygen species (ROS) generated during cellular metabolism are neutralized by the antioxidant system **(b)**. When there is excessive ROS production or a weakened antioxidant defense, oxidative stress develops **(c)**, accompanied by damage to membrane lipids, proteins, and DNA, thereby disrupting cellular structure and function.

One of the universal mechanisms of cellular damage induced by nanoplastics is oxidative stress (Fig. 4.13). Normally, reactive oxygen species (ROS) are continuously generated in the cell during energy metabolism but are maintained at safe levels by the antioxidant system—enzymes and molecules that neutralize excess radicals. When ROS production exceeds the capacity of this system, oxidative stress ensues—a state in which free radicals begin to damage membrane lipids, proteins, and DNA, compromising cellular structure and function.

Experimental data indicate that internalized plastic nanoparticles can sustain chronically elevated ROS levels and exacerbate inflammatory responses.[104] Under such conditions, cellular defense mechanisms are depleted, and damage gradually accumulates. If this state persists over a prolonged period, the function of vital cellular systems is impaired, the frequency of DNA mutations increases, and programmed cell death (apoptosis) may be triggered.

[103] *Ma, Q., Lei, J., Pang, Y., et al. Neurotoxicity of Micro- and Nanoplastics: A Comprehensive Review of Central Nervous System Impacts. Environ. Health 4, 4–24 (2026). https://doi.org/10.1021/envhealth.5c00087*
[104] *Milillo, C., Aruffo, E., Di Carlo, P., et al. Polystyrene nanoplastics mediate oxidative stress, senescence, and apoptosis in a human alveolar epithelial cell line. Front. Public Health 12, 1385387 (2024). https://doi.org/10.3389/fpubh.2024.1385387*

Neurons are particularly vulnerable to oxidative stress: they consume high levels of oxygen, maintain constant high metabolic activity, possess limited antioxidant reserves, and have virtually no regenerative capacity.[105] Therefore, additional oxidative stress induced by nanoplastics elevates the risk of neuronal inflammation, dysfunction, and death, potentially contributing to the pathogenesis of neurodegenerative diseases, including Parkinson's disease.[106,107]

Experimental studies utilizing planarians—small flatworms with remarkable regenerative capabilities—demonstrate that microplastics can induce oxidative stress, cellular damage, and systemic organismal disruptions.[108] Planarians are frequently employed as a model system because their regenerative capabilities are highly sensitive to early cellular-level perturbations, including toxicological exposures (Fig. 4.14).

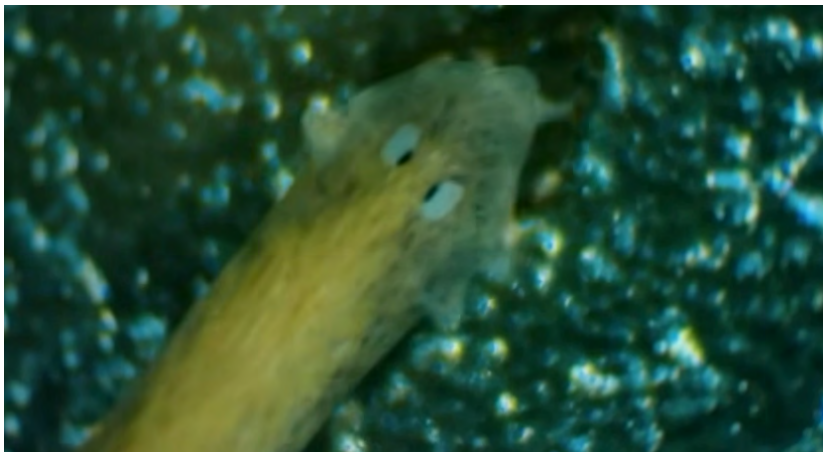


Figure 4.14.

Planaria — a model organism widely used in experimental studies on the toxicity of environmental pollutants. Due to their high regenerative capacity and sensitivity to cellular damage, planarians allow for the detection of early effects of microplastic exposure, including the development of oxidative stress and the disruption of physiological processes.

These experiments established that microplastics independently elevate reactive oxygen species levels, impair the antioxidant system, induce tissue damage, and disrupt normal physiological processes in planarians.[108] These effects become particularly pronounced when microplastics are co-exposed with other pollutants, such as heavy metals. Under such conditions, the deleterious impact is amplified, and the processes of tissue regeneration and repair in planarians are significantly impaired.

It has been further revealed that microplastic exposure alters the composition of the planarian microbiota. This indicates that the impact of micro- and nanoplastics extends beyond individual cells, affecting metabolic regulation, immune responses, and overall physiological stability.[108]

Thus, experimental data confirm that microplastics can induce oxidative stress, disrupt cellular functions, and destabilize systemic processes—especially synergistically with other toxic agents—serving as a model illustration of how such mechanistic pathways may manifest in more complex biological systems.

[105] *Exner, N., Lutz, A. K., Haass, C., et al. Mitochondrial dysfunction in Parkinson's disease: molecular mechanisms and pathophysiological consequences. EMBO J 31, 3038–3062 (2012). https://doi.org/10.1038/emboj.2012.170*

[106] *106 Kim, D. Y., Park, M. K., Yang, H. W., et al. Effects of Microplastic Accumulation on Neuronal Death After Global Cerebral Ischemia. Cells 14, 241 (2025). https://doi.org/10.3390/cells14040241*

[107] *Zhu, Y., Wu, J., Zhao, S., et al. Environmentally relevant concentrations of polystyrene nanoplastics induce Parkinson's-like neurotoxicity in C. elegans via oxidative stress. Environment International 206, 109926 (2025). https://doi.org/10.1016/j.envint.2025.109926*

[108] *Han, Y., Zhang, X., Liu, P., et al. Microplastics exposure causes oxidative stress and microbiota dysbiosis in planarian Dugesia japonica. Environ Sci Pollut Res 29, 28973–28983 (2022). https://doi.org/10.1007/s11356-022-18547-x*

MITOCHONDRIAL DYSFUNCTION AND ENERGY DEFICIT

The primary mechanistic pathway underlying the deleterious effects of MNPs at the cellular level is their impact on mitochondria[109,110,111]—the central hub of cellular energy production and the regulation of vital processes (Fig. 4.15). Without mitochondria, the cell loses its capacity for survival and repair.[112] Mitochondria are organelles that supply cells with energy via adenosine triphosphate (ATP) synthesis.[113] This process is driven by the electron transport chain (ETC), a series of protein complexes situated in the inner mitochondrial membrane that facilitate directed electron transfer and ATP synthesis. Owing to the high efficiency of this system, a single mitochondrion can synthesize up to tens of millions of ATP molecules per day, making it critical for maintaining cellular viability.[113]

Neurons exhibit exceptional vulnerability to energy crises.[114] Unlike many other cell types, they are post-mitotic (non-dividing) and have virtually no regenerative capacity, yet they continuously consume massive amounts of ATP to sustain electrical activity and signal transmission. Therefore, any perturbations in energy metabolism render neurons acutely susceptible to mitochondrial dysfunction and inflammation.

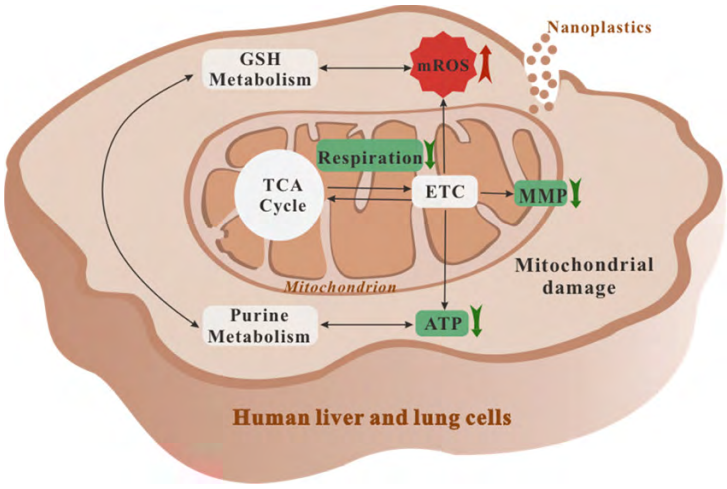


Figure 4.15.

Schematic representation of the effect of nanoplastics on mitochondrial function in human liver and lung cells. Nanoplastic particles cause an overproduction of mitochondrial reactive oxygen species (mROS), a decrease in mitochondrial membrane potential (MMP), and the suppression of mitochondrial respiration. This disrupts cellular energy metabolism, including the tricarboxylic acid (TCA) cycle, the electron transport chain (ETC), and ATP synthesis.

Source: Lin, S., Zhang, H., Wang, C., et al. Metabolomics Reveal Nanoplastic-Induced Mitochondrial Damage in Human Liver and Lung Cells. Environ. Sci. Technol. 56, 12483–12493 (2022). https://doi.org/10.1021/acs.est.2c03980 License: CC BY 4.0.

[109] *Liu, H., Li, H., Yao, X., et al. Environmental nanoplastics induce mitochondrial dysfunction: A review of cellular mechanisms and associated diseases. Environmental Pollution 382, 126695 (2025). https://doi.org/10.1016/j.envpol.2025.126695*

[110] *Lin, S., Zhang, H., Wang, C., et al. Metabolomics Reveal Nanoplastic-Induced Mitochondrial Damage in Human Liver and Lung Cells. Environ. Sci. Technol. 56, 12483–12493 (2022). https://doi.org/10.1021/acs.est.2c03980*

[111] *Maharana, T., Taranath, A., Fernandes, C. S. E., et al. Micro- and nanoplastic-induced mitochondrial dysfunction and organelle miscommunication: A toxicological perspective. Toxicology 519, 154306 (2026). https://doi.org/10.1016/j.tox.2025.154306*

[112] *Liu, Y., Huang, Y., Xu, C., et al. Mitochondrial Dysfunction and Therapeutic Perspectives in Cardiovascular Diseases. IJMS 23, 16053 (2022). https://doi.org/10.3390/ijms232416053*

[113] *Pizzorno, J. Mitochondria—Fundamental to Life and Health. Integr Med (Encinitas) 13, 8–15 (2014). https://pmc.ncbi.nlm.nih.gov/articles/PMC4684129/*

[114] *Eisen, A., Pioro, E. P., Goutman, S. A., et al. Nanoplastics and Neurodegeneration in ALS. Brain Sciences 14, 471 (2024). https://doi.org/10.3390/brainsci14050471*

[115] *Sun, Z., Wen, Y., Zhang, F., et al. Exposure to nanoplastics induces mitochondrial impairment and cytomembrane destruction in Leydig cells. Ecotoxicology and Environmental Safety 255, 114796 (2023). https://doi.org/10.1016/j.ecoenv.2023.114796*

Experimental data demonstrate that polystyrene nanoparticles (~20–50 nm) are capable of penetrating cells and accumulating within mitochondria.[115] For perspective: a mitochondrion is typically 5–10 µm in length and 0.3–1 µm in width. Nanoplastic particles measure less than 0.1 µm, enabling high intracellular mobility and the capacity for direct interaction with mitochondrial structures (Fig. 4.16).

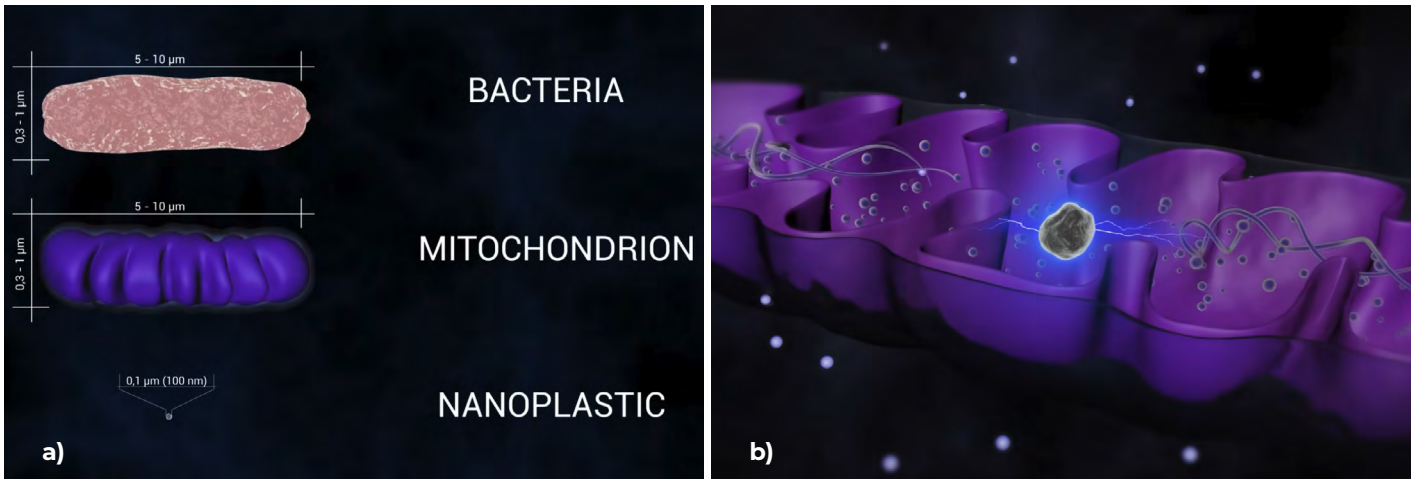


Figure 4.16.

Size comparison of a bacterium, a mitochondrion, and a nanoplastic particle (a) and the potential interaction of nanoplastics with mitochondria (b). Due to their small size, nanoplastics can penetrate the cell and interact with mitochondria.

This accumulation (Fig. 4.17, 4.18) is accompanied by a reduction in the mitochondrial membrane potential and a decline in ATP synthesis; simultaneously, the generation of reactive oxygen species (ROS) is amplified, triggering cascades of cellular stress and programmed cell death (apoptosis).[116]

[116] *Huang, Y., Liang, B., Li, Z., et al. Polystyrene nanoplastic exposure induces excessive mitophagy by activating AMPK/ULK1 pathway in differentiated SH-SY5Y cells and dopaminergic neurons in vivo. Part Fibre Toxicol 20, 44 (2023). https://doi.org/10.1186/s12989-023-00556-4*

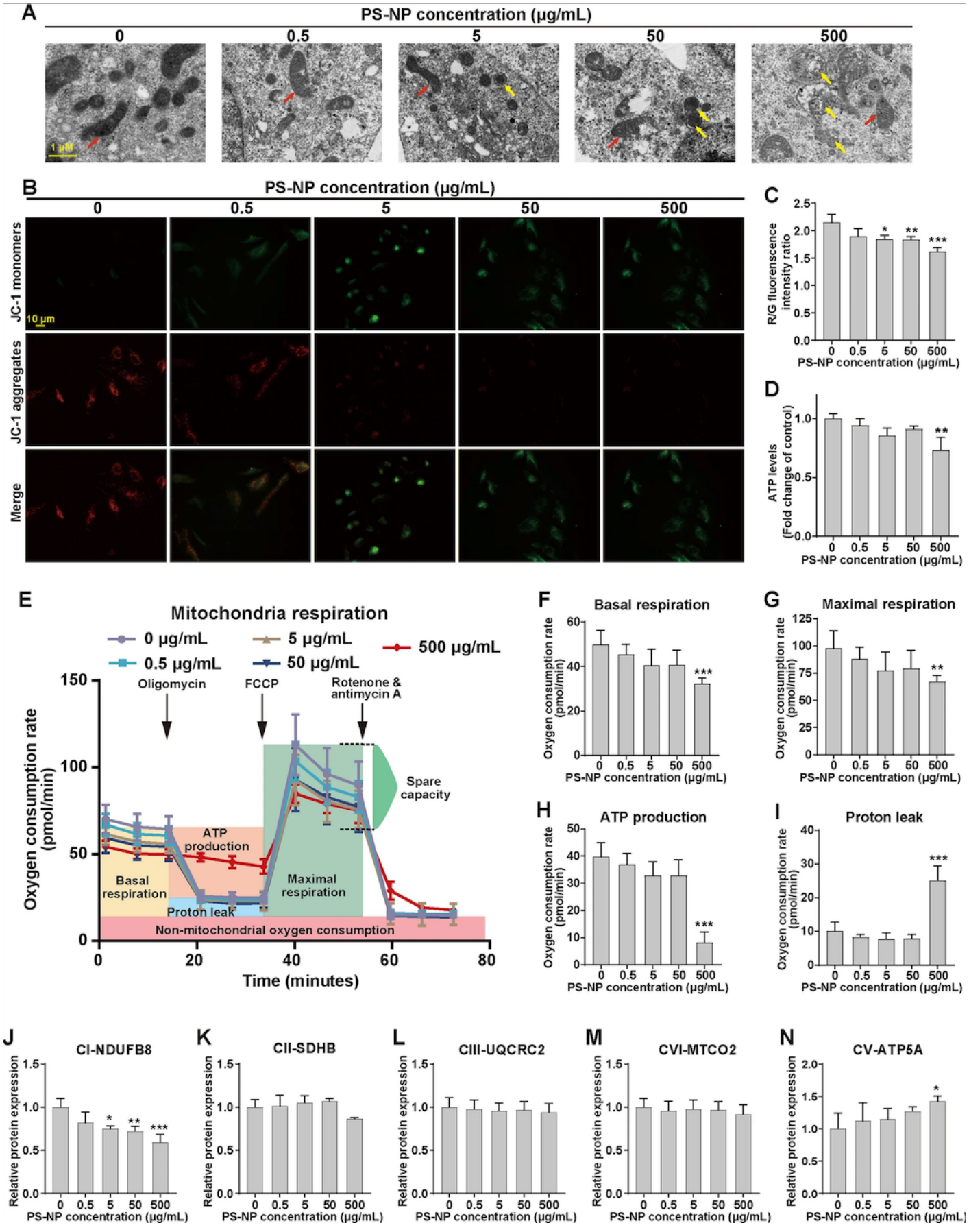

A
PS-NP concentration (μg/mL)
0
0.5
5
50
500
1 μM
B
PS-NP concentration (μg/mL)
JC-1 monomers
JC-1 aggregates
Merge
10 μm
C
R/G fluorenscence intensity ratio
PS-NP concentration (μg/mL)
D
ATP levels (Fold change of control)
E
Mitochondria respiration
0 μg/mL
0.5 μg/mL
5 μg/mL
50 μg/mL
500 μg/mL
Oligomycin
FCCP
Rotenone & antimycin A
Oxygen consumption rate (pmol/min)
Time (minutes)
Basal respiration
ATP production
Proton leak
Maximal respiration
Spare capacity
Non-mitochondrial oxygen consumption
F
Basal respiration
G
Maximal respiration
H
ATP production
I
Proton leak
J
CI-NDUFB8
K
CII-SDHB
L
CIII-UQCRC2
M
CVI-MTCO2
N
CV-ATP5A
Relative protein expression

Figure 4.17.

Impairment of mitochondria and mitochondrial stress by PS-NPs in differentiated SH-SY5Y cells (neuron model). **A** Representative TEM images of cells after PS-NP exposure. Red arrows indicate mitochondria, and yellow arrows indicate mitophagosomes. Impact of PS-NPs on **B**, **C** mitochondrial membrane potential (ΔΨm)and **D** cellular ATP levels. **E** Mitochondrial stress profiles. Relative changes in key parameters of mitochondrial function: **F** basal respiration, **G** maximal respiration, **H** mitochondrial ATP production, and I proton (H+) leakage. **J–N** The quantification of protein expression levels of electron transport chain complexes I-V (ETC CI-NDUFB8, CII-SDHB, CIII-UQCRC2, CVI-MTCO2, and CV-ATP5A). Results are presented as mean ± SD (n = 3). *P < 0.05, **P < 0.01, ***P < 0.001, compared to the 0 μg/mL group.

Source: Huang, Y., Liang, B., Li, Z., et al. Polystyrene nanoplastic exposure induces excessive mitophagy by activating AMPK/ULK1 pathway in differentiated SH-SY5Y cells and dopaminergic neurons in vivo. Part Fibre Toxicol 20, 44 (2023). https://doi.org/10.1186/s12989-023-00556-4 License: CC BY 4.0.

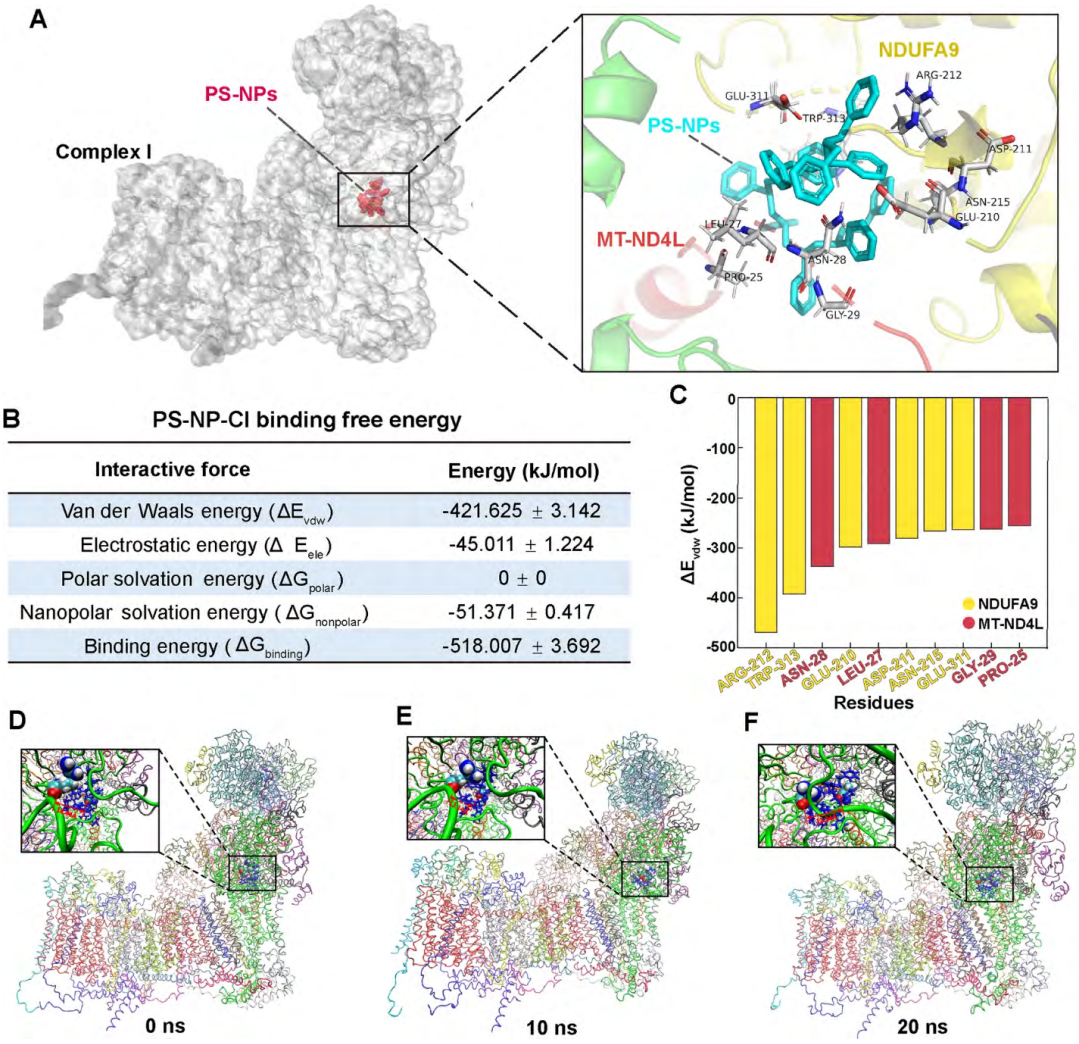


Figure 4.18.

Molecular docking and molecular dynamic (MD) simulation showing interaction between PS-NPs and CI. **A** Molecular docking model illustrating the binding of PS-NPs to Complex I (CI). Detailed interaction view (enlarged panel). **B** Binding free energy between PS-NPs and CI. **C** Van der Waals interactions between specific residues of CI and PS-NPs. **D-F** Conformational changes of the PS-NP- CI complex in MD simulation.

Source: Huang, Y., Liang, B., Li, Z., et al. Polystyrene nanoplastic exposure induces excessive mitophagy by activating AMPK/ULK1 pathway in differentiated SH-SY5Y cells and dopaminergic neurons in vivo. Part Fibre Toxicol 20, 44 (2023). https://doi.org/10.1186/s12989-023-00556-4 License: CC BY 4.0.

When reactive oxygen species become overwhelmingly abundant, a state known as mitochondrial dysfunction ensues. In this condition, mitochondria fail to manage the metabolic load, and the cell initiates defense mechanisms—ranging from inflammation to apoptosis. Oxidative stress acts as a pivotal driver in initiating and perpetuating mitochondrial dysfunction, playing a universal role in cellular damage induced by nanoplastics.

Mitochondrial DNA (mtDNA) is especially vulnerable to ROS.[117] Current evidence indicates that micro- and nanoplastics can disrupt mitochondrial operations and induce damage to mtDNA. Nanoplastics can penetrate mitochondria and accumulate within their matrix, provoking structural and functional aberrations (cristae deformation, membrane depolarization, and elevated reactive oxygen species (ROS) production), thereby establishing the conditions for mtDNA damage.[109] Initial studies examining exposure to ~100 nm particles have demonstrated intra-mitochondrial DNA damage accompanied by the release of DNA fragments into the cytosol and the activation of inflammatory cascades, alongside the compromised integrity of the mitochondrial genome (structural mtDNA damage) correlated with depleted ATP and diminished membrane potential.[118,119] Furthermore, dose-dependent alterations in the copy number of mtDNA have been documented, serving as a biomarker for mitochondrial stress and impaired mtDNA replication.[115]

Consequently, the cumulative data indicate that MNPs induce damage, fragmentation, and functional impairment of mtDNA, alongside alterations in its abundance. The integrity of mitochondrial DNA is paramount for the functional state of mitochondria. Because mtDNA encodes critical proteins comprising the electron transport chain and ATP synthesis machinery, its damage directly attenuates the efficiency of energy metabolism, exacerbates the generation of reactive oxygen species, and progressively degrades the overall capacity of mitochondria to sustain cellular energy.

The accumulation of mtDNA damage instigates a definitive vicious cycle: increased structural damage → decreased ATP synthesis → exacerbated oxidative stress → further deterioration of mitochondrial function. Thus, mtDNA damage not only reflects mitochondrial stress but actively amplifies it, emerging as a central node in the pathogenesis of mitochondrial dysfunction and long-term cellular injury.

Experimental data indicate that polystyrene nanoparticles (PS-NPs) can profoundly suppress mitochondrial respiration in the brain. This impairment affects both synaptic mitochondria located within neural terminals and non-synaptic mitochondria situated elsewhere in the cell. The mechanism of this damage is rooted in the fact that PS-NPs disrupt electron transfer between the principal complexes of the mitochondrial ETC, specifically between Complexes I–III and II–III (Fig. 4.19).[120]

[117] *Cha, M.-Y., Kim, D. K. & Mook-Jung, I. The role of mitochondrial DNA mutation on neurodegenerative diseases. Exp Mol Med 47, e150–e150 (2015).* https://doi.org/10.1038/emm.2014.122
[118] *Shen, R., Yang, K., Cheng, X., et al. Accumulation of polystyrene microplastics induces liver fibrosis by activating cGAS/STING pathway. Environmental Pollution 300, 118986 (2022).* https://doi.org/10.1016/j.envpol.2022.118986
[119] *Liu, T., Hou, B., Wang, Z., et al. Polystyrene microplastics induce mitochondrial damage in mouse GC-2 cells. Ecotoxicology and Environmental Safety 237, 113520 (2022).* https://doi.org/10.1016/j.ecoenv.2022.113520
[120] *Seward, D. M., Keegan, C. D. & Davey, G. P. Polystyrene nanoplastics target electron transport chain complexes in brain mitochondria. Journal of Hazardous Materials: Plastics 1, 100003 (2025).* https://doi.org/10.1016/j.hazmp.2025.100003

These sites are essential for the sequential transfer of electrons that ultimately drives ATP synthesis. This identified effect represents a novel mechanistic pathway of nanoplastic neurotoxicity, fundamentally based on the perturbation of mitochondrial function—a critically vital component of cellular metabolism, particularly in highly energy-demanding cells such as cerebral neurons.

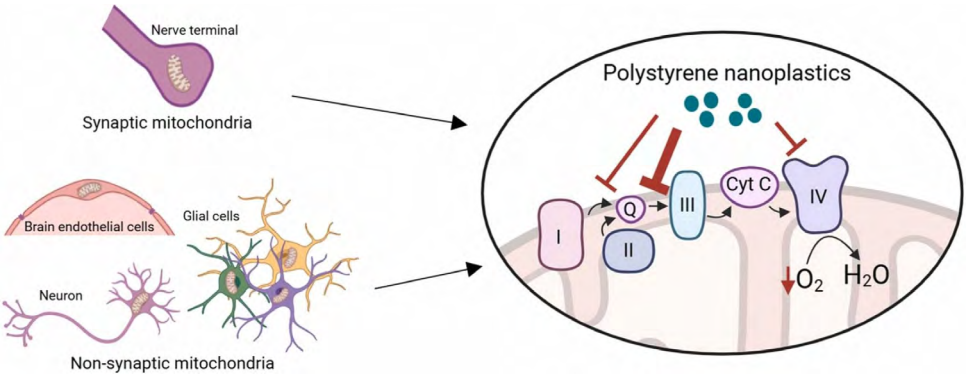


Figure 4.19.

Effect of polystyrene nanoparticles (PS-NPs) on brain cell mitochondria. Nanoplastics can affect the mitochondria of neurons, glial cells, and endothelial cells, disrupting the operation of the electron transport chain. The particles selectively impair electron transfer between complexes I–III and II–III of the respiratory chain, inhibit oxygen consumption and the efficiency of mitochondrial respiration.

Source: Seward, D. M., Keegan, C. D. & Davey, G. P. Polystyrene nanoplastics target electron transport chain complexes in brain mitochondria. Journal of Hazardous Materials: Plastics 1, 100003 (2025). https://doi.org/10.1016/j.hazmp.2025.100003 License: CC BY 4.0.

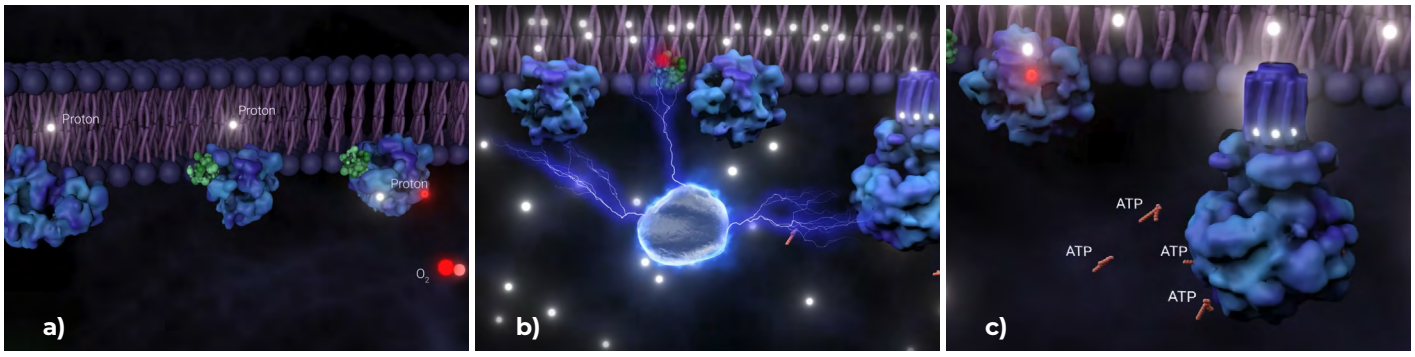


Figure 4.20.

Schematic representation of the effect of nanoplastics on the mitochondrial respiratory chain. a) Normal electron transfer through the protein complexes of the mitochondrial respiratory chain, which ensures the synthesis of adenosine triphosphate (ATP)—the cell's primary energy source. b) An electrostatically charged nanoplastic particle can disrupt electron transfer between the protein complexes of the respiratory chain. c) The disruption of electron transport reduces the efficiency of the mitochondrial ATP synthase enzyme complex and leads to a decrease in ATP production.

In conclusion, extensive data from experimental studies and reviews demonstrate that the primary mechanistic pathway of cellular damage induced by micro- and nanoplastics is intrinsically linked to the disruption of mitochondrial function. Their exposure (Fig. 4.20) leads to a decline in ATP synthesis, membrane depolarization, heightened oxidative stress, and mitochondrial DNA damage, collectively establishing a vicious cycle that continually exacerbates mitochondrial dysfunction. These processes are fundamental to cellular homeostasis, and their perturbation can propel the development of pathologies associated with chronic stress, inflammation, and compromised cellular resilience to environmental stressors.

STRUCTURAL PROTEIN DEGRADATION INDUCED BY NANOPLASTICS

One of the fundamental physicochemical mechanisms of cellular dysfunction in the presence of micro- and nanoplastics (MNPs) is the disruption of the spatial structure of biomolecules, driven by the electrostatic interaction between charged plastic particles and proteins. Nanoplastics can critically affect the secondary structure of proteins. In studies based on molecular dynamics (MD) simulations, it was demonstrated that nanoplastics, through their interaction with biomolecules, induce the transformation of α-helices into β-sheets and cause general denaturation (Fig. 4.21).[121,122] The authors note that the native configuration of the protein is destabilized under the influence of plastic. This structural transition (α-helix → β-sheet) has fatal consequences for biochemical activity: the protein transforms into a flat β-sheet or an unstructured random coil, completely "switching off" its normal function.

[121] *Hollóczki, O. & Gehrke, S. Nanoplastics can change the secondary structure of proteins. Sci Rep 9, 16013 (2019). https://doi.org/10.1038/s41598-019-52495-w*
[122] *Hollóczki, O. Evidence for protein misfolding in the presence of nanoplastics. International Journal of Quantum Chemistry 121, e26372 (2021). https://doi.org/10.1002/qua.26372*

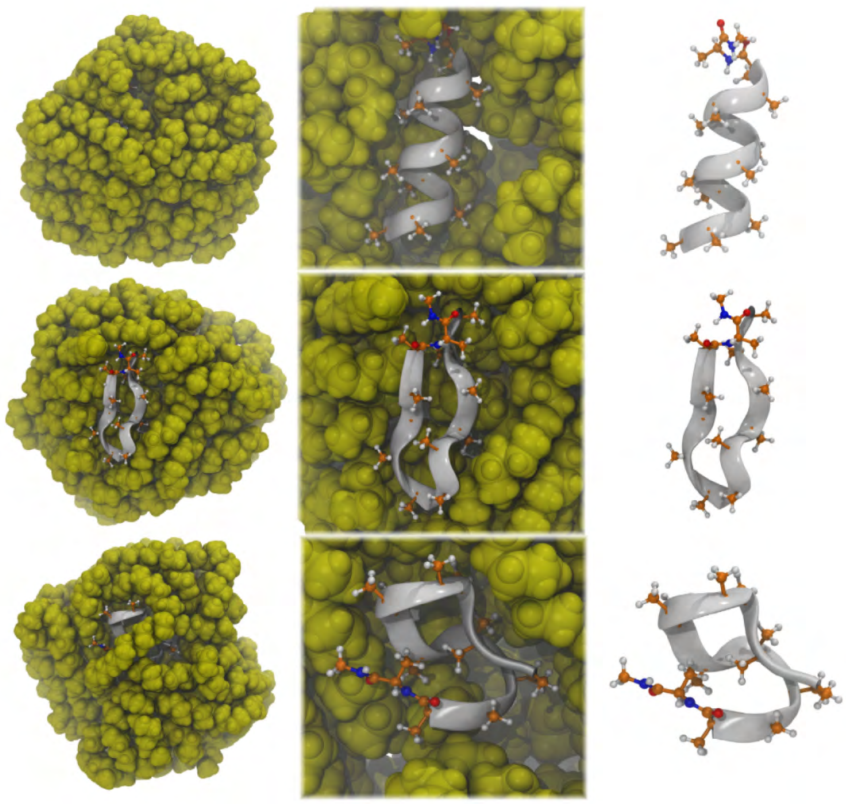


Figure 4.21.

Most stable relevant structures for the Ala12 peptide at the polyethylene nanoparticle (in yellow) and the peptide (gray ribbon and ball-and-stick with blue for N, red for O, orange for C, and white for H atoms) as obtained from simulated annealing and the subsequent GFN2-xTB geometry optimization. Top: with an α-helical peptide conformation, middle: with a β-hairpin peptide conformation, bottom: with the unconstrained peptide, folded together with the plastic in the corresponding simulated annealing runs. The middle column shows the orientation of the peptide in between the polymer chains, with the interfacial chains omitted for visibility. In the right column the plastic is fully omitted, showing only the peptide.

Source: Hollóczki, O. Evidence for protein misfolding in the presence of nanoplastics. International Journal of Quantum Chemistry 121, e26372 (2021). https://doi.org/10.1002/qua.26372
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This conformational rearrangement, resulting from electrostatic interactions with the charged plastic surface, disrupts normal cellular metabolism. This process is closely linked to the formation of a "protein corona" on the plastic surface, where the electrostatic forces of the particles cause the rearrangement of protein domains, as confirmed by the data of another study.[123]

The surface charge of the nanoplastic is the primary factor destroying the native architecture of biomolecules. The loss of natural protein structure leads to the impairment of catalytic and transport functions. These conformational changes can initiate a cascade of disruptions extending from the molecular level to the cellular, tissue, and systemic levels of biological organization.

[123] *Mahmoudi, M., Landry, M. P., Moore, A., et al. The protein corona from nanomedicine to environmental science. Nat Rev Mater 8, 422–438 (2023). https://doi.org/10.1038/s41578-023-00552-2*

CELLULAR LEVEL: ACCUMULATION OF MISFOLDED PROTEINS

When the native conformation of proteins is disrupted, the cell activates quality control systems, including molecular chaperones and the proteasome system, which ensure the refolding or degradation of damaged proteins.

However, a significant increase in the number of denatured molecules increases the load on these mechanisms, leading to the accumulation of misfolded proteins in the cytoplasm and the development of proteotoxic stress.[124] This is accompanied by the activation of the endoplasmic reticulum (ER) stress response, increased chaperone expression, temporary suppression of protein synthesis, and disruption of cellular metabolism.

If the restoration of protein homeostasis (proteostasis) proves impossible, signaling pathways leading to apoptosis—programmed cell death—are activated.

DISRUPTION OF CELLULAR FUNCTIONS

Since proteins participate in nearly all biochemical processes within the cell, their structural destabilization leads to multiple functional impairments.

ENZYME SYSTEMS

Changes in the spatial structure of enzymes destabilize their active sites, resulting in a reduction or complete loss of catalytic activity. This slows or blocks various metabolic reactions.

CYTOSKELETON

Damage to structural proteins of the cytoskeleton disrupts intracellular transport and the spatial organization of the cell.

[124] *Hetz, C., Zhang, K. & Kaufman, R. J. Mechanisms, regulation and functions of the unfolded protein response. Nat Rev Mol Cell Biol 21, 421–438 (2020).* https://doi.org/10.1038/s41580-020-0250-z

MEMBRANE RECEPTORS

Conformational changes in receptor proteins can impair intercellular signaling mechanisms and distort the transmission of signaling molecules.

ION CHANNELS

Disruption of ion channel protein structures can alter the cell's ionic balance, which is particularly critical for neurons and other electrically excitable cells.

TOXICITY OF PROTEIN AGGREGATES

A key consequence of protein conformational changes is aggregation. The formation of β-sheets enhances intermolecular interactions, leading to the creation of dense protein aggregates.

These aggregates exhibit pronounced cytotoxicity: they can damage intracellular structures, impair mitochondrial function, and stimulate the production of reactive oxygen species (ROS). Elevated ROS levels cause oxidative stress, which further damages membrane lipids, DNA, and other proteins, forming a resulting "vicious cycle" of cellular damage.[125]

TISSUE LEVEL: INFLAMMATORY RESPONSES

The death of damaged cells triggers the immune system. Innate immune cells respond to damage signals, leading to macrophage activation and the release of inflammatory mediators. In nervous tissue, microglial cells perform a similar function. Prolonged activation of the immune response can lead to chronic inflammation, exacerbating tissue damage.

[125] *Urani, C., Barbieri, R., Alloisio, S., et al. From the Environment to Molecular Interactions of Nanoplastics: Unraveling the Neurotoxic Impacts and the Implications in Neurodegenerative Processes. Applied Sciences 14, 7280 (2024).* https://doi.org/10.3390/app14167280

SYSTEMIC LEVEL: DEVELOPMENT OF PATHOLOGICAL PROCESSES

With long-term exposure to factors causing protein conformational changes, these processes can contribute to the development of various pathologies, including neurodegenerative diseases such as Alzheimer's,[126,127] and Parkinson's,[128] which are characterized by the accumulation of aggregated proteins and impaired neuronal function (Figs. 4.22, 4.23, 4.24). Moreover, chronic denaturation and aggregation of proteins can lead to metabolic disorders and sustain chronic inflammation.

> *"Perhaps, a neuron harboring negatively charged nanoplastic at the acidic lysosome in the cell body would be more likely to develop an α-synuclein inclusion than a healthy lysosome that readily degrades protein aggregates before they can propagate."*[128]

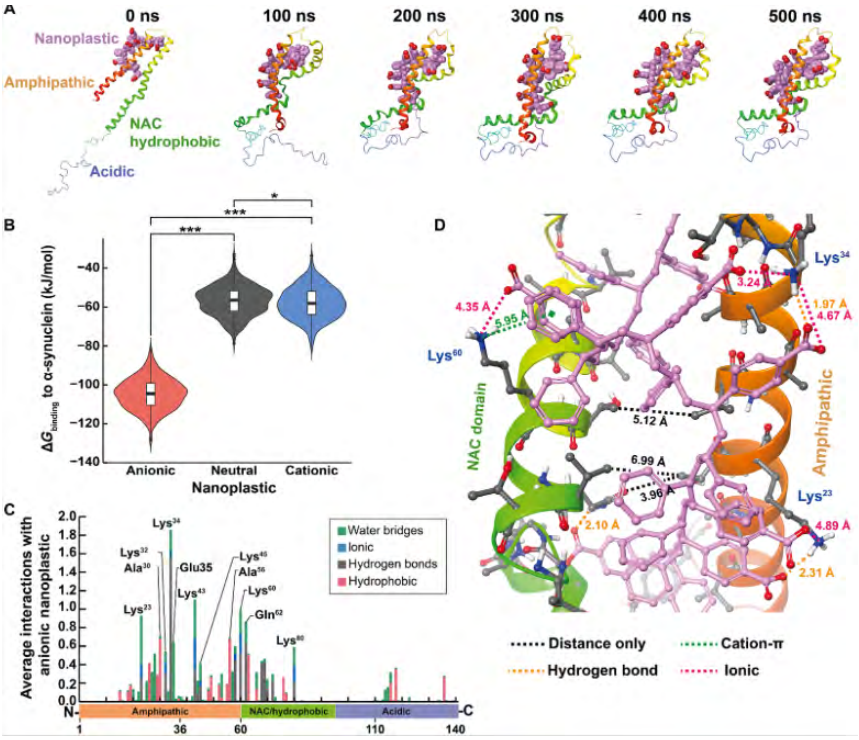


Figure 4.22.

In silico modeling identifies a strong interaction between the amphipathic and non-amyloid component (NAC) domains of α-synuclein and anionic nanoplastic. (A) Snapshots at 100-ns intervals of anionic nanoplastic with monomeric α-synuclein across MD simulations. (B) Violin plots overlaid with box-and-whisker plots representing the distribution of $\Delta G_{binding}$ for each nanoplastic. (C) Histogram demonstrating the average number of interactions between α-synuclein and anionic nanoplastics across converged MD simulation (200 to 500 ns), categorized by four intermolecular interaction types. (D) Representative image of anionic nanoplastic bridging the amphipathic and NAC domains of α-synuclein. Anionic nanoplastic, depicted as ball and stick, shows polar hydrogens only; pink = carbon, red = oxygen. α-Synuclein (ribbons) are colored by residue index; white = hydrogen, gray = carbon, blue = nitrogen, red = oxygen.

Source: Liu, Z., Sokratian, A., Duda, A. M., et al. Anionic nanoplastic contaminants promote Parkinson's disease–associated α-synuclein aggregation. Science Advances 9, eadi8716 (2023). https://doi.org/10.1126/sciadv.adi8716 License: CC BY 4.0.

[126] *Wang, Y., Huang, G., Liang, X., et al. Microglial Clearance of Alzheimer's Amyloid-Beta Obstructed by Nanoplastics. Environ Sci Nano 12, 3247–3260 (2025). https://doi.org/10.1039/D5EN00291E*

[127] *Gou, X., Fu, Y., Li, J., et al. Impact of nanoplastics on Alzheimer 's disease: Enhanced amyloid-β peptide aggregation and augmented neurotoxicity. Journal of Hazardous Materials 465, 133518 (2024). https://doi.org/10.1016/j.jhazmat.2024.133518*

[128] *Liu, Z., Sokratian, A., Duda, A. M., et al. Anionic nanoplastic contaminants promote Parkinson's disease–associated α-synuclein aggregation. Science Advances 9, eadi8716 (2023). https://doi.org/10.1126/sciadv.adi8716*

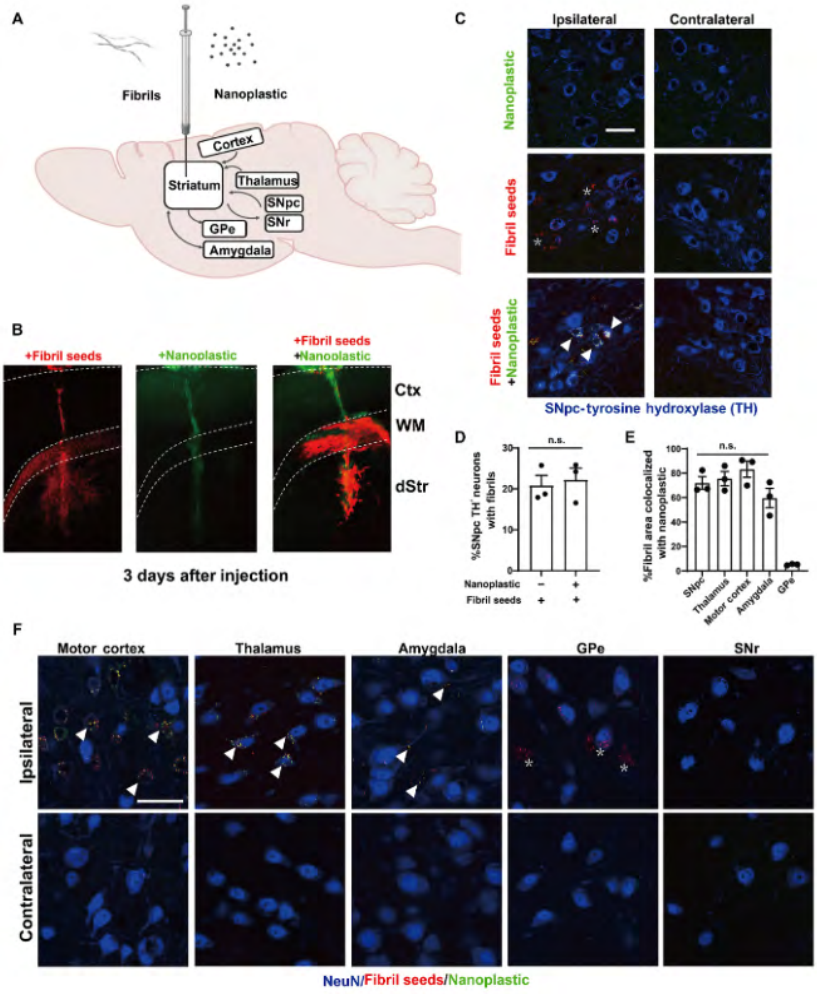


Figure 4.23.

Anionic nanoplastics contaminants traffic with α-synuclein fibrils along retrograde projections and accumulate in neuronal bodies. (A) Depiction of ~P90 male (nontransgenic) mice injected unilaterally in the dorsal striatum with 15 μg of FITC-conjugated nanoplastic, or 4.5 μg of Alexa Fluor 647–labeled human α-synuclein fibrils, or the injection of these particles together. (B) Representative light sheet microscopy of mouse brain, analyzed 3 days after injections, with α-synuclein fibril-only (red), nanoplastic-only (green), or co-particle injections. (C) Representative Airyscan images of dopaminergic cells in the SNpc, with arrowheads indicating cells with vesicles filled with both fibrils and nanoplastics (white signal). (D) Column graph showing the mean proportion of dopaminergic neurons positive for fibrils. (E) Column graph showing that the proportion of fibril-nanoplastic co-positivity is similar across retrograde-targeted brain regions, with the exception of the GPe (anterograde from injection site). (F) Representative Airyscan images of neurons analyzed in (E), with arrowheads highlighting neurons filled with vesicles co-labeled (yellow) with nanoplastic and fibrils, whereas asterisks highlight neurons filled with only fibrils (GPe).

Source: Liu, Z., Sokratian, A., Duda, A. M., et al. Anionic nanoplastic contaminants promote Parkinson's disease–associated α-synuclein aggregation. Science Advances 9, eadi8716 (2023). https://doi.org/10.1126/sciadv.adi8716

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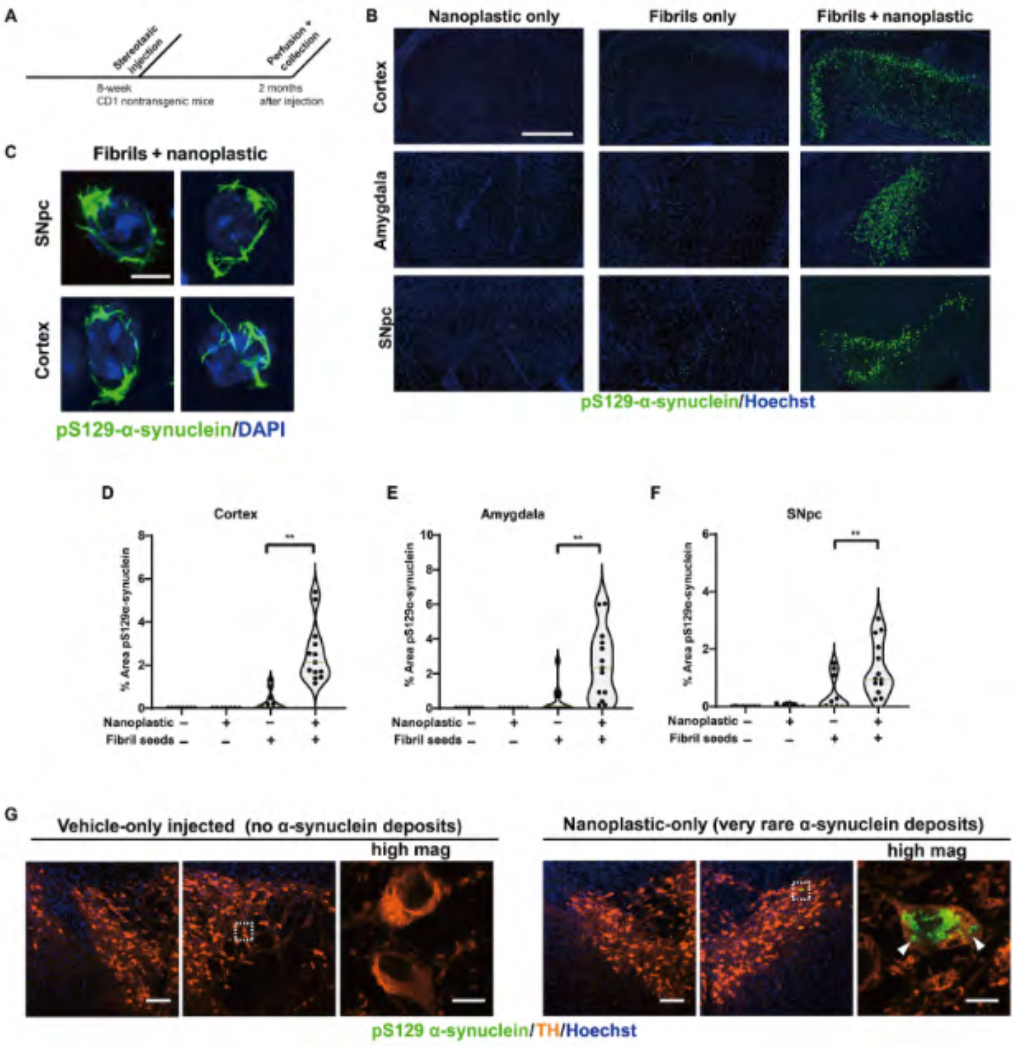


Figure 4.24.

Anionic nanoplastic contaminants in the brain synergize with α-synuclein fibrils to increase the deposition of pathology in the cortex, amygdala, and substantia nigra in nontransgenic mice. (A) Timeline for the injection of adult (nontransgenic) male mice with control saline (N = 8 mice), 4.5 μg of unlabeled αsynuclein fibrils (N = 12 mice), or 4.5 μg of unlabeled α-synuclein fibrils combined with 15 μg of unlabeled nanoplastic (N = 12 mice). (B) Representative immunofluorescence staining of α-synuclein pathology (pS129–α-synuclein, green) in the indicated brain region 2 months after injections. Scale bar, 0.5 mm. (C) Representative highmagnification Airyscan images of pS129–α-synuclein staining inside of dopaminergic neurons (SNpc) or pyramidal neurons in the motor cortex (cortex). Scale bar, 10 μm. (D) Violin plots showing the distributed α-synuclein inclusion burden (pS129–α-synuclein area in each image) in ipsilateral cortex, (E) amygdala, and (F) SNpc. (G) Two months after nanoplastic-only injections, 3 of 10 mice demonstrated robust but isolated αsynuclein inclusions (green) in dopaminergic neurons in the SNpc, suggesting that nanoplastic on its own can induce de novo mature inclusions. Bounding boxes are magnified to highlight these inclusions (arrowheads). Scale bars, 100 and 10 μm (for "high mag").

Source: Liu, Z., Sokratian, A., Duda, A. M., et al. Anionic nanoplastic contaminants promote Parkinson's disease–associated α-synuclein aggregation. Science Advances 9, eadi8716 (2023). https://doi.org/10.1126/sciadv.adi8716

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GENOTOXICITY

The destructive influence on biomolecular structures extends to the genetic apparatus. Studies in 2024 have found that nanoplastics (e.g. polyvinylchloride; PVC) suppress the mechanisms of homologous recombination (HR) in DNA.[129] HR is one of the most accurate ways to repair damaged DNA sections, especially critical after double-strand breaks that can lead to mutations or cell death.[130,131] Suppression of this pathway means cells are less capable of repairing severe DNA damage, increasing error generation (genomic instability) and potentially contributing to chronic inflammation, aging, and an increased risk of pathologies.

Thus, nanoplastics do not merely mechanically damage the cell but interfere with fundamental molecular biological processes, directly destroying the functional structure of proteins and blocking genome defense mechanisms.

CONCLUSION

This analysis indicates that micro- and nanoplastics not only accumulate in tissues but can also interact with biological processes across multiple levels of organization. Small particle size and surface electrostatic properties contribute to interference with key cellular mechanisms, including alterations in membrane potential, ion channel function, and neurotransmission, as well as the induction of oxidative stress and inflammatory responses, mitochondrial dysfunction, and disturbances in energy metabolism.

These processes may occur concurrently and potentially reinforce one another, giving rise to cumulative cellular stress that, under certain conditions, can lead to cell damage, impaired tissue function, and features associated with accelerated biological aging. The underlying mechanisms are thought to involve an initial physical interaction at the particle–cell interface, followed by chemical imbalances (e.g., ionic dysregulation and reactive oxygen species generation), and, in some cases, downstream molecular and structural alterations in biomolecules. As these effects involve fundamental cellular functions, they suggest a potential for systemic biological impact. However, current evidence remains incomplete, and further comprehensive investigation is required to clarify their extent and relevance under real-world exposure conditions.

[129] *Huang, Y., Li, X., Xu, S., et al. Polyvinyl chloride nanoplastics suppress homology-directed repair and promote oxidative stress to induce esophageal epithelial cellular senescence and cGAS-STING-mediated inflammation. Free Radic Biol Med 226, 288–301 (2025).* https://doi.org/10.1016/j.freeradbiomed.2024.11.012

[130] *Jasin, M. & Rothstein, R. Repair of Strand Breaks by Homologous Recombination. Cold Spring Harbor Perspectives in Biology 5, a012740–a012740 (2013).* https://pubmed.ncbi.nlm.nih.gov/24097900/

[131] *Ceccaldi, R., Rondinelli, B. & D'Andrea, A. D. Repair Pathway Choices and Consequences at the Double-Strand Break. Trends Cell Biol 26, 52–64 (2016).* https://doi.org/10.1016/j.tcb.2015.07.009

PART 5.

MICRO- AND NANOPLASTICS AS A POTENTIAL **NEUROBIOLOGICAL RISK FACTOR**

BIOACCUMULATION AND PENETRATION INTO THE CNS

Current experimental and clinical data indicate that micro- and nanoplastic particles (MNPs) can cross physiological barriers and are detected in various human tissues, including the central nervous system (CNS).[132] The highest concentrations of nanoplastics have been found specifically in brain tissue (Fig. 5.1). A research team from the University of New Mexico analyzed brain, liver, and kidney samples from individuals who died in 2016 and 2024. It was found that the concentration of micro- and nanoplastics in the brain samples of healthy individuals was 7 to 30 times higher than in the liver or kidneys. In the brain tissue of patients

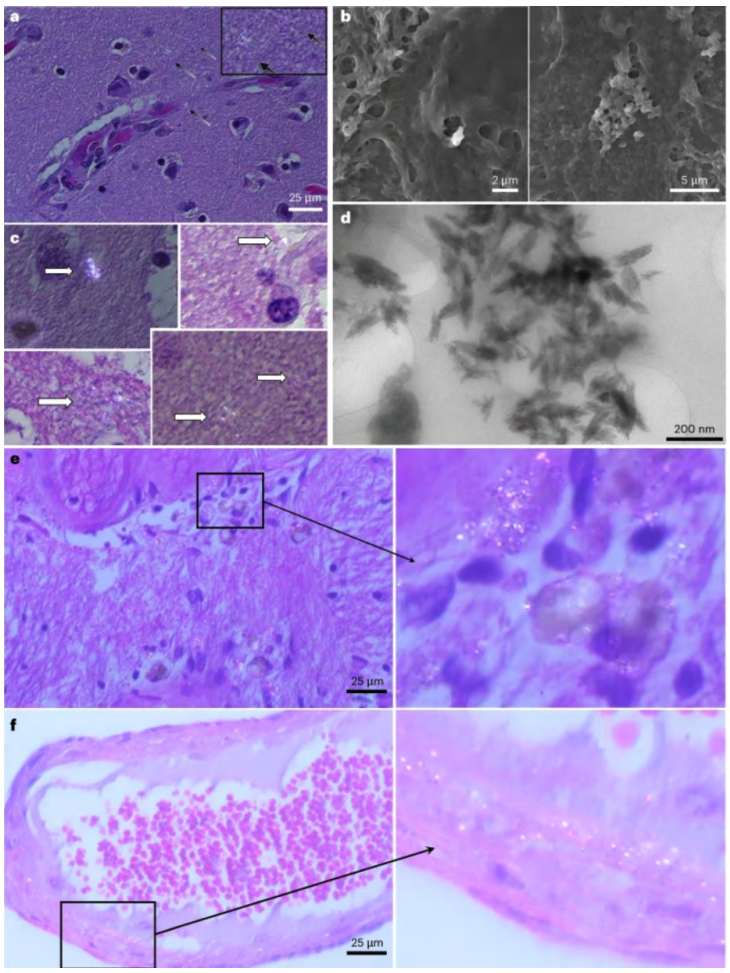


Figure 5.1.

Visualization of putative plastics in the brain. a,b, Polarization wave microscopy (a, black arrows indicate refractory inclusions; inset is a digital magnification for clarity) and scanning electron microscope (SEM) (b, visual fields are 15.4 and 20.1 µm wide) were used to scan sections of brain from decedent human samples. c, Large (>1 µm) inclusions were not observed; additional polarization wave examples are highlighted (white arrows highlight submicron refractory inclusions). Resolution limitations of these technologies drove the use of Transmission electron microscopy (TEM) to examine the extracts from the pellets used for pyrolysis gas chromatography–mass spectrometry. d, Example of TEM images resolved innumerable shard- or flake-like solid particulates following dispersion, with dimensions largely <200 nm in length and <40 nm in width. e,f, Polarization wave microscopy reveals substantially more refractile inclusions in dementia cases, especially in regions with associated immune cell accumulation (e) and along the vascular walls (f). All images were collected on a small subset of participants (n = 10 for normal brains; n = 3 for dementia cases) to provide visual evidence to support analytical chemistry.

Source: Nihart, A. J., Garcia, M. A., El Hayek, E., et al. Bioaccumulation of microplastics in decedent human brains. Nat Med 31, 1114–1119 (2025). https://doi.org/10.1038/s41591-024-03453-1

License: CC BY-NC-ND 4.0.

[132] *Pei, Z., Zhong, H., Li, X., et al. Micro-nanoplastics in the central nervous system: Evidence, mechanisms and perspectives. Toxicology 522, 154424 (2026). https://doi.org/10.1016/j.tox.2026.154424*

[133] *Nihart, A. J., Garcia, M. A., El Hayek, E., et al. Bioaccumulation of microplastics in decedent human brains. Nat Med 31, 1114–1119 (2025). https://doi.org/10.1038/s41591-024-03453-1*

Furthermore, plastic levels in the 2024 brain tissue samples were 50% higher compared to the 2016 samples, indicating an escalating trajectory of bioaccumulation. Quantitative assessments underscore the magnitude of this phenomenon. According to researcher Matthew Campen (University of New Mexico), "In normal people around 45 to 50 years old, we found an average of 4900 micrograms of plastic particles per gram of brain tissue."He noted that this is "equivalent to an entire standard plastic spoon."[134]

According to a mathematical model the concentration of particles in organs is directly proportional to their lipid content.,[135] This explains why contamination levels in the central nervous system are exponentially higher than in the liver or kidneys. This effect is driven by the hydrophobic nature of nanoplastics, which determines their high affinity for lipid structures and therefore makes the brain — an organ composed of approximately 60% lipids[136] — a primary target for bioaccumulation. This process is inherently cumulative: due to a high rate of absorption and exceedingly low elimination efficiency, the concentration of plastic particles in the brain progressively increases over a lifespan. The universality of this mechanism, corroborated across various biological species, allows for a highly reliable projection of neurotoxic burden in humans.

Experimental studies demonstrate that polystyrene nanoparticles (MNPs) disrupt the integrity of the blood-brain barrier (BBB), which is the critical defensive structure regulating permeability and the transport of substances between the blood and CNS tissue to maintain homeostasis. Exposure to nanoparticles damages the tight junction proteins between endothelial cells, which leads to increased BBB permeability and compromises its protective function (Fig. 5.2, 5.3).[137]

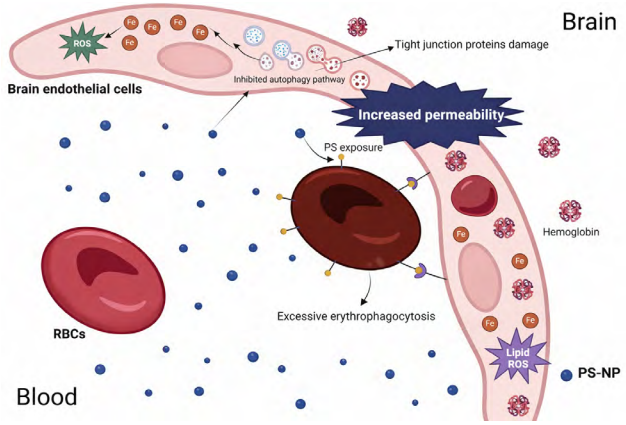


Figure 5.2.

Schematic representation of the impact of polystyrene nanoplastic particles (PS-NPs) on the blood-brain barrier. Nanoplastics disrupt the function of brain vascular endothelial cells, damage tight junction proteins, and increase barrier permeability. Concurrently, autophagy is suppressed, the accumulation of iron and reactive oxygen species (ROS) increases, and the erythrophagocytosis of damaged red blood cells is enhanced. These interconnected processes compromise the integrity of the blood-brain barrier and lead to the development of cellular damage.

Source: Kim, E.-H., Baek, S. M., Park, H. J., et al. Polystyrene nanoplastics promote the blood-brain barrier dysfunction through autophagy pathway and excessive erythrophagocytosis. Ecotoxicology and Environmental Safety 289, 117471 (2025). https://doi.org/10.1016/j.ecoenv.2024.117471

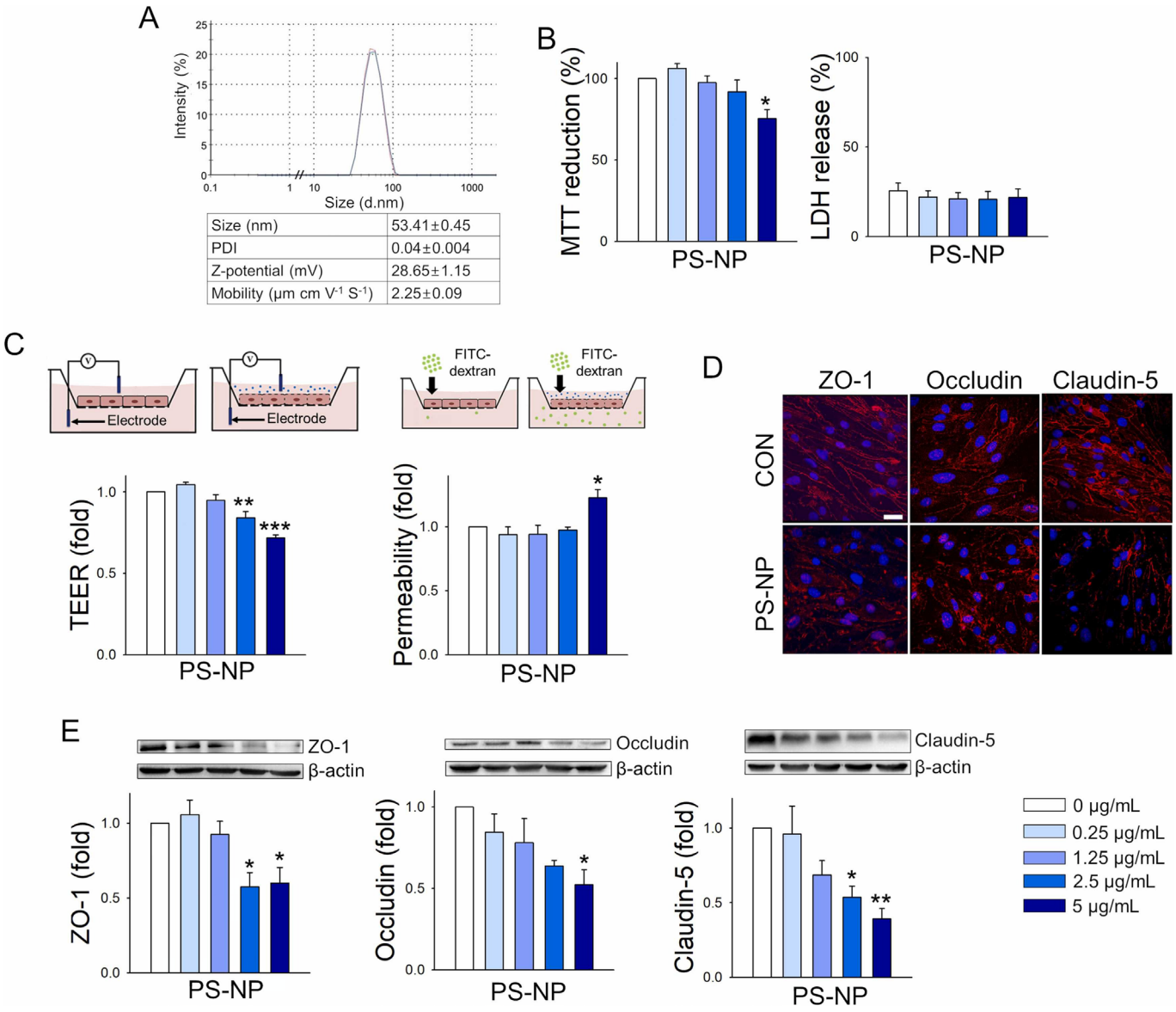
License: CC BY-NC-ND 4.0.

[134] *VRT NWS. Microplastics in de hersenen. (2025). https://www.vrt.be/vrtnws/nl/2025/02/04/microplastics-in-de-hersenen (Accessed 20 March 2026).*

[135] *Ganan-Calvo, A. M. Analytical review of nanoplastic bioaccumulation data and a unified toxicokinetic model: from teleosts to human brain. Preprint (2026). https://doi.org/10.48550/arXiv.2512.22593*

[136] *Hussain, G., Wang, J., Rasul, A., et al. Role of cholesterol and sphingolipids in brain development and neurological diseases. Lipids Health Dis 18, 26 (2019). https://doi.org/10.1186/s12944-019-0965-z*

[137] *Kim, E.-H., Baek, S. M., Park, H. J., et al. Polystyrene nanoplastics promote the blood-brain barrier dysfunction through autophagy pathway and excessive erythrophagocytosis. Ecotoxicology and Environmental Safety 289, 117471 (2025). https://doi.org/10.1016/j.ecoenv.2024.117471*



Size (nm)	53.41±0.45
PDI	0.04±0.004
Z-potential (mV)	28.65±1.15
Mobility (μm cm V^{-1} S^{-1})	2.25±0.09

Figure 5.3.

Disrupted BBB function in PS-NP-treated brain endothelial cell (bEnd.3 cell). (A) The characterization of 50 nm-sized PS-NP was analyzed through DLS. (B) The cell viability was measured using MTT and LDH assay after 24 hours of exposure to 50 nm-sized PS-NP (n = 3). (C) To quantify the permeability of bEnd.3 cells, the TEER and FITC-dextran were used 24 hours after PS-NP exposure (n = 3–4). (D) The intensity of TJ proteins was identified using confocal in bEnd.3 cells expose to 5 μg/mL of PS-NP for 24 hours (n = 3). (E) The TJ protein levels including ZO-1, occludin and claudin-5 were detected by western blot 24 hours after treatment of various concentrations of PS-NP (n = 4–6). Scale bar = 30 μm. All data are presented as mean ± SEM. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ vs. 0 μg/mL of PS-NP.

Source: Kim, E.-H., Baek, S. M., Park, H. J., et al. Polystyrene nanoplastics promote the blood-brain barrier dysfunction through autophagy pathway and excessive erythrophagocytosis. Ecotoxicology and Environmental Safety 289, 117471 (2025). https://doi.org/10.1016/j.ecoenv.2024.117471

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The mechanism underlying this disruption is associated with autophagy inhibition — a critical cellular process for degrading damaged components. Autophagy inhibition induced by PS-NPs leads to an accumulation of iron within endothelial cells. This iron overload exacerbates oxidative stress and promotes the induction of ferroptosis — a form of programmed cell death associated with lipid metabolism dysfunction and excess iron. Thus, the impairment of autophagy serves as the key link explaining why BBB permeability increases.[137]

An additional mechanism involves the impact of PS-NPs on red blood cells (erythrocytes). As nanoparticles damage these blood cells, the endothelial cells begin to actively engulf them (a process known as erythrophagocytosis). This process results in a supplementary influx of iron into the endothelial cells, further amplifying oxidative stress damage.[137] Consequently, nanoplastics can compromise the blood-brain barrier through two distinct pathways: directly, by damaging the endothelium (the cells lining the blood vessels), and indirectly, by altering the properties of circulating blood cells.

Furthermore, morphological studies indicate an alternative pathway for plastic particles to infiltrate the brain (Fig. 5.4). Specifically, analysis of the olfactory bulbs in 15 deceased individuals revealed the presence of microplastics in 8 of them (Fig. 5.5, 5.6).[138] These findings suggest that the inhalational route may serve as a viable physiological pathway for plastic particles to enter the brain, completely bypassing the blood-brain barrier. This occurs because olfactory neurons and their nerve fibers directly connect the nasal cavity to the olfactory bulb through the cribriform plate of the ethmoid bone.

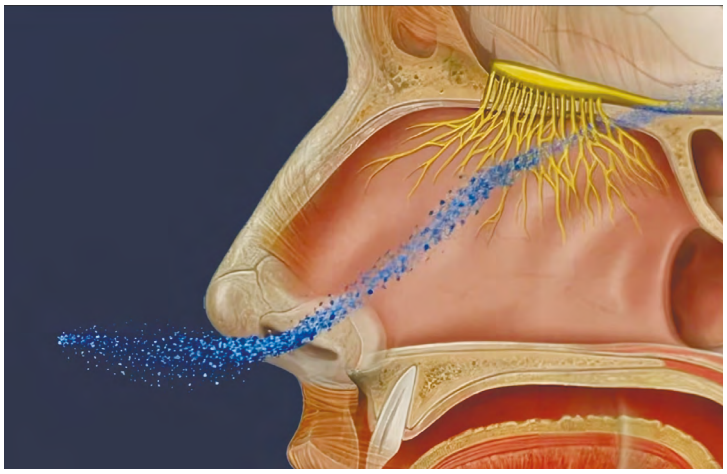


Figure 5.4.

Illustration of a potential entry pathway for microplastic particles into the brain. Olfactory neurons provide a direct connection between the nasal cavity and the olfactory bulb, potentially allowing inhaled particles to bypass the blood-brain barrier and reach the brain directly.

[138] *Amato-Lourenço, L. F., Dantas, K. C., Júnior, G. R., et al. Microplastics in the Olfactory Bulb of the Human Brain. JAMA Netw Open 7, e2440018 (2024). https://doi.org/10.1001/jamanetworkopen.2024.40018*

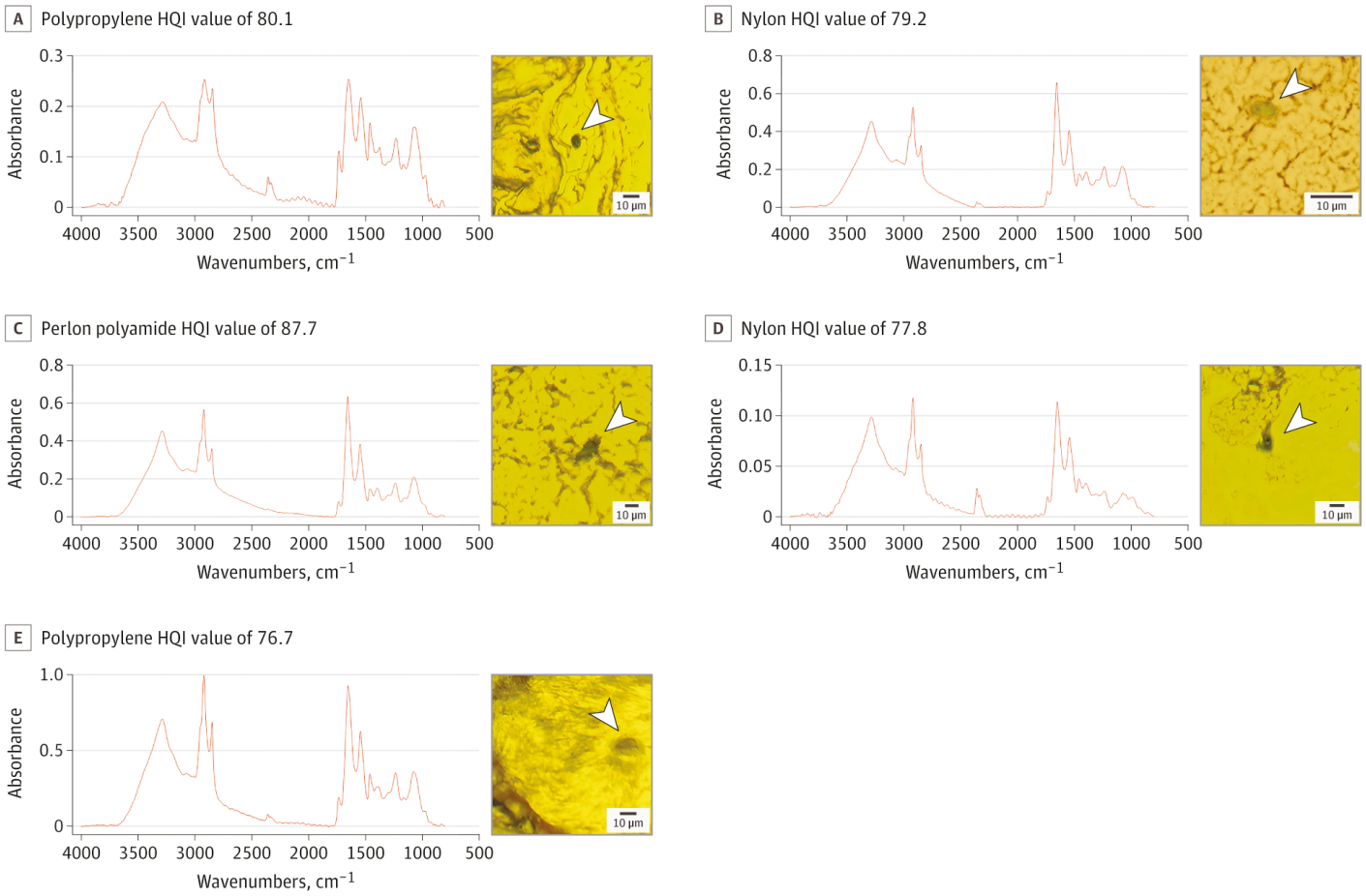


Figure 5.5.

Microphotographs and micro-Fourier transform infrared (µFTIR) spectra of the microplastics found in the olfactory bulb tissue.

Source: Amato-Lourenço, L. F., Dantas, K. C., Júnior, G. R., et al. Microplastics in the Olfactory Bulb of the Human Brain. JAMA Netw Open 7, e2440018 (2024). https://doi.org/10.1001/jamanetworkopen.2024.40018

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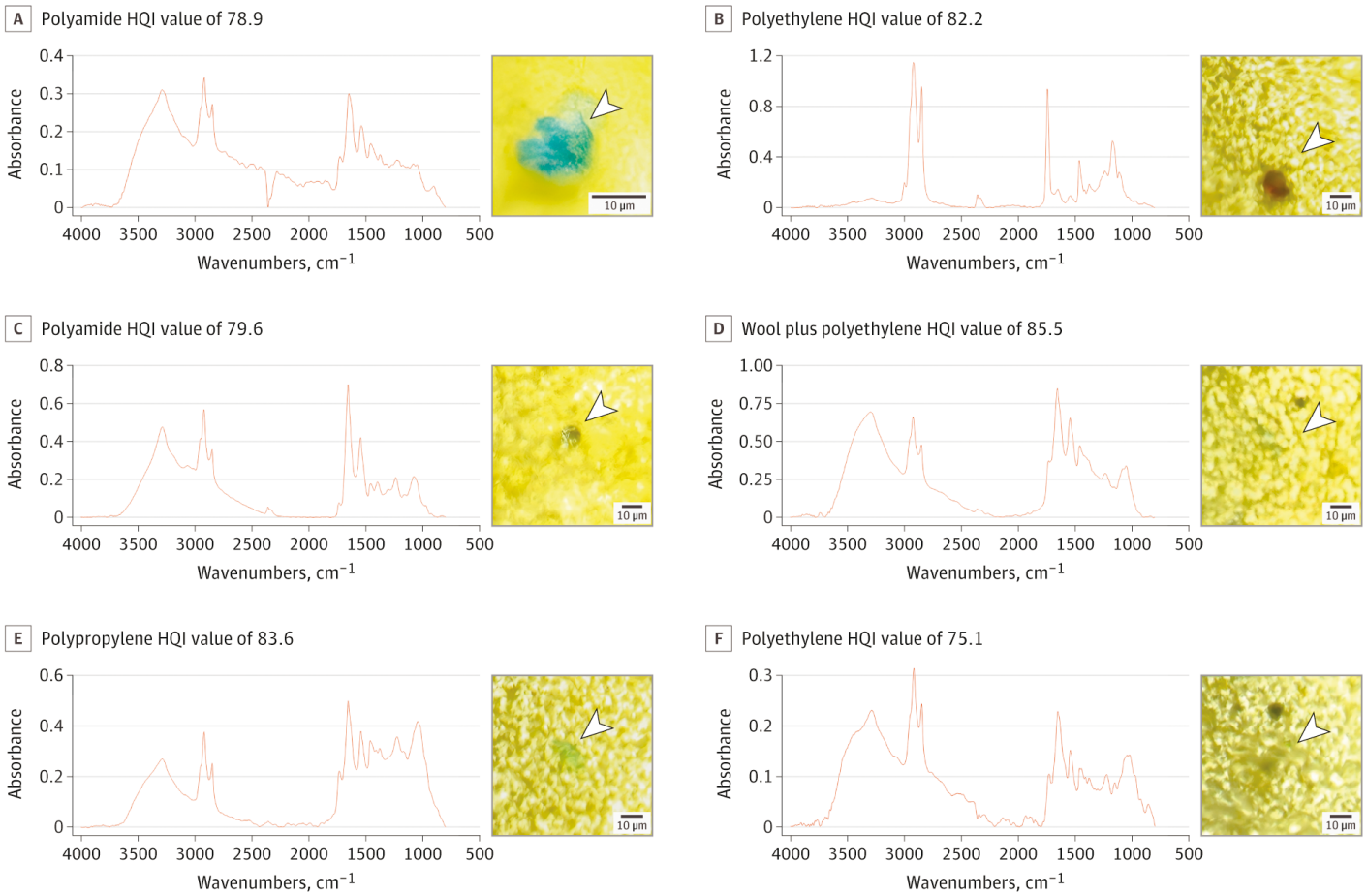


Figure 5.6.

Microphotographs and micro-Fourier transform infrared (µFTIR) spectra of the main microplastics found in the digested olfactory bulb.

Source: Amato-Lourenço, L. F., Dantas, K. C., Júnior, G. R., et al. Microplastics in the Olfactory Bulb of the Human Brain. JAMA Netw Open 7, e2440018 (2024). https://doi.org/10.1001/jamanetworkopen.2024.40018 License: CC BY.

These findings suggest that micro- and nanoplastics can be considered an emerging potential neurobiological risk factor.[139]

NEUROINFLAMMATION AND NEURODEGENERATION

Neuronal degradation induced by nanoplastic exposure,[140,141,142] is viewed as a primary mechanism (Fig. 5.7, 5.8) through which cellular damage translates into clinically significant systemic dysfunctions (see Part 4).

[139] *Ma, Q., Lei, J., Pang, Y., et al. Neurotoxicity of Micro- and Nanoplastics: A Comprehensive Review of Central Nervous System Impacts. Environ. Health 4, 4–24 (2026). https://doi.org/10.1021/envhealth.5c00087*

[140] *Seward, D. M., Keegan, C. D. & Davey, G. P. Polystyrene nanoplastics target electron transport chain complexes in brain mitochondria. Journal of Hazardous Materials: Plastics 1, 100003 (2025). https://doi.org/10.1016/j.hazmp.2025.100003*

[141] *Araújo, A. M., Mota, C., Ramos, H., et al. The neurotoxic threat of micro- and nanoplastics: evidence from In Vitro and In Vivo models. Arch Toxicol 99, 3505–3525 (2025). https://doi.org/10.1007/s00204-025-04091-3*

[142] *Mota, C., Araújo, A. M., Enea, M., et al. Are all nanoplastics equally neurotoxic? Influence of size and surface functionalization on the toxicity of polystyrene nanoplastics in human neuronal cells. Environmental Pollution 390, 127445 (2026). https://doi.org/10.1016/j.envpol.2025.127445*

Because neurons are integral to both higher cognitive functions and autonomic processes, their dysfunction can disrupt the regulation of cardiovascular, respiratory, digestive, and other physiological systems. Consequently, the impact of nanoplastics extends far beyond localized cellular toxicity, acquiring profound systemic clinical significance.[139,143]

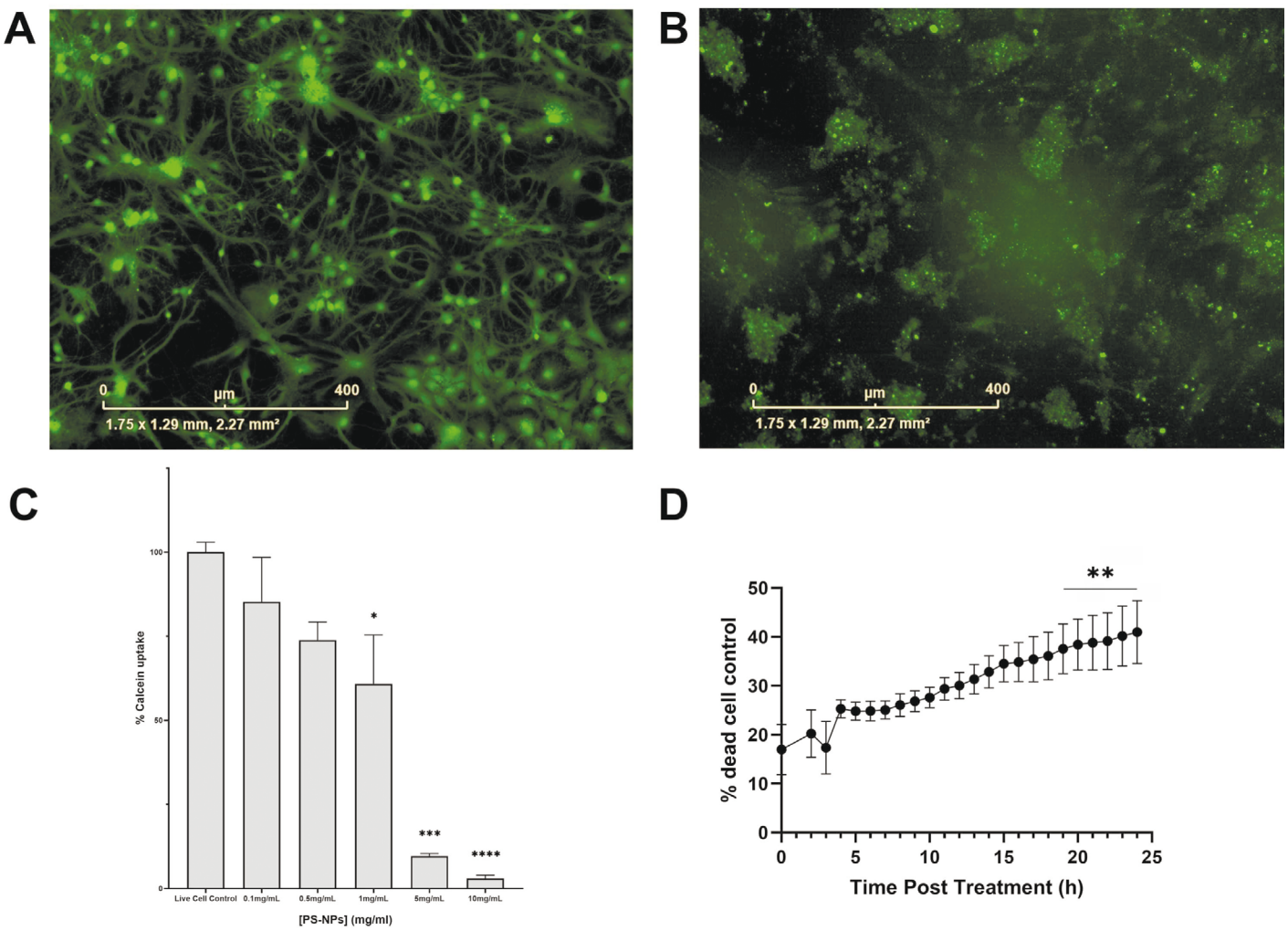


Figure 5.7.

Polystyrene nanoplastics are toxic to primary cortical neurons. Live cell control (A) and dead cell control (B). % Calcein uptake (C). Propidium iodide staining of cells treated with 1 mg/ml of PS-NPs (D), n = 6. Results are displayed as mean ± SEM. One-Way ANOVA (C) or Friedman test (D): ns = not significant, * = $p \leq 0.05$, ** = $p \leq 0.01$, *** = $p \leq 0.001$, and **** = $p \leq 0.0001$.

Source: Seward, D. M., Keegan, C. D. & Davey, G. P. Polystyrene nanoplastics target electron transport chain complexes in brain mitochondria. Journal of Hazardous Materials: Plastics 1, 100003 (2025). https://doi.org/10.1016/j.hazmp.2025.100003 License: CC BY 4.0.

[143] *Zhang, S., Wu, J., Wu, M., et al. From environment to brain: the role of microplastics in neurobehavioral disorders. Front. Neurosci. 19, 1691461 (2025). https://doi.org/10.3389/fnins.2025.1691461*

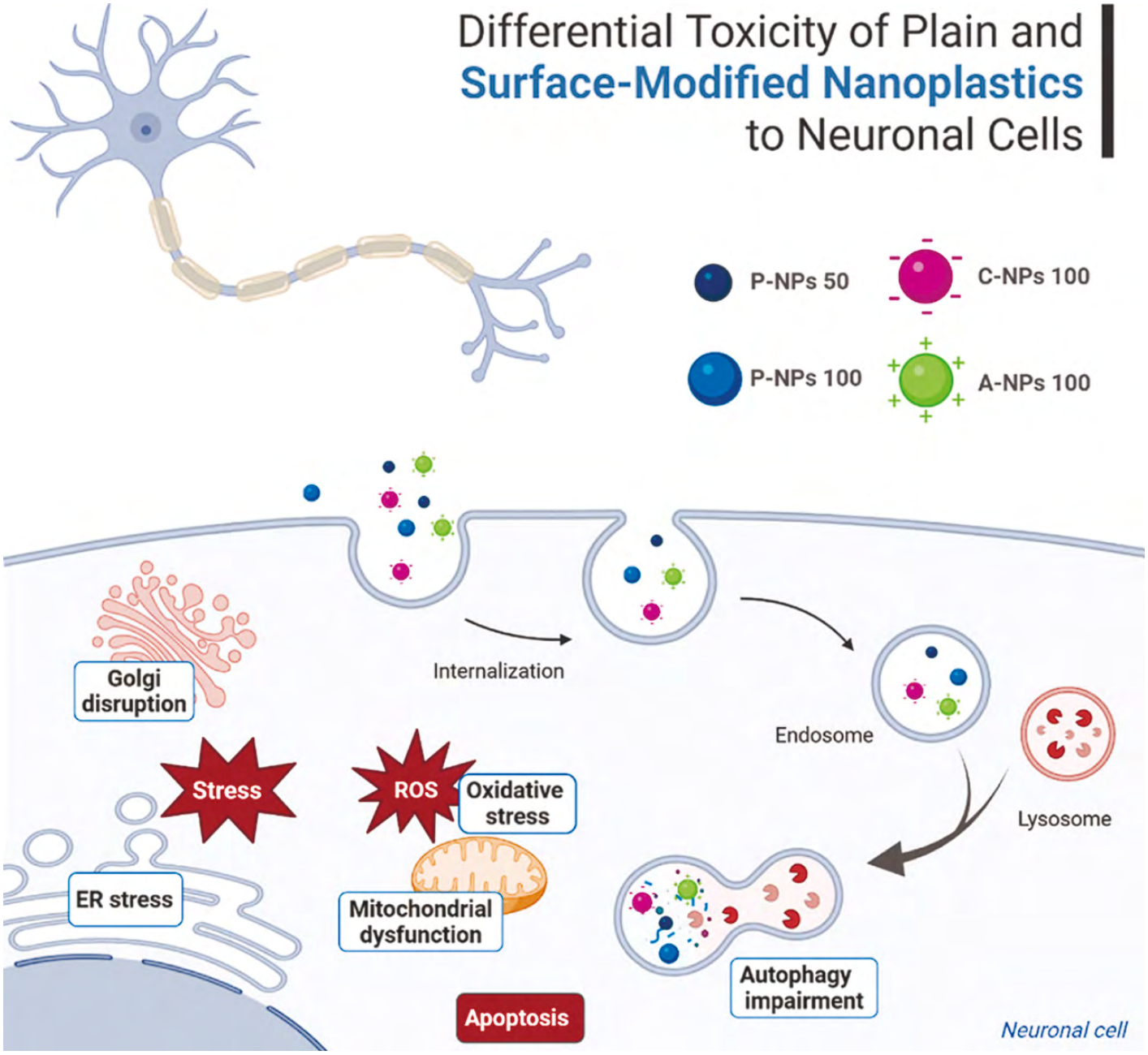


Figure 5.8.

Putative mechanisms of PS-NPs-induced toxicity in neuronal SH-SY5Y cells.

Source: Mota, C., Araújo, A. M., Enea, M., et al. Are all nanoplastics equally neurotoxic? Influence of size and surface functionalization on the toxicity of polystyrene nanoplastics in human neuronal cells. Environmental Pollution 390, 127445 (2026). https://doi.org/10.1016/j.envpol.2025.127445 License: CC BY 4.0.

Upon penetrating brain tissue, micro- and nanoplastic particles accumulate in both neurons and glial cells. Given that glial cells outnumber neurons in the central nervous system (CNS) by 1.5 to 10 times,[144] their dysfunction can have critical consequences for the entire neural network.[145] Glial cells provide essential metabolic and trophic support to neurons, participate in the formation and maintenance of synaptic networks, and regulate inflammatory processes. Therefore, damage to glial cells directly compromises neuronal survival and functional activity.

At the cellular level, MNP exposure initiates oxidative stress in both neurons and glia. This damage potentially activates microglia — specialized immune cells of the brain that represent a specific type of glia. Under normal conditions, their primary function is to clear out damaged structures and protect neural tissue. However, under continuous exposure to nanoplastics — as new MNP particles continually enter the brain — microglia potentially remain in a state of prolonged activation. As a result, this defensive response may gradually transition into chronic neuroinflammation, which subsequently amplifies oxidative stress and cellular damage. This establishes a pathological feedback loop in which cellular damage triggers chronic inflammation, further amplifying oxidative stress and ultimately leading to progressive neural tissue degeneration(Fig. 5.9).

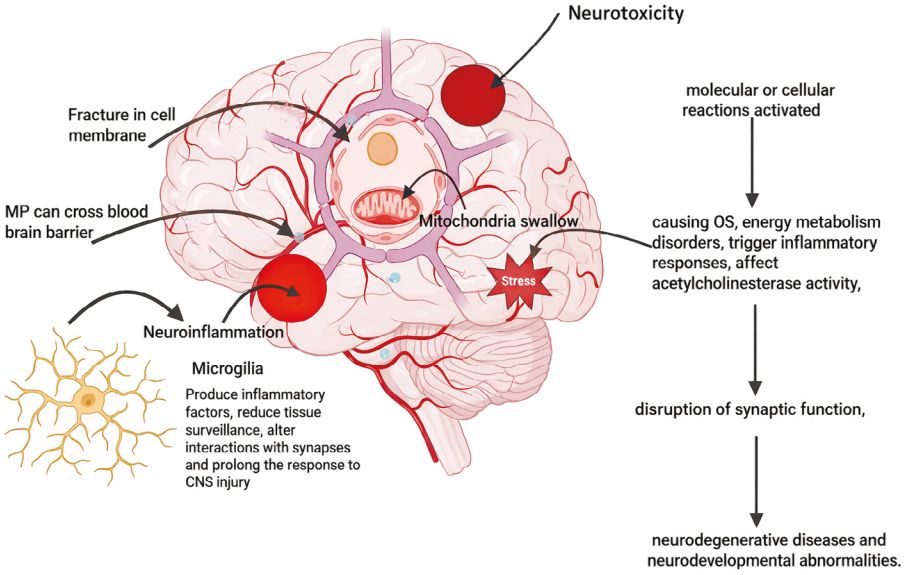


Figure 5.9.

MPs and NPs causing ROS stress, neuroinflammation, neurotoxicity, fracture in cell membrane, neurodegenerative disease, and neurodevelopmental abnormalities.

Source: Thapliyal, C., Negi, S., Nagarkoti, S., et al. Mechanistic insight into potential toxic effects of microplastics and nanoplastics on human health. Discov Appl Sci 7, 645 (2025). https://doi.org/10.1007/s42452-025-07214-8

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[144] *von Bartheld, C. S., Bahney, J. & Herculano-Houzel, S. The search for true numbers of neurons and glial cells in the human brain: A review of 150 years of cell counting. Journal of Comparative Neurology 524, 3865–3895 (2016). https://doi.org/10.1002/cne.24040*

[145] *Thapliyal, C., Negi, S., Nagarkoti, S., et al. Mechanistic insight into potential toxic effects of microplastics and nanoplastics on human health. Discov Appl Sci 7, 645 (2025). https://doi.org/10.1007/s42452-025-07214-8*

POTENTIAL CLINICAL AND EPIDEMIOLOGICAL IMPLICATIONS

Current data indicate that the cascade of pathological processes (oxidative stress, mitochondrial dysfunction, and neuroinflammation) positions micro- and nanoplastics as a significant risk factor not only for classical neurodegenerative diseases but also for a broader spectrum of inflammatory and autoimmune CNS pathologies.[143,146,147] Consequently, MNP neurotoxicity is potentially linked to a much wider array of disorders than previously recognized.

Primarily, neuronal damage is viewed through the lens of neurodegenerative diseases, such as Alzheimer's disease, Parkinson's disease, Amyotrophic Lateral Sclerosis (ALS), and Multiple Sclerosis (MS).[148] All these pathologies are characterized by the progressive loss of neurons and the disruption of neural networks, leading to a decline in cognitive, motor, and other bodily functions. The loss of nerve cells in these conditions causes irreversible structural and functional changes in the nervous system, ultimately impairing an individual's ability to think, move, and sustain normal daily living activities.

Another distinct group of consequences involves conditions like epilepsy[149,150] and stroke.[151] In these clinical scenarios, neuronal destruction and impaired electrical conductivity lead to either pathological electroencephalographic activity or acute cerebrovascular events followed by neural tissue damage. The destabilization of neural networks creates the underlying substrate for functional impairments, which can manifest as either acute or chronic conditions.

Neurological disorders currently constitute the leading cause of physical and cognitive disability worldwide, affecting an estimated 3.4 billion people.[152] Over the past three decades, their prevalence has surged significantly, and projections indicate that the number of patients could double within the next 20 years.[153]

The parallel rise in neurological disorders alongside escalating plastic pollution suggests a potential link between the two. Specifically, a growing body of studies evaluate micro- and nanoplastics as an emerging risk factor for neurological health.

[146] *Baroni, A., Moulton, C., Cristina, M., et al. Nano- and Microplastics in the Brain: An Emerging Threat to Neural Health. Nanomaterials 15, 1361 (2025). https://doi.org/10.3390/nano15171361*

[147] *Liu, S., He, Y., Yin, J., et al. Neurotoxicities induced by micro/nanoplastics: A review focusing on the risks of neurological diseases. Journal of Hazardous Materials 469, 134054 (2024). https://doi.org/10.1016/j.jhazmat.2024.134054*

[148] *Eisen, A., Pioro, E. P., Goutman, S. A., et al. Nanoplastics and Neurodegeneration in ALS. Brain Sciences 14, 471 (2024). https://doi.org/10.3390/brainsci14050471*

[149] *Xie, R., Xiao, X., Zhao, W., et al. Association between long-term exposure of polystyrene microplastics and exacerbation of seizure symptoms: Evidence from multiple approaches. Ecotoxicology and Environmental Safety 302, 118741 (2025). https://doi.org/10.1016/j.ecoenv.2025.118741*

[150] *Huang, H., Hou, J., Li, M., et al. Microplastics in the bloodstream can induce cerebral thrombosis by causing cell obstruction and lead to neurobehavioral abnormalities. Sci. Adv. 11, eadr8243 (2025). https://doi.org/10.1126/sciadv.adr8243*

[151] *Marfella, R., Prattichizzo, F., Sardu, C., et al. Microplastics and Nanoplastics in Atheromas and Cardiovascular Events. N Engl J Med 390, 900–910 (2024). https://doi.org/10.1056/NEJMoa2309822*

[152] *Steinmetz, J. D., Seeher, K. M., Schiess, N., et al. Global, regional, and national burden of disorders affecting the nervous system, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. The Lancet Neurology 23, 344–381 (2024). https://doi.org/10.1016/S1474-4422(24)00038-3*

[153] *Van Schependom, J. & D'haeseleer, M. Advances in Neurodegenerative Diseases. Journal of Clinical Medicine 12, 1709 (2023). https://doi.org/10.3390/jcm12051709*

A study led by Dr. Sarju Ganatra and presented at the American Academy of Neurology analyzed data from residents of 218 US coastal counties. Health metrics derived from CDC reports were cross-referenced with regional microplastic pollution levels. The findings revealed that in areas with high concentrations of micro- and nanoplastics, the incidence of memory and cognitive impairments was 9% higher; motor disorders were 6% higher; difficulties with self-care were 16% higher; and the inability to maintain functional autonomy was 8% higher compared to regions with low pollution levels.[154]

Furthermore, populations in coastal areas with high levels of microplastic pollution showed a higher prevalence of cardiovascular diseases, stroke, and metabolic dysfunction. This points toward a multifaceted toxicity paradigm, simultaneously impacting the nervous, cardiovascular, and metabolic systems.[154]

In conclusion, the presented data indicate a statistically significant correlation between environmental micro-/nanoplastic pollution levels and a decline in both cognitive performance and functional independence among the population.

It is crucial to recognize that neurons are distributed throughout the organism, providing coordination for internal visceral organs. Damage to neural structures can lead to autonomic nervous system dysfunction, disrupting the regulation of the cardiovascular, digestive, and other vital systems. Consequently, the impact of nanoplastics on neurons is inherently systemic and extends far beyond the central nervous system alone. If nanoplastics can trigger neuronal damage mechanisms at the cellular level, their downstream effects could manifest as a broad spectrum of neurological, psychiatric, and somatic disorders.

The overlapping timelines of rising neurological disease rates and escalating plastic pollution underscore the urgent need for comprehensive, interdisciplinary research. Although definitive causal mechanisms require further investigation, the magnitude of these correlations strongly positions micro- and nanoplastics as a critical emerging risk factor for global public health.

[154] *Makwana, B., Desai, B., Srinivasan, J., et al. Impact of Marine Microplastics on Neurologic and Functional Disabilities: A Population-Level Study. European Journal of Neurology 32, e70144 (2025). https://doi.org/10.1111/ene.70144*

PART 6.

SYSTEMIC IMPACT OF MICRO- AND NANOPLASTICS ON THE HUMAN BODY THROUGH THE **DESTABILIZATION OF THE GUT–BRAIN AXIS** AND **DISRUPTION OF INTESTINAL BARRIER FUNCTIONS**

Modern physiology views the gut not merely as a digestive organ, but as a fundamental regulatory center, often referred to as the "second brain." It harbors approximately 70 %[155] of the immune system's cells, roughly 200–600 million neurons [156] of the enteric nervous system, and a complex microbiota ecosystem comprising hundreds of trillions of microorganisms. By affecting the intestinal epithelium and the microbiome, plastic particles may contribute to a cascade of pathological responses, potentially linking local disturbances to systemic inflammatory processes.

These effects may involve alterations in neuroimmune signaling along the gut–brain axis and have been hypothesized to contribute to the development or progression of certain neurodegenerative and metabolic disorders. However, current evidence remains limited, and causal relationships have not yet been conclusively established.

IMPAIRMENT OF PHYSICAL INTEGRITY AND SELECTIVE PERMEABILITY OF THE INTESTINAL BARRIER

The intestinal barrier represents a key site of interaction for nanoplastics—a highly specialized system that enables nutrient absorption while simultaneously preventing the passage of toxins and pathogens. (Fig. 6.1). The integrity of this structure is maintained by the epithelium, the mucus layer, and a system of tight junctions. Micro- and nanoplastic particles act as a destructive factor: their accumulation leads to damage of epithelial cells and functional weakening of tight junctions.[157]

As a result, increased intestinal permeability develops: the barrier loses its selectivity, opening a pathway for the uncontrolled translocation of foreign agents into the submucosal layer (Fig. 6.2).

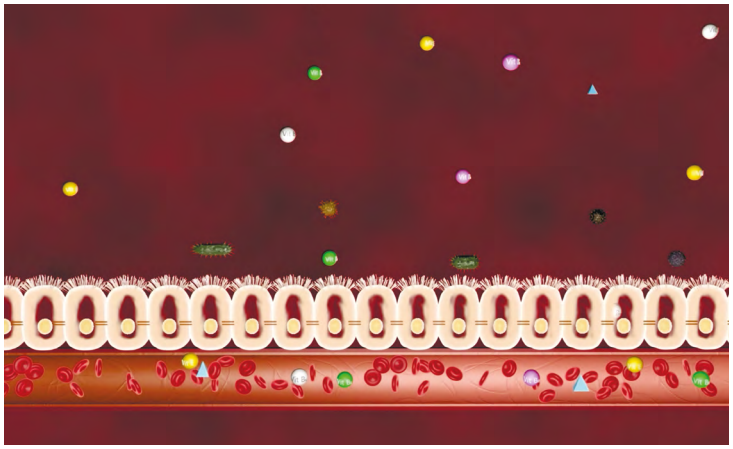


Figure 6.1.

Illustration of the normal state of the intestinal barrier that facilitates nutrient absorption while simultaneously blocking toxins and pathogens.

[155] *Wiertsema, S. P., Bergenhenegouwen, J. van, Garssen, J., et al. The Interplay between the Gut Microbiome and the Immune System in the Context of Infectious Diseases throughout Life and the Role of Nutrition in Optimizing Treatment Strategies. Nutrients 13, 886 (2021). https://doi.org/10.3390/nu13030886*

[156] *Fleming, M. A., Ehsan, L., Moore, S. R., et al. The Enteric Nervous System and Its Emerging Role as a Therapeutic Target. Gastroenterology Research and Practice 2020, 1–13 (2020). https://doi.org/10.1155/2020/8024171*

[157] *Liang, B., Zhong, Y., Huang, Y., et al. Underestimated health risks: polystyrene micro- and nanoplastics jointly induce intestinal barrier dysfunction by ROS-mediated epithelial cell apoptosis. Part Fibre Toxicol 18, 20 (2021). https://doi.org/10.1186/s12989-021-00414-1*



Figure 6.2.

Illustration of the disruption of intestinal barrier integrity under the influence of MNPs, which leads to the uncontrolled translocation of foreign agents.

ROLE OF SURFACE CHARGE AND FORMATION OF THE "PROTEIN CORONA"

The mechanism of interaction between plastic particles and living tissue is largely determined by their physicochemical properties, particularly their surface charge. Upon entering the gastrointestinal tract, nanoplastics instantly acquire a layer of adsorbed proteins and lipids, forming a so-called "protein corona"which determines how they are recognized by cells.[158] Electrostatic interaction is a key factor in their toxicity: protective mucus components[159] (mucins) possess a negative charge; therefore, positively charged[160] (cationic) particles bind more actively to the mucus and are retained longer near the epithelium, thereby increasing the exposure time to the toxic agent.

At the epithelial level, charged nanoparticles, especially cationic ones, interact more actively with cell membranes and undergo endocytosis more frequently.[160] Experimental studies on human intestinal epithelium models in vitro, as well as in animal models, demonstrate that such particles cause cellular stress, induce an inflammatory response, and reduce the expression of proteins responsible for the density of tight junctions.[161,162] Consequently, the permeability of the intestinal barrier increases, and its selectivity is weakened (Fig. 6.3).

[158] *Brouwer, H., Porbahaie, M., Boeren, S., et al. The in vitro gastrointestinal digestion-associated protein corona of polystyrene nano- and microplastics increases their uptake by human THP-1-derived macrophages. Part Fibre Toxicol 21, 4 (2024). https://doi.org/10.1186/s12989-024-00563-z*

[159] *Bandi, S. P., Kumbhar, Y. S. & Venuganti, V. V. K. Effect of particle size and surface charge of nanoparticles in penetration through intestinal mucus barrier. J Nanopart Res 22, 62 (2020). https://doi.org/10.1007/s11051-020-04785-y*

[160] *Liu, X., Wang, J., Borghi, A., et al. Functionalized polystyrene nanoplastics induce distinct toxicity and transcriptomic changes in human intestinal Caco-2 cells. Micropl.&Nanopl. 6, 11 (2026). https://doi.org/10.1186/s43591-025-00170-5*

[161] *Jin, Y., Lu, L., Tu, W., et al. Impacts of polystyrene microplastic on the gut barrier, microbiota and metabolism of mice. Sci Total Environ 649, 308–317 (2019) . https://doi.org/10.1016/j.scitotenv.2018.08.353*

[162] *Hsu, W.-H., Chen, Y.-Z., Chiang, Y.-T., et al. Polystyrene nanoplastics disrupt the intestinal microenvironment by altering bacteria-host interactions through extracellular vesicle-delivered microRNAs. Nat Commun 16, 5026 (2025). https://doi.org/10.1038/s41467-025-59884-y*

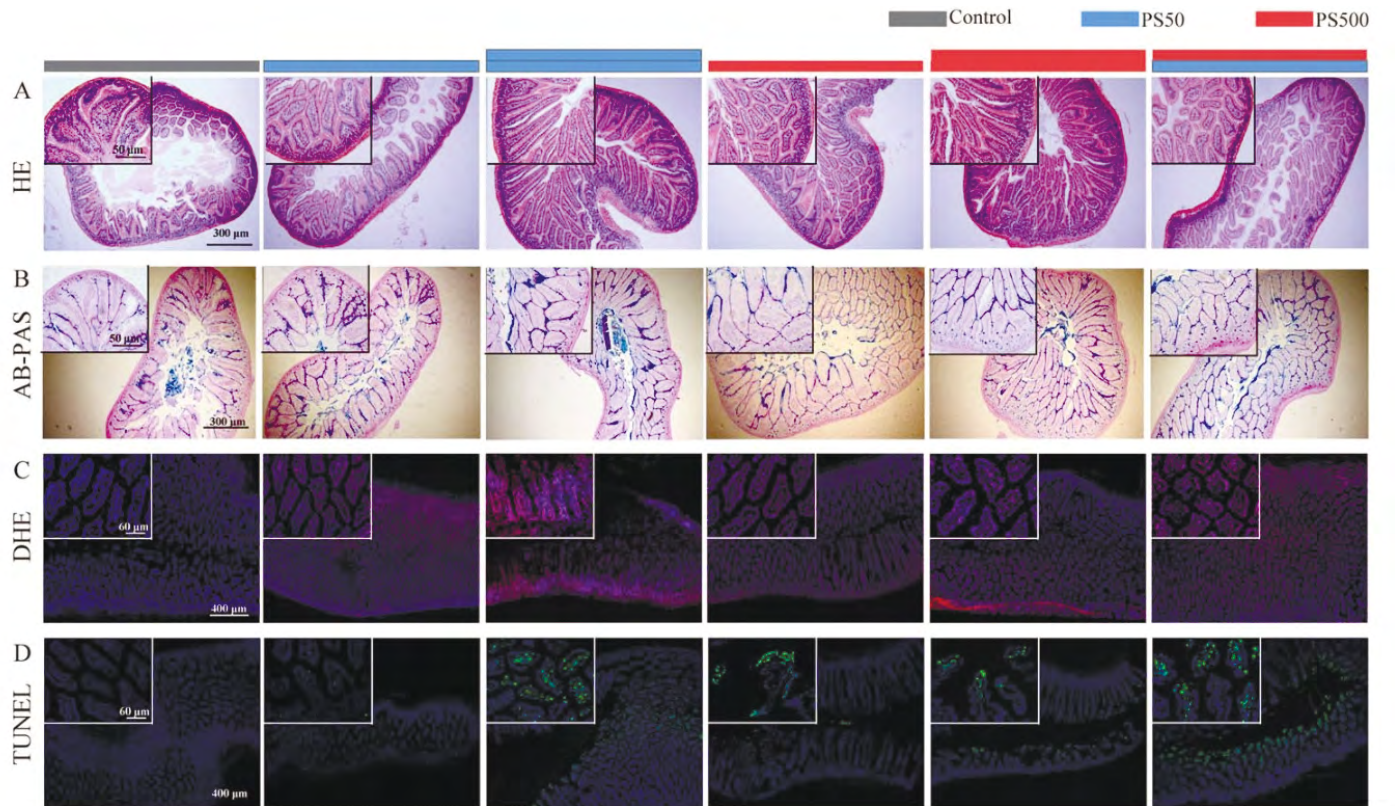


Figure 6.3.

Mixture of polystyrene micro- and nanoplastics PS aggravated dysfunction of intestinal barrier in mice. Representative merged images of the jejunum sections A) stained with H&E to assess the pathological changes; B) stained with AB-PAS to assess the change of mucin secretion, the mucin was stained in blue; C) stained with DHE to assess the ROS generation, the ROS was stained in red and the nucleus was stained with DAPI in blue; D) stained with TUNEL to assess cell apoptosis, the apoptosis cells were stained in green and the nucleus was stained with DAPI in blue.

Source: Liang, B., Zhong, Y., Huang, Y., et al. Underestimated health risks: polystyrene micro- and nanoplastics jointly induce intestinal barrier dysfunction by ROS-mediated epithelial cell apoptosis. Part Fibre Toxicol 18, 20 (2021). https://doi.org/10.1186/s12989-021-00414-1 License: CC BY 4.0.

IMMUNE ACTIVATION AND THE CHRONICITY OF INFLAMMATION

Increased barrier permeability leads to the penetration of bacterial antigens, toxins, and plastic particles themselves into the localization zones of the gut-associated lymphoid tissue (GALT). Instead of maintaining immune tolerance, a shift toward chronic, low-level activation of the immune system potentially occurs.[163] Nanoplastics provoke systemic inflammation: macrophages and other cells of the innate immune system engulf particles coated with a "protein corona," thereby continuously sustaining the production of pro-inflammatory signals. Since the intake of toxins and plastics is regular, the inflammatory response does not subside but transitions into a chronic phase, spreading throughout the body via the bloodstream.

[163] *Hirt, N. & Body-Malapel, M. Immunotoxicity and intestinal effects of nano- and microplastics: a review of the literature. Part Fibre Toxicol 17, 57 (2020). https://doi.org/10.1186/s12989-020-00387-7*

CLINICAL EVIDENCE FROM INFLAMMATORY BOWEL DISEASE

Studies of patients with inflammatory bowel disease (IBD) have revealed a direct correlation between the severity of the pathology and microplastic concentration. Empirical data demonstrate significantly elevated levels: in patients with IBD, the average concentration of microplastics in feces is 41.8 particles/g of dry matter, compared to 28.0 in healthy individuals.[164] Preclinical animal models confirm that inflamed intestinal mucosa becomes a "trap" for microplastics. The particles become lodged, hindering the healing process and contributing to prolonged intestinal epithelial dysfunction even after their external intake has ceased.[165]

DESTABILIZATION OF MICROBIOTA AS A KEY REGULATOR

A key component of communication within the gut-brain axis is the gut microbiota,[166] which consists of approximately 100 trillion microorganisms[167] that regulate metabolism, synthesize neuroactive metabolites, and help modulate neuroendocrine and immune processes.

Micro- and nanoplastics disrupt the fragile balance of this ecosystem, causing dysbiosis (the death of beneficial bacteria and the inducement of opportunistic flora growth).[168,169] The consequence of dysbiosis is a distortion of signals along the gut-brain axis (Fig. 6.4): changes in microbial metabolic activity disrupt the synthesis of neurotransmitter precursors (serotonin, GABA, dopamine) and distort information transmitted via the vagus nerve (nervus vagus), which serves as the primary communication channel between the gastrointestinal (GI) tract and the CNS (Fig. 6.5).

[164] *Yan, Z., Liu, Y., Zhang, T., et al. Analysis of Microplastics in Human Feces Reveals a Correlation between Fecal Microplastics and Inflammatory Bowel Disease Status. Environ. Sci. Technol. 56, 414–421 (2022). https://doi.org/10.1021/acs.est.1c03924*

[165] *Xie, S., Zhang, R., Li, Z., et al. Microplastics perturb colonic epithelial homeostasis associated with intestinal overproliferation, exacerbating the severity of colitis. Environmental Research 217, 114861 (2023). https://doi.org/10.1016/j.envres.2022.114861*

[166] *Thursby, E. & Juge, N. Introduction to the human gut microbiota. Biochem J 474, 1823–1836 (2017). https://doi.org/10.1042/BCJ20160510*

[167] *Ferranti, E., Dunbar, S. B., Dunlop, A. L., et al. 20 Things you Didn't Know About the Human gut Microbiome. J Cardiovasc Nurs 29, 479–481 (2014). https://doi.org/10.1097/JCN.0000000000000166*

[168] *Thin, Z. S., Chew, J., Ong, T. Y. Y., et al. Impact of microplastics on the human gut microbiome: a systematic review of microbial composition, diversity, and metabolic disruptions. BMC Gastroenterol 25, 583 (2025). https://doi.org/10.1186/s12876-025-04140-2*

[169] *Bora, S. S., Gogoi, R., Sharma, M. R., et al. Microplastics and human health: unveiling the gut microbiome disruption and chronic disease risks. Front. Cell. Infect. Microbiol. 14, 1492759 (2024). https://doi.org/10.3389/fcimb.2024.1492759*

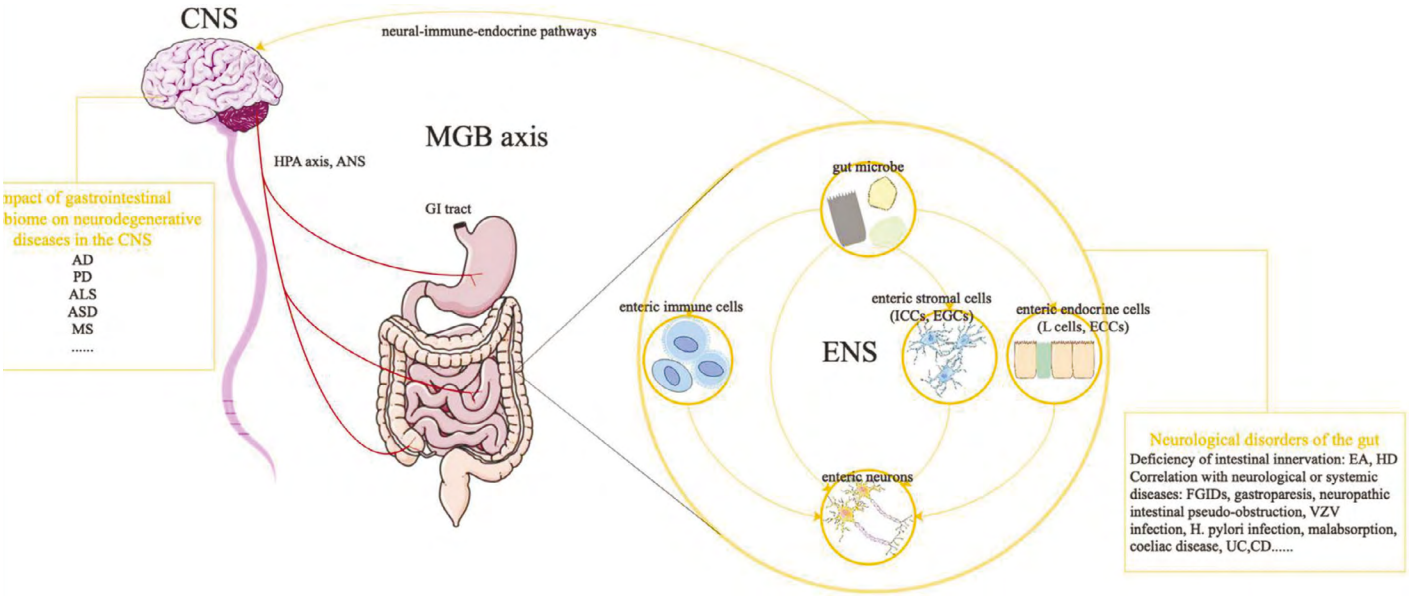


Figure 6.4.

Effects of GI microbiota on the enteric nervous system (ENS) and microbiota-gut-brain (MGB) axis. The autonomic nervous system (ANS), in combination with the hypothalamic-pituitary-adrenal (HPA) axis and neuroendocrine signaling, can induce CNS-modulated changes in the gut. Meanwhile, bacterial molecules and neural-immune-endocrine pathways impact the development of the CNS, giving rise to a dynamic communication network in the MGB axis.

Source: Geng, Z.-H., Zhu, Y., Li, Q.-L., et al. Enteric Nervous System: The Bridge Between the Gut Microbiota and Neurological Disorders. Front. Aging Neurosci. 14, 810483 (2022). https://doi.org/10.3389/fnagi.2022.810483

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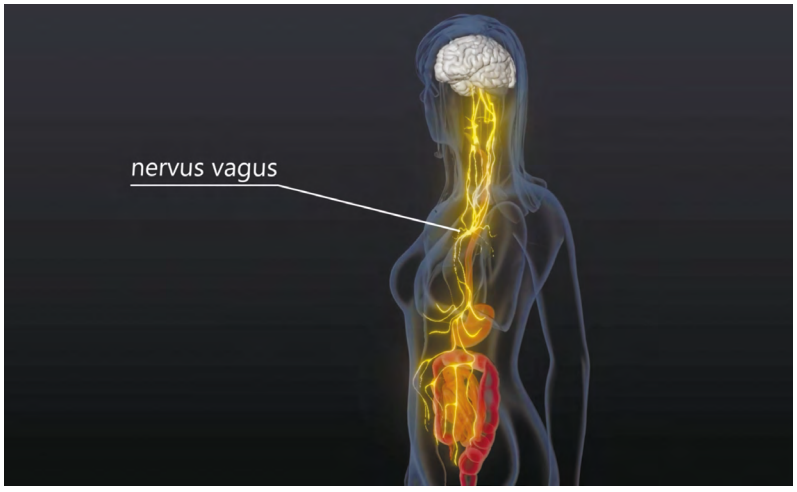


Figure 6.5.

Illustration of the nervus vagus (yellow), which serves as the primary communication channel between the gastrointestinal (GI) tract and the central nervous system (CNS).

SYSTEMIC EFFECTS OF DYSBIOSIS: IMPACT ON THE CENTRAL NERVOUS SYSTEM

Pathological processes (Fig. 6.6) are not confined to the digestive system but extend to the central nervous system (Fig. 6.7). Pro-inflammatory cytokines and bacterial metabolites, penetrating the compromised intestinal barrier into the bloodstream, can cross the blood-brain barrier. This creates prerequisites for neuroinflammation and neurodegeneration. Disrupted bidirectional communication between the microbiome and the brain is associated with the development of depression, anxiety disorders, as well as severe pathologies such as Alzheimer's and Parkinson's diseases.[170,171] Notably, children with autism spectrum disorder (ASD), frequently present with a combination of dysbiosis and gastrointestinal dysfunction, further corroborating the systemic nature of these processes.[172,173,174]

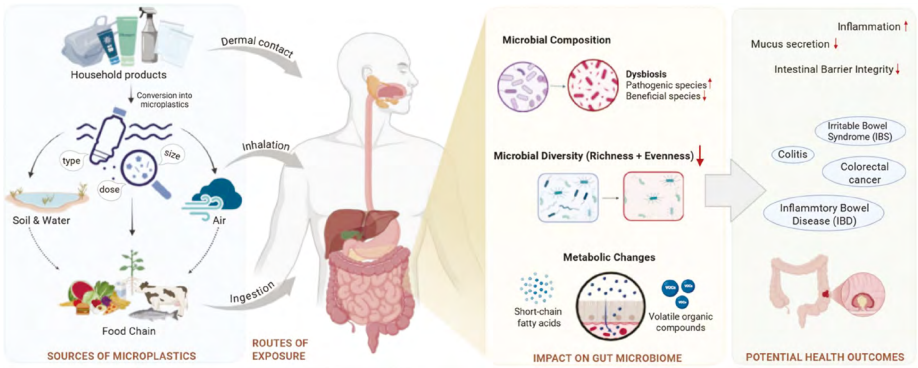


Figure 6.6.

The figure presents a graphical summary of changes in the microbial metabolic functions and signaling pathways caused by MPs exposure.

Source: Thin, Z. S., Chew, J., Ong, T. Y. Y., et al. Impact of microplastics on the human gut microbiome: a systematic review of microbial composition, diversity, and metabolic disruptions. BMC Gastroenterol 25, 583 (2025). https://doi.org/10.1186/s12876-025-04140-2

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[170] *Chen, Y., Xu, J. & Chen, Y. Regulation of Neurotransmitters by the Gut Microbiota and Effects on Cognition in Neurological Disorders. Nutrients 13, 2099 (2021). https://doi.org/10.3390/nu13062099*

[171] *Liang, X., Huang, G., Wang, Y., et al. Polystyrene Nanoplastics Hitch-Hike the Gut–Brain Axis to Exacerbate Parkinson's Pathology. ACS Nano 19, 5475–5492 (2025). https://doi.org/10.1021/acsnano.4c13914*

[172] *Kang, D.-W., Park, J. G., Ilhan, Z. E., et al. Reduced Incidence of Prevotella and Other Fermenters in Intestinal Microflora of Autistic Children. PLOS ONE 8, e68322 (2013). https://doi.org/10.1371/journal.pone.0068322*

[173] *Su, Q., Wong, O. W. H., Lu, W., et al. Multikingdom and functional gut microbiota markers for autism spectrum disorder. Nat Microbiol 9, 2344–2355 (2024). https://doi.org/10.1038/s41564-024-01739-1*

[174] *Yu, R., Hafeez, R., Ibrahim, M., et al. The complex interplay between autism spectrum disorder and gut microbiota in children: A comprehensive review. Behavioural Brain Research 473, 115177 (2024). https://doi.org/10.1016/j.bbr.2024.115177*

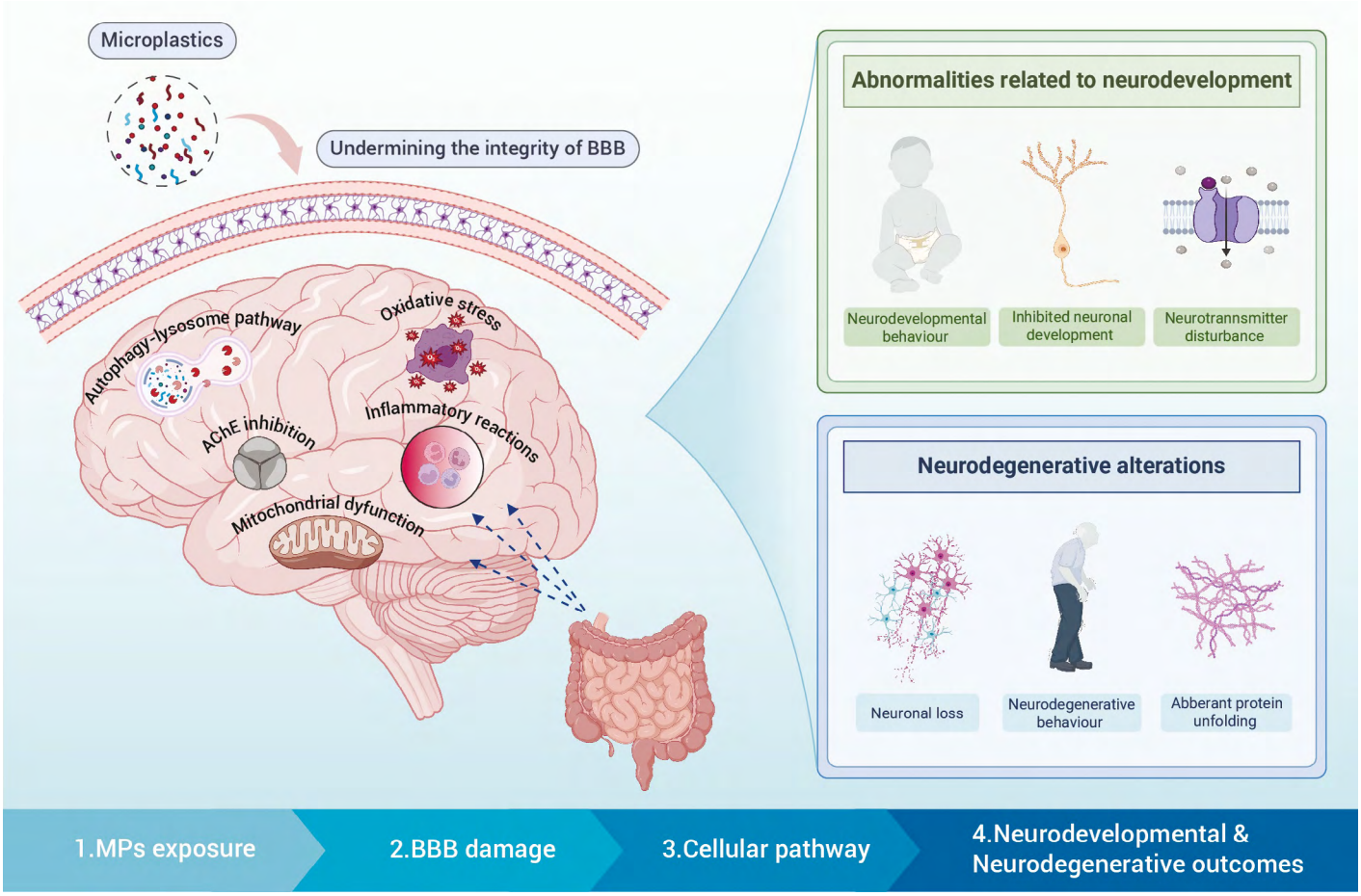


Figure 6.7.

Risks of neurological diseases induced by MPs and possible mechanisms.

Source: Farooq, U., Muhammad, Z., Yang, Q., et al. Microplastics and the gut-brain axis: Unraveling neurotoxic mechanisms and health implications. TIME 4, 100190 (2026). https://doi.org/10.59717/j.xinn-med.2026.100190

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Thus, the impact of micro- and nanoplastics forms a self-sustaining pathological mechanism — a "vicious cycle." Plastics destroy barriers and the microbiota, eventually leading to penetration of toxins into the blood and brain.[175] The resulting neuroinflammation and stress response, via neuroendocrine pathways, further compromise the protective functions of the intestine.[176,177]

Ultimately, nanoplastic-induced alterations in the gut may represent a systemic risk factor: rather than being limited to local gastrointestinal effects, they potentially contribute to processes associated with biological aging, metabolic disorders (e.g., obesity, diabetes), cardiovascular disease, and neuropsychiatric diseases. Nanoplastics may simultaneously affect the digestive, immune, nervous, and endocrine systems, thereby potentially compromising key physiological functions.

[175] *Farooq, U., Muhammad, Z., Yang, Q., et al. Microplastics and the gut-brain axis: Unraveling neurotoxic mechanisms and health implications. TIME 4, 100190 (2026). https://doi.org/10.59717/j.xinn-med.2026.100190*

[176] *Liang, B., Deng, Y., Huang, Y., et al. Fragile Guts Make Fragile Brains: Intestinal Epithelial Nrf2 Deficiency Exacerbates Neurotoxicity Induced by Polystyrene Nanoplastics. ACS Nano 18, 24044–24059 (2024). https://doi.org/10.1021/acsnano.4c03874*

[177] *Vanuytsel, T., Bercik, P. & Boeckxstaens, G. Understanding neuroimmune interactions in disorders of gut–brain interaction: from functional to immune-mediated disorders. Gut 72, 787–798 (2023). https://doi.org/10.1136/gutjnl-2020-320633*

PART 7.

MICRO- AND NANOPLASTICS AS A FACTOR IN **SYSTEMIC DESTABILIZATION OF IMMUNE DEFENSE** AND **AMPLIFICATION OF INFECTIOUS RISKS**

MNPs should currently not be viewed as an inert component of the environment, but as a physically-chemically active agent capable of directly interfering with the functioning of the immune system and altering the nature of the organism's interaction with infectious agents.

"Micro- and nanoplastics (MNPs) are emerging environmental immunotoxins with widespread human exposure through ingestion, inhalation, and dermal contact. Detected in the placenta, lungs, blood, bone marrow, and brain, MNPs accumulate in immune organs where they disrupt innate and adaptive cell functions."[178]

The immune system plays a key role in protecting the body against foreign materials by participating in their recognition, elimination, and the repair of damaged tissues.

Impairment of its functions due to MNP exposure potentially reduces the efficacy of these protective mechanisms. Accumulated data indicate the participation of MNPs in disrupting immune regulation, enhancing the pathogenic properties of microorganisms, and accelerating processes underlying the formation of antimicrobial resistance (AMR). Immunodeficiency caused by chronic MNP exposure may contribute to the development of a wide spectrum of diseases, increasing the risk of infections, inflammatory processes, and chronic pathologies.

IMMUNOTOXICITY OF MICRO- AND NANOPLASTICS

Experimental studies have shown that upon contact with micro- and nanoplastics, immune cells under *in vitro* conditions die more rapidly compared to control cells that were not exposed to plastic particles. Accelerated immune cell death under the influence of MNPs potentially reduces the efficacy of immune defense and weakens the organism's ability to withstand infectious agents.[179]

Beyond direct toxic effects on cells, MNPs can disrupt the functioning of the immune system, altering the ability of immune cells to perform their protective functions.[180,181,182]

[178] *Fan, J. & Ha, Y. Micro- and Nanoplastics and the Immune System: Mechanistic Insights and Future Directions. Immuno 5, 52 (2025). https://doi.org/10.3390/immuno5040052*

[179] *Park, K.-M., Kim, B., Woo, W., et al. Polystyrene microplastics induce activation and cell death of neutrophils through strong adherence and engulfment. Journal of Hazardous Materials 480, 136100 (2024). https://doi.org/10.1016/j.jhazmat.2024.136100*

[180] *Hirt, N. & Body-Malapel, M. Immunotoxicity and intestinal effects of nano- and microplastics: a review of the literature. Part Fibre Toxicol 17, 57 (2020). https://doi.org/10.1186/s12989-020-00387-7*

[181] *Giustarini, G., Skrabanja, T. L. P., Van Den Berg, A. E. T., et al. Polystyrene microplastics cross the murine intestine and induce inflammatory cell death after phagocytosis by human monocytes and neutrophils. iScience 28, 114076 (2025). https://doi.org/10.1016/j.isci.2025.114076*

[182] *Ahmadi, P., Doyle, D., Mojarad, N., et al. Effects of micro- and nanoplastic exposure on macrophages: a review of molecular and cellular mechanisms. Toxicology Mechanisms and Methods, 35, 823–846 (2025). https://doi.org/10.1080/15376516.2025.2500546*

Specifically, experiments have demonstrated that MNP exposure (Fig. 7.1):

- reduces the phagocytic activity of macrophages (diminishing their ability to engulf and destroy microbes);[180,181,182]
- impairs dendritic cell maturation (cells that initiate the adaptive immune response);
- induce robust formation of neutrophil extracellular traps;[178]
- alters the activation of T and B lymphocytes (the primary cells of adaptive immunity involved in targeted defense).

The immunotoxicity of MNPs is influenced by surface properties, including electrostatic charge, functional groups, oxidation state, and shape. These factors determine the extent to which particles interact with immune system cells and induce these functional changes.

Collectively, the indicated processes create prerequisites for an imbalance between innate and adaptive immunity and increase the risk of chronic and systemic inflammatory reactions affecting various organs and systems.

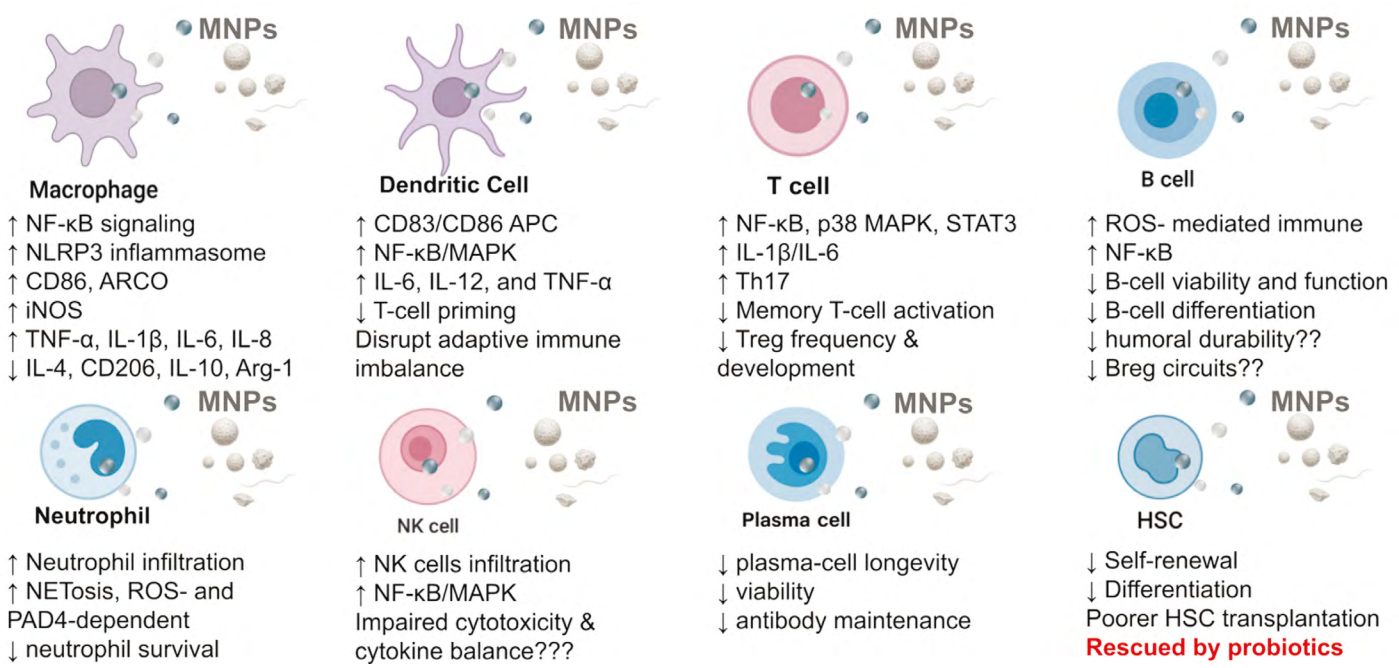


Figure 7.1.

Immunotoxic effects of MNPs on immune cells. MNPs impair both innate and adaptive immune functions, including macrophage and dendritic cell activation, neutrophil NETosis, and NK cell dysfunction. They also disrupt T and B cell balance, reduce plasma-cell viability, and impair hematopoietic stem cell renewal, collectively promoting chronic inflammation and immune dysregulation. ↑ indicates upregulation; ↓ indicates downregulation; ?? indicates that further validation is required; ??? indicates that the direction of change (upregulation or downregulation) remains uncertain and needs confirmation.

Source: Fan, J. & Ha, Y. Micro- and Nanoplastics and the Immune System: Mechanistic Insights and Future Directions. Immuno 5, 52 (2025). https://doi.org/10.3390/immuno5040052 License: CC BY 4.0.

MNPs AND DAMAGE TO LYMPHOID ORGANS

Nanoplastics are capable of penetrating the bone marrow[183] and thymus[184] — key organs ensuring the formation and maturation of blood cells and the immune system (Fig. 7.2).

The bone marrow performs two fundamental functions critically important for maintaining organismal homeostasis. First, it is the primary organ of hematopoiesis, ensuring the formation of erythrocytes, leukocytes, and platelets, which are necessary for oxygen transport, immune defense, and hemostasis (blood clotting). Second, bone marrow stem cells participate in the regeneration of various tissues, including bone, cartilage, muscle, and adipose tissue (Fig. 7.3).

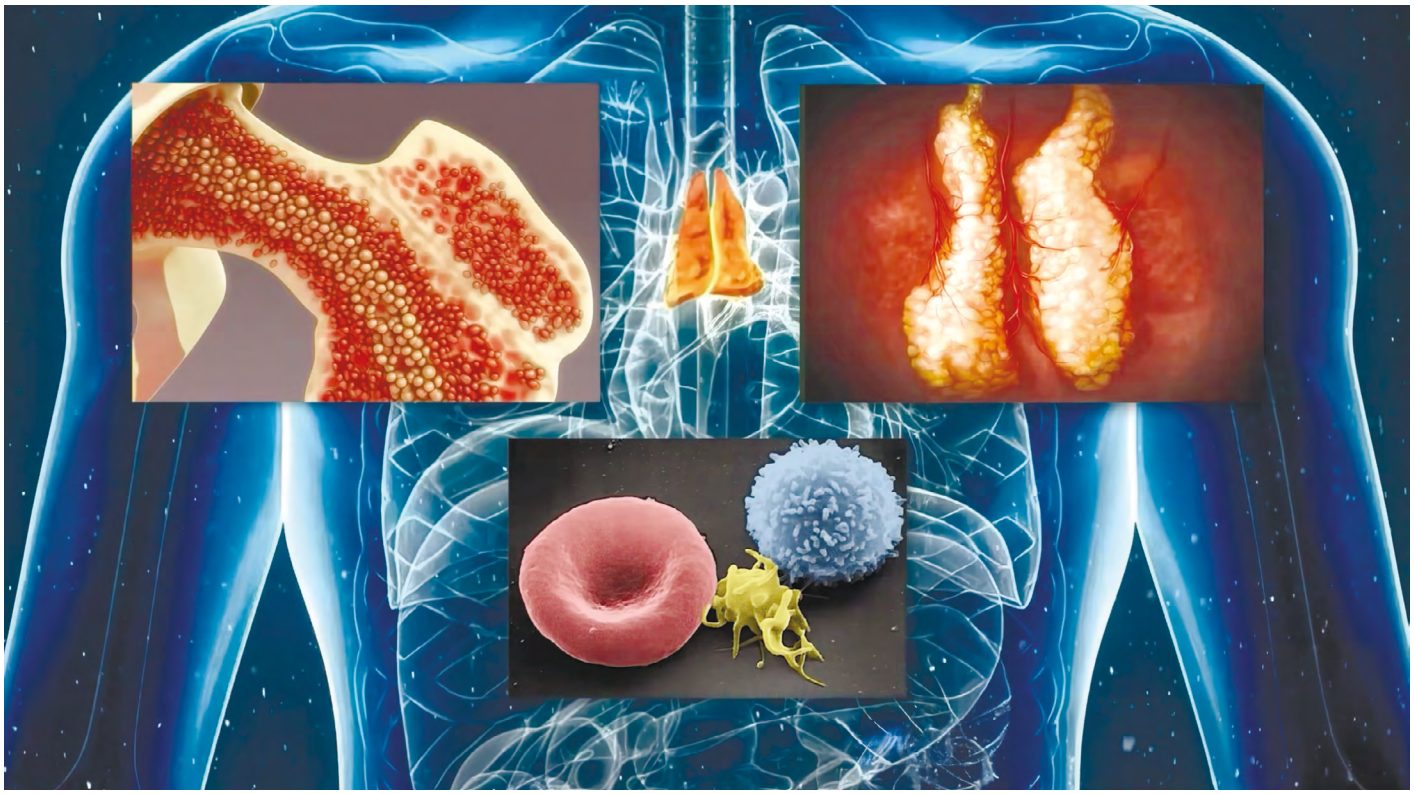


Figure 7.2.

Illustration of the bone marrow and thymus — organs where the formation and differentiation of blood and immune defense cells occur.

[183] *Guo, X., Wang, L., Wang, X., et al. Discovery and analysis of microplastics in human bone marrow. Journal of Hazardous Materials 477, 135266 (2024). https://doi.org/10.1016/j.jhazmat.2024.135266*

[184] *Li, J., Yin, K., Hou, L., et al. Polystyrene microplastics mediate inflammatory responses in the chicken thymus by Nrf2/NF-κB pathway and trigger autophagy and apoptosis. Environmental Toxicology and Pharmacology 100, 104136 (2023). https://doi.org/10.1016/j.etap.2023.104136*

Exposure of stem cells and hematopoietic bone marrow cells to nanoplastics may potentially lead to disruptions of both blood cell renewal processes and tissue repair mechanisms. Disruption of these functions has a number of clinically significant consequences: deterioration of hematopoiesis may lead to the development of anemia due to erythrocyte deficiency, reduced immune defense given a decrease in leukocyte count, and an increased risk of bleeding due to platelet shortage. Damage to bone tissue cells renders bones more fragile, increases the probability of fractures, and slows their recovery processes.

Thus, penetration of MNPs into key immune organs exerts a systemic impact on the organism, affecting several vital physiological systems and basic mechanisms of renewal and recovery simultaneously.[185]



Figure 7.3.

Illustration of the bone marrow (center) — a hematopoietic organ where erythrocytes, leukocytes, and platelets are produced (left). Concurrently, its stem cells participate in the regeneration of bone, cartilage, muscle, and adipose tissue (right).

[185] *Sun, R., Xu, K., Yu, L., et al. Preliminary study on impacts of polystyrene microplastics on the hematological system and gene expression in bone marrow cells of mice. Ecotoxicology and Environmental Safety 218, 112296 (2021). https://doi.org/10.1016/j.ecoenv.2021.112296*

ELECTROSTATIC PROPERTIES OF MNPs AND INCREASED PATHOGENICITY OF MICROORGANISMS

Micro- and nanoplastics exert a negative bidirectional impact: they not only affect the human immune system, but also facilitate the survival, proliferation, and increased resistance of pathogenic microorganisms due to their physicochemical properties.

According to experimental data, positively charged nanoplastic particles intensify physiological stress in gut bacteria, including Escherichia coli. Under conditions of such stress, microorganisms may exhibit signs of increased virulence — the ability to cause host cell damage or provoke infectious processes. The electrostatic charge of nanoplastic particles plays a key role in this, promoting the growth of bacterial pathogenicity toward human cells. Meanwhile, nanoplastic particles lacking a pronounced electrostatic charge do not induce significant physiological stress in microorganisms and do not increase their pathogenic potential (Fig. 7.4).[186]

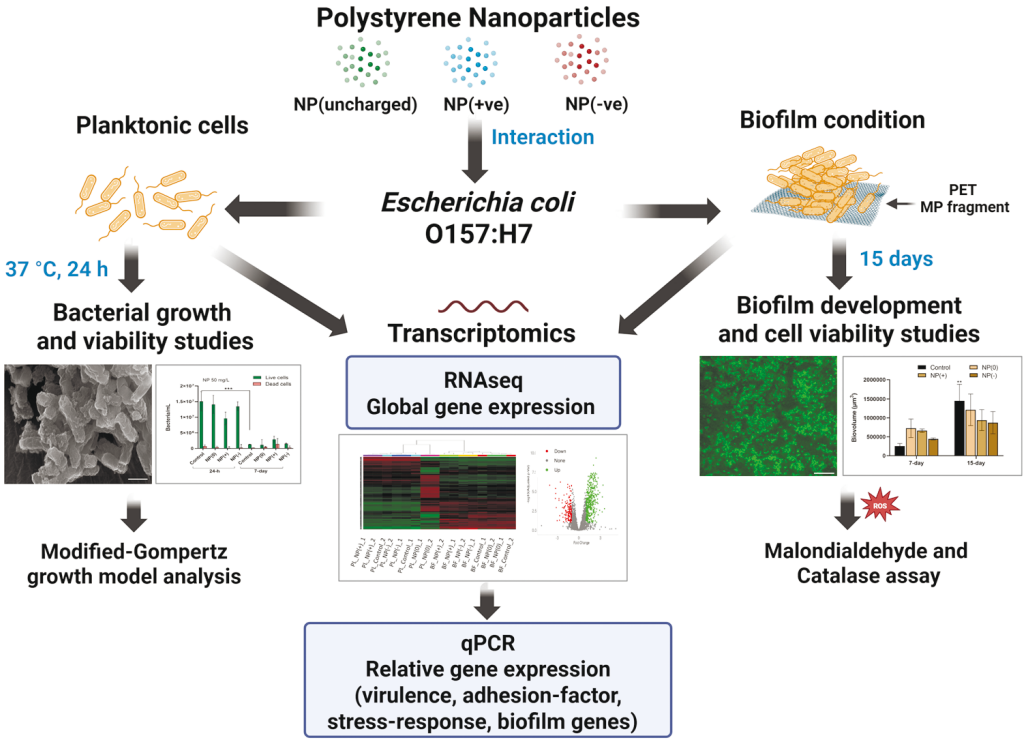


Figure 7.4.

PS-NPs with different surface charges affect the viability and pathogenicity of E. coli O157:H7 bacteria.

Source: Nath, J., Banerjee, G., De, J., et al. Nanoplastics-mediated physiologic and genomic responses in pathogenic Escherichia coli O157:H7. J Nanobiotechnol 23, 304 (2025). https://doi.org/10.1186/s12951-025-03369-z License: CC BY 4.0.

[186] Nath, J., Banerjee, G., De, J., et al. Nanoplastics-mediated physiologic and genomic responses in pathogenic Escherichia coli O157:H7. J Nanobiotechnol 23, 304 (2025). https://doi.org/10.1186/s12951-025-03369-z

THE ROLE OF MICROPLASTICS IN THE SPREAD OF VIRAL AND BACTERIAL PATHOGENS

The obtained experimental data point to the ability of microplastics, particularly polystyrene particles, to effectively adsorb viruses on their surface. As noted in research, electrostatic attraction is the primary virus adsorption mechanism on microplastics.[187]

Under laboratory conditions, it has been established that in freshwater, up to 98.6% of viral particles can be adsorbed on such particles.[187]

While many viruses are inactivated by ultraviolet radiation and oxidative factors within several hours, they may persist for 2 to 7 days when adsorbed onto plastic surfaces.[188,189,190] This creates conditions for virus accumulation on everyday items, including handrails, bottles, and door handles, potentially increasing the risk of viral infection transmission.

Aerosol transport represents an additional factor in infection spread. A droplet expelled during coughing or sneezing by an infected person can contain up to 200 million viral particles into the environment,[191] which are capable of settling on plastic surfaces or binding with micro- and nanoplastic particles present in the air and water. Thus, microplastics effectively fulfill the function of a vector, facilitating the dissemination of infectious agents in air and aquatic environments.

PROTEIN CORONA AND IMMUNE RECOGNITION OF MNPs

After entering the bloodstream, electrostatically charged particles of micro- and nanoplastics are rapidly coated with plasma proteins, forming a so-called biomolecular corona (Fig. 7.5).[192] The resulting protein shell can change the biological identity of particles and reduce their recognition by the immune system.[193]

[187] *Lu, J., Yu, Z., Ngiam, L., et al. Microplastics as potential carriers of viruses could prolong virus survival and infectivity. Water Research 225, 119115 (2022). https://doi.org/10.1016/j.watres.2022.119115*
[188] *Nauheim, R. C., Romanowski, E. G., Araullo-Cruz, T., et al. Prolonged Recoverability of Desiccated Adenovirus Type 19 from Various Surfaces. Ophthalmology 97, 1450–1453 (1990). https://doi.org/10.1016/S0161-6420(90)32389-8*
[189] *Corpet, D. E. Why does SARS-CoV-2 survive longer on plastic than on paper? Medical Hypotheses 146, 110429 (2021). https://doi.org/10.1016/j.mehy.2020.110429*
[190] *Van Doremalen, N., Bushmaker, T., Morris, D. H., et al. Aerosol and Surface Stability of SARS-CoV-2 as Compared with SARS-CoV-1. N Engl J Med 382, 1564–1567 (2020). https://doi.org/10.1056/NEJMc2004973*
[191] *Pawłowski, P. H. & Zielenkiewicz, P. The role of electric charge in SARS-CoV-2 and other viral infections. AIMS Biophysics 11, 166–188 (2024). https://doi.org/10.3934/biophy.2024011*
[192] *Sadiq, A., Litti, L., Bogialli, S., et al. The influence of nanoplastics' surface charge on the formation of protein corona and the subsequent sorption of Cd2+ and Pb2+ ions. Colloids and Surfaces C: Environmental Aspects 4, 100088 (2026). https://doi.org/10.1016/j.colsuc.2025.100088*
[193] *Kopatz, V., Wen, K., Kovács, T., et al. Micro- and Nanoplastics Breach the Blood–Brain Barrier (BBB): Biomolecular Corona's Role Revealed. Nanomaterials 13, 1404 (2023). https://doi.org/10.3390/nano13081404*

"The features of NPs such as the size, surface chemistry, charge are the critical factors influencing the behavior of protein corona. Molecular properties and protein corona composition affect the cellular uptake of NPs."[194]

This mechanism potentially contributes to a longer presence of MNPs in the body and enhances their effects on immune and infectious processes.

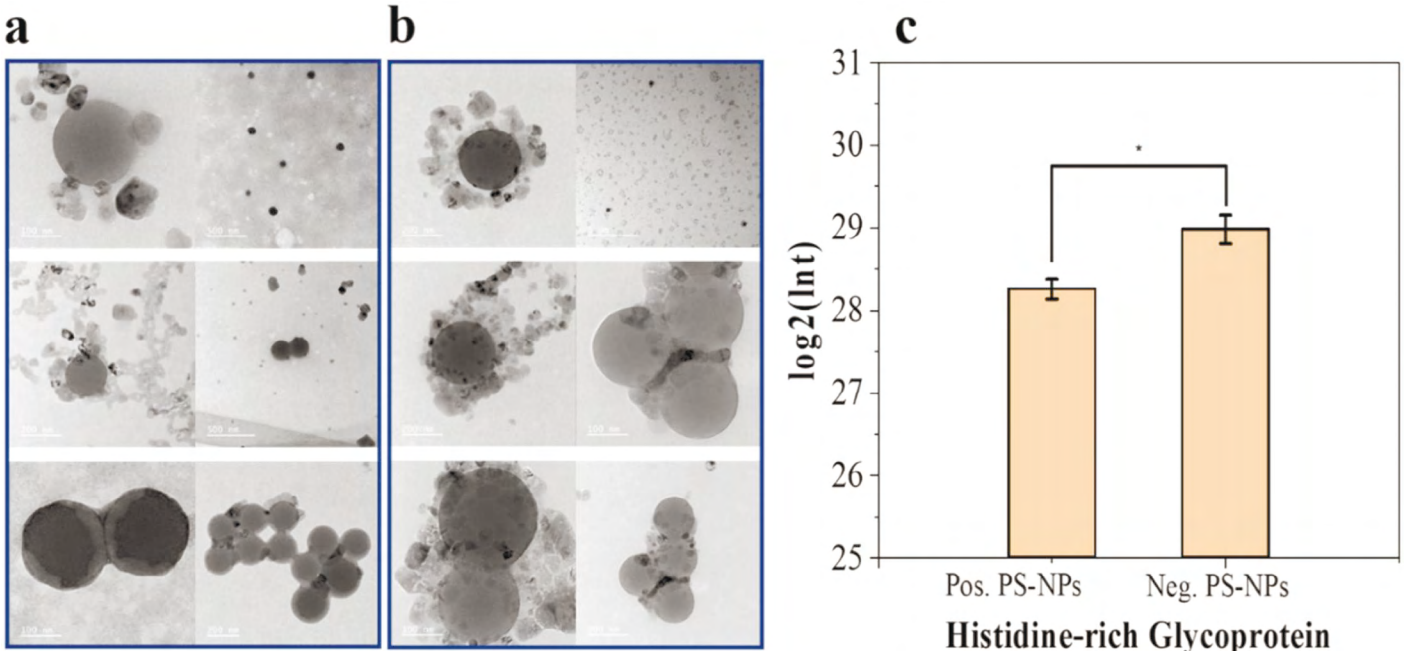


Figure 7.5.

Formation and characterization of the NPs-biological corona complex.

Transmission electron microscopy images of biologically covered positive (a) and negatively (b) charged PS-NPs. Comparative adsorption profile of histidine-rich-glycoprotein adsorption on charged PS-NPs (c).

Source: Sadiq, A., Litti, L., Bogialli, S., et al. The influence of nanoplastics' surface charge on the formation of protein corona and the subsequent sorption of Cd^{2+} and Pb^{2+} ions. Colloids and Surfaces C: Environmental Aspects 4, 100088 (2026). https://doi.org/10.1016/j.colsuc.2025.100088 License: CC BY 4.0.

[194] *Kopac, T. Protein corona, understanding the nanoparticle–protein interactions and future perspectives: A critical review. International Journal of Biological Macromolecules 169, 290–301 (2021). https://doi.org/10.1016/j.ijbiomac.2020.12.108*

MICROPLASTICS AS A FACTOR OF INCREASING ANTIBIOTIC RESISTANCE

Antimicrobial resistance (AMR) has acquired the scale of a global public health threat in recent years. According to 2019 data, resistance to antimicrobial drugs was the direct cause of death for more than 1.27 million people. In total, antimicrobial resistance was associated with approximately 5 million lethal outcomes, i.e., deaths where resistance was present and worsened the outcome, but may not have been the sole cause of death.[195]

Recent studies indicate that environmental pollution with MNPs is one of the factors contributing to the development of bacterial resistance, exacerbating the AMR crisis.[196]

Plastic surfaces create a favorable environment for bacterial proliferation. On these surfaces, bacteria form biofilms — dense multilayered communities encased in a sticky protective layer (Extracellular Polymeric Substances, EPS), which holds cells together and protects them from external influences.

Biofilms provide microorganisms with:

- protection against desiccation and ultraviolet radiation;
- protection against host immune response factors upon entry into the organism;
- limitation of antibiotic penetration to bacterial cells.

Thus, bacteria in biofilm form are characterized by enhanced survival and reduced sensitivity to antimicrobial therapy.

Bacteria are capable of persisting on plastic surfaces longer than on most other materials, sometimes for up to three days or more.[196] This increases the probability of their transmission and spread.

Collectively, these processes allow for plastics of all sizes, from macro- to micro- and nano-fractions, to be regarded as a significant substrate facilitating the transport and dissemination of potential pathogens in aquatic ecosystems, soils, and food chains.

[195] *World Health Organization. Antimicrobial resistance. (2023) https://www.who.int/news-room/fact-sheets/detail/antimicrobial-resistance (Accessed 20 March 2026).*

[196] *Gross, N., Muhvich, J., Ching, C., et al. Effects of microplastic concentration, composition, and size on Escherichia coli biofilm-associated antimicrobial resistance. Appl Environ Microbiol 91, e02282-24 (2025). https://doi.org/10.1128/aem.02282-24*

SORPTION PROPERTIES OF MICROPLASTICS AND ACCUMULATION OF POLLUTANTS

It has been discovered that on “weathered” (environmentally aged) plastic surfaces, toxic chemicals and pathogens can accumulate at least 10 times faster than previously thought.[197] Laboratory studies indicate that microplastic particles are capable of sorbing metal ions[198] and organic pollutants[199,200] through a combination of physical and chemical processes, ensuring their retention on the particle surface (Fig. 7.6).

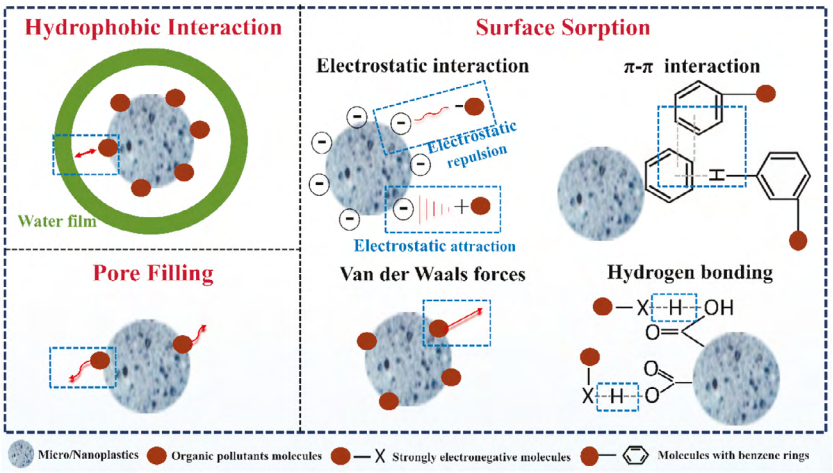


Figure 7.6.

Schematic representation of the primary interaction between MNPs and organic pollutants (OPs).

Source: Xie, Y., Lyu, Y., Irshad, S., et al. Combined interactions and ecotoxicological effects of micro/nanoplastics and organic pollutants in soil–plant systems: a critical overview. Environ. Sci.: Adv. 4, 1166–1180 (2025). https://doi.org/10.1039/D5VA00072F

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The formation of bacterial biofilms on microplastics additionally alters particle surface properties, enhancing their ability to bind metals and leading to the accumulation of higher concentrations compared to MP particles without biofilms.[201]

The nature and intensity of interactions between microplastics and pollutants depend on the polymer material type and particle size. These parameters determine the surface properties of microplastics, including electrostatic charge that influences the ability of particles to bind additional environmental components.

EXTERNAL ELECTRON TRANSFER AS A MECHANISM OF BACTERIAL METABOLIC ACTIVATION

Under laboratory conditions, it has been shown that bacterial contact with microplastics activates specific metabolic processes associated with electron transfer.[202] In this scenario, microplastic particles can serve as electron donors, providing bacteria with an additional energy resource (Fig. 7.7).[196]

[197] *Maddison, C., Sathish, C. I., Lakshmi, D., et al. An advanced analytical approach to assess the long-term degradation of microplastics in the marine environment. npj Mater Degrad 7, 59 (2023). https://doi.org/10.1038/s41529-023-00377-y*

[198] *Richard, H., Carpenter, E. J., Komada, T., et al. Biofilm facilitates metal accumulation onto microplastics in estuarine waters. Science of The Total Environment 683, 600–608 (2019). https://doi.org/10.1016/j.scitotenv.2019.04.331*

[199] *Bhagat, K., Doussiemo, D. R. B., Mushro, N., et al. Effect of Biofouling on the Sorption of Organic Contaminants by Microplastics. Environmental Toxicology and Chemistry 43, 1973–1981 (2024). https://doi.org/10.1002/etc.5938*

[200] *Xie, Y., Lyu, Y., Irshad, S., et al. Combined interactions and ecotoxicological effects of micro/nanoplastics and organic pollutants in soil–plant systems: a critical overview. Environ. Sci.: Adv. 4, 1166–1180 (2025). https://doi.org/10.1039/D5VA00072F*

[201] *Johansen, M. P., Cresswell, T., Davis, J., et al. Biofilm-enhanced adsorption of strong and weak cations onto different microplastic sample types: Use of spectroscopy, microscopy and radiotracer methods. Water Research 158, 392–400 (2019). https://doi.org/10.1016/j.watres.2019.04.029*

[202] *Yang, F.-A., Cao, C., Guo, H., et al. Decoding Microplastic-Induced Adaptive Strategies in Electroactive Biofilms: Stress Resistance Pathways and Enhanced Extracellular Electron Transfer. ACS EST Eng. 5, 2680–2691 (2025). https://doi.org/10.1021/acsestengg.5c00471*

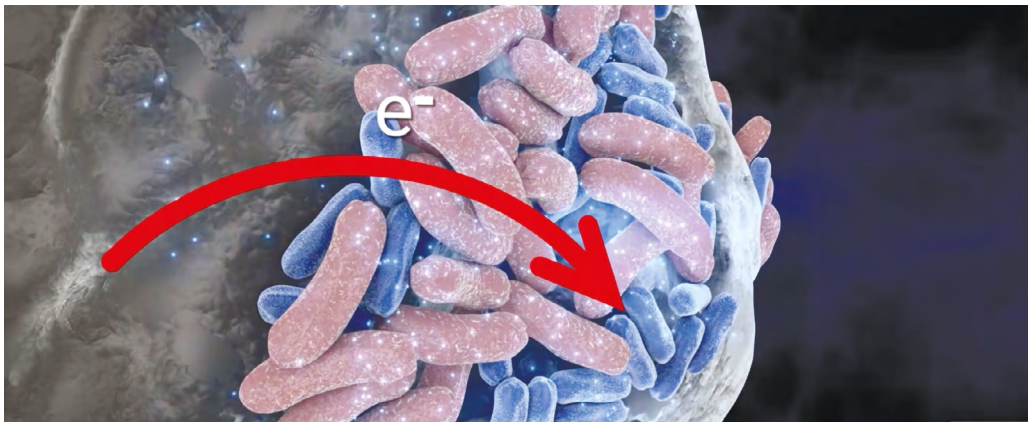


Figure 7.7.

Illustration of electron transfer from a plastic particle to bacteria.

This mechanism is known as extracellular electron transfer (EET). Its activation can enhance energy metabolism, which increases their viability, promotes more intensive division and mutational activity, and facilitates the formation of particularly dense multilayered biofilms. Such biofilms possess high resistance and significantly limit antibiotic penetration to bacterial cells. Furthermore, the additional energy supply obtained by bacteria via extracellular electron transfer may directly contribute to the development and fixation of resistance to antimicrobial drugs.

It is particularly important that the acquired properties persist in bacteria even after the removal of microplastics from the experimental environment. Upon cessation of contact, microorganisms continue to demonstrate increased resistance,[196] high mutational activity, and the ability to form robust biofilms on other surface types.

Overall, current evidence suggests that micro- and nanoplastics can promote increased antibiotic resistance across multiple classes of antibiotics.[196]

These facts confirm the presence of prolonged changes in bacterial populations under the influence of micro- and nanoplastics. Findings from studies testify that plastic pollution may accelerate the formation and spread of so-called “superbugs” — microorganisms resistant to the action of medicinal drugs.[203] This process acquires special significance in the context of the global antimicrobial resistance threat.

According to World Health Organization estimates, by 2050, antibiotic-resistant infections could lead to the death of up to 10 million people annually.[204] Concurrently, the rates of new antimicrobial drug development significantly lag behind the speed of evolution and adaptation of bacterial populations, which amplifies the scale and complexity of this problem.

If current trends persist, there is a risk of further reduction in the clinical value of antibiotics, which may substantially limit the capabilities of modern medicine in combating infectious diseases.

[203] *Dick, L., Batista, P. R., Zaby, P., et al. The adsorption of drugs on nanoplastics has severe biological impact. Sci Rep 14, 25853 (2024). https://doi.org/10.1038/s41598-024-75785-4*

[204] *World Health Organization. New report calls for urgent action to avert antimicrobial resistance crisis. (2019) https://www.who.int/news/item/29-04-2019-new-report-calls-for-urgent-action-to-avert-antimicrobial-resistance-crisis (Accessed 20 March 2026).*

PART 8.

INHALATION EXPOSURE TO MICRO- AND NANOPLASTICS: RISKS TO THE RESPIRATORY SYSTEM

Despite the advanced achievements of modern medicine, lower respiratory tract infections remain the deadliest infectious pathology in the world, consistently ranking among the top five leading causes of death (Fig. 8.1).[205] Simultaneously, cancer of the trachea, bronchi, and lungs ranks sixth in global mortality. This points to the presence of an unaccounted yet potentially key etiological factor. Findings from recent studies suggest that chronic inhalation of MNPs may act as a systemic trigger for structural, biochemical, and microbiological disruptions in the respiratory system.[206,207] Such exposure creates conditions for prolonged inflammation, the development of fibrosis, and an increased risk of oncological processes.

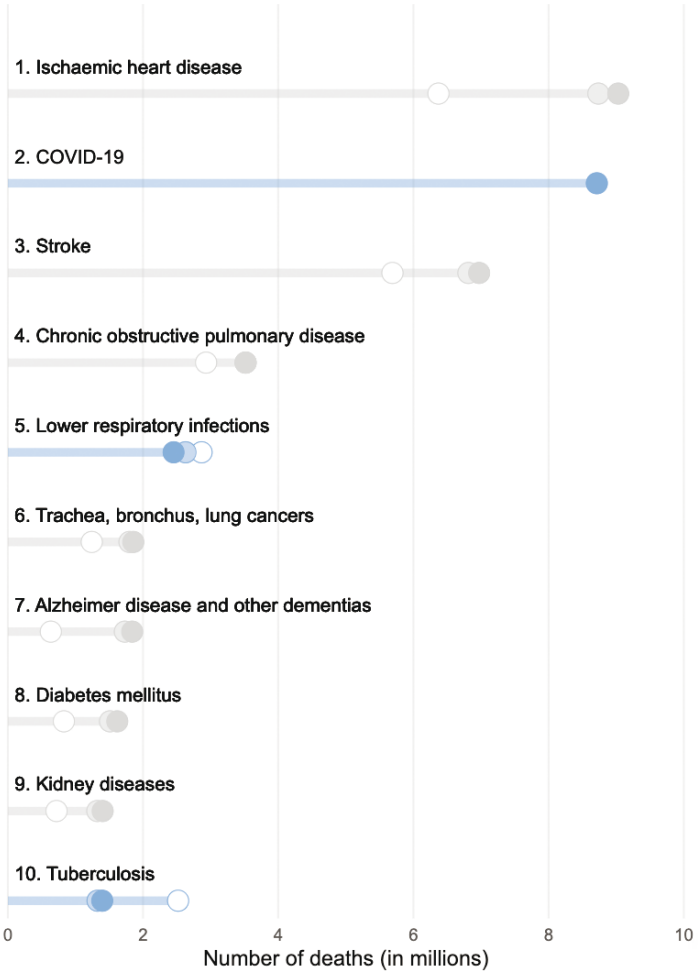


Figure 8.1.

Leading causes of death in 2021 globally.

Source: World Health Organization (WHO). The Top 10 Causes of Death. (2024) https://www.who.int/news-room/fact-sheets/detail/the-top-10-causes-of-death (Accessed 20 March 2026)

License: CC BY-NC-SA 3.0 IGO.

[205] *World Health Organization. The Top 10 Causes of Death. (2024) https://www.who.int/news-room/fact-sheets/detail/the-top-10-causes-of-death (Accessed 20 March 2026).*

[206] *Epeslidou, E., Scott, J. S., de Klein, B., et al. Microplastics as environmental modifiers of lung disease. EMBO Mol Med 18, 381–395 (2026). https://doi.org/10.1038/s44321-025-00353-w*

[207] *Chen, Q., Gao, J., Yu, H., et al. An emerging role of microplastics in the etiology of lung ground glass nodules. Environ Sci Eur 34, 25 (2022). https://doi.org/10.1186/s12302-022-00605-3*

SCALE OF IMPACT AND PHYSICAL PENETRATION CAPABILITY

The health risk posed by the widespread presence of plastic microparticles in the air should not be underestimated, as the maximum annual exposure dose outdoors can reach 1 million units. At the same time, the concentration of microparticles in indoor air is often significantly higher than in open spaces.[208]

It is worth noting that exposure through inhalation poses a particular risk to children. Given their lower body weight and higher respiratory rate, the concentration of inhaled plastic per kilogram of body weight in children is approximately 3-4 times higher than in adults. Thus, the physiological characteristics of a child's body determine their particular vulnerability to this factor.

Settling deep within the human lung tissue, these particles initiate a cascade of pathological reactions. Nanoparticles smaller than 0.1 µm pose the highest threat.[206] Unlike larger microplastics that settle primarily in the upper respiratory tract, nanoplastics penetrate unimpeded into all sections of the respiratory tract, reaching the alveoli.[209,210,211,212] Given the enormous alveolar surface area of the human lungs (50-140 m^2),[213] it's a significant area for nanoplastic accumulation.

ACCUMULATION IN TISSUES AND THE MECHANISM OF CELL DAMAGE

Analysis of human lung tissue samples demonstrates a widespread distribution of plastic particles. Microparticles ranging in size from 4 µm to over 2 mm were detected in all lung tissue samples (Fig. 8.2).[214]

Micro-IR spectroscopy data quantitatively confirm their distribution: in the upper region (0.80 ± 0.96 MP/g), middle/lingular (0.41 ± 0.37 MP/g), and with significantly higher levels detected in the lower (3.12 ± 1.30 MP/g) region.

[208] *Liao, Z., Ji, X., Ma, Y., et al. Airborne microplastics in indoor and outdoor environments of a coastal city in Eastern China. Journal of Hazardous Materials 417, 126007 (2021). https://doi.org/10.1016/j.jhazmat.2021.126007*

[209] *Kahan, M. S., Bleier, B. S., Amiji, M. M., et al. The Effects of Microplastics and Nanoplastics in the Nasal Airway and Upper Respiratory Tract. Sinusitis 10, 1 (2025). https://doi.org/10.3390/sinusitis10010001*

[210] *Gou, Z., Wu, H., Li, S., et al. Airborne micro- and nanoplastics: emerging causes of respiratory diseases. Particle and Fibre Toxicology 21, 50 (2024). https://doi.org/10.1186/s12989-024-00613-6*

[211] *Paplińska-Goryca, M., Misiukiewicz-Stępień, P., Wróbel, M., et al. The impaired response of nasal epithelial cells to microplastic stimulation in asthma and COPD. Sci Rep 15, 4242 (2025). https://doi.org/10.1038/s41598-025-87242-x*

[212] *Tokito, T., Kido, T., Nagafuchi, O., et al. Microplastics in Human Bronchoalveolar Lavage Fluid. Respirology 30, 1141–1152 (2025) . https://doi.org/10.1111/resp.70107*

[213] *Ananda Rao, A. & Johncy, S. Tennis Courts in the Human Body: A Review of the Misleading Metaphor in Medical Literature. Cureus (2022). https://doi.org/10.7759/cureus.21474*

[214] *Jenner, L. C., Rotchell, J. M., Bennett, R. T., et al. Detection of microplastics in human lung tissue using µFTIR spectroscopy. Science of The Total Environment 831, 154907 (2022). https://doi.org/10.1016/j.scitotenv.2022.154907*

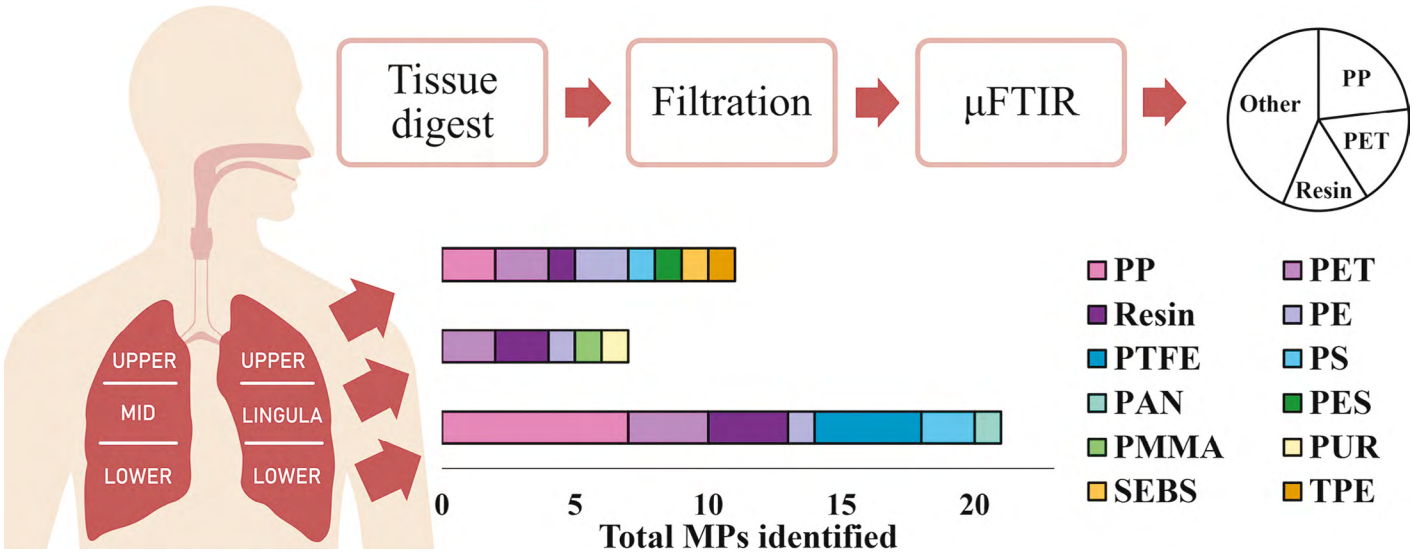


Figure 8.2.

Graphical abstract of research: Detection of microplastics in human lung tissue.

Source: Jenner, L. C., Rotchell, J. M., Bennett, R. T., et al. Detection of microplastics in human lung tissue using μFTIR spectroscopy. Science of The Total Environment 831, 154907 (2022). https://doi.org/10.1016/j.scitotenv.2022.154907

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In a study carried out using laser infrared spectroscopy, microplastics were identified in all tested sputum samples from patients with respiratory tract diseases (Fig. 8.3, 8.4).[214,215] The number of detected particles in the sputum ranged from 18 to 92 particles per 10 ml, testifying that microplastic inhalation is a common occurrence among patients with respiratory diseases.

Morphological characteristics of the particles are of significant importance when assessing the biological impact of microplastics on the respiratory tract. Fibrous and angular particles not only penetrate small bronchi and alveoli more easily, but are also extremely difficult to remove through mucociliary clearance (the self-cleaning mechanism of the airways).[216] Macrophages ("cleaning" cells) poorly handle long fibers. As a result, these particles can persist in lung tissue for a long time, triggering a cascade of cellular damage, sustaining chronic inflammation, and creating conditions for fibrous scarring as well as oncogenic changes in bronchial and lung cells.

[215] *Huang, S., Huang, X., Bi, R., et al. Detection and Analysis of Microplastics in Human Sputum. Environ. Sci. Technol. 56, 2476–2486 (2022). https://doi.org/10.1021/acs.est.1c03859*

[216] *Amato-Lourenço, L. F., Carvalho-Oliveira, R., Júnior, G. R., et al. Presence of airborne microplastics in human lung tissue. Journal of Hazardous Materials 416, 126124 (2021). https://doi.org/10.1016/j.jhazmat.2021.126124*

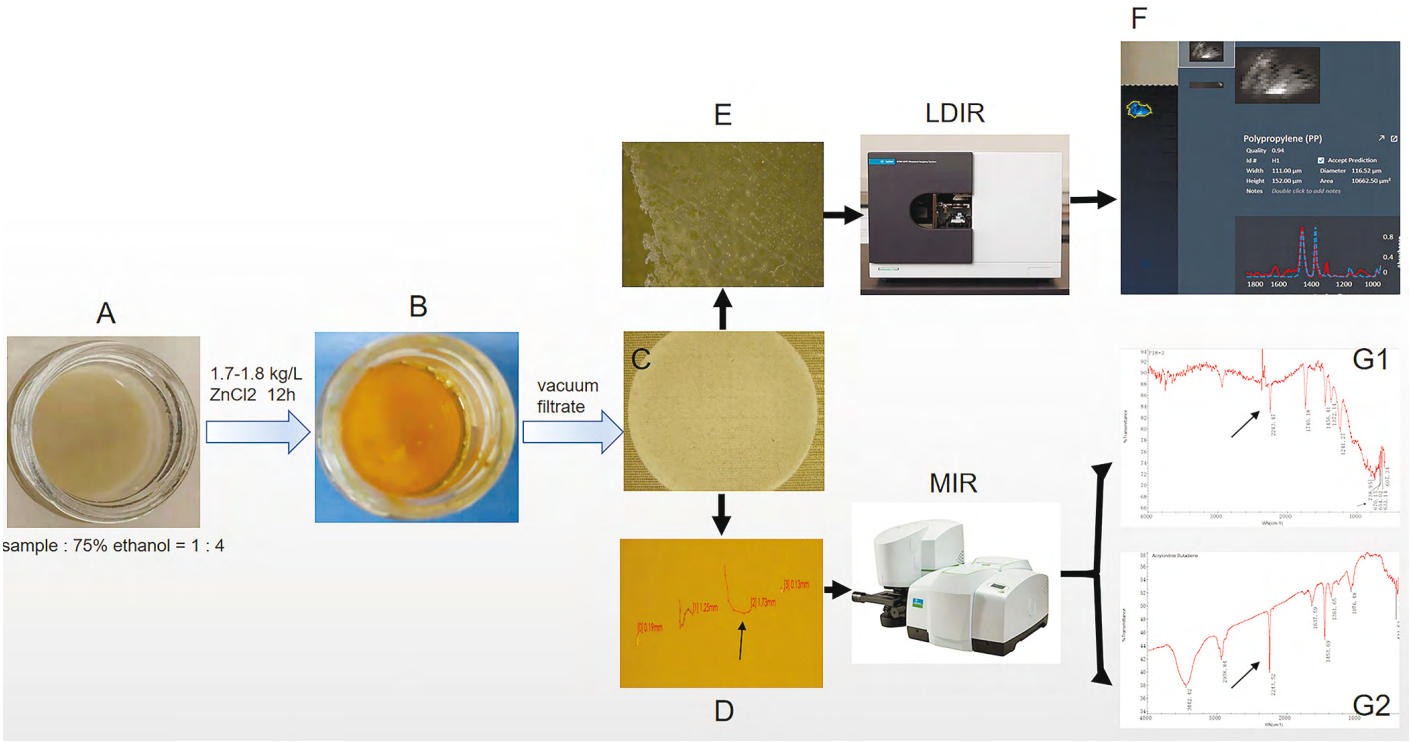


Figure 8.3.

Experimental procedure of sputum treatment and detection of microplastics.

Source: Huang, S., Huang, X., Bi, R., et al. Detection and Analysis of Microplastics in Human Sputum. Environ. Sci. Technol. 56, 2476–2486 (2022). https://doi.org/10.1021/acs.est.1c03859 License: CC BY-NC-ND 4.0.

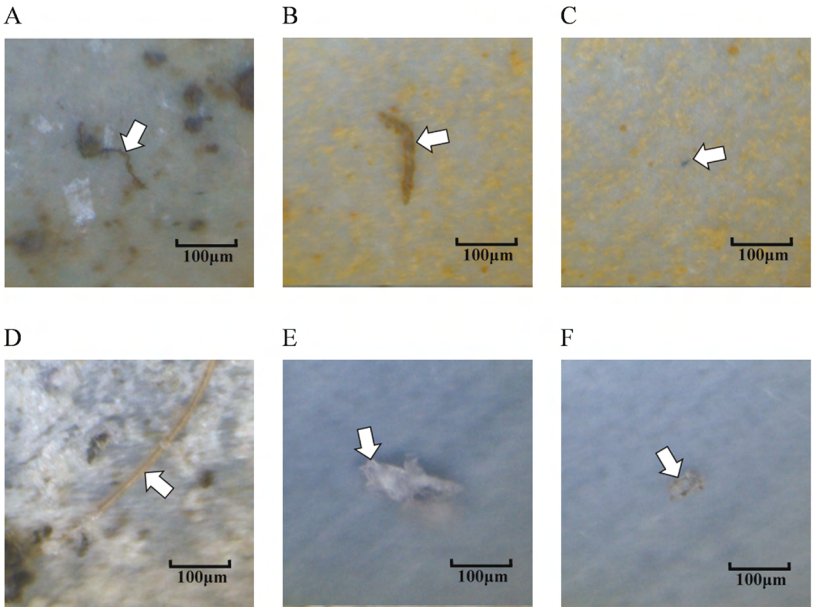


Figure 8.4.

mages of MPs identified from human lung tissue samples. A, B, C and D = (A = PET, polyethylene terephthalate,) (B = polyurethane, PUR) (C = Resin) (D = polyacrylonitrile, PAN). E and F = MPs identified within blanks. (E = polystyrene, PS) (F = polypropylene, PP).

Source: Jenner, L. C., Rotchell, J. M., Bennett, R. T., et al. Detection of microplastics in human lung tissue using μFTIR spectroscopy. Science of The Total Environment 831, 154907 (2022). https://doi.org/10.1016/j.scitotenv.2022.154907

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CELLULAR AND INTRACELLULAR TOXICITY

At the tissue level, MNP particles have been shown to damage the epithelium,[217] potentially disrupting the barrier function of mucous membranes.

"Microplastics are able to internalize into epithelial cells, and the rate of internalization is influenced by physicochemical properties such as particle size and surface charge. Microplastics with small particle size are internalized more rapidly than larger particles. The internalization and intracellular accumulation of microplastics with positive surface charge is greater than that of microplastics with negative surface charge." [218]

This damage also affects the ciliated epithelium (Fig. 8.5).[218] Suppression of ciliary activity blocks mucus clearance, provoking the obstruction of small bronchi and significantly increasing the risk of secondary infections.

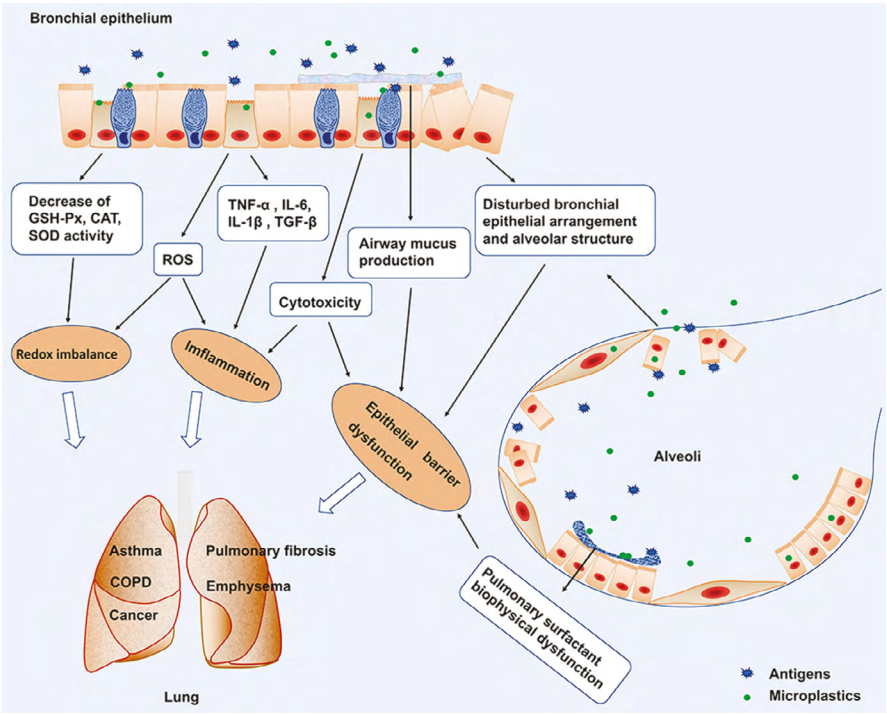


Figure 8.5.

Potential effects and possible mechanisms of microplastics on respiratory. Microplastic exposure interferes with the biophysical function of pulmonary surfactants and disrupts alveolar structure and airway barrier function. Exposure of microplastics to lung epithelial cells inhibits epithelial cell proliferation and induces epithelial cell apoptosis.

Source: Lu, K., Zhan, D., Fang, Y., et al. Microplastics, potential threat to patients with lung diseases. Front. Toxicol. 4, 958414 (2022). https://doi.org/10.3389/ftox.2022.958414

License: CC BY 4.0.

[217] *Milillo, C., Aruffo, E., Di Carlo, P., et al. Polystyrene nanoplastics mediate oxidative stress, senescence, and apoptosis in a human alveolar epithelial cell line. Front. Public Health 12, 1385387 (2024). https://doi.org/10.3389/fpubh.2024.1385387*

[218] *Lu, K., Zhan, D., Fang, Y., et al. Microplastics, potential threat to patients with lung diseases. Front. Toxicol. 4, 958414 (2022). https://doi.org/10.3389/ftox.2022.958414*

The surface electrostatic charge and small sizes of micro- and nanoplastics enhance their interaction with cell membranes. The electrostatic attraction between the charged surface of MNP particles and the phospholipid layer of the membrane facilitates their adsorption and endocytosis (cellular uptake). After penetrating a cell, the particles can accumulate in the cytoplasm and interact with intracellular organelles.[219]

At the intracellular level, as already discussed in this report, mitochondria become one of the primary targets of nanoplastics.[220] Damage to them (Fig. 8.6) leads to the disruption of cellular respiration processes, causing an acute cellular energy deficit and increasing oxidative stress (generation of reactive oxygen species, ROS).[221] Since micro- and nanoplastics enter the respiratory system continuously with every breath, the processes of damage to cells and mitochondria become continuous. As a result, chronic inflammation[222] develops and persists, which contributes to further disruption of the integrity of the respiratory tract's epithelial barriers and creating conditions for easier penetration by viruses and bacteria.

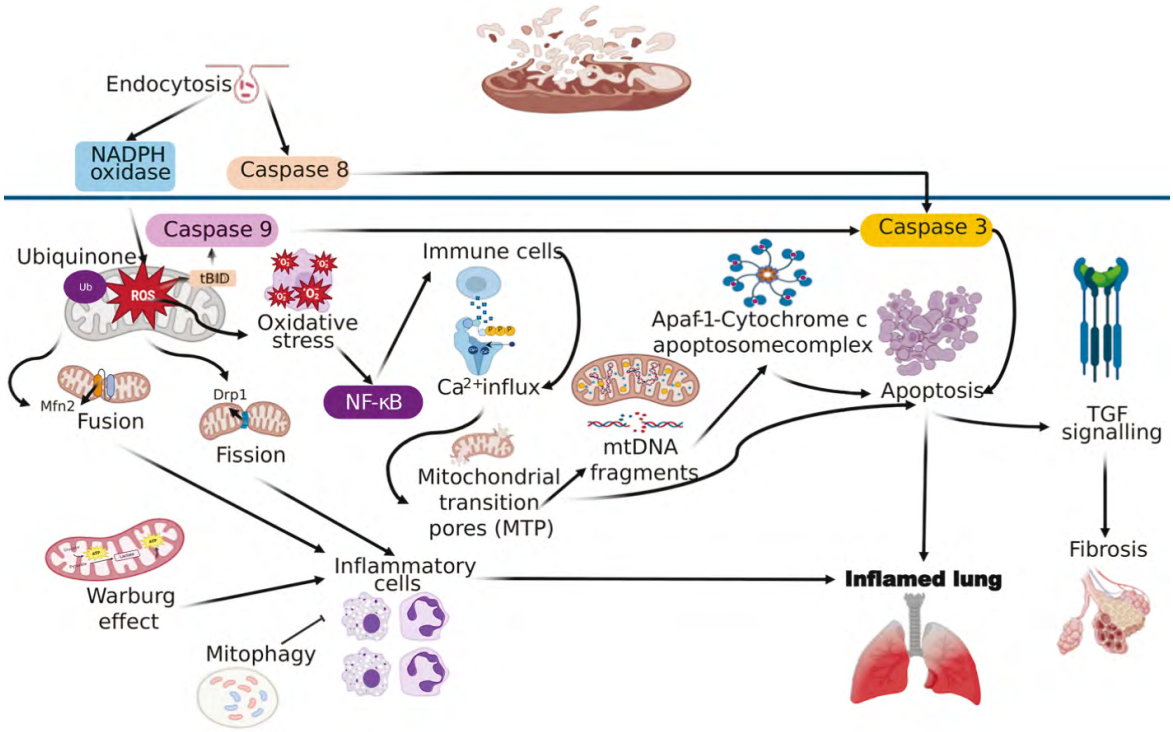


Figure 8.6.

General mechanisms of ROS production by mitochondrial dysfunction leading to lung inflammation and fibrosis.

Source: Narala, V. R., Narala, S. R., Aiya Subramani, P., et al. Role of mitochondria in inflammatory lung diseases. Front. Pharmacol. 15, 1433961 (2024). https://doi.org/10.3389/fphar.2024.1433961 License: CC BY 4.0.

[219] *Moscatiello, G. Y., Natale, C., Inserra, M., et al. The surface charge both influences the penetration and safety of polystyrene nanoparticles despite the protein corona formation. Environ. Sci.: Nano 12, 2857–2870 (2025). https://doi.org/10.1039/D4EN00962B*

[220] *Ji, Y., Chen, L., Wang, Y., et al. Realistic Nanoplastics Induced Pulmonary Damage via the Crosstalk of Ferritinophagy and Mitochondrial Dysfunction. ACS Nano 18, 16790–16807 (2024). https://doi.org/10.1021/acsnano.4c02335*

[221] *Narala, V. R., Narala, S. R., Aiya Subramani, P., et al. Role of mitochondria in inflammatory lung diseases. Front. Pharmacol. 15, 1433961 (2024). https://doi.org/10.3389/fphar.2024.1433961*

[222] *Yang, S., Zhang, T., Ge, Y., et al. Inhalation exposure to polystyrene nanoplastics induces chronic obstructive pulmonary disease-like lung injury in mice through multi-dimensional assessment. Environmental Pollution 347, 123633 (2024). https://doi.org/10.1016/j.envpol.2024.123633*

Experimental data reveal the cytotoxic potential of these particles at the molecular level (Fig. 8.7).[217] "At the tested size (800 nm), PS-NPs can enter lung epithelial cells, triggering oxidative stress, senescence, and cell death. Furthermore, these airborne particles may incite a senescent phenotype, elevating basal inflammation levels and diminishing lung tissue repair capabilities, potentially leading to prolonged lung diseases following chronic exposure."[217]

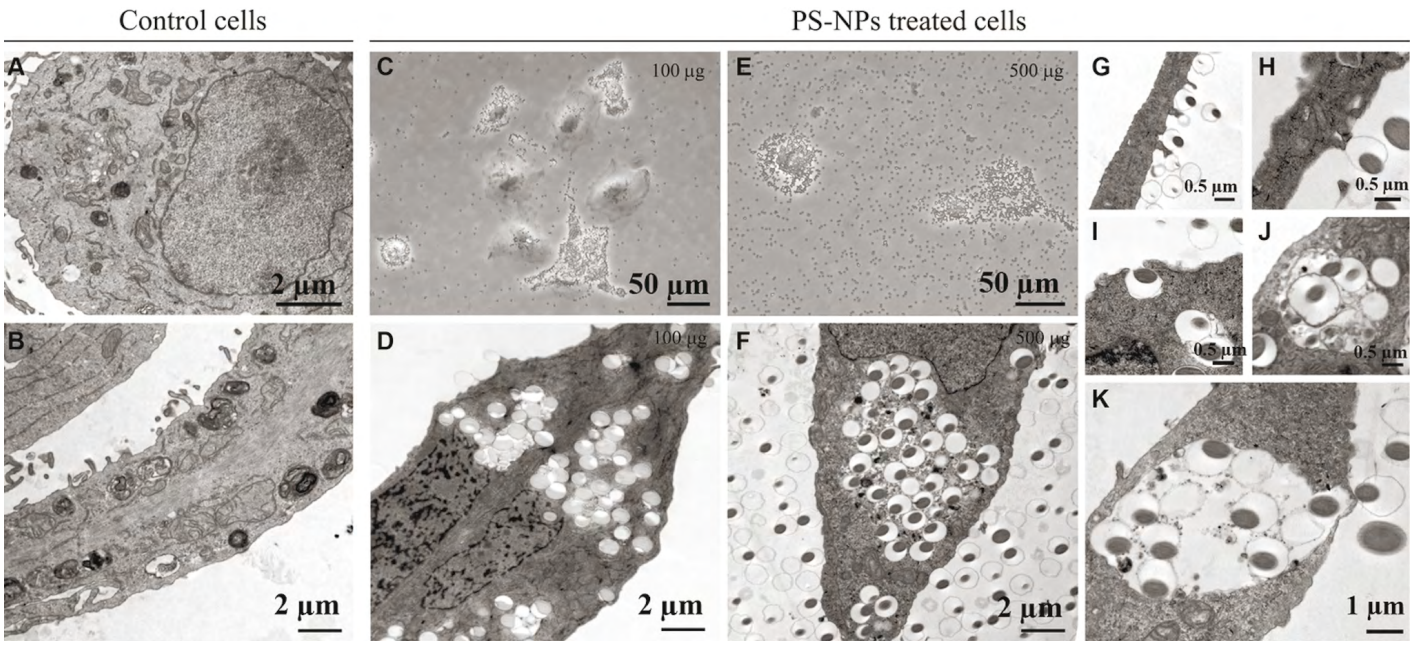


Figure 8.7.

Cellular intake of PS-NPs in A549 cells after 100 µg/mL or 500 µg/mL treatment. Transmission electron microscopy (TEM) analysis revealed the movement of nano-sized plastics into membrane structures (A,B,D,F,G–K). Active transport through the cell membrane (insets in G–K) may represent a significant gateway into the cell after treatment with different PS-NP concentrations (100 and 500 µg). Panels (C,E) reported images acquired from optical microscopy.

Source: Milillo, C., Aruffo, E., Di Carlo, P., et al. Polystyrene nanoplastics mediate oxidative stress, senescence, and apoptosis in a human alveolar epithelial cell line. Front. Public Health 12, 1385387 (2024). https://doi.org/10.3389/fpubh.2024.1385387
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DISRUPTION OF LOCAL HOMEOSTASIS AND RESPIRATORY TRACT DYSBIOSIS

In addition to systemic toxic effects, micro- and nanoplastics have a devastating effect on the local ecosystem of the respiratory tract that plays an important role in maintaining the barrier function of the lungs.[223] MNPs can accumulate in the airway surface liquid, including bronchial secretions, as

[223] *Bardawil, C. E., Dobbins, J., Lankford, S., et al. Micro- and Nanoplastics and Pulmonary Health: The Current State of Research. Microplastics 5, 29 (2026). https://doi.org/10.3390/microplastics5010029*

well as change the composition and functional activity of the lung microbiota, reducing its protective potential.

In vivo studies in animal models have shown that chronic inhalation of micro- and nanoplastics leads to dysbiosis (pronounced changes in the microbial composition of the respiratory tract).[224] This is accompanied by a reduction in microbiota diversity in the nasal cavity and lungs, along with increased growth of opportunistic pathogens such as Pseudomonas.

In the airway dysbiosis, the mucous membrane loses part of its protective function. The epithelial layer becomes more permeable, facilitating the penetration of microorganisms into the tissues. Local immunity weakens, reducing the body's ability to effectively resist bacterial and viral agents. As a result, the likelihood of their penetration into the tissues increases, sustaining chronic inflammation and contributing to the development of respiratory diseases.

EXPERIMENTAL AND CLINICAL EVIDENCE BASE

A number of authoritative studies confirm the link between MNP accumulation and the morphofunctional disorders in lung tissue.

- **Inflammatory reactions:** The authors of the study note that exposure to microplastics causes a persistent inflammatory response and potentially increases the risk of respiratory diseases. An experimental model has shown that inhalation exposure to polypropylene microplastics leads to dose-dependent lung inflammation in rats. The number of neutrophils and macrophages increased in the bronchoalveolar lavage fluid, while inflammatory markers (CINC-1, CINC-2, MPO) indicated cell damage. A persistent increase in inflammatory markers (CINC-1, CINC-2, MPO) and prolonged gene activation (including IL-17 — a cytokine that induces inflammatory reactions) were observed, persisting for up to 6 months.[225]
- **Modification of chronic diseases:** A review by an international group of scientists integrates data from multiple studies and shows that micro- and nanoplastics accumulate in lung tissue, causing inflammation, oxidative stress, and cellular damage (Fig. 8.8). They induce epithelial-mesenchymal transition (a pathological transformation of cells), disrupt genetic regulation, and activate processes associated with chronic lung diseases, including bronchial asthma, chronic obstructive pulmonary disease (COPD), and pulmonary fibrosis. The data also indicate that MNPs are found more frequently in precancerous and malignant areas of the lungs than in healthy tissue.[226]

[224] *Zha, H., Xia, J., Li, S., et al. Airborne polystyrene microplastics and nanoplastics induce nasal and lung microbial dysbiosis in mice. Chemosphere 310, 136764 (2023). https://doi.org/10.1016/j.chemosphere.2022.136764*
[225] *Tomonaga, T., Higashi, H., Izumi, H., et al. Investigation of pulmonary inflammatory responses following intratracheal instillation of and inhalation exposure to polypropylene microplastics. Part Fibre Toxicol 21, 29 (2024). https://doi.org/10.1186/s12989-024-00592-8*
[226] *Epeslidou, E., Scott, J. S., de Klein, B., et al. Microplastics as environmental modifiers of lung disease. EMBO Mol Med 18, 381–395 (2026). https://doi.org/10.1038/s44321-025-00353-w*

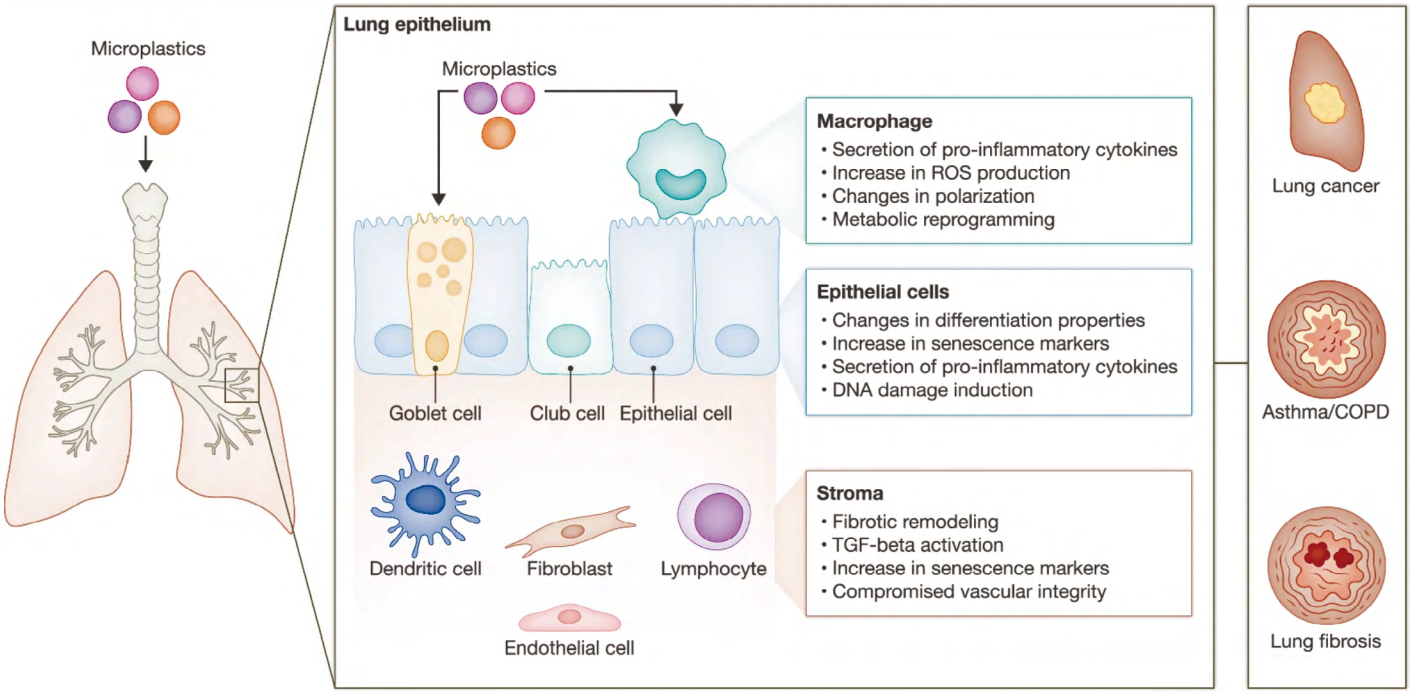


Figure 8.8.

Overview of the impact of inhaled microplastics on lung cellular homeostasis and associated disease outcomes.

Source: Epeslidou, E., Scott, J. S., de Klein, B., et al. Microplastics as environmental modifiers of lung disease. EMBO Mol Med 18, 381–395 (2026). https://doi.org/10.1038/s44321-025-00353-w License: CC BY 4.0.

- **Structural anomalies:** An empirical study identified a link between microplastics and the formation of ground-glass opacity nodules (spots with increased lung density found on computed tomography scans) in the lungs (Fig. 8.9). The frequency of their detection was higher in samples from older patients, indicating a possible cumulative nature of the pollutant's impact. This confirms the assumption that long-term contact with microplastics may contribute to the development of structural pathologies in respiratory tissue, potentially increasing the risk of developing precancerous or chronic changes.[227]

[227] Chen, Q., Gao, J., Yu, H., et al. An emerging role of microplastics in the etiology of lung ground glass nodules. Environ Sci Eur 34, 25 (2022). https://doi.org/10.1186/s12302-022-00605-3

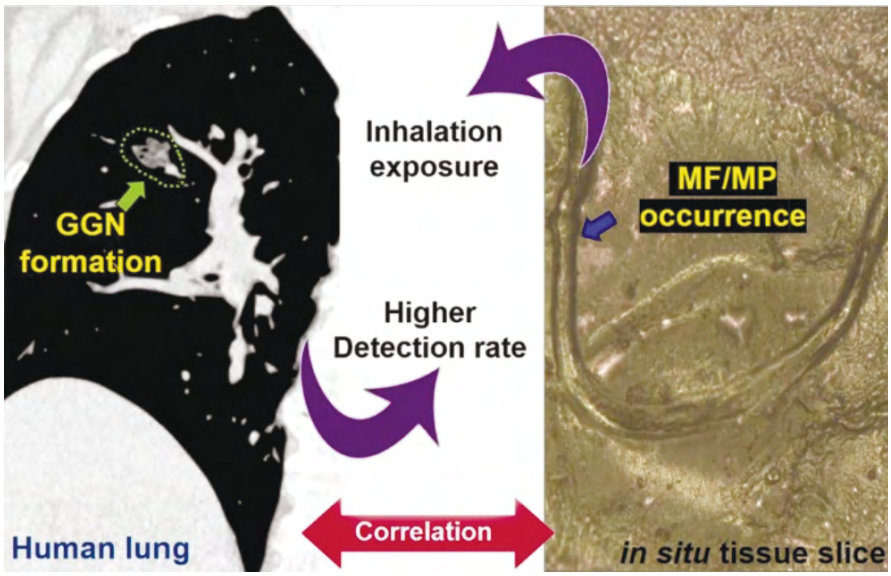


Figure 8.9.

Microplastics correlation in human lung tissues with ground-glass nodules formation was preliminarily explored.

Source: Chen, Q., Gao, J., Yu, H., et al. An emerging role of microplastics in the etiology of lung ground glass nodules. Environ Sci Eur 34, 25 (2022). https://doi.org/10.1186/s12302-022-00605-3

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- **Systemic disruptions:** Researchers established that the presence of MNPs in lung tissue is associated with changes in the blood clotting system: "The high MPs group had significantly higher levels of platelet (PLT), thrombocytocrit, and fibrinogen (FIB)," which points to a potential risk of peripheral thrombosis.[228]

  In addition, the inhalation route represents a systemic threat that extends beyond the respiratory tract. Due to their small size and electrostatic charge, inhaled particles are capable of overcoming protective barriers and penetrate the central nervous system through two independent pathways. First, nanoparticles can translocate from the lungs into the systemic bloodstream, from which they are capable of passing through the blood-brain barrier and entering the brain. Second, a direct penetration mechanism has been proven: moving along the olfactory nerve fibers, MNPs reach brain structures, bypassing protective barriers altogether.[229] This reinforces the exceptional biological and clinical significance of the inhalation route for delivering MNPs to the body.[230]

[228] *Wang, S., Lu, W., Cao, Q., et al. Microplastics in the Lung Tissues Associated with Blood Test Index. Toxics 11, 759 (2023). https://doi.org/10.3390/toxics11090759*
[229] *Amato-Lourenço, L. F., Dantas, K. C., Júnior, G. R., et al. Microplastics in the Olfactory Bulb of the Human Brain. JAMA Netw Open 7, e2440018 (2024). https://doi.org/10.1001/jamanetworkopen.2024.40018*
[230] *Huang, X., Saha, S. C., Saha, G., et al. Transport and deposition of microplastics and nanoplastics in the human respiratory tract. Environmental Advances 16, 100525 (2024). https://doi.org/10.1016/j.envadv.2024.100525*

FUNCTIONAL CONSEQUENCES:

The combination of the described structural changes (inflammation, fibrosis, tissue densification) leads to alveolar degradation and impaired gas exchange.[231] This causes persistent tissue hypoxia, which, in turn, aggravates mitochondrial dysfunction and oxidative stress in lung tissue cells. Mitochondrial impairment reduces the energy potential of cells, which compromises the lungs' ability to maintain normal regeneration and resist inflammation. As a result, structural damage to the alveoli increases, gas exchange becomes further limited, and the cycle of pathological changes repeats, forming a closed, vicious circle of progressive lung dysfunction.

CARCINOGENIC POTENTIAL OF MICRO- AND NANOPLASTICS

One of the most severe consequences of MNP accumulation in the respiratory system is the creation of conditions for cancer development.[232] Research data show that micro- and nanoplastics are capable of interfering with key cellular processes, including viability, migration,[232] metastatic activity, and apoptosis, as well as participate in the regulation of genes associated with the progression of tumor conditions.[226,232,233]

> *"Markedly, both asthma and COPD display elevated expression of IL-19 and BCL2L15 genes upon microplastic exposure, linking these pollutants to a shared axis of Th2-associated inflammation and potentially carcinogenic signaling."*[226]

A number of experimental studies emphasize that micro- and nanoplastics can induce inflammation, oxidative stress, and DNA damage, as well as interact with biomolecules.[234] Taken together, these effects allow MNPs to be viewed as a potential factor involved in the mechanisms of the tumor development. Microplastics have been shown to act not only as a physical irritant but also as a carrier of chemical additives and adsorbed carcinogens, amplifying their potential biological impact.[233] This supports the hypothesis of MNPs' role in mechanisms that promote tumor initiation and progression.

[231] *Vadász, I. & Sznajder, J. I. Gas Exchange Disturbances Regulate Alveolar Fluid Clearance during Acute Lung Injury. Front. Immunol. 8, 757 (2017). https://doi.org/10.3389/fimmu.2017.00757*
[232] *Brynzak-Schreiber, E., Schögl, E., Bapp, C., et al. Microplastics role in cell migration and distribution during cancer cell division. Chemosphere 353, 141463 (2024). https://doi.org/10.1016/j.chemosphere.2024.141463*
[233] *Bogani, G., Trimarchi, E., Chiappa, V., et al. Microplastics and cancer. European Journal of Cancer Prevention 10.1097/CEJ.0000000000000999 (2025). https://doi.org/10.1097/CEJ.0000000000000999*
[234] *Joseph, M. M., B. Nair, J. & Mary Joseph, A. Microscopic menace: exploring the link between microplastics and cancer pathogenesis. Environmental Science: Processes & Impacts 27, 1768–1795 (2025). https://doi.org/10.1039/D5EM00232J*

An analysis of 100 lung tissue samples detected microfibers in 58% of tumor samples (compared to 46% in healthy tissue), whereby two-thirds of the total mass of microplastic particles found concentrated in malignant neoplasms.[227] This provides biologically plausible evidence that MNPs do not simply accumulate in tissues but are also involved in the mechanisms of tumor initiation and progression.

In addition, spectroscopy was used to detect submicron plastic particles, with higher levels of microplastic exposure correlating with more frequent detection in precancerous and cancerous tissues.

Experimental data further indicate that the surface properties of particles determine the extent of their biological activity:

"PS-NPs with smaller size and positive surface-charge have stronger toxic effects, and it triggered epithelial-to-mesenchymal transition, the prelude to lung fibrosis, in human alveolar epithelial A549 cells."[235]

Thus, the inhalation of micro- and nanoplastics must be considered an aggressive, multi-level risk factor that potentially contributes to the pathogenesis of leading causes of death such as COPD, lung fibrosis, and malignant neoplasms of the respiratory tract.

The physicochemical properties of the particles are especially important. In particular, their and surface electrostatic charge determine the dynamics of their deposition in the airways and the efficiency of cellular uptake (internalization). Despite the obvious role of these parameters in sustaining chronic inflammation and inducing fibrosis, the mechanisms of electrostatic interaction between micro- and nanoplastics and biological membranes remain insufficiently studied.

[235] *Halimu, G., Zhang, Q., Liu, L., et al. Toxic effects of nanoplastics with different sizes and surface charges on epithelial-to-mesenchymal transition in A549 cells and the potential toxicological mechanism. Journal of Hazardous Materials 430, 128485 (2022). https://doi.org/10.1016/j.jhazmat.2022.128485*

PART 9.

THE SYSTEMIC IMPACT OF MICRO- AND NANOPLASTICS **ON THE MUSCULOSKELETAL SYSTEM:** FROM CELLULAR TOXICITY TO THE GLOBAL DISEASE BURDEN

Growing evidence suggests that micro- and nanoplastics may play an active role in driving pathological processes within the musculoskeletal system.[236] Recent studies confirm the ability of plastic particles to cross biological barriers (Fig. 9.1), accumulate in tissues, and exert a chronic cumulative effect (an impact that intensifies with prolonged presence in the body).[237,238]

It is fundamentally important to consider that:

"The surface charge of MNPs also plays a key role in cellular uptake efficiency, with zeta potential showing a positive correlation with the number of particles that are internalised."[239]

Their small size facilitates the penetration of these particles into tissues and cells, while surface characteristics, including electrostatic charge, determine the intensity of their interaction with cell membranes, proteins, and intracellular structures. Therefore, the physicochemical properties of MNPs are directly linked to the magnitude of their biological impact.

In the context of systemic MNP exposure, bone tissue is considered a potential target for their biological effects. Bone tissue is a mineralized connective tissue (containing mineral components that provide strength) that not only provides structural support and facilitates movement, but also plays a central role in regulating calcium levels in the body, protecting the bone marrow (the tissue responsible for blood cell production), and regulating metabolic processes.

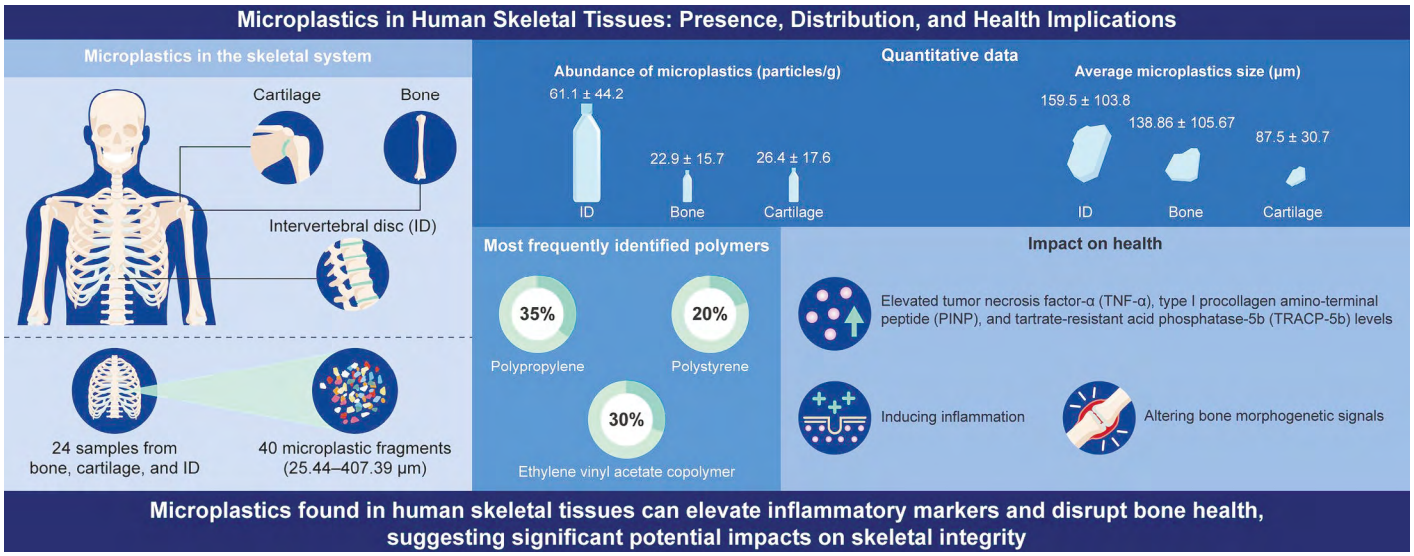


Figure 9.1.

Microplastics in human skeletal tissues: presence, distribution and health implications. Forty microplastic fragments were observed in 24 samples from the bone, cartilage, and intervertebral disc, ranging from 25.44 to 407.39 μm in diameter. The deposition abundance of microplastics in the human intervertebral disc (61.1 ± 44.2 particles/g) was higher than those in the bone (22.9 ± 15.7 particles/g) and cartilage tissue (26.4 ± 17.6 particles/g). The average sizes of microplastics in intervertebral discs (159.5 ± 103.8 μm) and bone (138.86 ± 105.67 μm) were larger than that in the cartilage tissue (87.5 ± 30.7 μm).

Source: Yang, Q., Peng, Y., Wu, X., et al. Microplastics in human skeletal tissues: Presence, distribution and health implications. Environment International 196, 109316 (2025). https://doi.org/10.1016/j.envint.2025.109316 License: CC BY-NC-ND 4.0.

[236] *Lu, S., Wei, Y., Xu, R., et al. New insights: Discovery of microplastics in human bone and skeletal muscle. The Innovation Medicine 2, 100100 (2024). https://doi.org/10.59717/j.xinn-med.2024.100100*

[237] *Yang, Q., Peng, Y., Wu, X., et al. Microplastics in human skeletal tissues: Presence, distribution and health implications. Environment International 196, 109316 (2025). https://doi.org/10.1016/j.envint.2025.109316*

[238] *Ali, N., Katsouli, J., Marczylo, E. L., et al. The potential impacts of micro-and-nano plastics on various organ systems in humans. eBioMedicine 99, 104901 (2024). https://doi.org/10.1016/j.ebiom.2023.104901*

[239] *Ali, N., Katsouli, J., Auyang, E., et al. Microplastic and nanoplastic pollution and associated potential disease risks. The Lancet Planetary Health 9, 101390 (2025). https://doi.org/10.1016/j.lanplh.2025.101390*

IDENTIFICATION OF MNPs IN HUMAN BONE AND SKELETAL MUSCLE

Unlike the acute toxic effects of chemicals or drugs, the damaging impact of micro- and nanoplastics on the musculoskeletal system may develop gradually. It may lead to long-term, persistent metabolic disruptions in bone, cartilage, and muscle tissues.[240]

MNPs can actively interfere with metabolic and cellular processes, causing an imbalance between the formation of new bone tissue (osteogenesis) and its natural breakdown (resorption), thereby triggering oxidative stress and chronic inflammation.[240,241,242] Collectively, these processes[243] contribute to the accelerated development of degenerative changes in bones, joints, intervertebral discs,[244] and muscles,[236,245] increasing the risk of osteoporosis, osteoarthritis,[246] sarcopenia (muscle loss), and pathologic fractures (Fig. 9.2, 9.3).[247]

[240] *Zhao, H., Mu, S., Wang, W., et al. Potential threats of environmental microplastics to the skeletal system: current insights and future directions. Front. Endocrinol. 16, 1658056 (2025). https://doi.org/10.3389/fendo.2025.1658056*

[241] *Zhang, Y.-W., Zhou, D.-Y., Wang, S.-C., et al. Bridging relevance between microplastics, human health and bone metabolism: Emerging threats and research directions. Environmental Chemistry and Ecotoxicology 6, 422–435 (2024). https://doi.org/10.1016/j.enceco.2024.08.006*

[242] *Shengchen, W., Jing, L., Yujie, Y., et al. Polystyrene microplastics-induced ROS overproduction disrupts the skeletal muscle regeneration by converting myoblasts into adipocytes. Journal of Hazardous Materials 417, 125962 (2021). https://doi.org/10.1016/j.jhazmat.2021.125962*

[243] *Giannandrea, D., Parolini, M., Citro, V., et al. Nanoplastic impact on bone microenvironment: A snapshot from murine bone cells. Journal of Hazardous Materials 462, 132717 (2024). https://doi.org/10.1016/j.jhazmat.2023.132717*

[244] *Sun, Y., Zhang, Y., Wang, Y., et al. From accumulation to degeneration: Microplastics as emerging risk factors for intervertebral disc health. Environmental Pollution 388, 127379 (2026). https://doi.org/10.1016/j.envpol.2025.127379*

[245] *Fusagawa, H., Youn, A., Wilkerson, E., et al. The Effects of Microplastics on Musculoskeletal Disorder; A Narrative Review. Curr Rev Musculoskelet Med 18, 39–47 (2024). https://doi.org/10.1007/s12178-024-09932-9*

[246] *Pan, C., Hong, R., Wang, K., et al. Chronic exposure to polystyrene microplastics triggers osteoporosis by breaking the balance of osteoblast and osteoclast differentiation. Toxicology 510, 154017 (2025). https://doi.org/10.1016/j.tox.2024.154017*

[247] *Pelepenko, L. E., de Oliveira, M. C., Masaro, D. A., et al. Effects of microplastics on the bones: a comprehensive review. Osteoporos Int 36, 1327–1345 (2025). https://doi.org/10.1007/s00198-025-07580-4*

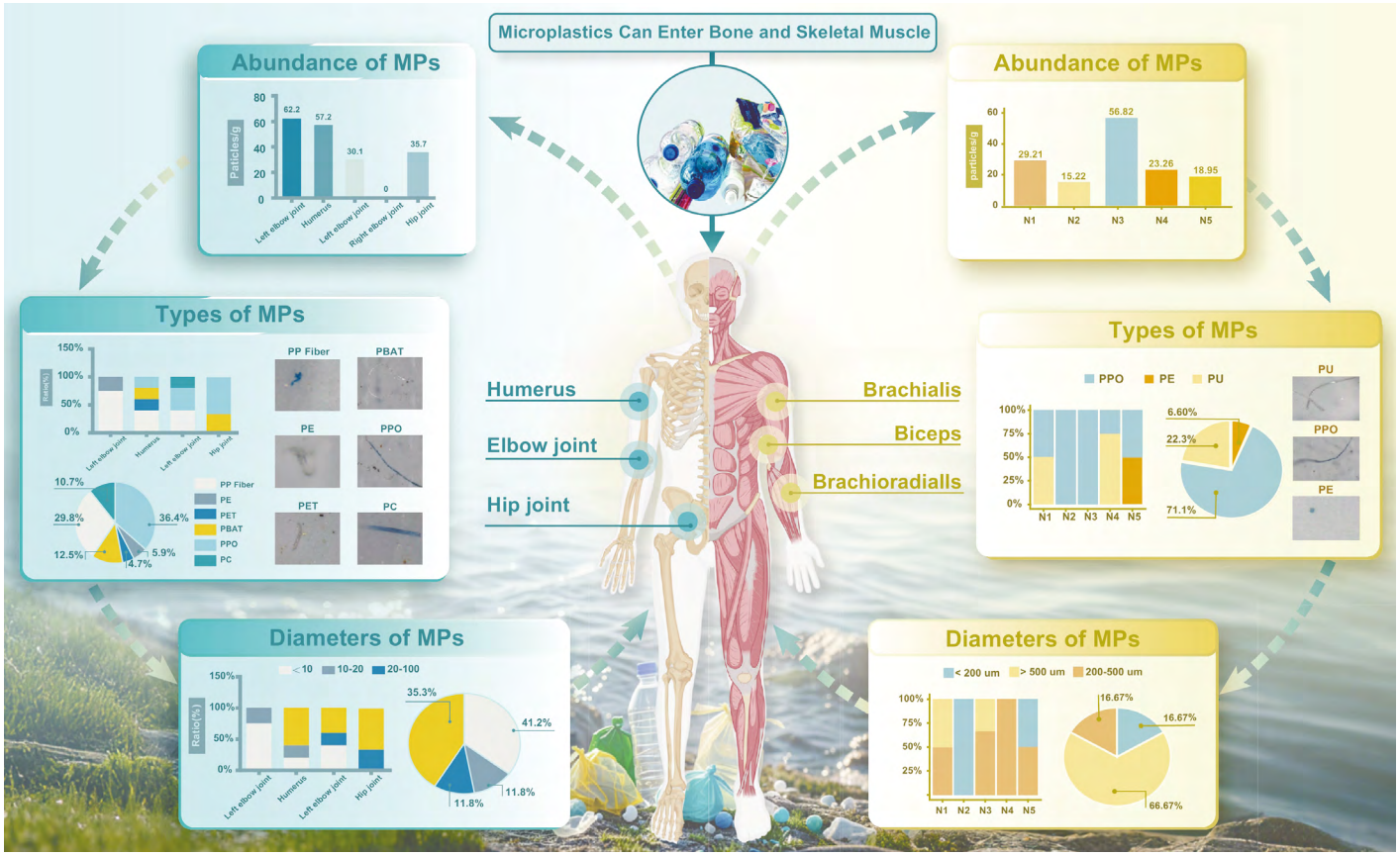


Figure 9.2.

Identification of MPs in human bone and skeletal muscle.
MPs were measured and identified using the Raman Spectrometer. In bone, the abundance of the detectable MPs in ranged from 30.1 particles/g to 62.2 particles/g; MPs with particle diameters larger than 100 μm and smaller than 10 μm account for approximately 70% of the total; six polymer types were identified, including polypropylene PP Fiber, polyethylene PE, polyethylen tereftale PET, poly butyleneadipate-co-terephthalate PBAT, polyphenylene oxide PPO, polycarbonate PC, and polyproylene PP. Fiber was the most frequently detected polymer in the bone, comprising 36.4% of the total. In skeletal muscle, the abundance of the detectable MPs ranged from 15.22–56.82 particles/g; MPs with particle diameters larger than 500 μm accounted for 66.67% of the total; three polymer types are detectable in skeletal muscle, including PPO, PE and PU, with PPO being the most prevalent.

Source: Lu, S., Wei, Y., Xu, R., et al. New insights: Discovery of microplastics in human bone and skeletal muscle. The Innovation Medicine 2, 100100 (2024). https://doi.org/10.59717/j.xinn-med.2024.100100 License: CC BY 4.0.

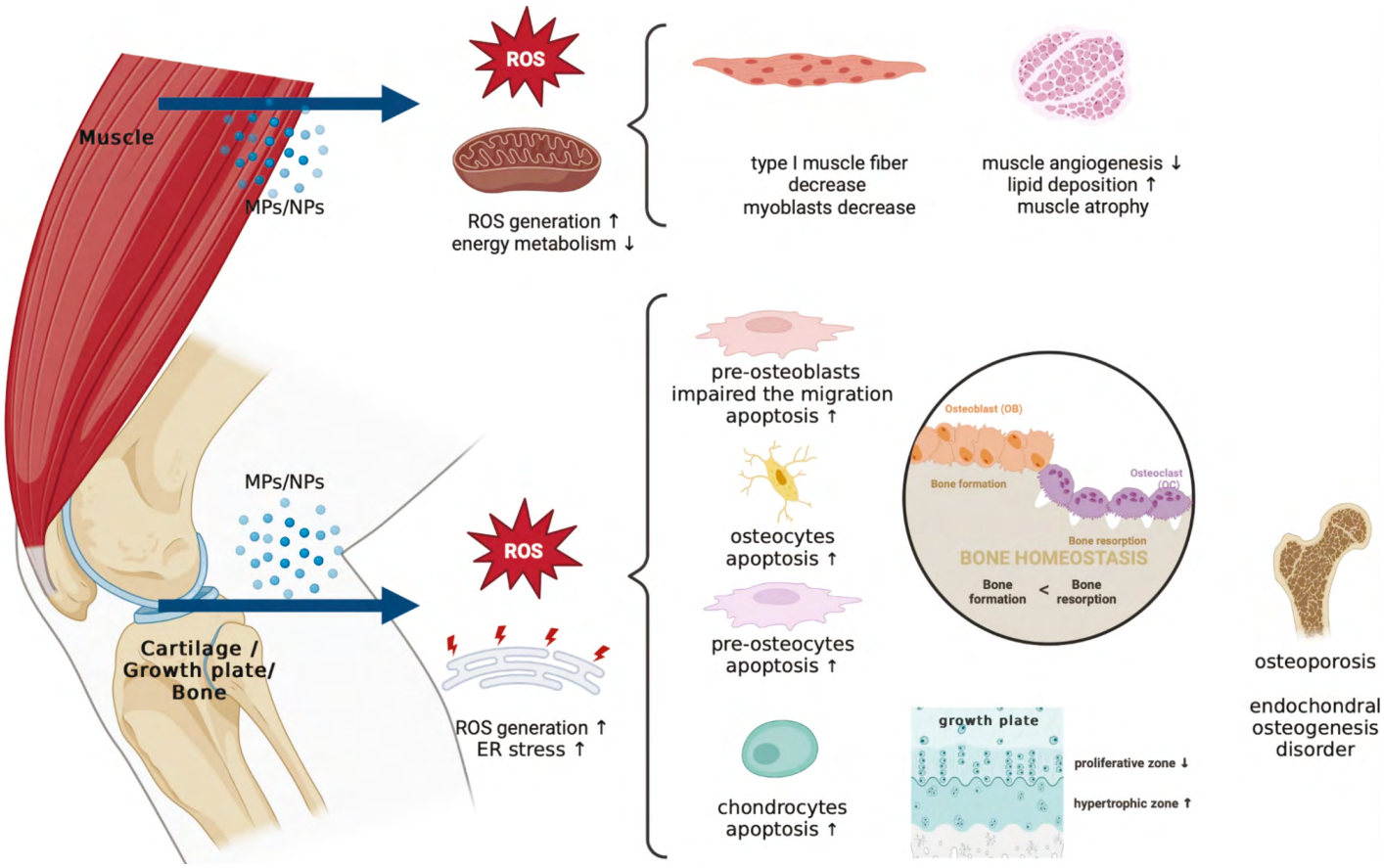


Figure 9.3.

Effects of microplastics on musculoskeletal system, including the cartilage, growth plates, bones, and muscles. MPs = microplastics; NPs = nanoplastics; ROS = reactive oxygen species; ER = endoplasmic reticulum.

Source: Fusagawa, H., Youn, A., Wilkerson, E., et al. The Effects of Microplastics on Musculoskeletal Disorder; A Narrative Review. Curr Rev Musculoskelet Med 18, 39–47 (2024). https://doi.org/10.1007/s12178-024-09932-9 License: CC BY 4.0.

MECHANISMS OF SPINAL DEGENERATION

A significant area of research has focused on the effects of microplastics on intervertebral discs.[244] Experimental data has shown that polyethylene microplastics can penetrate the cells of the nucleus pulposus — the central part of the intervertebral disc providing shock absorption and spinal flexibility.[248]

Inside the cells, MNPs can cause oxidative stress, accelerated aging, and damage to the extracellular matrix. This leads to the accelerated degeneration of intervertebral discs — a key pathological process underlying chronic back pain and degenerative disc disease.[244,248,249]

Given the limited regenerative capacity of spinal tissues, this type of micro- and nanoplastic exposure could have particularly severe consequences.

[248] *Zhang, W., Liu, K., Li, Z., et al. Polyethylene microplastics transported to nucleus pulposus cells via macrophages-derived exosomes, and promote intervertebral disc degeneration through NAT10/SLC7A11 and ac4C modification. Ecotoxicology and Environmental Safety 306, 119324 (2025). https://doi.org/10.1016/j.ecoenv.2025.119324*

[249] *Zhang, W., Li, Z., Wang, Z., et al. Polyethylene microplastics promote nucleus pulposus cell senescence by inducing oxidative stress via TLR4/NOX2 axis. Ecotoxicology and Environmental Safety 292, 117950 (2025). https://doi.org/10.1016/j.ecoenv.2025.117950*

A NEW PARADIGM FOR BONE LOSS: OSTEOCYTIC OSTEOLYSIS

Studies of bone tissue surrounding endoprostheses (joint implants) have revealed an alarming and previously unknown pathogenic mechanism for bone loss. Previously, it was believed that bone loss triggered by foreign particles was primarily driven by the inflammatory activation of osteoclasts (the cells responsible for breaking down bone tissue). However, research has shown that particles of polyethylene (a material frequently used in the construction of artificial joints) can stimulate osteocytic osteolysis. This is a process where osteocytes, which are mature bone tissue cells, begin to break down the surrounding tissue. This phenomenon represents a new form of pathological process that is qualitatively different from the classic mechanisms of bone loss.[250]

THE IMPACT OF MNPS ON BONE METABOLISM AND STRUCTURE

Micro- and nanoplastics can affect bone biomineralization processes.[251] Their surfaces can bind ions and act as a "platform" for the deposition of minerals that make up bone tissue. This potentially disrupts the formation of the mineralized matrix, which is the foundation for normal bone hardness and strength.

The accumulation of micro- and nanoplastic particles within the developing bone matrix can lead to the disruption of normal bone metabolism, the disruption of osteoblast and osteoclast activity, and the disruption of calcium-phosphorus metabolism — a key mechanism for maintaining bone health. This leads to an increase in the incidence of osteoporosis, bone density, and higher bone fragility.

Experimental data show that microplastic particles can slow bone growth, lead to the shortening of long bones, and cause the thinning of trabecular bone. In trabecular bone (the spongy, inner part of the bone), an accumulation of aging osteoblasts, a deterioration of bone microarchitecture, and a decline in autophagy (the intracellular mechanism for removing damaged structures) within these cells have been identified.[252]

Microplastic particles can induce endoplasmic reticulum stress, affecting the intracellular

[250] *Ormsby, R. T., Cantley, M., Kogawa, M., et al. Evidence that osteocyte perilacunar remodelling contributes to polyethylene wear particle induced osteolysis. Acta Biomaterialia 33, 242–251 (2016). https://doi.org/10.1016/j.actbio.2016.01.016*

[251] *Zhang, Y.-W., Zhou, D.-Y., Wang, S.-C., et al. Bridging relevance between microplastics, human health and bone metabolism: Emerging threats and research directions. Environmental Chemistry and Ecotoxicology 6, 422–435 (2024). https://doi.org/10.1016/j.enceco.2024.08.006*

[252] *Pelepenko, L. E., de Oliveira, M. C., Masaro, D. A., et al. Effects of microplastics on the bones: a comprehensive review. Osteoporos Int 36, 1327–1345 (2025). https://doi.org/10.1007/s00198-025-07580-4*

structure involved in protein synthesis and "assembly," thereby disrupting the natural mechanisms of bone formation. In cell models, micro- and nanoplastic particles have been shown to affect bone tissue precursor cells (pre-osteoblasts, pre-osteoclasts, and osteocyte-like cells) by triggering oxidative stress and apoptosis (programmed cell death). As a result, oxidative stress and apoptosis developed, which led to impaired cell migration and a shift in the differentiation balance between osteoblasts (cells responsible for building bone) and osteoclasts (cells responsible for breaking down bone).[253] Ultimately, these cellular and tissue changes can compromise bone strength and significantly increase the risk of fractures (Fig. 9.4).[251]

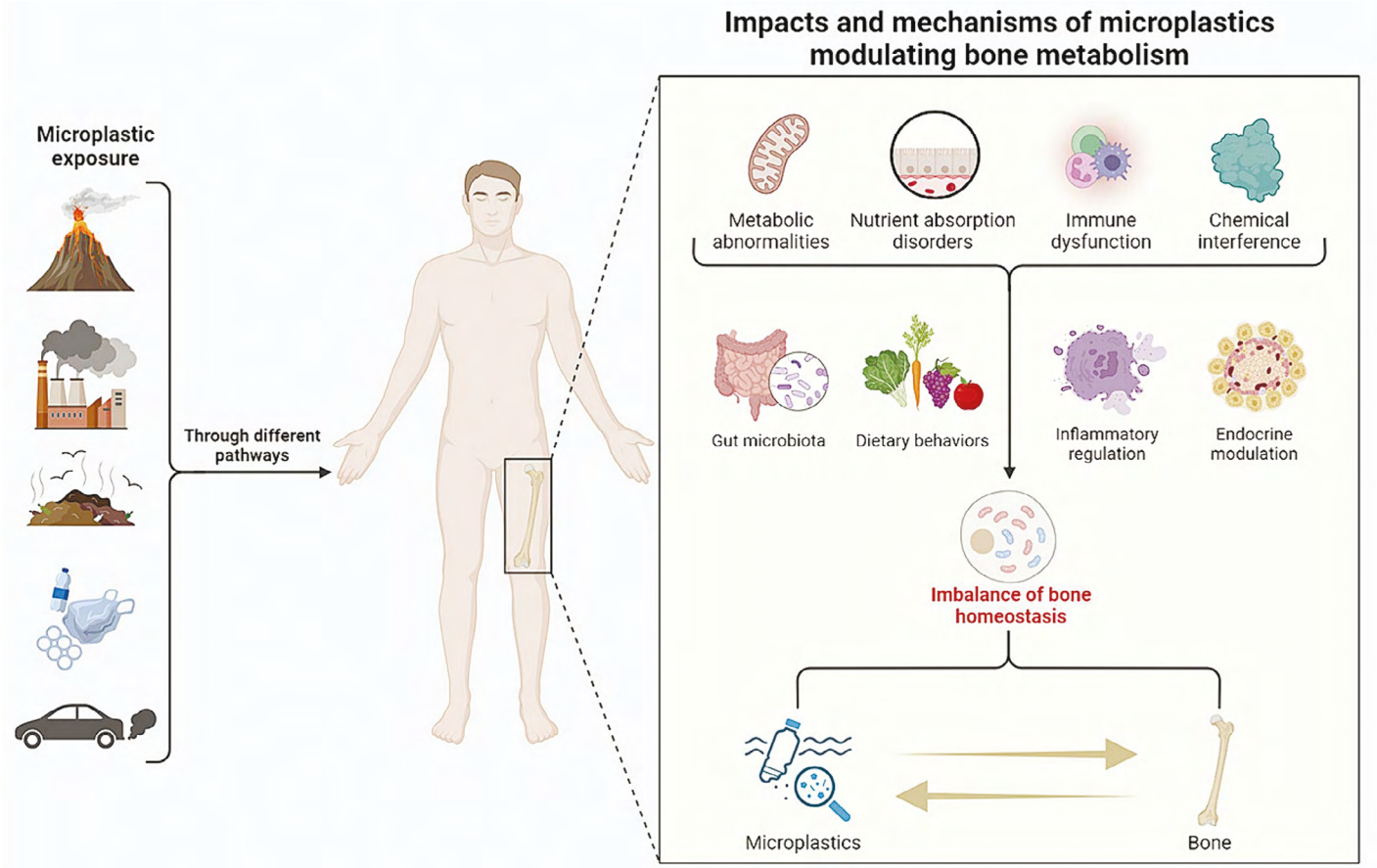


Figure 9.4.

Impacts and mechanisms of microplastics modulating the bone metabolism.

Source: Zhang, Y.-W., Zhou, D.-Y., Wang, S.-C., et al. Bridging relevance between microplastics, human health and bone metabolism: Emerging threats and research directions. Environmental Chemistry and Ecotoxicology 6, 422–435 (2024). https://doi.org/10.1016/j.enceco.2024.08.006 License: CC BY-NC-ND 4.0.

[253] *Fusagawa, H., Youn, A., Wilkerson, E., et al. The Effects of Microplastics on Musculoskeletal Disorder; A Narrative Review. Curr Rev Musculoskelet Med 18, 39–47 (2024). https://doi.org/10.1007/s12178-024-09932-9*

THE SPECIFICS OF NANOTOXICITY

Studies using zebrafish (Danio rerio) models have shown that nanoplastics are capable of causing changes in the expression of genes involved in bone formation by disrupting the primary bone morphogenetic protein (BMP) pathway, which controls the processes of bone development and repair. This results in pronounced skeletal deformities in fish larvae.[254]

Upon penetrating the bone marrow, nanoplastics reduce the survival rate of mesenchymal stem cell precursors (young cells) while stimulating the process of accelerated bone destruction (osteoclastogenesis). This creates a pathological vicious cycle: the formation of new bone is suppressed, while its destruction is accelerated (Fig. 9.5).[255]

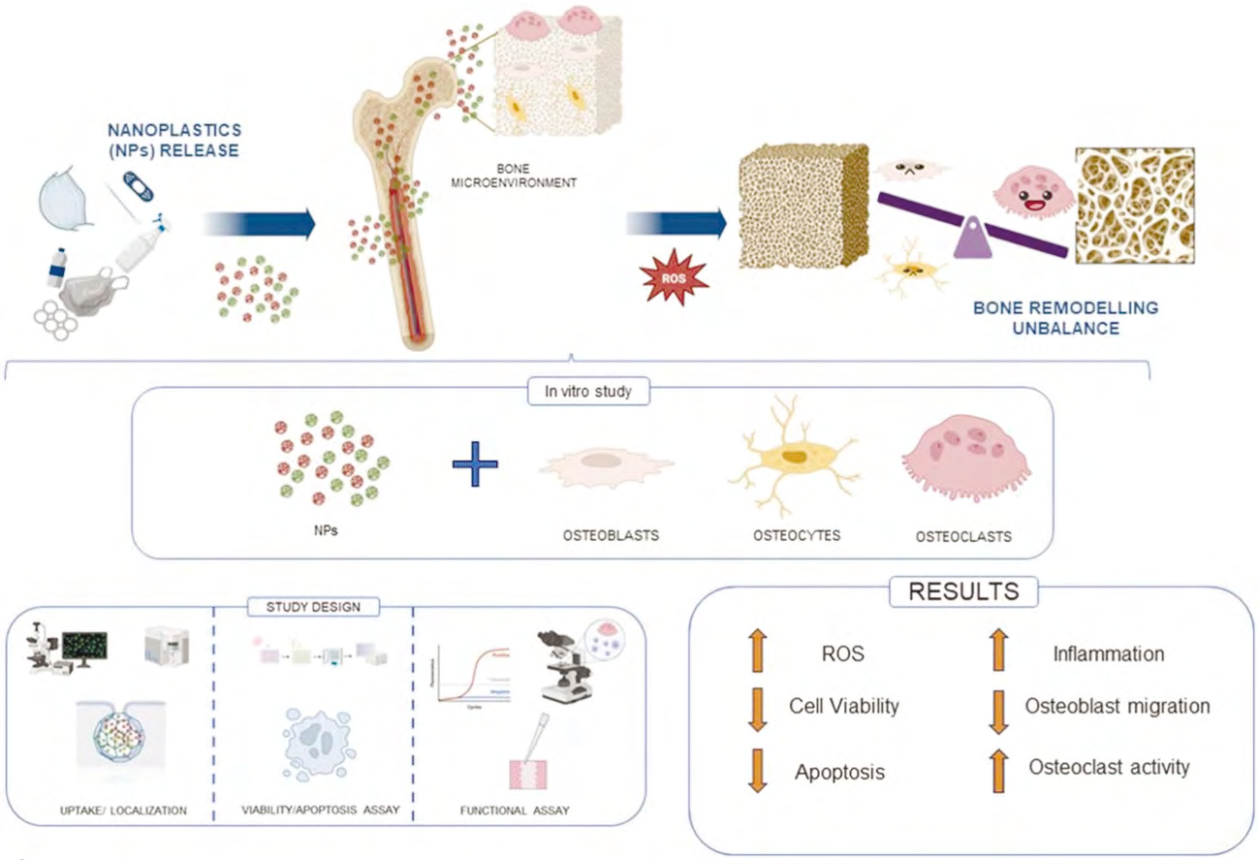


Figure 9.5.

The impact of nanoplastics on the bone microenvironment: an analysis based on murine bone cells.
NPs affect cell viability and induce ROS production and apoptosis in pre-osteoblasts, osteocytes, and pre-osteoclasts. NPs impair the migration capability of pre-osteoblasts and potentiate the osteoclastogenesis of preosteoclasts. NPs affected the expression of genes related to inflammatory and osteoblastogenic pathways in pre-osteoblasts and osteocytes, related to the osteoclastogenic commitment of pre-osteoclasts.

Source: Giannandrea, D., Parolini, M., Citro, V., et al. Nanoplastic impact on bone microenvironment: A snapshot from murine bone cells. Journal of Hazardous Materials 462, 132717 (2024). https://doi.org/10.1016/j.jhazmat.2023.132717

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[254] *Liu, W., Zeng, M., Li, Y., et al. Polystyrene nanoplastics mediate skeletal toxicity through oxidative stress and the BMP pathway in zebrafish (Danio rerio). Ecotoxicology and Environmental Safety 285, 117096 (2024). https://doi.org/10.1016/j.ecoenv.2024.117096*

[255] *Giannandrea, D., Parolini, M., Citro, V., et al. Nanoplastic impact on bone microenvironment: A snapshot from murine bone cells. Journal of Hazardous Materials 462, 132717 (2024). https://doi.org/10.1016/j.jhazmat.2023.132717*

SKELETAL MUSCLE DEGRADATION AND MITOCHONDRIAL DYSFUNCTION

Alongside bone tissue, the skeletal muscle is severely compromised. Skeletal muscles perform vital functions: they produce movement, maintain joint stability, and participate in the regulation of metabolism. Any disruptions to their structure or function immediately impact the condition of bones, joints, and overall physical mobility.

Studies have established that micro- and nanoplastics within skeletal muscles can disrupt energy and lipid metabolism. A decrease in the proportion of Type I muscle fibers (slow, endurance fibers), a deterioration of blood supply, the development of muscle atrophy, and an accumulation of lipids are noted, which leads to fatty degeneration of the tissue — the gradual replacement of muscle structures with fat.[253]

The functioning of skeletal musculature directly depends on the function of mitochondria — cellular organelles providing energy (ATP) production and the regulation of muscle regeneration. Muscle tissue regeneration is mediated by muscle stem cells (satellite cells), which require robust energy support.[256]

Exposure to electrostatically charged micro- and nanoplastics can cause mitochondrial dysfunction and a reduction in ATP synthesis.[257] The resulting energy deficit impairs the recovery of muscle tissue, hastens the onset of fatigue, slows down regeneration after exertion or injury, and contributes to the premature onset of age-related degenerative changes in the skeletal muscles.[258]

CLINICAL PICTURE: OSTEOARTHRITIS AND EPIDEMIOLOGICAL EVIDENCE

The possible association between MNPs and osteoarthritis is particularly alarming. MNP particles are detected across various components of the human musculoskeletal system: in cartilage (averaging 26 particles/g), bone (23 particles/g), and intervertebral discs (61 particles/g). The most frequently encountered particles are those of polypropylene, polystyrene, and ethylene-vinyl acetate copolymers.[259]

[256] *Alway, S. E., Paez, H. G. & Pitzer, C. R. The Role of Mitochondria in Mediation of Skeletal Muscle Repair. Muscles 2, 119–163 (2023).* https://doi.org/10.3390/muscles2020011

[257] *Bhattacharjee, S., Ershov, D., Islam, M. A., et al. Role of membrane disturbance and oxidative stress in the mode of action underlying the toxicity of differently charged polystyrene nanoparticles. RSC Adv. 4, 19321–19330 (2014).* https://doi.org/10.1039/C3RA46869K

[258] *Cui, J., Yue, X., Zhang, Y., et al. Polystyrene nanoplastics promote muscle cell senescence through microtubule hyper-stabilization-mediated mitophagy dysfunction and cGAS-Sting activation. Journal of Hazardous Materials 496, 139232 (2025).* https://doi.org/10.1016/j.jhazmat.2025.139232

[259] *Malik, M. A., Wu, S., Zhang, W., et al. Microplastics as an emerging driver of osteoarthritis: a translational synthesis of environmental exposure, patho-mechanisms, and public health implications. J Transl Med 23, 1061 (2025).* https://doi.org/10.1186/s12967-025-07081-2

"Microplastics might trigger an inflammatory response, causing occurrence and aggravation of osteoarthritis, and inflammatory reactions can damage joint cartilage, and bone structures, resulting in the joint pain, stiffness, and dysfunction."[260]

MNPs are capable of activating the inflammatory process via the NLRP3 inflammasome (an intracellular multiprotein complex that triggers the production of pro-inflammatory molecules) and amplifying the generation of reactive oxygen species. These processes disrupt the balance between bone-forming cells and bone-resorbing cells, provoking the development of chronic inflammation.[260]

The combination of these disruptions contributes to the progression of osteoarthritis, increases the risk of mobility loss, and heightens the likelihood of long-term disability.

"Epidemiological data revealed that populations in coastal and industrial regions exhibited up to 1.85-fold increased OA risk."[259]

Such statistical correlations demonstrate that the biological effects of exposure to micro- and nanoplastics identified in laboratory settings manifest in real-world population disease rates and carry true clinical significance.

DEGENERATIVE BONE DISEASES: CHALLENGES FOR SOCIETY AND HEALTHCARE

Osteoarthritis represents a massive socioeconomic burden, ranking as the fourth leading cause of disability worldwide. Direct medical costs for osteoarthritis treatment exceed $460 billion annually. In the US, $185 billion is spent each year on osteoarthritis treatment, primarily driven by knee replacements and disability payments.[259]

[260] *Zhang, Y.-W., Zhou, D.-Y., Wang, S.-C., et al. Bridging relevance between microplastics, human health and bone metabolism: Emerging threats and research directions. Environmental Chemistry and Ecotoxicology 6, 422–435 (2024). https://doi.org/10.1016/j.enceco.2024.08.006*

Osteoporotic vertebral fractures are highly prevalent: globally, one such fracture occurs every 22 seconds in men and women over the age of 50.[261] The most severe consequences are observed with hip fractures, which are accompanied by a high mortality rate, reaching 20–24% within the first year following the fracture. Among survivors, there is a severe loss of functional independence: 40% are unable to walk independently, and 60% require assistance one year later.

Projections indicate that by 2050, the number of hip fractures worldwide will increase by 310% in men and 240% in women compared to 1990 levels.[262]

These data indicate that MNPs have the potential to turn the already heavy burden of osteoarthritis and fractures into an even more acute socioeconomic challenge for healthcare systems and society as a whole.

In this context, micro- and nanoplastics can be considered as a hidden biological factor that systemically disrupts the function of the musculoskeletal system. Their impact heightens the vulnerability of bones, joints, and muscles, creating a chronic background that contributes to accelerated tissue aging and an increased risk of injury.

[261] *Epidemiology | International Osteoporosis Foundation. https://www.osteoporosis.foundation/health-professionals/fragility-fractures/epidemiology (Accessed 20 March 2026)*
[262] *Burden of Osteoporosis | International Osteoporosis Foundation. https://www.osteoporosis.foundation/policy-makers/burden-osteoporosis (Accessed 20 March 2026)*

PART 10.

THE IMPACT OF MICRO- AND NANOPLASTICS **ON HUMAN REPRODUCTIVE HEALTH**

The growing environmental pollution caused by micro- and nanoplastics is being considered a potentially significant risk factor for human reproductive health.

Experimental and clinical studies demonstrate the ability of these particles to accumulate in the tissues of the reproductive organs.[263,264]

Exposure to MNPs is associated with damage to mitochondria (the key organelles responsible for cellular energy metabolism).[265] The energy produced by mitochondria is indispensable at every stage of the reproductive process. Mitochondrial dysfunction can lead to disruptions in gametogenesis (the maturation of germ cells), steroidogenesis (the synthesis of sex hormones), and the neurohumoral regulation of reproductive processes.[266,267,268] The combined effect of these disturbances are linked to reduced fertility, the development of erectile dysfunction, and other reproductive disorders in both men[267] and women[268].

Furthermore, they can negatively impact conception, the course of pregnancy, and early embryonic development.[269] Overall, these findings point to the emergence of a potentially unfavorable global trend that requires further in-depth, interdisciplinary research.

DAMAGE TO MALE REPRODUCTIVE CELLS AND IMPAIRMENT OF SPERMATOGENESIS

The production of viable, motile sperm is a critical prerequisite for male fertility, and this process relies heavily on the energy produced by mitochondria in the form of ATP (Fig. 10.1).[265,270] Mitochondrial damage or dysfunction may result in reduced sperm motility and potential morphological abnormalities (Fig. 10.2), directly affecting fertility.[267,271]

[263] *Hong, Y., Wu, S. & Wei, G. Adverse effects of microplastics and nanoplastics on the reproductive system: A comprehensive review of fertility and potential harmful interactions. Science of The Total Environment 903, 166258 (2023). https://doi.org/10.1016/j.scitotenv.2023.166258*

[264] *Montano, L., Raimondo, S., Piscopo, M., et al. First evidence of microplastics in human ovarian follicular fluid: An emerging threat to female fertility. Ecotoxicology and Environmental Safety 291, 117868 (2025). https://doi.org/10.1016/j.ecoenv.2025.117868*

[265] *Grillo, G., Falvo, S., Latino, D., et al. Polystyrene microplastics impair the functions of cultured mouse Leydig (TM3) and Sertoli (TM4) cells by inducing mitochondrial-endoplasmic reticulum damage. Ecotoxicology and Environmental Safety 274, 116202 (2024). https://doi.org/10.1016/j.ecoenv.2024.116202*

[266] *CIFAR IVF. Does Mitochondrial Dysfunction Affect Fertility? (2024) https://www.cifarivf.com/does-mitochondrial-dysfunction-affect-fertility/ (Accessed 20 March 2026)*

[267] *Mai, Z., Yang, D., Wang, D., et al. A narrative review of mitochondrial dysfunction and male infertility. Transl Androl Urol 13, 2134–2145 (2024). https://doi.org/10.21037/tau-24-262*

[268] *Wang, T., Xu, P., Yuan, J., et al. Mitochondrial dysfunction in oocytes: implications for fertility and ageing. J Ovarian Res 18, 186 (2025). https://doi.org/10.1186/s13048-025-01764-6*

[269] *Dumollard, R., Duchen, M. & Carroll, J. The Role of Mitochondrial Function in the Oocyte and Embryo. in Current Topics in Developmental Biology vol. 77 21–49 (Academic Press, 2007). https://doi.org/10.1016/S0070-2153(06)77002-8*

[270] *Sun, Z., Wen, Y., Zhang, F., et al. Exposure to nanoplastics induces mitochondrial impairment and cytomembrane destruction in Leydig cells. Ecotoxicology and Environmental Safety 255, 114796 (2023). https://doi.org/10.1016/j.ecoenv.2023.114796*

[271] *Amaral, A., Lourenço, B., Marques, M., et al. Mitochondria functionality and sperm quality. Reproduction 146, R163–R174 (2013). https://doi.org/10.1530/REP-13-0178*

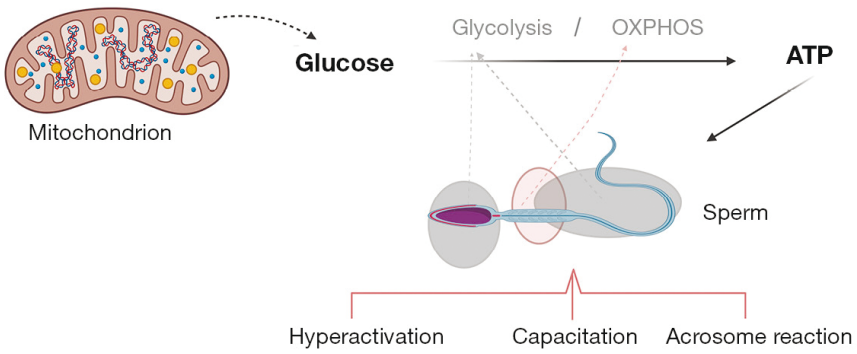


Figure 10.1.

ATP in sperm is produced by only two metabolic pathways, glycolysis and oxidative phosphorylation. The former occurs mainly in the sperm head and the main part of the flagellum, whereas the latter occurs mainly in the region of mitochondrial distribution in the middle segment of the flagellum. Sperm consumes ATP provided by mitochondria primarily to maintain motility, but also for various cellular events, including hyperactivation, capacitation, and acrosome reaction. ATP, adenosine triphosphate.

Source: Mai, Z., Yang, D., Wang, D., et al. A narrative review of mitochondrial dysfunction and male infertility. Transl Androl Urol 13, 2134–2145 (2024). https://doi.org/10.21037/tau-24-262 License: CC BY-NC-ND 4.0.

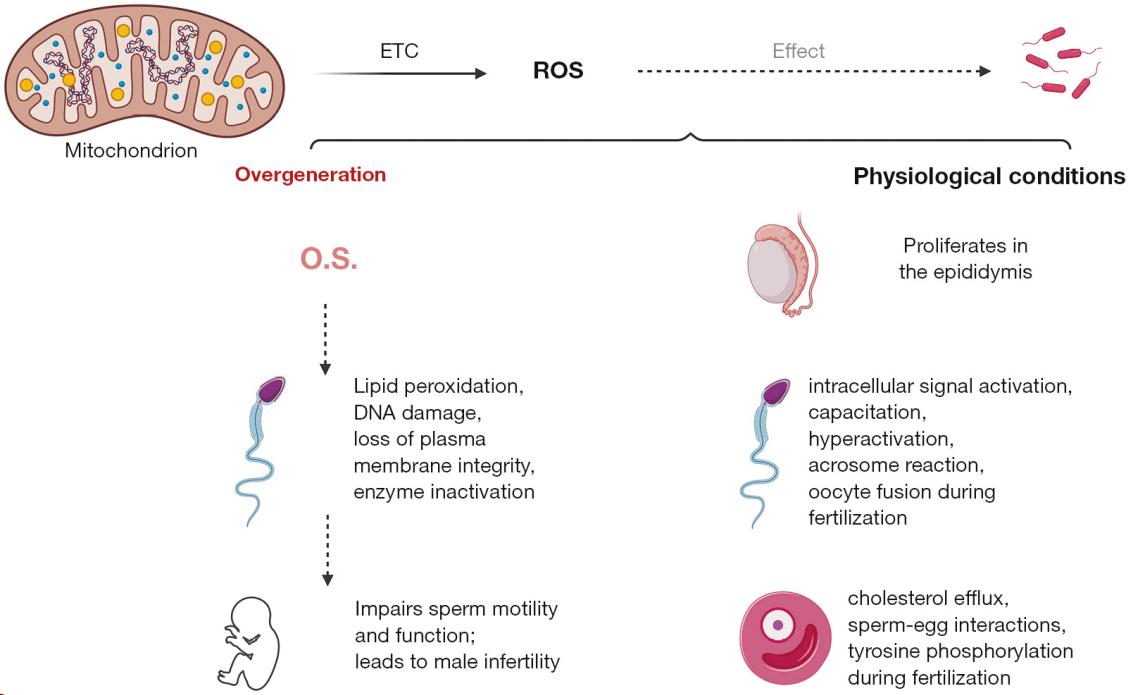


Figure 10.2.

Under physiological conditions, sperm produce appropriate levels of ROS, which are required for sperm proliferation and maturation in the epididymis, important for intracellular signaling activation, capacitation, hyperactivation, acrosome reaction and fusion with oocytes during fertilization, and critical for cholesterol efflux, sperm-egg interaction and tyrosine phosphorylation during fertilization. However, excessive accumulation of ROS in sperm may lead to oxidative stress, which in turn leads to sperm lipid peroxidation and DNA damage, or loss of plasma membrane integrity, or enzyme inactivation. All of these may further impair sperm motility and function and lead to male infertility. ETC, electron transport chain; ROS, reactive oxygen species; O.S., oxidative stress.

Source: Mai, Z., Yang, D., Wang, D., et al. A narrative review of mitochondrial dysfunction and male infertility. Transl Androl Urol 13, 2134–2145 (2024). https://doi.org/10.21037/tau-24-262 License: CC BY-NC-ND 4.0.

Data from confocal Raman microspectroscopy indicate the presence of microplastic particles in all analyzed samples of human semen.[272]

They exert a rapid and pronounced damaging effect on spermatozoa.

In one in vitro study on human spermatozoa, reactive oxygen species (ROS) overgeneration, mitochondrial dysfunction, and DNA fragmentation were observed to be induced by 30 min of exposure to 50 and 100 nm PS-NPs.[263]

These types of damage can lead to reduced sperm motility, viability, and functional activity, thereby directly hindering fertilization. Long-term consequences are confirmed by animal experiments, which have demonstrated the accumulation of plastic particles in the testes (Fig. 10.3, 10.4), leading to the disruption of spermatogenesis.[273,274]

> *"Significant reductions in volume, motility, epididymal sperm count and serum testosterone level were observed. Histological examination of testicular architecture showed distorted testes with vacuolated seminiferous tubules at the highest percentage, together with increased catalase and decreased superoxide dismutase activities. This study showed that ingestion of PS-MPs caused reproductive dysfunction in male rats and contributes to understanding the potential toxicity of microplastics in terrestrial animals."*[274]

[272] *Li, N., Yang, H., Dong, Y., et al. Prevalence and implications of microplastic contaminants in general human seminal fluid: A Raman spectroscopic study. Science of The Total Environment 937, 173522 (2024). https://doi.org/10.1016/j.scitotenv.2024.173522*

[273] *Jin, H., Yan, M., Pan, C., et al. Chronic exposure to polystyrene microplastics induced male reproductive toxicity and decreased testosterone levels via the LH-mediated LHR/cAMP/PKA/StAR pathway. Part Fibre Toxicol 19, 13 (2022). https://doi.org/10.1186/s12989-022-00453-2*

[274] *Ilechukwu, I., Ehigiator, B. E., Ben, I. O., et al. Chronic toxic effects of polystyrene microplastics on reproductive parameters of male rats. Environ Anal Health Toxicol 37, e2022015 (2022). https://doi.org/10.5620/eaht.2022015*

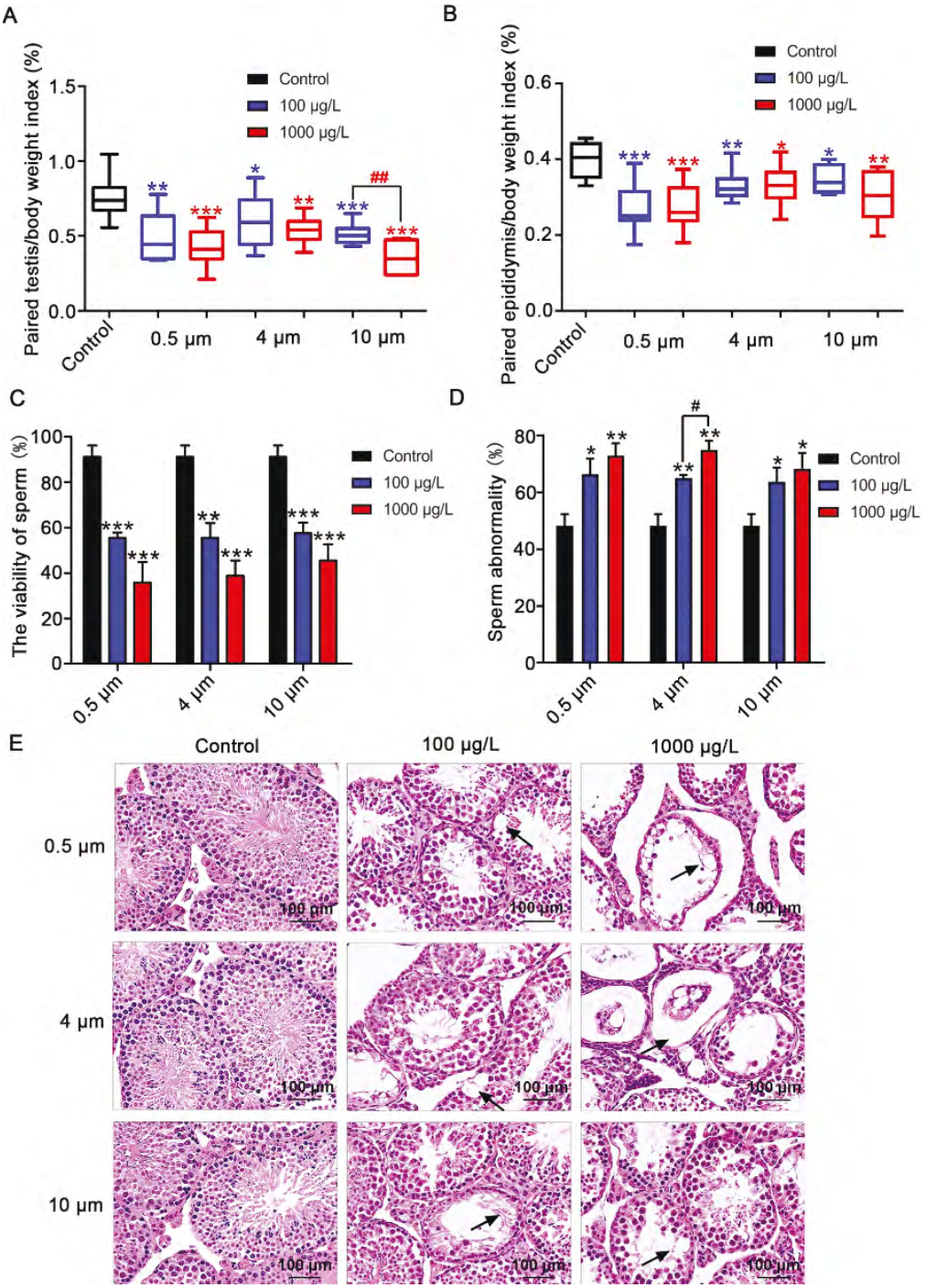


Figure 10.3.

PS-MPs exposure induced reproductive toxicity in male mice. Mice were given drinking water containing different sizes of PS-MPs at 100 μg/L and 1000 μg/L for 180 continuous days. A The paired testis/body weight index was assessed (n = 15). B The paired epididymis/body weight index was analyzed (n = 15). C The viability of sperm was detected (n = 15). D The rate of sperm abnormality was calculated (n = 15). E The impact of PS-MPs on testis structures was examined by H&E staining. Arrows: abnormal structure. Data represent means ± SD. *P < 0.05, **P < 0.01, ***P < 0.001 vs. control. #P < 0.05, ##P < 0.01 vs. 100 μg/L group.

Source: Jin, H., Yan, M., Pan, C., et al. Chronic exposure to polystyrene microplastics induced male reproductive toxicity and decreased testosterone levels via the LH-mediated LHR/cAMP/PKA/StAR pathway. Part Fibre Toxicol 19, 13 (2022). https://doi.org/10.1186/s12989-022-00453-2 License: CC BY 4.0.

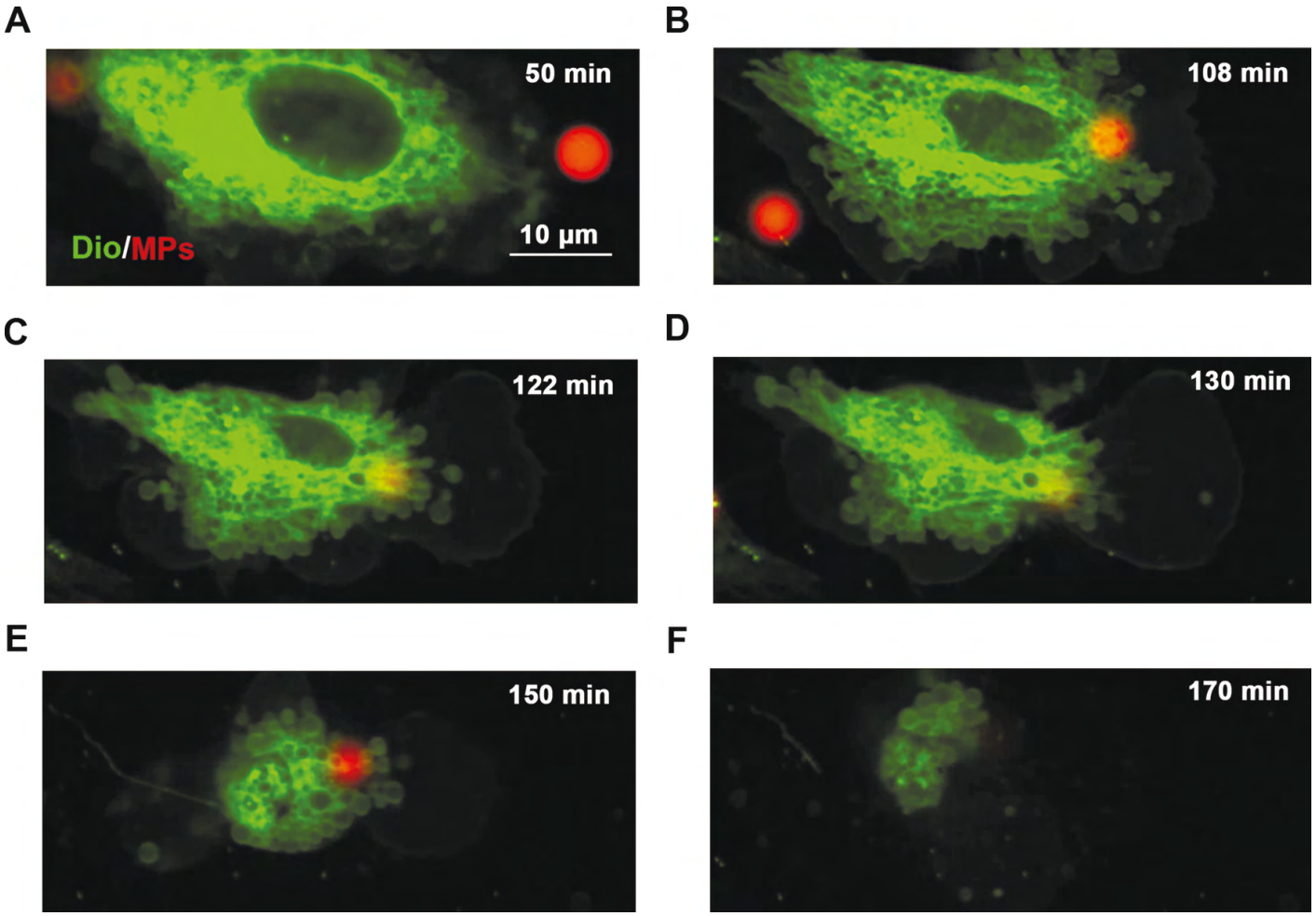


Figure 10.4.

Images of particle-cell interactions of PS-MPs. Leydig cells were exposed to 4 μm PS-MPs. Spinning disc confocal images of the cells with fluorescently labeled filamentous actin (green) and PS-MPs (red) (scale bar = 10 μm). The pictures were captured at 50 min (A), 108 min (B), 122 min (C), 130 min (D), 150 min (E), and 170 min (F) to show obvious changes.

Source: Jin, H., Yan, M., Pan, C., et al. Chronic exposure to polystyrene microplastics induced male reproductive toxicity and decreased testosterone levels via the LH-mediated LHR/cAMP/PKA/StAR pathway. Part Fibre Toxicol 19, 13 (2022). https://doi.org/10.1186/s12989-022-00453-2 License: CC BY 4.0.

The available data may provide a potential explanation for an alarming trend—a decline in total sperm count in men by more than 60%, as reported in a meta-analysis covering the period from 1973 to 2018.[275]

[275] *Levine, H., Jørgensen, N., Martino-Andrade, A., et al. Temporal trends in sperm count: a systematic review and meta-regression analysis of samples collected globally in the 20th and 21st centuries. Human Reproduction Update 29, 157–176 (2023). https://doi.org/10.1093/humupd/dmac035*

HORMONAL IMBALANCE AND ITS CONSEQUENCES

In addition to their direct effect on spermatozoa, micro- and nanoplastics indirectly affect fertility through the disruption of endocrine regulation. The synthesis of sex hormones, particularly testosterone, is an energy-dependent process requiring significant ATP expenditure. Studies in animal models demonstrate that chronic exposure to MNP particles induces mitochondrial dysfunction, causing impaired ATP production. This disruption, in turn, contributes (among other effects) to a reduction in testosterone production levels.[273,274, 276] Research has shown that under high-dose microplastic exposure, hormone concentrations decreased by as much as 78%.[277] The resulting hormonal deficiency has multifaceted clinical manifestations: from infertility and reduced libido to delayed sexual development in adolescents. Furthermore, a major consequence of this hormonal deficiency is erectile dysfunction. Decreased testosterone levels negatively affect vascular function within the corpora cavernosa of the penis, disrupting the mechanisms that enable an erection.

NEUROGENIC MECHANISMS OF ERECTILE DYSFUNCTION

Another pathway of MNP impact may involve the disruption of neurogenic and neurovascular regulation[278,279] of erection. Erection is a complex physiological process that depends on the coordinated function of the autonomic nervous system and the endothelium: nerve impulses trigger the release of nitric oxide (NO) and initiate the relaxation of smooth muscles in the cavernous tissue. Experimental data in animals confirm that chronic exposure to polystyrene nanoparticles can disrupt this mechanism.[280]

"The findings indicated that PS-NPs could accumulate within the corpus cavernosum and high dose but not low dose of PS-NPs triggered erectile dysfunction. Moreover, the toxicological effects of PS-NPs on erectile function include fibrosis in the corpus cavernous, endothelial dysfunction, reduction in testosterone levels, elevated oxidative stress and apoptosis."[280]

These effects potentially reduce the efficiency of the NO-dependent neurogenic mechanism. In a rat model, 28-day exposure to high doses of polystyrene nanoparticles was associated with erectile dysfunction within the first month of exposure.[280]

Thus, MNPs can negatively affect erectile function through a complex impact, acting via both hormonal and neurological pathways.

[276] *Qu, J., Wu, L., Mou, L., et al. Polystyrene microplastics trigger testosterone decline via GPX1. Science of The Total Environment 947, 174536 (2024). https://doi.org/10.1016/j.scitotenv.2024.174536*

[277] *Kehinde, S. A., Fatokun, T. P., Praveena, S. M., et al. Testicular mitochondrial redox imbalance and impaired oxidative phosphorylation underlie microplastic-induced testicular dysfunction in Wistar rats. Front. Toxicol. 8, 1699288 (2026). https://doi.org/10.3389/ftox.2026.1699288*

[278] *Baroni, A., Moulton, C., Cristina, M., et al. Nano- and Microplastics in the Brain: An Emerging Threat to Neural Health. Nanomaterials 15, 1361 (2025). https://doi.org/10.3390/nano15171361*

[279] *Balistreri, C. R., Magro, D. & Jadavji, N. M. Insights into the toxic effects of micro-nano-plastics on the human brain and their relationship with the onset of neurological diseases: A narrative review. Ageing Research Reviews 111, 102836 (2025) . https://doi.org/10.1016/j.arr.2025.102836*

[280] *Wang, M., Dai, B., Liu, Q., et al. Polystyrene nanoplastics exposure causes erectile dysfunction in rats. Ecotoxicology and Environmental Safety 280, 116551 (2024). https://doi.org/10.1016/j.ecoenv.2024.116551*

EPIDEMIOLOGY OF ERECTILE DYSFUNCTION: CURRENT TRENDS

The clinical consequences of the described mechanisms are characterized by high prevalence. According to a national study held in Japan, the prevalence of erectile dysfunction (ED) has reached a level where one in every three men in the country experiences corresponding symptoms.[281] The most pronounced negative trend was observed in the 20 to 24-year age group, where the ED rate (26.6%) has nearly leveled with the rate seen in men aged 50 to 54 years (27.8%) (Fig. 10.5).

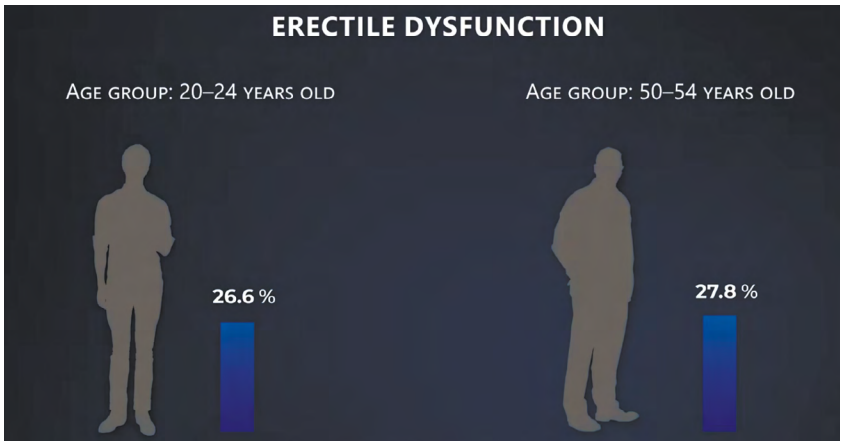


Figure 10.5.

The most pronounced negative trend was observed in the 20 to 24-year age group, where the ED rate (26.6%) has nearly leveled with the rate seen in men aged 50 to 54 years (27.8%).

Data source: Tsujimura, A., Fukuhara, S., Chiba, K., et al. Erectile Function and Sexual Activity Are Declining in the Younger Generation: Results from a National Survey in Japan. The World Journal of Men's Health 43, 239–248 (2025). https://doi.org/10.5534/wjmh.240137

A meta-analysis of global data indicates that symptoms of ED affect, to varying degrees, up to 76.5% of men worldwide.[282] Existing demographic projections,[283] which predicted an increase in the number of men with ED from 152 million in 1995 to 322 million by 2025, may underestimate the scale of the problem in light of the rapid accumulation of MNPs in the environment.

IMPACT OF MNPS ON THE FEMALE REPRODUCTIVE SYSTEM

The female reproductive system is also vulnerable to the effects of MNPs. Recent studies have demonstrated, for the first time, the presence of microplastic particles in female ovarian follicular fluid (Fig. 10.6, 10.7).Particles were detected in 14 out of 18 women examined, with an average concentration of 2,191 particles/mL (ranging from 0 to 7,181 particles/mL). These findings provide direct evidence that microplastics are capable of crossing the blood-follicle barrier.[284]

[281] *Tsujimura, A., Fukuhara, S., Chiba, K., et al. Erectile Function and Sexual Activity Are Declining in the Younger Generation: Results from a National Survey in Japan. The World Journal of Men's Health 43, 239–248 (2025). https://doi.org/10.5534/wjmh.240137*

[282] *Kessler, A., Sollie, S., Challacombe, B., et al. The global prevalence of erectile dysfunction: a review. BJU International 124, 587–599 (2019). https://doi.org/10.1111/bju.14813*

[283] *Aytaç, Mckinlay, & Krane. The likely worldwide increase in erectile dysfunction between 1995 and 2025 and some possible policy consequences. BJU International 84, 50–56 (1999). https://doi.org/10.1046/j.1464-410x.1999.00142.x*

[284] *Montano, L., Raimondo, S., Piscopo, M., et al. First evidence of microplastics in human ovarian follicular fluid: An emerging threat to female fertility. Ecotoxicology and Environmental Safety 291, 117868 (2025). https://doi.org/10.1016/j.ecoenv.2025.117868*

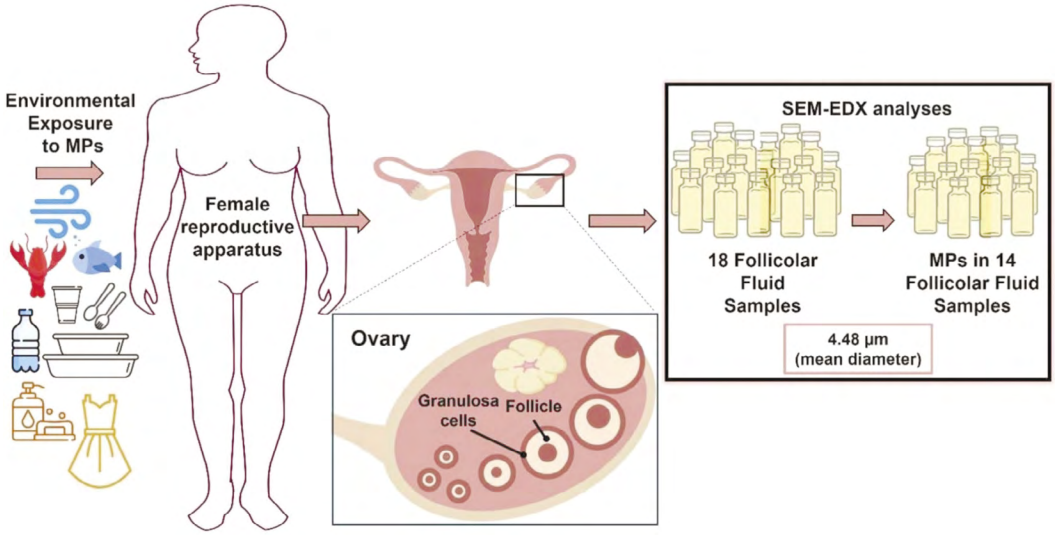


Figure 10.6.

Microplastics were found in human follicular fluid samples. Plastic particles < 10 µm were measured using Scanning Electron Microscopy with EDX (X Energy Dispersion) detection. Microplastics found in 14 out of 18 samples, average 2191 p/mL, mean diameter 4.48 µm.

Source: Montano, L., Raimondo, S., Piscopo, M., et al. First evidence of microplastics in human ovarian follicular fluid: An emerging threat to female fertility. Ecotoxicology and Environmental Safety 291, 117868 (2025). https://doi.org/10.1016/j.ecoenv.2025.117868 License: CC BY-NC-ND 4.0.

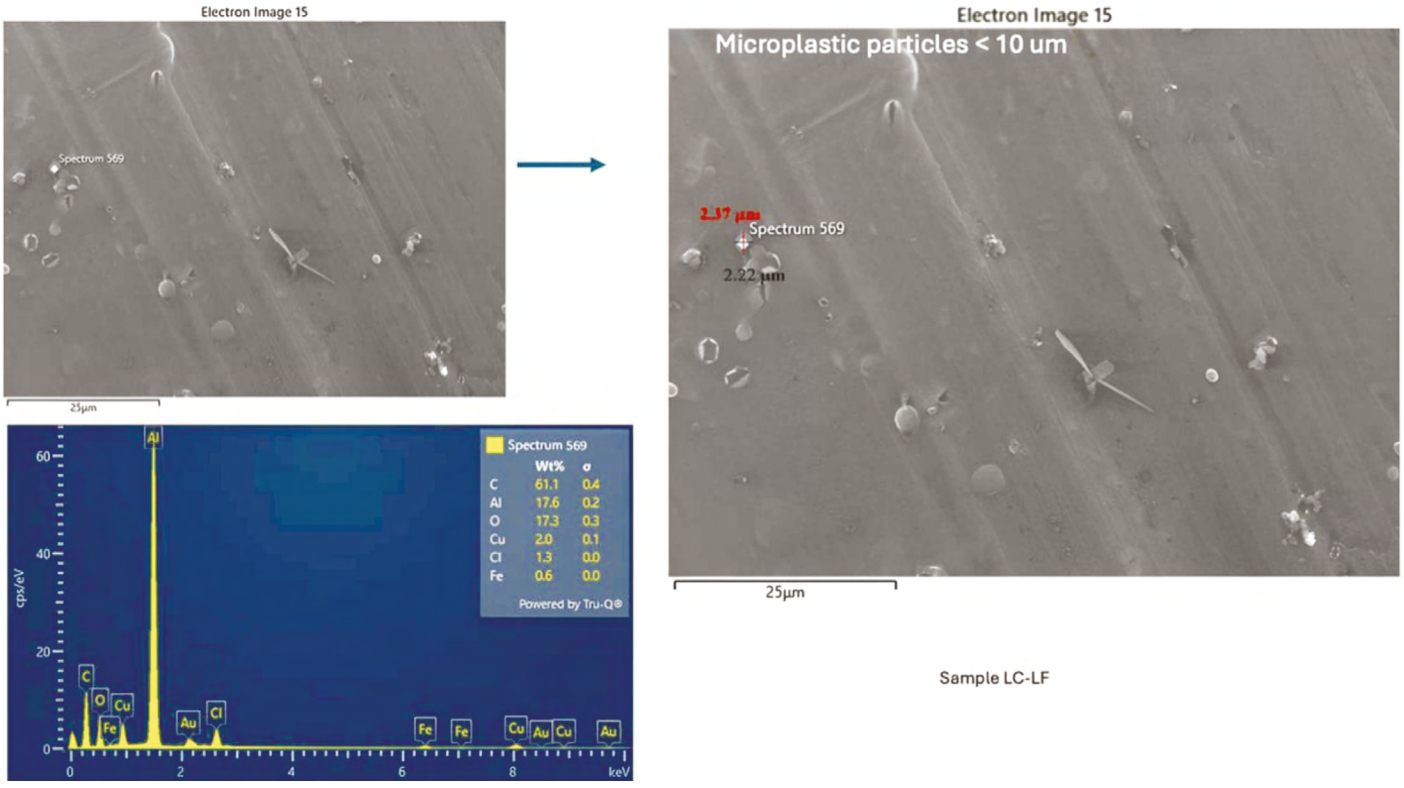


Figure 10.7.

Selected Scanning Electron Microscopy SEM-EDX image MPs in follicular fluid.

Source: Montano, L., Raimondo, S., Piscopo, M., et al. First evidence of microplastics in human ovarian follicular fluid: An emerging threat to female fertility. Ecotoxicology and Environmental Safety 291, 117868 (2025). https://doi.org/10.1016/j.ecoenv.2025.117868 License: CC BY-NC-ND 4.0.

The accumulation of micro- and nanoplastics in ovarian follicular fluid creates conditions for a potential impact on the developing oocyte and may disrupt key mechanisms of female fertility.[284,285,286,287,288]

Studies in animal models demonstrate: "In vivo, exposure to 1 mg/day PS-NPs resulted in decreased fertility, abnormal ovarian function, and increased ovarian apoptosis in female mice."[287]

The energy supply for female reproduction depends entirely on mitochondria, the number of which in a human oocyte is estimated to range from 100,000 to 600,000.[289,290,291]

Nanoplastic particles are capable of inducing mitochondrial dysfunction (see Part 4). Mitochondrial dysfunction, in turn, can lead to impaired oocyte maturation, reduced probability of successful fertilization, and even early embryonic developmental arrest.[292,293,294]

An additional risk is associated with the chemical additives within the plastics,[295] many of which are known endocrine disruptors [296] capable of directly interfering with the regulatory mechanisms of the reproductive system.[297,298] Furthermore, MNPs can act as potential transporters ("Trojan Horse effect") of toxic substances.[299,300] Plastic particles adsorb toxic compounds from the environment, significantly increasing their bioavailability and the efficiency of their penetration into the body, which creates a synergistic damaging effect.[301,302,303,304,305]

[285] *Hou, J., Lei, Z., Cui, L., et al. Polystyrene microplastics lead to pyroptosis and apoptosis of ovarian granulosa cells via NLRP3/Caspase-1 signaling pathway in rats. Ecotoxicology and Environmental Safety 212, 112012 (2021).* https://doi.org/10.1016/j.ecoenv.2021.112012

[286] *Xue, Y., Cheng, X., Ma, Z.-Q., et al. Polystyrene nanoplastics induce apoptosis, autophagy, and steroidogenesis disruption in granulosa cells to reduce oocyte quality and fertility by inhibiting the PI3K/AKT pathway in female mice. J Nanobiotechnol 22, 460 (2024).* https://doi.org/10.1186/s12951-024-02735-7

[287] *Zeng, L., Zhou, C., Xu, W., et al. The ovarian-related effects of polystyrene nanoplastics on human ovarian granulosa cells and female mice. Ecotoxicology and Environmental Safety 257, 114941 (2023).* https://doi.org/10.1016/j.ecoenv.2023.114941

[288] *Cheng, X., Xue, Y., Wang, H., et al. Maternal exposure to polystyrene nanoplastics during gestation and lactation caused fertility decline in female mouse offspring. Ecotoxicology and Environmental Safety 289, 117632 (2025).* https://doi.org/10.1016/j.ecoenv.2024.117632

[289] *CIFAR IVF. Does Mitochondrial Dysfunction Affect Fertility? (2024)* https://www.cifarivf.com/does-mitochondrial-dysfunction-affect-fertility/ *(Accessed 20 March 2026)*

[290] *Fragouli, E. & Wells, D. Mitochondrial DNA Assessment to Determine Oocyte and Embryo Viability. Semin Reprod Med 33, 401–409 (2015).* https://doi.org/10.1055/s-0035-1567821

[291] *Kofinas, J. D., Seth-Smith, M. L., Kramer, Y., et al. Oocytes with impaired meiotic maturation contain increased mtDNA deletions. J Assist Reprod Genet 42, 753–762 (2025).* https://doi.org/10.1007/s10815-025-03393-w

[292] *Wang, T., Xu, P., Yuan, J., et al. Mitochondrial dysfunction in oocytes: implications for fertility and ageing. J Ovarian Res 18, 186 (2025).* https://doi.org/10.1186/s13048-025-01764-6

[293] *Dumollard, R., Duchen, M. & Carroll, J. The Role of Mitochondrial Function in the Oocyte and Embryo. in Current Topics in Developmental Biology vol. 77 21–49 (Academic Press, 2007).* https://doi.org/10.1016/S0070-2153(06)77002-8

[294] *Yang, J., Wang, L., Ma, J., et al. Endometrial proteomic profile of patients with repeated implantation failure. Front. Endocrinol. 14, 1144393 (2023).* https://doi.org/10.3389/fendo.2023.1144393

[295] *IPEN (International Pollutants Elimination Network). Plastics, EDCs & Health.* https://ipen.org/documents/plastics-edcs-health *(Accessed 20 March 2026)*

[296] *Maradonna, F., Vandenberg, L. N. & Meccariello, R. Editorial: Endocrine-Disrupting Compounds in Plastics and Their Effects on Reproduction, Fertility, and Development. Front. Toxicol. 4, 886628 (2022).* https://doi.org/10.3389/ftox.2022.886628

[297] *Hannon, P. R. & Flaws, J. A. The Effects of Phthalates on the Ovary. Front. Endocrinol. 6, 8 (2015).* https://doi.org/10.3389/fendo.2015.00008

[298] *Welch, B. M., Keil, A. P., Buckley, J. P., et al. Associations Between Prenatal Urinary Biomarkers of Phthalate Exposure and Preterm Birth: A Pooled Study of 16 US Cohorts. JAMA Pediatr 176, 895–905 (2022).* https://doi.org/10.1001/jamapediatrics.2022.2252

[299] *Gruber, E. S., Stadlbauer, V., Pichler, V., et al. To Waste or Not to Waste: Questioning Potential Health Risks of Micro- and Nanoplastics with a Focus on Their Ingestion and Potential Carcinogenicity. Expo Health 15, 33–51 (2023).* https://doi.org/10.1007/s12403-022-00470-8

[300] *Hildebrandt, L., Nack, F. L., Zimmermann, T., et al. Microplastics as a Trojan horse for trace metals. Journal of Hazardous Materials Letters 2, 100035 (2021).* https://doi.org/10.1016/j.hazl.2021.100035

[301] *Munoz, M., Ortiz, D., Nieto-Sandoval, J., et al. Adsorption of micropollutants onto realistic microplastics: Role of microplastic nature, size, age, and NOM fouling. Chemosphere 283, 131085 (2021).* https://doi.org/10.1016/j.chemosphere.2021.131085

[302] *Wang, F., Wong, C. S., Chen, D., et al. Interaction of toxic chemicals with microplastics: A critical review. Water Research 139, 208–219 (2018).* https://doi.org/10.1016/j.watres.2018.04.003

[303] *Ateia, M., Zheng, T., Calace, S., et al. Sorption behavior of real microplastics (MPs): Insights for organic micropollutants adsorption on a large set of well-characterized MPs. Science of The Total Environment 720, 137634 (2020).* https://doi.org/10.1016/j.scitotenv.2020.137634

[304] *Brennecke, D., Duarte, B., Paiva, F., et al. Microplastics as vector for heavy metal contamination from the marine environment. Estuarine, Coastal and Shelf Science 178, 189–195 (2016).* https://doi.org/10.1016/j.ecss.2015.12.003

[305] *Shi, Y., Almuhtaram, H. & Andrews, R. C. Adsorption of Per- and Polyfluoroalkyl Substances (PFAS) and Microcystins by Virgin and Weathered Microplastics in Freshwater Matrices. Polymers 15, 3676 (2023).* https://doi.org/10.3390/polym15183676

Thus, the combined experimental, clinical, and epidemiological data allows us to view the accumulation of MNPs in reproductive tissues as a significant pathogenetic factor, potentially contributing to a global decline in fertility and an increase in reproductive disorders.[306,307,308,309,310,311]

It is important to note that the size of an MNP and the electrostatic charge on its surface plays a key role in the initial interaction of micro- and nanoplastic particles with biological systems. Electrostatic attraction between the charged MNP surface and the cell membrane facilitates particle adsorption and cellular entry. Once inside the cells, micro- and nanoplastic particles can trigger mitochondrial dysfunction, hormonal imbalance, and neurogenic disorders, which directly reduce fertility and impair sexual function in both men and women.

According to current WHO statistics, approximately 17.5% of the adult population – roughly 1 in 6 worldwide – experience infertility.[312] This may represent the early stage of a broader adverse trend.

Predictive assessments by experts, notably Professor Shanna Swan, suggest that neuroendocrine disruptions caused by plastic additives could lead to a total reproductive crisis by 2045.[313] Thus, the problem of MNP exposure transcends environmental issues and constitutes a fundamental threat to demographic stability and the future of humanity.

[306] *Zhang, C., Zhang, G., Sun, K., et al. Association of mixed exposure to microplastics with sperm dysfunction: a multi-site study in China. eBioMedicine 108, (2024). https://doi.org/10.1016/j.ebiom.2024.105369*

[307] *EarthDay.org. Plastics: The Kingpin of the Fertility Crisis. (2024). https://www.earthday.org/plastics-the-kingpin-of-the-fertility-crisis/ (Accessed 20 March 2026)*

[308] *Grillo, G., Falvo, S., Latino, D., et al. Polystyrene microplastics impair the functions of cultured mouse Leydig (TM3) and Sertoli (TM4) cells by inducing mitochondrial-endoplasmic reticulum damage. Ecotoxicology and Environmental Safety 274, 116202 (2024). https://doi.org/10.1016/j.ecoenv.2024.116202*

[309] *Zhang, Z., Meng, J., Tian, J., et al. Reproductive and developmental implications of micro- and nanoplastic internalization: Recent advances and perspectives. Ecotoxicology and Environmental Safety 286, 117245 (2024). https://doi.org/10.1016/j.ecoenv.2024.117245*

[310] *Wei, Z., Wang, Y., Wang, S., et al. Comparing the effects of polystyrene microplastics exposure on reproduction and fertility in male and female mice. Toxicology 465, 153059 (2022). https://doi.org/10.1016/j.tox.2021.153059*

[311] *Hou, B., Wang, F., Liu, T., et al. Reproductive toxicity of polystyrene microplastics: In vivo experimental study on testicular toxicity in mice. Journal of Hazardous Materials 405, 124028 (2021). https://doi.org/10.1016/j.jhazmat.2020.124028*

[312] *World Health Organization (WHO). 1 in 6 People Globally Affected by Infertility. (2023). https://www.who.int/news/item/04-04-2023-1-in-6-people-globally-affected-by-infertility (Accessed 20 March 2026)*

[313] *The Guardian. Shanna Swan: 'Most couples may have to use assisted reproduction by 2045' (2021). https://www.theguardian.com/society/2021/mar/28/shanna-swan-fertility-reproduction-count-down (Accessed 20 March 2026)*

PART 11.

THE IMPACT OF MICRO- AND NANOPLASTICS ON **PRENATAL** AND **POSTNATAL DEVELOPMENT**

Current data indicate that micro- and nanoplastics (MNPs) can exert a systemic effect on the human body, starting from the stage of intrauterine development. The detection of microparticles in maternal and fetal tissues identifies MNPs as a potentially significant risk factor capable of disrupting fundamental processes of embryogenesis, intrauterine development, and the postnatal formation of vital body systems.

The urgency of this issue is particularly pronounced given the documented rise in congenital anomalies and developmental disorders in children across many countries worldwide.

PENETRATION OF MICRO- AND NANOPLASTICS THROUGH THE PLACENTAL BARRIER

During pregnancy, a complex physiological system between the mother and fetus develops to ensure normal growth, nutrition, and protection for the developing organism. A central role in this system is played by the placenta, which performs gas exchange, transport of nutrients and metabolites, as well as barrier and immunoregulatory functions. For a long time, it was believed that the placental barrier possesses high selective permeability and reliably limits the penetration of foreign substances from the mother's organism to the fetus. However, experimental data indicate that plastic micro- and nanoparticles are capable of breaching placental defense mechanisms and accumulating in placental tissue. A significant milestone was the study by the scientific group led by Antonio Ragusa, which first detected microplastics in the placentas of women (Fig. 11.1).[314] This discovery demonstrated that synthetic polymer particles can penetrate tissues previously considered protected from the influence of artificial materials.

[314] *Ragusa, A., Svelato, A., Santacroce, C., et al. Plasticenta: First evidence of microplastics in human placenta. Environment International 146, 106274 (2021). https://doi.org/10.1016/j.envint.2020.106274*

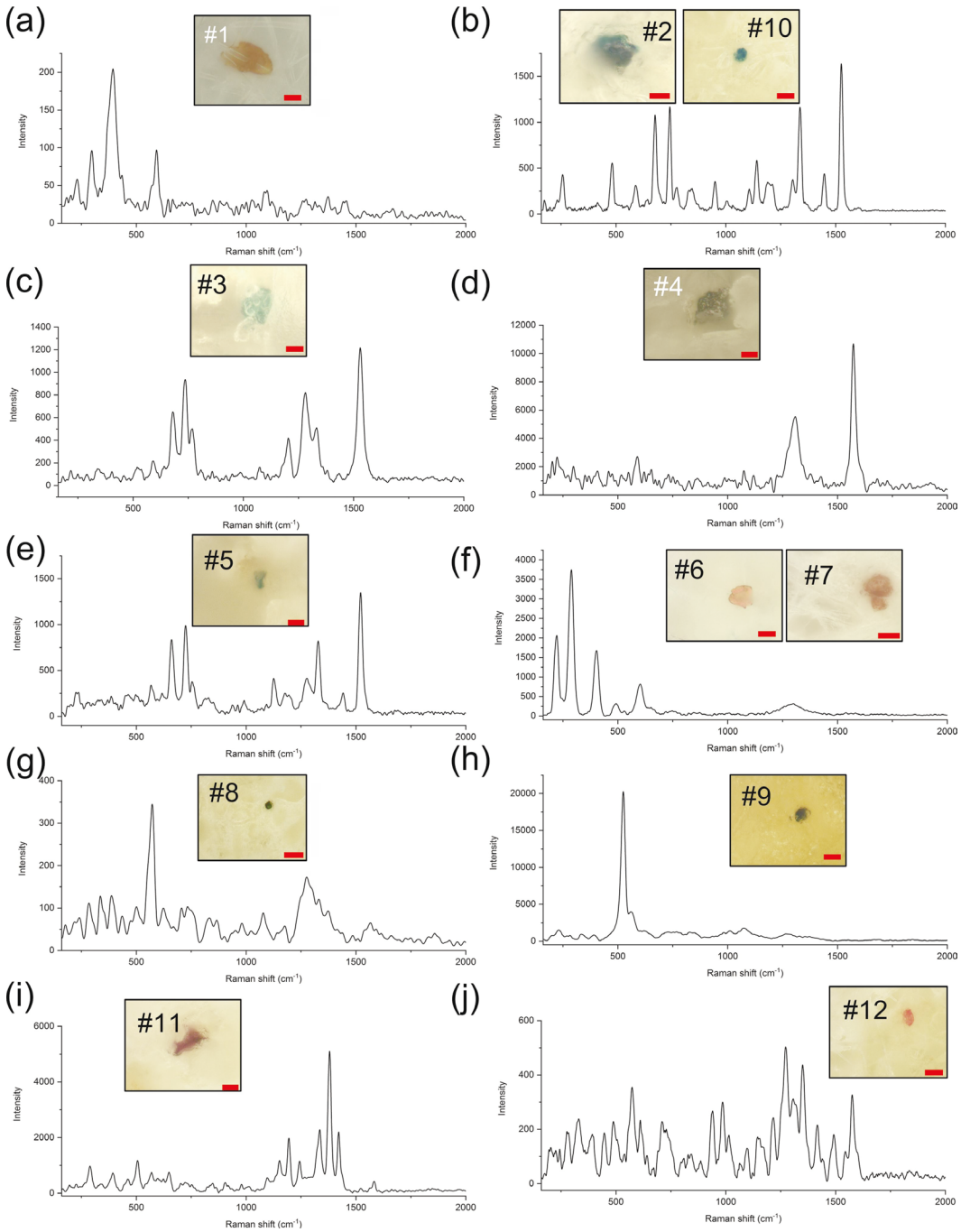


Figure 11.1.

Microphotographs and Raman spectra of the microplastics found in human placenta: (a) Particle #1 (scale bar 5 μm); (b) Particles #2 and #10 (scale bar 5 μm for #2 and 10 μm for #10); (c) Particle #3 (scale bar 5 μm); (d) Particle #4 (scale bar 5 μm); (e) Particle #5 (scale bar 5 μm); (f) Particles #6 and #7 (scale bar 10 μm for #6 and 5 μm for #7); (g) Particle #8 (scale bar 10 μm); (h) Particle #9 (scale bar 10 μm); (i) Particle #11 (scale bar 5 μm), and (j) Particle #12 (scale bar 10 μm).

Source: Ragusa, A., Svelato, A., Santacroce, C., et al. Plasticenta: First evidence of microplastics in human placenta. Environment International 146, 106274 (2021). https://doi.org/10.1016/j.envint.2020.106274 License: CC BY-NC-ND 4.0.

MECHANISMS OF PLACENTAL TOXICITY: CELLULAR AND MOLECULAR EFFECTS

Experimental data on animal models show that exposure to microplastics can disrupt placental function and retard embryonic development. Specifically, chronic exposure to microplastic particles caused **fetal growth restriction and functional placental impairments** associated with the activation of stress signaling pathways, endoplasmic reticulum stress, and apoptosis of placental cells (Fig. 11.2).[315] These processes led to a decrease in the efficiency of placental tissue in supporting fetal development and may reflect potential risks for normal pregnancy given similar exposure in other species (Fig. 11.3, 11.4, 11.5).[316]

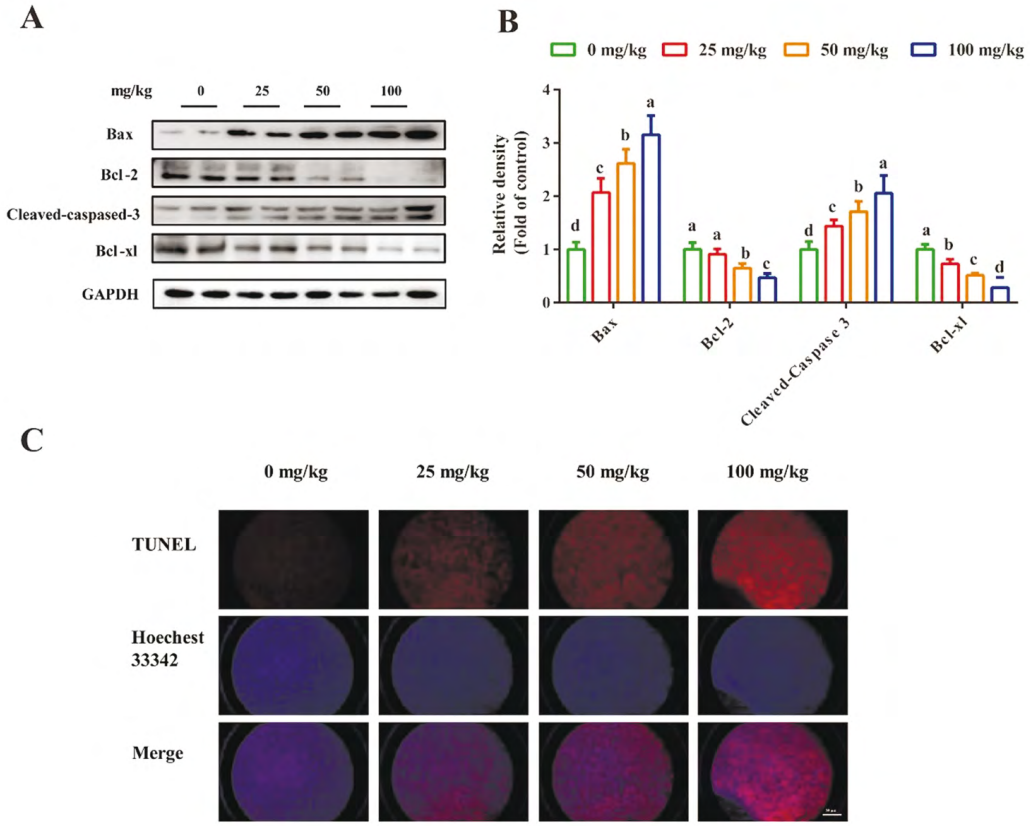


Figure 11.2.

Effects of microplastics on placental apoptosis in pregnant mice. (A-B) Western blot was used to analyze the expression of apoptosis related proteins in placental tissue. n = 3 (C) Representative picture of TUNEL staining of placental tissue (Labyrinth zone) with 50 μm scale bar. Letters a-d indicate significant differences ($P < .05$).

Source: Bai, J., Wang, Y., Deng, S., et al. Microplastics caused embryonic growth retardation and placental dysfunction in pregnant mice by activating GRP78/IRE1α/JNK axis induced apoptosis and endoplasmic reticulum stress. Part Fibre Toxicol 21, 36 (2024). https://doi.org/10.1186/s12989-024-00595-5 License: CC BY-NC-ND 4.0.

[315] *Bai, J., Wang, Y., Deng, S., et al. Microplastics caused embryonic growth retardation and placental dysfunction in pregnant mice by activating GRP78/IRE1α/JNK axis induced apoptosis and endoplasmic reticulum stress. Part Fibre Toxicol 21, 36 (2024). https://doi.org/10.1186/s12989-024-00595-5*

[316] *Ragusa, A., Cristiano, L., Di Vinci, P., et al. Artificial plasticenta: how polystyrene nanoplastics affect in-vitro cultured human trophoblast cells. Front. Cell Dev. Biol. 13, 1539600 (2025). https://doi.org/10.3389/fcell.2025.1539600*

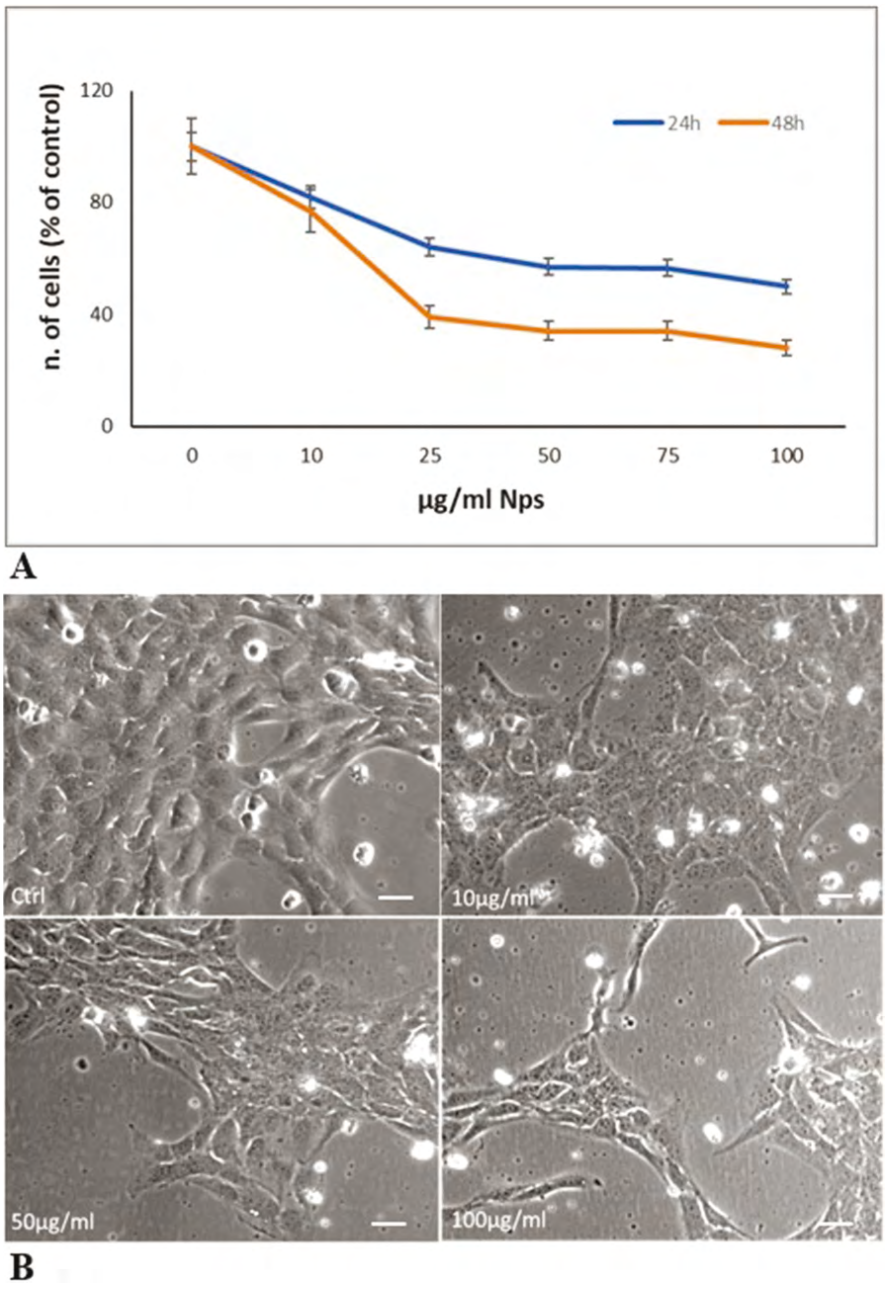


Figure 11.3.

PS-Ps treatment of trophoblast cells (A) Cell viability evaluation with CCK-8 assay. Cells were treated with increasing concentrations of 0.05 µm NPs (10, 25, 50 75, and 100 µg/mL). Reported data represent the mean ± SD of three experiments. The differences in values compared to the control are significant ($p < 0.05$ at 10 µg/mL; $P < 0.01$ at 25–100 µg/mL) (B) Representative contrast phase images of cells after 24 h of treatment. Cells treated for 24 h with 10, 50, and 100 µg/mL microspheres decrease their proliferation and undergo morphological changes becoming flatter and longer and losing contact with each other. Bar, 25 µm.

Source: Ragusa, A., Cristiano, L., Di Vinci, P., et al. Artificial plasticenta: how polystyrene nanoplastics affect in-vitro cultured human trophoblast cells. Front. Cell Dev. Biol. 13, 1539600 (2025). https://doi.org/10.3389/fcell.2025.1539600

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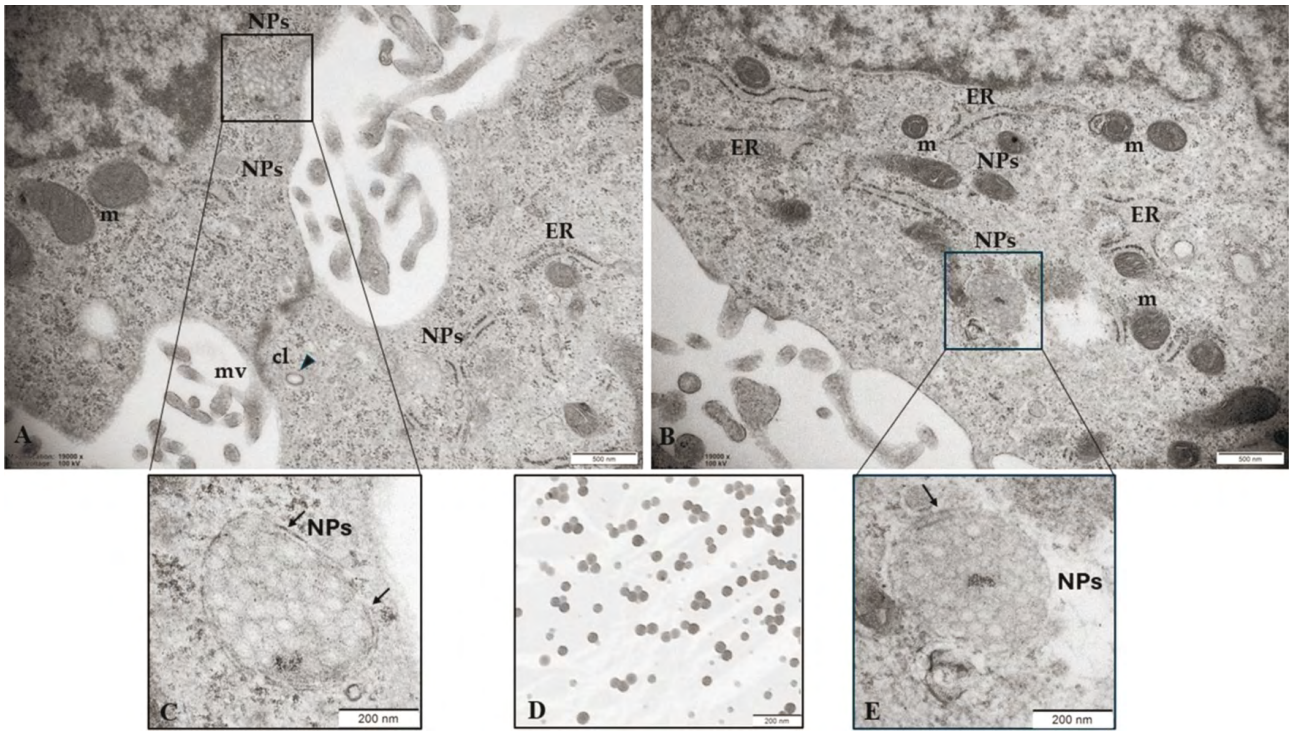


Figure 11.4.

NPs in human trophoblast cells (A, B) NPs (50 µg/mL) were able to enter the cells, probably via endocytosis and (C, E) accumulate in structures constituted by a double membrane (arrows) resembling lysosomes (D) PS-NPs in cell medium (100 µg/mL) observed by TEM were used as a positive control, confirming the commercial particle size of 0.05 µm. Bar, 200nm. CL, clathrin vesicles; m, mitochondria; mv, microvilli. Bar, 500 µm (A, B). Bar = 200 µm (C–E).

Source: Ragusa, A., Cristiano, L., Di Vinci, P., et al. Artificial plasticenta: how polystyrene nanoplastics affect in-vitro cultured human trophoblast cells. Front. Cell Dev. Biol. 13, 1539600 (2025). https://doi.org/10.3389/fcell.2025.1539600 License: CC BY 4.0.

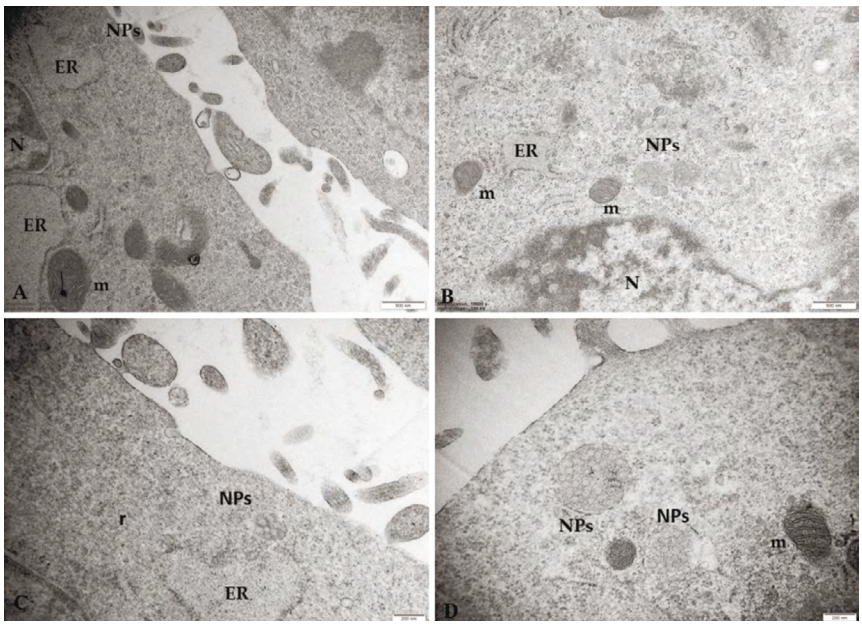


Figure 11.5.

Microphotographs of trophoblast cells treated with 50 µg/mL NPs (A–C) NPs aggregates induce morphological changes in ER and mitochondria. The ER appears dilated, with sparse ribosomes on the outer surface. Degranulation and disaggregation of polyribosomes (r) free in the cytoplasm also occur (D) Microphotographs of placental cells treated with 100 µg/mL NPs in which swollen electrondense mitochondria (m) appear in the cytoplasm. Note that the size of aggregates is enhanced by increasing NP concentration. Bar, 500 nm (A, B); Bar, 200 nm (C, D). N, nucleus.

Source: Ragusa, A., Cristiano, L., Di Vinci, P., et al. Artificial plasticenta: how polystyrene nanoplastics affect in-vitro cultured human trophoblast cells. Front. Cell Dev. Biol. 13, 1539600 (2025). https://doi.org/10.3389/fcell.2025.1539600 License: CC BY 4.0.

An experimental study testing the toxicity of different PS-NPs concentration on human placental cells demonstrated:

> *"PS-NPs also induced size- and surface charge-dependent expression profiles of genes involved in various and interrelated toxicity pathways. In particular, PS-NPs increased intracellular reactive oxygen species in human placental cells, which can induce DNA damage and lead to cell cycle arrest in G1 or G2 phase, inflammation and apoptosis."*[317]

The results showed that upon entering placental cells, plastic nanoparticles increase the amount of reactive oxygen species (ROS) inside the cell—this causes oxidative stress capable of damaging DNA. Such stress, in turn, disrupts the normal cell cycle (delays cell division), induces inflammatory reactions, and triggers apoptosis—programmed cell death. These molecular disturbances affect the expression of genes involved in several interconnected toxicity pathways, indicating a complex impact of PS-NPs on placental cell functions.[317]

Thus, polystyrene nanoparticles can disrupt placental function at the cellular level, which reduces the functional competence of placental cells and potentially impairs their ability to support normal embryonic development.

Accumulated data from human and animal studies show that the presence of microplastics in the placenta is associated with adverse perinatal outcomes.[318] Microplastic particles were detected in the placentas of women who gave birth to children with intrauterine growth restriction. Higher concentration of MNP particles was associated with reduced birth weight, body length, and head circumference of newborns.[319] Such changes indicate a possible disruption of the functional properties of the placenta, which may reflect impaired transport of oxygen and nutrients to the fetus—a key condition for normal growth and development.

[317] *Shen, F., Li, D., Guo, J., et al. Mechanistic toxicity assessment of differently sized and charged polystyrene nanoparticles based on human placental cells. Water Research 223, 118960 (2022). https://doi.org/10.1016/j.watres.2022.118960*

[318] *Cheng, Y., Li, Y., Zhang, Y., et al. Gestational exposure to micro- and nanoplastics leads to poor pregnancy outcomes by impairing placental trophoblast syncytialization. Environmental Pollution 381, 126520 (2025). https://doi.org/10.1016/j.envpol.2025.126520*

[319] *Amereh, F., Amjadi, N., Mohseni-Bandpei, A., et al. Placental plastics in young women from general population correlate with reduced foetal growth in IUGR pregnancies. Environmental Pollution 314, 120174 (2022). https://doi.org/10.1016/j.envpol.2022.120174*

PRETERM BIRTH AND THE GLOBAL BURDEN OF PERINATAL PATHOLOGY

A study conducted by scientists from the USA showed that concentrations of microplastics and nanoplastics in the placenta were significantly higher in women who delivered prematurely compared to those who delivered at term.

Polyvinyl chloride (PVC), polyethylene terephthalate (PET), polyurethane (PU), and polycarbonate (PC) were significantly elevated in PTB, and PET, PU, and PC inversely correlated with gestational age and birth weight. These observations allowed to hypothesize that MNP accumulation may influence physiological processes, including inflammatory reactions and placental functions involved in the regulation of pregnancy duration.[320]

Antonio Ragusa metaphorically described such findings as follows:

"It is like having a cyborg baby: no longer composed only of human cells, but a mixture of biological and inorganic entities."[321]

Prematurity remains one of the most serious global public health problems. In 2020, preterm birth rates ranged from 4 to 16% of total births across different countries. 13.4 million children were born prematurely—more than one in ten. Complications of preterm birth are the leading cause of death among children under 5 years of age. Many surviving children face lifelong disabilities, including visual and hearing impairments and learning difficulties. [322]

IMPACT ON EMBRYONIC TISSUES AND ORGANOGENESIS

The detection of plastic particles not only in the placenta[323] (Fig. 11.6) but also in embryonic tissues is of serious concern.

[320] *Jochum, M., Garcia, M., Hammerquist, A., et al. Elevated Micro- and Nanoplastics Detected in Preterm Human Placentae. Preprint (2025). https://doi.org/10.21203/rs.3.rs-5903715/v1*

[321] *ThePrint. 'It's like having a cyborg baby' — in a first, microplastics found in human placenta. (2021). https://theprint.in/science/its-like-having-a-cyborg-baby-in-a-first-microplastics-found-in-human-placenta/586956/ (Accessed 20 March 2026)*

[322] *World Health Organization (WHO). Preterm Birth. https://www.who.int/news-room/fact-sheets/detail/preterm-birth (Accessed 20 March 2026)*

[323] *Medley, E. A., Spratlen, M. J., Yan, B., et al. A Systematic Review of the Placental Translocation of Micro- and Nanoplastics. Curr Envir Health Rpt 10, 99–111 (2023). https://doi.org/10.1007/s40572-023-00391-x*

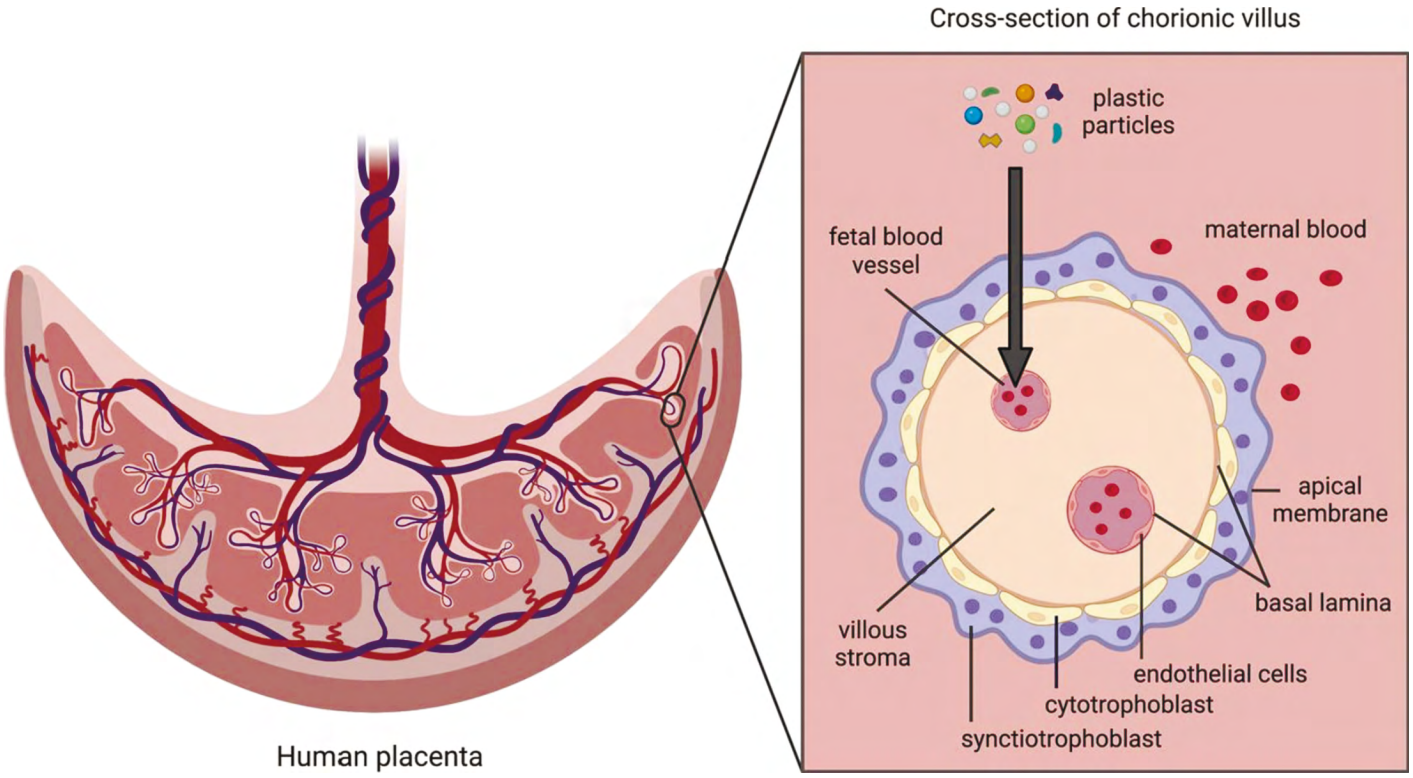


Figure 11.6.

Structure of placental barrier and translocation route for plastic particles. Schematic diagram of human placenta with a labeled cross section of a chorionic villus and arrow showing route for plastic particles to be transported from maternal to fetal circulation.

Source: Medley, E. A., Spratlen, M. J., Yan, B., et al. A Systematic Review of the Placental Translocation of Micro- and Nanoplastics. Curr Envir Health Rpt 10, 99–111 (2023). https://doi.org/10.1007/s40572-023-00391-x

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Nanoplastics may pose a particular risk during the early stages of embryogenesis, when major organs and systems are forming. Research on cellular and animal models and cellular systems has shown that exposure to nanoplastics disrupts the migration, proliferation, and survival of embryonic stem cells, including neural crest cells, leading to defects in organogenesis and an increased frequency of congenital anomalies (Fig. 11.7, 11.8, 11.9).[324]

[324] *Wang, M., Rücklin, M., Poelmann, R. E., et al. Nanoplastics causes extensive congenital malformations during embryonic development by passively targeting neural crest cells. Environment International 173, 107865 (2023). https://doi.org/10.1016/j.envint.2023.107865*

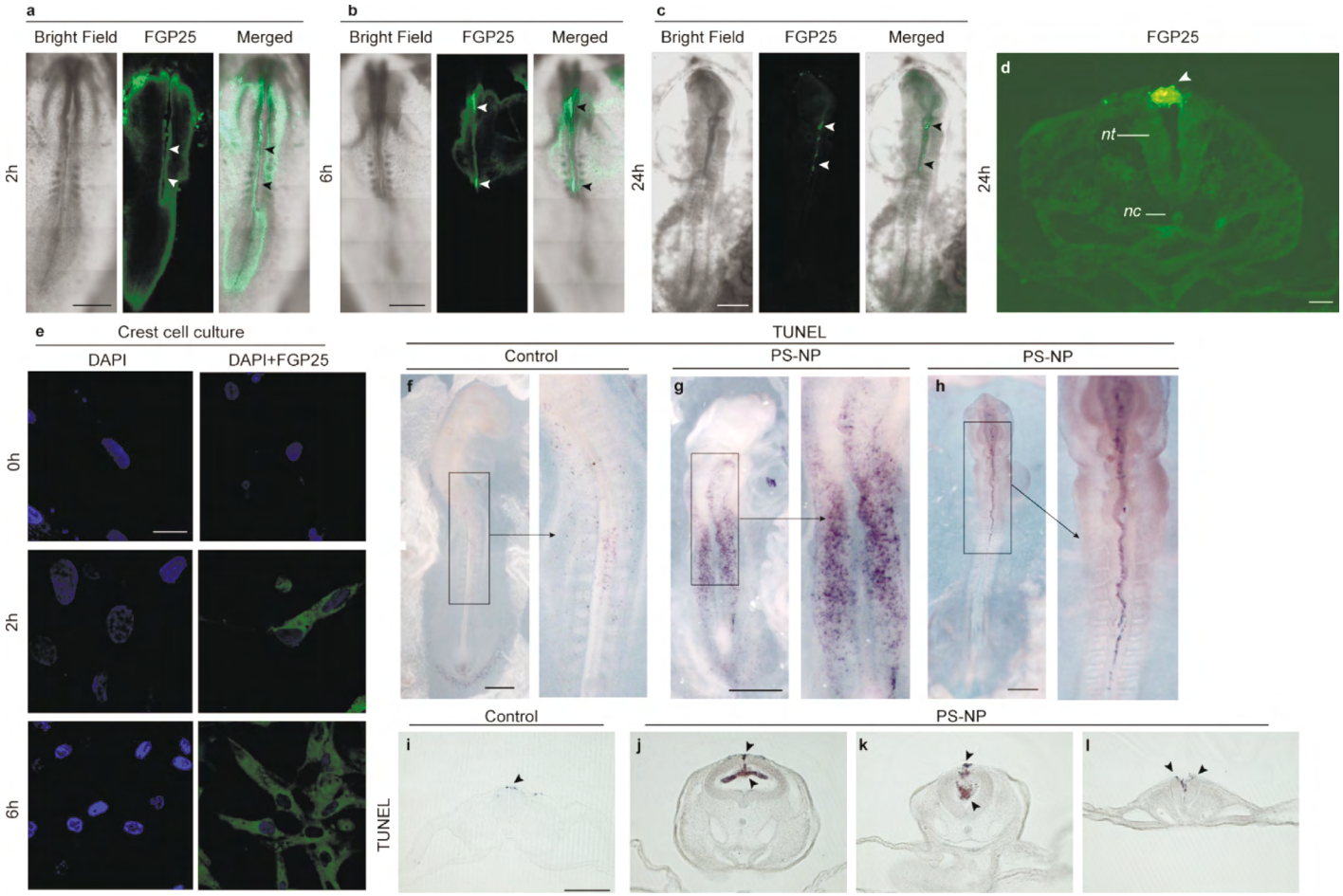


Figure 11.7.

Tissue distribution, selective localization, and induction of cell death in neural crest cells, by PS-NPs. a-c, chick embryos exposed to fluorescent PS-NPs (FGP, green florescence) examined at 2, 6 and 24 h, respectively. n = 2 for all different time series. **a**, 2 hpe PS-NPs treated chick embryos, stage 8. **b**, 6 hpe PS-NPs treated chick embryos, stage 9. **c**, 24 hpe PS-NPs treated chick embryos, stage 13. **d**, transverse cryosection (25 µm). **e**, neural crest cells stain with DAPI (blue florescence) exposed in vitro to fluorescent PS-NPs (green florescence) and examined at 0, 2, and 6 h, respectively. TUNEL staining of control (j) and PS-NP-treated (g and h) chick embryos. n = 2 for control and n = 3 for PS-NP treated group. (**f**), stage 14, (**g**), stage 12, (**h**), stage 12 (N.B. all embryos are 2 d incubation, but g and h show a developmental delay caused by PS-NP treatment). In g and h, note the extensive cell death, and dead cells in the dorsal midline. **i-l**, TUNEL staining on vibratome sections. n = 2 for both control and PS-NPs-treated chick embryos. **i**, control, stage 14. **j-l**, PS-NPs-treated, stage 12. PS-NPs treatment resulted in clumps of dead cells in the neural folds and in the neural tube lumen (black arrowhead).

Source: Wang, M., Rücklin, M., Poelmann, R. E., et al. Nanoplastics causes extensive congenital malformations during embryonic development by passively targeting neural crest cells. Environment International 173, 107865 (2023). https://doi.org/10.1016/j.envint.2023.107865 License: CC BY 4.0.

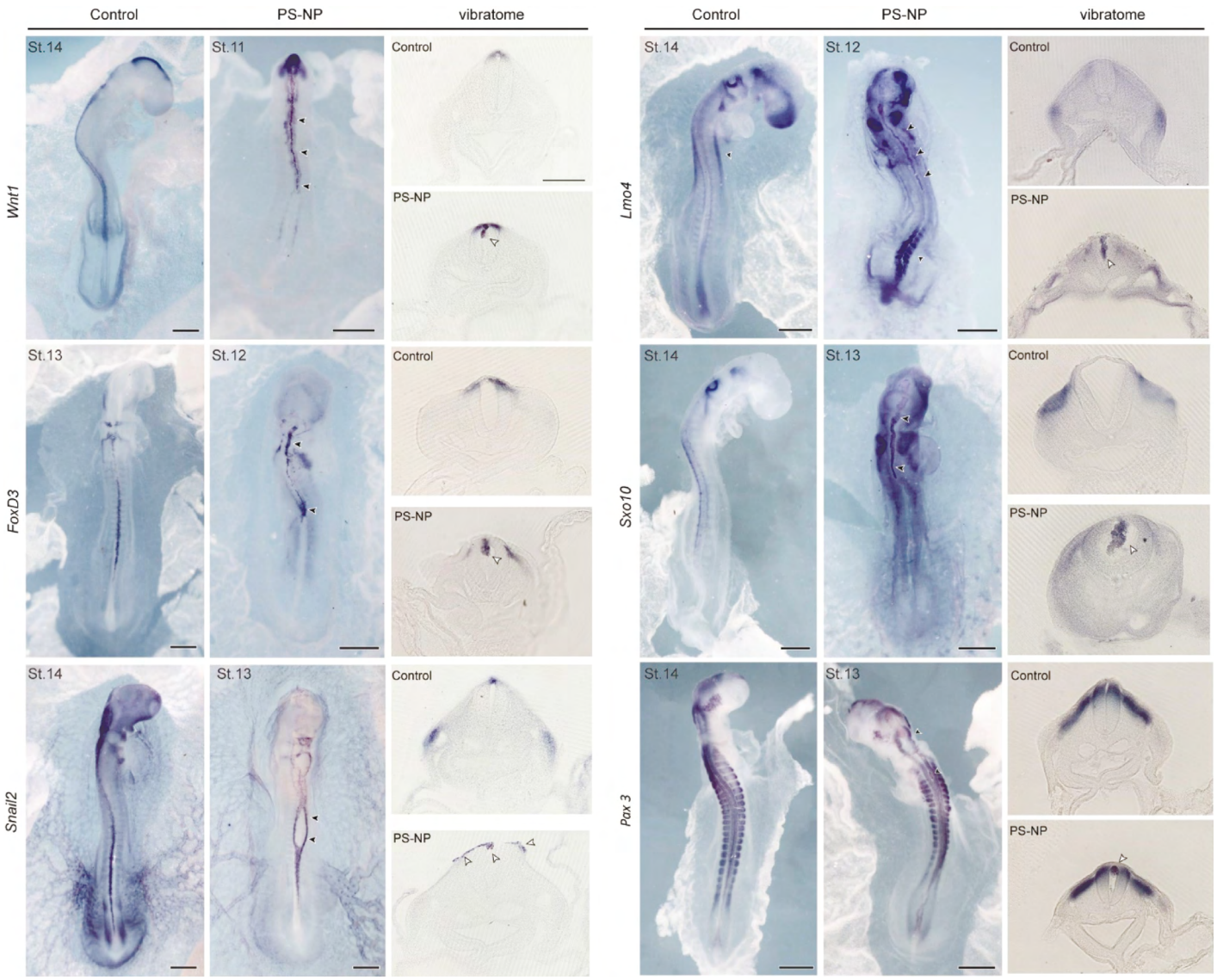


Figure 11.8.

Cells disrupted by 25 nm PS-NPs express neural crest markers. All embryos were exposed at stage 8 to 50 µL plain PS-NPs (5 mg/mL). n = 3 for both control and PS-NP treated group of all genes. Note, all embryos are two days incubation; the treated embryos all showed developmental delay relative to the controls. Cellular debris of neural crest cells (arrowheads). Key: PS-NP, embryos treated with polystyrene nanoparticles, 25 nm, 5 mg/mL. Arrowheads in the treated embryos indicate putative neural crest cells. In WNT1 and FOXD3 in situs, the cells are clumped. In SNAI2 in situs, there are putative neural crest cells at the margins of the neural tube defect. In all cases except the SNAI2 in situ shown, there are putative neural crest cell clumps inside the neural tube lumen. Scale bars, 500 µm in wholemount embryos, 200 µm in vibratome transverse sections.

Source: Wang, M., Rücklin, M., Poelmann, R. E., et al. Nanoplastics causes extensive congenital malformations during embryonic development by passively targeting neural crest cells. Environment International 173, 107865 (2023). https://doi.org/10.1016/j.envint.2023.107865 License: CC BY 4.0.

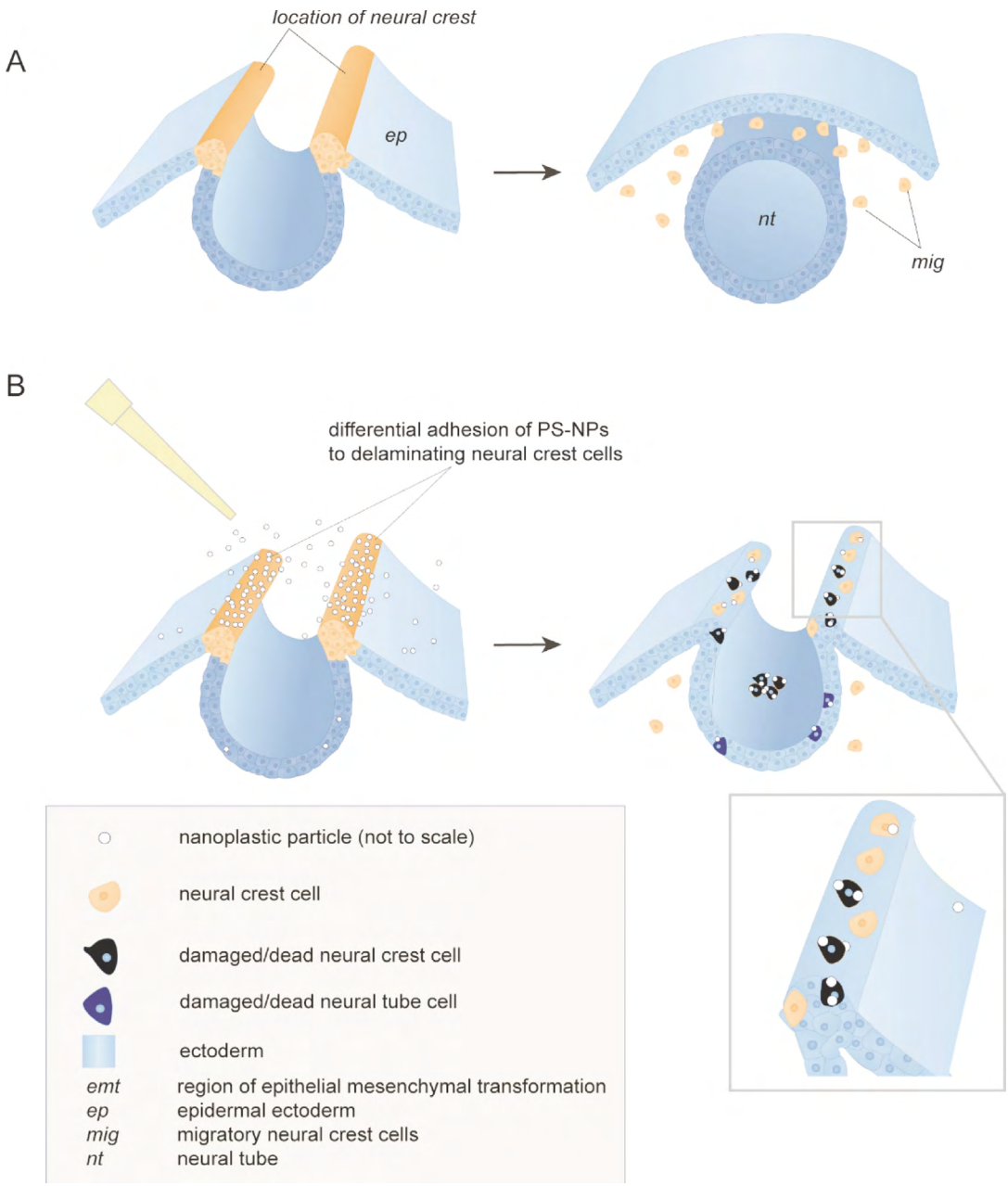


Figure 11.9.

Hypothesis of PS-NP-induced developmental toxicity, based on disruption of neural crest development. A, normal development of the neural tube and neural crest. B, development of the neural tube and crest after exposure of the embryo to 25 nm PS-NPs. Authors hypothesize that the nanoparticles adhere selectively to the neural crest cells undergoing epithelial-mesenchymal transition, and induce cell death in at least a subpopulation of those neural crest cells. This reduces the number of normal neural crest cells, disrupts their migration and interferes with the normal closure of the neural tube. Authors suggest that these events lead to neural tube defects, microphthalmia, craniofacial and cardiac malformations. Authors noted that we saw some evidence of very limited cell death in the neural tube induced by PS-NPs (as shown in this figure).

Source: Wang, M., Rücklin, M., Poelmann, R. E., et al. Nanoplastics causes extensive congenital malformations during embryonic development by passively targeting neural crest cells. Environment International 173, 107865 (2023). https://doi.org/10.1016/j.envint.2023.107865 License: CC BY 4.0.

Experiments on *Danio rerio* (zebrafish) embryos showed that exposure to polystyrene nanoparticles causes pronounced toxicity in early development. Morphological anomalies, decreased survival, and growth retardation were observed, as well as impaired development of primary motor neurons (Fig. 11.10, 11.11). These effects were accompanied by the activation of oxidative stress, altered expression of genes associated with neurogenesis, and increased levels of apoptosis, which collectively led to reduced motor activity in the embryos.[325]

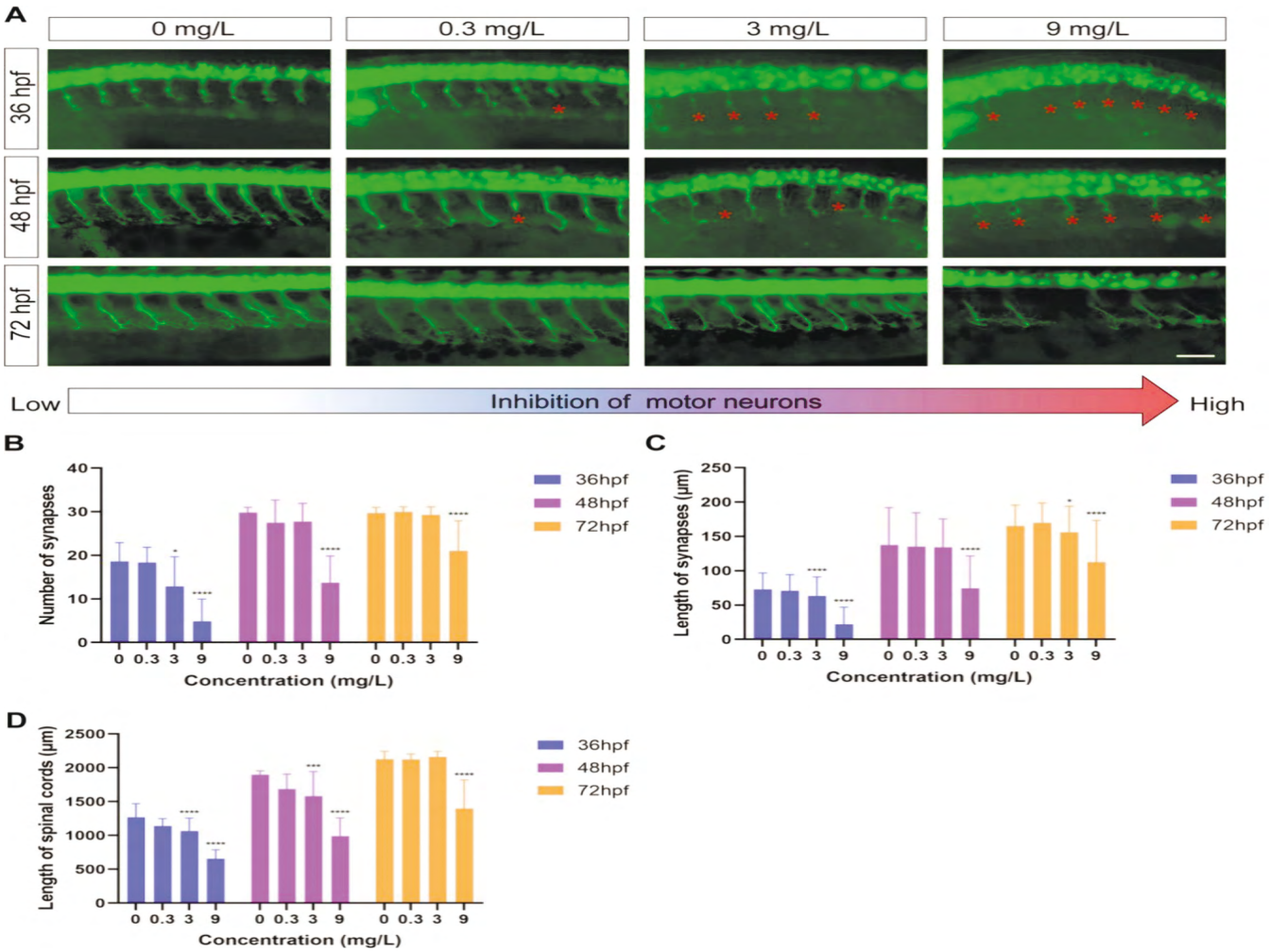


Figure 11.10.

Effects of NPs exposure on neural growth in zebrafish. (A) Neuronal morphology of zebrafish embryos/larvae at 36, 48, and 72 hpf of NP exposure. Scale bar, 200 um. (B) Quantification of the number of motor neurons in zebrafish embryos/larvae at 36, 48, and 72 hpf of NP exposure (n = 20 per group). (C) Quantification of the axon lengths of motor neurons in NP-treated zebrafish embryos/larvae at 36, 48, and 72 hpf (n = 20 per group). (D) Quantification of the spinal cord lengths of motor neurons in NPs-treated zebrafish embryos/larvae at 36, 48, and 72 hpf (n = 20 per group). Data are presented as the means ± SD *P < 0.05, ***P < 0.001, and ****P < 0.0001. Red asterisks represent axons.

Source: Cao, X., Xie, W., Feng, M., et al. Nanoplastic Exposure Mediates Neurodevelopmental Toxicity by Activating the Oxidative Stress Response in Zebrafish (Danio rerio). ACS Omega 9, 16508–16518 (2024). https://doi.org/10.1021/acsomega.4c00231 License: CC BY-NC-ND 4.0.

[325] *Cao, X., Xie, W., Feng, M., et al. Nanoplastic Exposure Mediates Neurodevelopmental Toxicity by Activating the Oxidative Stress Response in Zebrafish (Danio rerio). ACS Omega 9, 16508–16518 (2024). https://doi.org/10.1021/acsomega.4c00231*

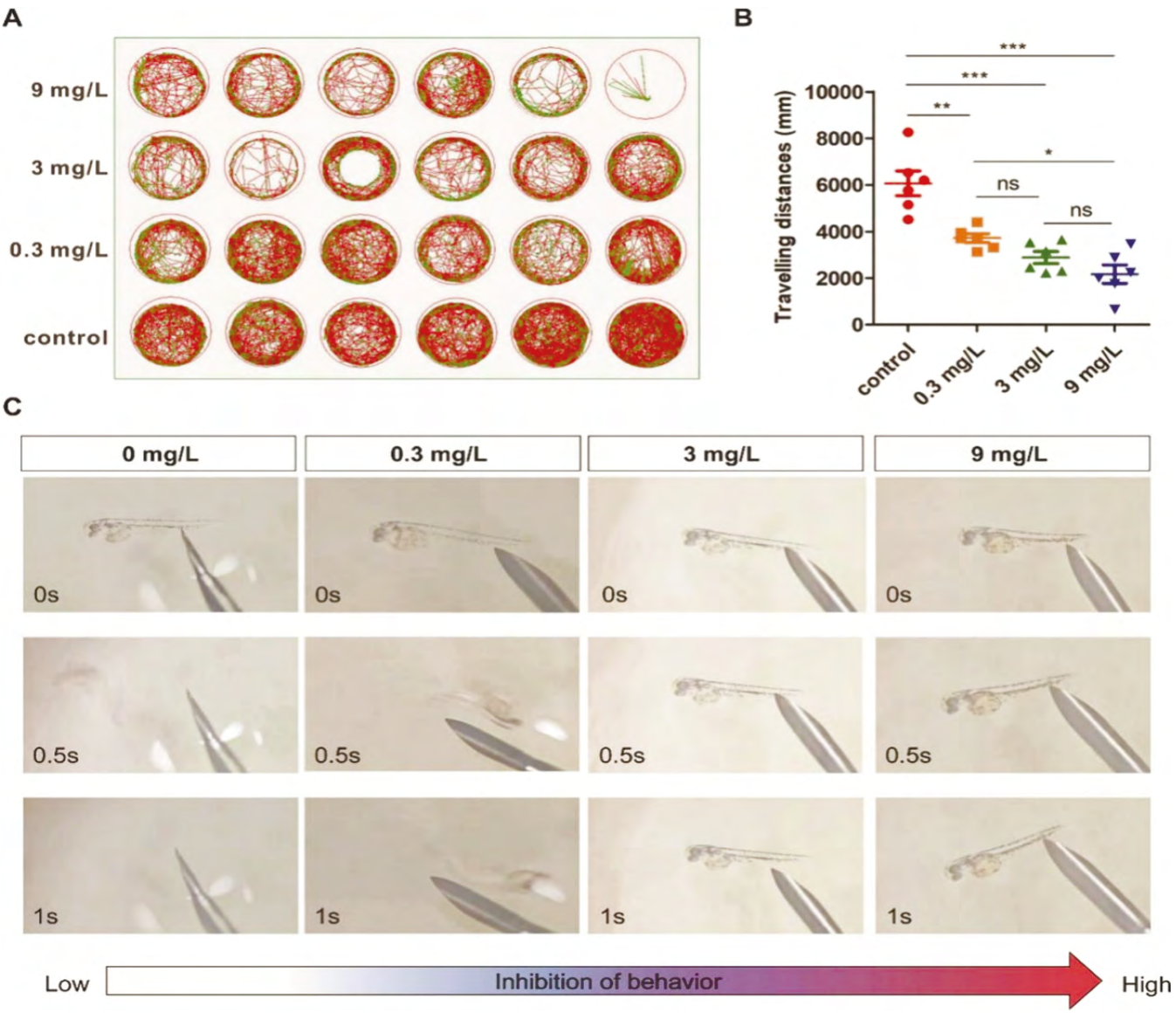


Figure 11.11.

NP exposure inhibits locomotor behavior in zebrafish larvae. (A) Moving track graphs of zebrafish larvae at 72 hpf of NPs exposure at different concentrations (n = 6 for each group). (B) Traveling distances of zebrafish larvae (n = 6 for each group). (C) Stereotypic escape response to different concentrations of NPs treatment (n = 6 per group). Data are presented as the mean ± SD *P < 0.05, *P < 0.01, ***P < 0.001 and ****P < 0.0001 compared with the control.

Source: Cao, X., Xie, W., Feng, M., et al. Nanoplastic Exposure Mediates Neurodevelopmental Toxicity by Activating the Oxidative Stress Response in Zebrafish (Danio rerio). ACS Omega 9, 16508–16518 (2024). https://doi.org/10.1021/acsomega.4c00231. License: CC BY-NC-ND 4.0.

Experimental data show that polystyrene nanoparticles disrupt the differentiation of human embryonic stem cells. Under the influence of nanoparticles, the efficiency of stem cell conversion into fully functional cardiomyocytes decreased. As a result, smaller cardiac organoids with weakened contractile activity were formed. The observed effects are accompanied by the development of mitochondrial oxidative stress, disruption of intracellular defense mechanisms (autophagy), and activation of stress-signaling pathways, which collectively indicate the potential ability of nanoplastic particles to interfere with the early stages of cardiac tissue formation.[326]

These findings are also supported by *in vivo* study in fish embryos, where exposure to nanoplastics has been associated with reduced heart rate and impaired blood flow.[326]

RISING PREVALENCE OF CONGENITAL ANOMALIES

As already indicated, experimental and histological evidence demonstrates that MNPs can traverse the placental barrier and enter fetal circulation, thereby extending their potential impact beyond placental function to direct effects on embryonic tissues.[320,323,327]

The observed association between micro- and nanoplastic (MNP) accumulation in the placenta and adverse pregnancy outcomes, including preterm birth and impaired fetal growth, indicates that these particles are not biologically inert but potentially actively interact with key regulatory systems of gestation.

This transition is of particular importance when considering the temporal sensitivity of embryogenesis. While placental dysfunction predominantly affects later stages of pregnancy—manifesting as growth restriction or prematurity—direct exposure of the embryo during early development introduces a fundamentally different level of risk. During organogenesis, tightly regulated processes such as cell proliferation, migration, differentiation, and programmed cell death determine the structural integrity of emerging organs. Disruption of these processes, even transiently, may result in permanent morphological abnormalities.

Experimental studies provide biologically plausible mechanisms for such effects. Nanoplastic particles have been shown to interfere with embryonic stem cell function, impair neural crest cell survival and migration, and disrupt the differentiation of cardiac and neural tissues. These effects are consistently associated with mitochondrial dysfunction, oxidative stress, and activation of apoptotic pathways—mechanisms that are known to play a central role in teratogenesis across a wide spectrum of congenital anomalies.[324,325]

[326] *Li, J., Weng, H., Liu, S., et al. Embryonic exposure of polystyrene nanoplastics affects cardiac development. Science of The Total Environment 906, 167406 (2024). https://doi.org/10.1016/j.scitotenv.2023.167406*

[327] *Ragusa, A., Cristiano, L., Di Vinci, P., et al. Artificial plasticenta: how polystyrene nanoplastics affect in-vitro cultured human trophoblast cells. Front. Cell Dev. Biol. 13, 1539600 (2025). https://doi.org/10.3389/fcell.2025.1539600*

In this context, the distinction between placental pathology and congenital malformations becomes less discrete and more continuous. Both may represent different manifestations of a shared upstream disturbance affecting the intrauterine environment. While placental dysfunction reflects impaired support of the fetus, direct interference with embryonic developmental programs may lead to structural anomalies. Importantly, these processes are not mutually exclusive and may occur concurrently within the same pregnancy.

Given the global and continuous exposure of human populations to micro- and nanoplastics, and their demonstrated ability to reach fetal tissues, the question arises whether these particles may contribute—directly or indirectly—to the background risk of congenital anomalies.

LIMITATIONS OF GLOBAL DATA AND THE NEED FOR HIGH-QUALITY REGISTRY SURVEILLANCE

The interpretation of epidemiological data on congenital anomalies requires considerable caution, as registry-based estimates are inherently influenced by multiple methodological and clinical factors. Reported prevalence does not solely reflect true embryonic incidence, but is shaped by differences in case ascertainment, diagnostic capabilities, and inclusion criteria across regions and over time. Advances in prenatal imaging and genetic testing have led to increased detection of anomalies—particularly milder or previously unrecognized forms—while at the same time enabling earlier termination of affected pregnancies. The inclusion or exclusion of terminations of pregnancy for fetal anomaly (TOPFA), stillbirths, and postnatally diagnosed cases varies between surveillance systems and can substantially alter observed trends.

Additional sources of bias include changes in classification systems, evolving diagnostic thresholds, and improvements in follow-up and reporting completeness. Rare anomalies are especially susceptible to statistical fluctuation, making short-term trends difficult to interpret. Furthermore, aggregated datasets may obscure heterogeneity between specific defect types, some of which are highly sensitive to diagnostic practices, while others are more robust indicators of underlying biological risk. As a result, apparent increases or decreases in prevalence cannot be directly equated with changes in true incidence.

Within this context, high-quality registries that apply standardized definitions and include comprehensive case ascertainment—particularly those incorporating TOPFA—provide a more reliable basis for identifying long-term patterns. However, even in such systems, observed trends should be regarded as signals requiring careful interpretation rather than definitive evidence of changing risk.[328,329]

[328] *European Commission Joint Research Centre (JRC). EUROCAT Data Collection: Guidelines for Data Registration. https://eu-rd-platform.jrc.ec.europa.eu/eurocat/data-collection/guidelines-for-data-registration_en (Accessed 20 March 2026).*

[329] *Morris, J. K., Springett, A. L., Greenlees, R., et al. Trends in congenital anomalies in Europe from 1980 to 2012. PLOS ONE 13, e0194986 (2018). https://doi.org/10.1371/journal.pone.0194986*

EUROCAT DATA AND EMERGING SIGNALS IN NON-GENETIC CONGENITAL ANOMALIES

Within the framework of these methodological considerations, data from the European Surveillance of Congenital Anomalies (EUROCAT) provide one of the most robust available sources for evaluating long-term trends. [KH1] EUROCAT registries apply standardized case definitions, include live births, stillbirths, and terminations of pregnancy for fetal anomaly (TOPFA), and maintain consistent surveillance across multiple populations.[330] This allows for a more comprehensive approximation of true embryofetal occurrence compared to datasets restricted to live births alone.

Analysis of EUROCAT registry data (2005–2023), restricted to non-genetic cases and including TOPFA, reveals modest but consistent upward trends in the prevalence of several severe congenital anomalies. These include central nervous system defects (arhinencephaly/holoprosencephaly and agenesis of the corpus callosum), congenital pulmonary airway malformations, and congenital cataract (Fig. 11.12 – 11.16).[330,331]



Figure 11.12.

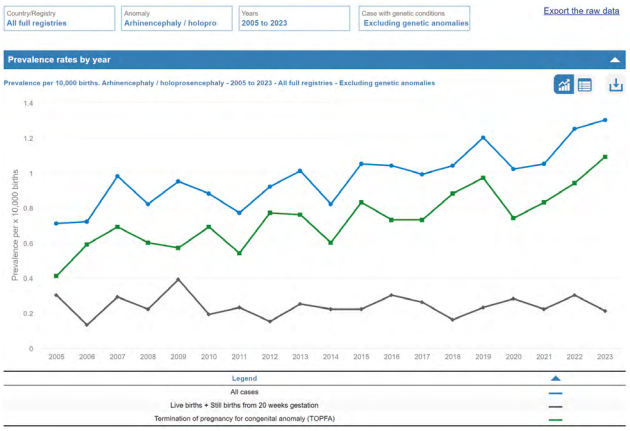
Pictures showing fronto-nasal encephalocoele and facial dysmorphism.

Source: Obanife, O. H., Adekanye, A. G., Ekpo, R. G., et al. Management of alobar holoprosencephaly associated with fronto-nasal encephalocoele and type I (closed-lips) schizencephaly at the university of calabar teaching Hospital: A case report and literature review. Interdisciplinary Neurosurgery 27, 101410 (2022). https://doi.org/10.1016/j.inat.2021.101410. License: CC BY-NC-ND 4.0.

Figure 11.13.

Arhinencephaly / holoprosencephaly prevalence per 10,000 births (EUROCAT, 2005–2023). The overall prevalence increased from approximately 0.4–0.5 per 10,000 births in the mid-2000s to around 1.0–1.2 per 10,000 births in recent years. Genetic anomalies are excluded, only data from full registries included. Given the rarity of the condition and potential improvements in prenatal detection and classification, the observed trend should be interpreted cautiously as a hypothesis-generating signal rather than evidence of a true increase in incidence.

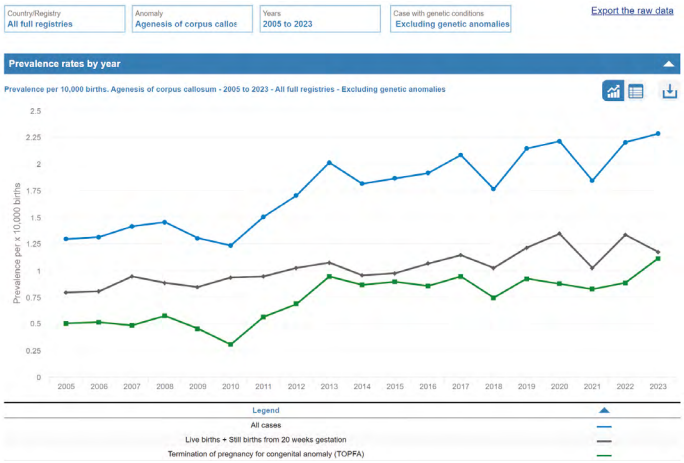
Source: European Commission Joint Research Centre (JRC). EUROCAT Data: Prevalence charts and tables. https://eu-rd-platform.jrc.ec.europa.eu/eurocat/eurocat-data/prevalence_en (Accessed 20 March 2026). License: CC BY 4.0.



[330] European Commission Joint Research Centre (JRC). EUROCAT Data: Prevalence charts and tables. https://eu-rd-platform.jrc.ec.europa.eu/eurocat/eurocat-data/prevalence_en (Accessed 20 March 2026).

[331] Obanife, O. H., Adekanye, A. G., Ekpo, R. G., et al. Management of alobar holoprosencephaly associated with fronto-nasal encephalocoele and type I (closed-lips) schizencephaly at the university of calabar teaching Hospital: A case report and literature review. Interdisciplinary Neurosurgery 27, 101410 (2022). https://doi.org/10.1016/j.inat.2021.101410

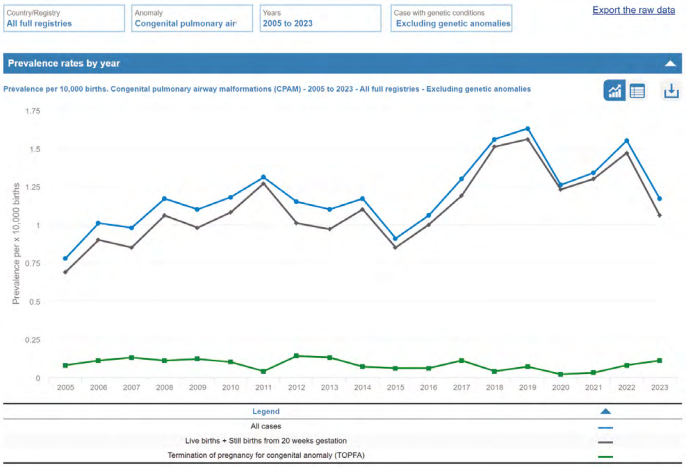
Figure 11.14.



Agenesis of the corpus callosum prevalence per 10,000 births (EUROCAT, 2005–2023). The total prevalence increased from approximately 1.2–1.4 per 10,000 births in the early study period to over 2.0–2.3 per 10,000 births in recent years. Genetic anomalies are excluded, only data from full registries included. Although improvements in neuroimaging and diagnostic awareness may contribute to this increase, the persistence and magnitude of the trend support its consideration as a potential epidemiological signal.

Source: European Commission Joint Research Centre (JRC). EUROCAT Data: Prevalence charts and tables. https://eu-rd-platform.jrc.ec.europa.eu/eurocat/eurocat-data/prevalence_en (Accessed 20 March 2026). License: CC BY 4.0.

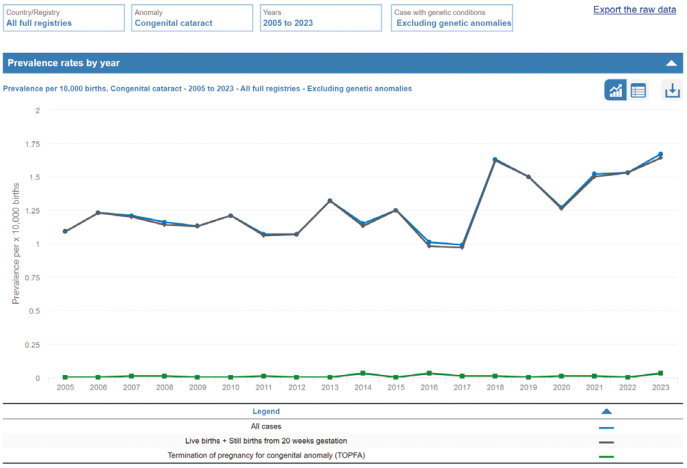
Figure 11.15.



Congenital Pulmonary Airway Malformations prevalence per 10,000 births (EUROCAT, 2005–2023). The prevalence increased from approximately 0.5–0.7 per 10,000 births in the mid-2000s to peaks of around 1.2–1.5 per 10,000 births during the late 2010s, followed by some fluctuation in recent years. Genetic anomalies are excluded, only data from full registries included. Interpretation is limited by the high sensitivity of CPAM diagnosis to advances in prenatal imaging and evolving classification criteria, which may substantially influence observed prevalence.

Source: European Commission Joint Research Centre (JRC). EUROCAT Data: Prevalence charts and tables. https://eu-rd-platform.jrc.ec.europa.eu/eurocat/eurocat-data/prevalence_en (Accessed 20 March 2026). License: CC BY 4.0.

Figure 11.16.



Congenital Cataract prevalence per 10,000 births (EUROCAT, 2005–2023). The prevalence increased from approximately 1.0–1.2 per 10,000 births in the early period to around 1.5–1.8 per 10,000 births after 2016, with a sustained higher level thereafter. Genetic anomalies are excluded, only data from full registries included. As congenital cataract is less dependent on prenatal imaging compared to other anomalies, the observed increase may be less affected by detection bias. Nevertheless, changes in screening practices and diagnostic awareness should be considered when interpreting this trend.

Source: European Commission Joint Research Centre (JRC). EUROCAT Data: Prevalence charts and tables. https://eu-rd-platform.jrc.ec.europa.eu/eurocat/eurocat-data/prevalence_en (Accessed 20 March 2026). License: CC BY 4.0.

The observed increases are apparent in non-genetic categories. Importantly, the inclusion of TOPFA reduces bias related to prenatal detection and termination, allowing a closer approximation of true embryofetal occurrence. Notably, the upward trend appears even more pronounced when all registries are included, rather than full registries alone. This may reflect differences in case ascertainment and reporting practices, as registries with varying levels of completeness and diagnostic intensity can introduce additional variability, including enhanced detection of milder or borderline cases and differences in follow-up and data capture.

These findings should be interpreted with appropriate caution. The anomalies examined are relatively rare and therefore inherently susceptible to statistical fluctuation, while several—particularly those affecting the central nervous system and lungs—are strongly influenced by advances in prenatal imaging and evolving diagnostic classification. As a result, the observed trends cannot be taken as evidence of a definitive increase in true incidence.

Nonetheless, the presence of concordant upward patterns across multiple, biologically distinct organ systems within a high-quality surveillance framework supports their consideration as hypothesis-generating signals. Such consistency warrants increased attention, particularly in the context of long-term population health monitoring. It also highlights the need for continued refinement and harmonization of congenital anomaly surveillance systems, including standardized case definitions, comprehensive inclusion of cases across pregnancy outcomes, and stable longitudinal data collection.

Experimental evidence indicates that MNPs as pervasive environmental agents are capable of crossing the placental barrier, interacting with embryonic tissues, and interfering with fundamental processes of organogenesis, including cell migration, differentiation, and survival. Within this context, the observed epidemiological patterns may be viewed as compatible with a potential contribution of chronic environmental exposures to the background risk of severe congenital anomalies. However, further interdisciplinary research and improved surveillance strategies are required to clarify these relationships and to distinguish true changes in incidence from artefacts of detection and reporting.

POSTNATAL EXPOSURE TO MNPs

Following birth, the exposure of the child's organism to micro- and nanoplastics continues. Plastic bottles, baby food packaging, and toys are additional sources of MNPs. Experimental data show that shaking a plastic bottle with warm water can release up to 16 million microplastic particles per liter.[332] Exposure to hot water and sterilization intensify this process. As a result, infants may ingest millions of microplastic particles and trillions of nanoplastic particles daily.

Breast milk, a key source of nutrients and immune protection for infants, has also been identified as a potential route of microplastic exposure. In one study, microplastics were detected in approximately 40% of analyzed breast milk samples. (Fig. 11.17).[333]

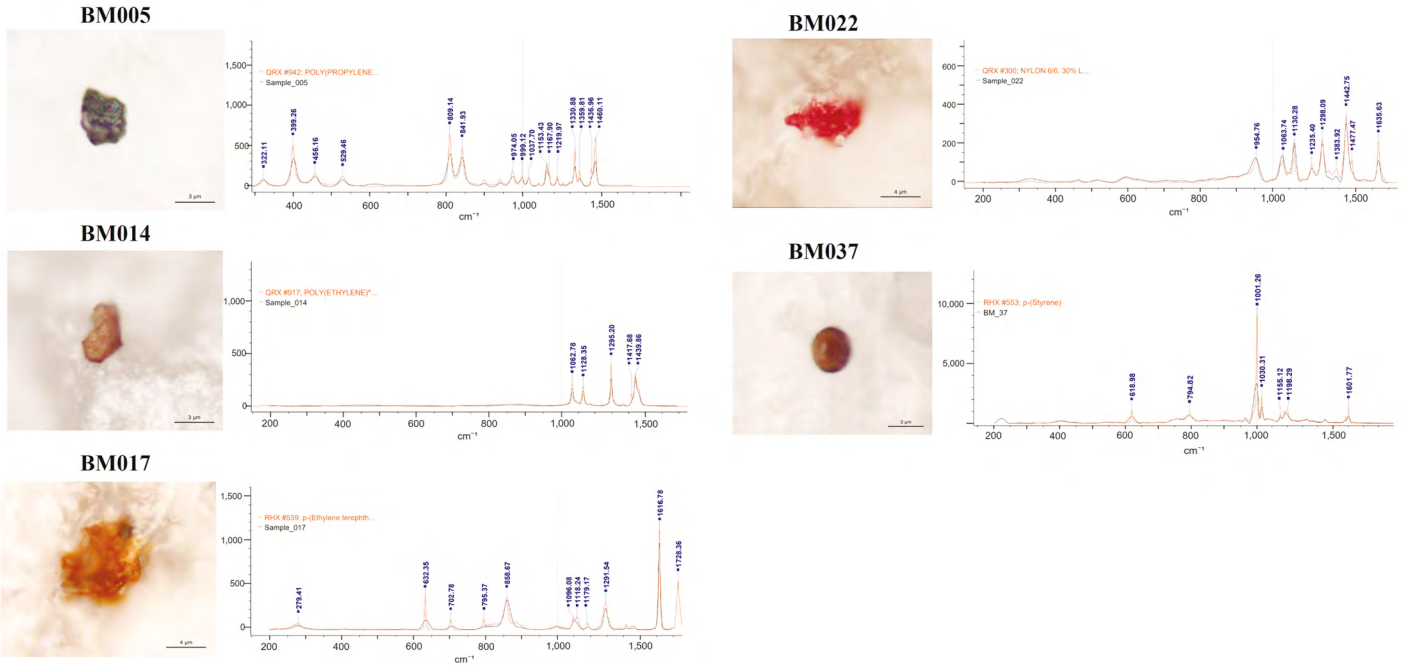


Figure 11.17.

Microphotographs and Raman spectra (wave numbers, cm^{-1}) of some selected MPs found in the analyzed breastmilk samples. PE: polyethylene; p-(Ethylene terephthalate); PVC: polyvinyl chloride; PP: polypropylene; NL: Nylon; PS: polystyrene.

Source: Saraluck, A., Techarang, T., Bunyapipat, P., et al. Detection of Microplastics in Human Breast Milk and Its Association with Changes in Human Milk Bacterial Microbiota. Journal of Clinical Medicine 13, 4029 (2024). https://doi.org/10.3390/jcm13144029 License: CC BY 4.0.

[332] *Li, D., Shi, Y., Yang, L., et al. Microplastic release from the degradation of polypropylene feeding bottles during infant formula preparation. Nat Food 1, 746–754 (2020). https://doi.org/10.1038/s43016-020-00171-y*

[333] *Saraluck, A., Techarang, T., Bunyapipat, P., et al. Detection of Microplastics in Human Breast Milk and Its Association with Changes in Human Milk Bacterial Microbiota. Journal of Clinical Medicine 13, 4029 (2024). https://doi.org/10.3390/jcm13144029*

The heightened vulnerability of the pediatric organism to micro- and nanoplastic exposure is conditioned by several physiological features of early age. Due to a more intensive metabolism and higher respiratory rate, children consume more air, water, and food per unit of body mass compared to adults. Given the same level of environmental pollution, this leads to a relatively higher dose of plastic particles entering the child's body.

An additional risk factor is the functional and structural immaturity of the body's protective barriers. At an early age, barrier systems are still in the formation stage, which may facilitate penetration of MNP particles into tissues and promote their accumulation. Specifically, in children under two years of age, the ethmoid bone of the skull is not fully formed; wider anatomical openings may potentially facilitate the transport of particles via the olfactory nerves into brain structures.[334] Thus, in conditions of the ubiquitous presence of micro- and nanoplastics, children are considered the most vulnerable population group.

ENDOCRINE, IMMUNE, AND EPIGENETIC EFFECTS

Experimental and epidemiological data indicate that micro- and nanoplastics, as well as plastic-associated chemical additives (including bisphenols and phthalates), are capable of interfering with the development of the reproductive and immune systems,[335] potentially increasing the risk of fertility disorders later in life. MNP exposure in early life stages is associated with changes in immune regulation,[336] raising the risk of developing allergic[337] and autoimmune conditions. Furthermore, a number of studies point to the ability of MNPs to induce epigenetic changes[338] (alterations in gene activity regulation), which may increase predisposition to non-communicable diseases in adulthood, including obesity, arterial hypertension, diabetes mellitus, and metabolic syndrome.

Simultaneously, MNPs can modify immune responses, including through the intensification of oxidative stress and pro-inflammatory signals. These effects are hypothesized to contribute to the disruption of immune tolerance and the persistence of chronic inflammation, potentially increasing susceptibility to immune dysregulation, including autoimmune processes.

[334] *Amato-Lourenço, L. F., Dantas, K. C., Júnior, G. R., et al. Microplastics in the Olfactory Bulb of the Human Brain. JAMA Netw Open 7, e2440018 (2024). https://doi.org/10.1001/jamanetworkopen.2024.40018*
[335] *Ullah, S., Ahmad, S., Guo, X., et al. A review of the endocrine disrupting effects of micro and nano plastic and their associated chemicals in mammals. Front. Endocrinol. 13, 1084236 (2023). https://doi.org/10.3389/fendo.2022.1084236*
[336] *Tan, Y.-T., Wang, W.-W., Xu, S.-Z., et al. Unraveling the impact of microplastics on autoimmune diseases: hidden dangers and environmental triggers. Environ Geochem Health 48, 95 (2026). https://doi.org/10.1007/s10653-026-02976-5*
[337] *Hu, J., Zheng, Y. & Gao, Y. The Impact of Microplastics on Allergy: Current Status and Future Research Directions. Int Arch Allergy Immunol (2025). https://doi.org/10.1159/000547873*
[338] *Subramanian, D., Ponnusamy Manogaran, G. & Dharmadurai, D. A systematic review on the impact of micro-nanoplastics on human health: Potential modulation of epigenetic mechanisms and identification of biomarkers. Chemosphere 363, 142986 (2024). https://doi.org/10.1016/j.chemosphere.2024.142986*

VULNERABILITY OF THE DEVELOPING BRAIN

The first years of a child's life are a critical period for the development of the central nervous system. During this stage, neurogenesis, neuronal differentiation, the formation of synaptic connections, and the establishment of functional neural networks determining cognitive, emotional, and behavioral characteristics in subsequent life periods occur actively. Due to high plasticity and the immaturity of regulatory mechanisms, any adverse external influences during this period can have long-term consequences.

Studies indicate that plastic nanoparticles are capable of interfering with processes of neuronal development.Their exposure is associated with the development of inflammatory reactions, intensification of oxidative stress, damage to immature neurons, and disruption of their differentiation and maturation.[339] Potential functional consequences include impairments in memory, attention, and learning, as well as alterations in emotional regulation manifesting as increased anxiety and behavioral instability.

A number of scientific publications point to the possible link between micro- and nanoplastic exposure and the risk of developing autism spectrum disorders (ASD).[340] Statistical data from the USA reflect a substantial increase in the number of diagnosed cases: while in 2000 autism spectrum disorders were identified in one in 150 children, by 2020 it was one in 36, and in 2022—one in 31 children.[341,342] Collectively, this corresponds to an approximately fivefold increase over the indicated period (Fig. 11.18).[343] Given the rapid increase in autism prevalence over recent decades, exposure to micro- and nanoplastics should be considered a potential factor requiring systematic and detailed scientific study.

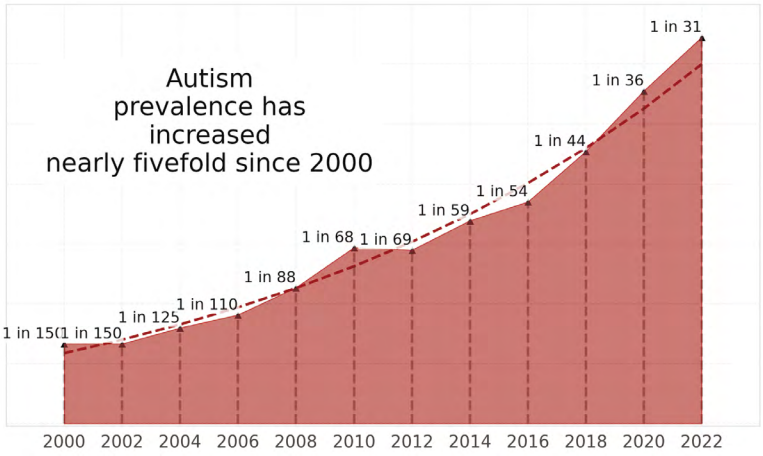


Figure 11.18.

Trends in autism prevalence (2000–2022) based on data from the CDC's Autism and Developmental Disabilities Monitoring (ADDM) Network.

Data source: U.S. Centers for Disease Control and Prevention (CDC). Autism Data and Research. https://www.cdc.gov/autism/data-research/index.html (Accessed 20 March 2026).

[339] *Eisen, A., Pioro, E. P., Goutman, S. A., et al. Nanoplastics and Neurodegeneration in ALS. Brain Sciences 14, 471 (2024). https://doi.org/10.3390/brainsci14050471*

[340] *Zaheer, J., Kim, H., Ko, I. O., et al. Pre/post-natal exposure to microplastic as a potential risk factor for autism spectrum disorder. Environment International 161, 107121 (2022). https://doi.org/10.1016/j.envint.2022.107121*

[341] *National Institute of Mental Health (NIMH). Autism Spectrum Disorder (ASD) Statistics. https://www.nimh.nih.gov/health/statistics/autism-spectrum-disorder-asd (Accessed 20 March 2026)*

[342] *Shaw, K. A., Williams, S., Patrick, M. E., et al. Prevalence and Early Identification of Autism Spectrum Disorder Among Children Aged 4 and 8 Years — Autism and Developmental Disabilities Monitoring Network, 16 Sites, United States, 2022. MMWR Surveill Summ 74, 1–22 (2025). https://doi.org/10.15585/mmwr.ss7402a1*

[343] *U.S. Centers for Disease Control and Prevention (CDC). Autism Data and Research. https://www.cdc.gov/autism/data-research/index.html (Accessed 20 March 2026).*

SUDDEN INFANT DEATH SYNDROME

The number of registered cases of sudden infant death syndrome (SIDS) in the USA rose between 2020 and 2022 by approximately 12%.[344] According to epidemiological data, SIDS remains one of the leading causes of mortality[345] among children under one year of age.[346] A diagnosis of SIDS is established in cases where an outwardly healthy infant dies suddenly, and the cause of death remains undetermined even after a comprehensive clinical and post-mortem examination.

Growing evidence therefore suggests that nanoplastics may be a potentially relevant, yet understudied, factor contributing to pathways implicated in the pathogenesis of SIDS.

Aside from mechanisms that may lead to sudden death in adults, it cannot be excluded that in children processes contributing to the pathogenesis of SIDS may be initiated already during prenatal development. As discussed earlier in this chapter, minute plastic particles are capable of penetrating the placenta—the barrier intended to protect the developing fetus. Once in the embryonic circulation, nanoplastics can interfere with the formation of vital systems: the nervous, cardiovascular, respiratory, and immune systems. Thus, they begin to disrupt the child's organism during its formation stage.

As discussed in this report, nanoplastics have been shown to induce inflammatory responses in various tissues under experimental conditions, including both in vitro and in vivo models.[347] Biological plausibility suggests that, if exposure occurs during prenatal development, such inflammatory and oxidative stress-related processes may influence organogenesis. In particular, the developing nervous and cardiorespiratory systems may be especially susceptible to disruption, including potential effects on the formation and maturation of neuronal networks involved in the autonomic regulation of respiration and cardiac rhythm.

Exposure to nanoplastics is associated with the activation of immune cells and inflammatory processes, increased oxidative stress, and mitochondrial damage. Such mechanisms include the activation of microglia and astrocytes, the release of pro-inflammatory cytokines, and a reduction in antioxidant defense; under experimental conditions, these processes have been associated with impaired neurogenesis and synapse formation, as well as alterations in neurochemistry.[339] These processes are particularly critical in the initial stages of brain development, as they may affect structures involved in the automatic regulation of respiration and cardiac activity in newborns. Disruptions in the formation of these organs and systems may weaken the infant's ability to control breathing and heart rhythm, particurarly during sleep.

[344] *Wolf, E. R., Rivara, F. P., Sen, A., et al. Sudden Unexpected Infant Death and Disparities in Infant Mortality in the US, 1999-2022. JAMA Pediatr 179, 344–346 (2025). https://doi.org/10.1001/jamapediatrics.2024.6200*
[345] *U.S. Centers for Disease Control and Prevention (CDC). Infant Mortality. https://www.cdc.gov/maternal-infant-health/infant-mortality/index.html (Accessed 20 March 2026)*
[346] *U.S. Centers for Disease Control and Prevention (CDC). Sudden Infant Death Syndrome (SIDS): Data and Research. https://www.cdc.gov/sudden-infant-death/data-research/data/ (Accessed 20 March 2026)*
[347] *Thapliyal, C., Negi, S., Nagarkoti, S., et al. Mechanistic insight into potential toxic effects of microplastics and nanoplastics on human health. Discov Appl Sci 7, 645 (2025). https://doi.org/10.1007/s42452-025-07214-8*

Although direct evidence for humans is currently limited and requires further confirmation, the aggregate of experimental data suggests that prenatal nanoplastic exposure may form a chronic inflammatory and oxidative background capable of adversely affecting the maturation of the nervous, respiratory, and cardiovascular systems, thereby increasing the child's vulnerability to disruptions of vital regulatory functions.

PATHOPHYSIOLOGY OF SIDS. HYPOPLASIA OF THE ARCUATE NUCLEUS OF THE MEDULLA OBLONGATA

In an extensive pathomorphological series of 217 cases of sudden and unexplained infant death, anomalies of the nuclei and structures of the brainstem and cerebellum associated with vital functions were identified in 84% of cases.[348] One of the most frequently described structural anomalies in infants who died from SIDS is hypoplasia of the arcuate nucleus of the medulla oblongata, i.e., the congenital underdevelopment of this structure.[349,350] This structure plays a key role in central chemosensitivity to CO_2/H^+ and cardiorespiratory regulation during sleep.

Morphometric studies have shown that arcuate nucleus hypoplasia occurs in approximately 30–56% of SIDS cases, with the nucleus being nearly completely underdeveloped in some infants, showing reduced volume and neuronal density.[349,351] In individual cases, severe hypoplasia or aplasia of the nucleus has been described with the preservation of other brainstem sections, suggesting a possible substrate for the impairment of autonomic regulation of respiration and cardiac activity during sleep.[350] Beyond complete hypoplasia, a number of studies have identified morphometric signs of neuronal immaturity in the arcuate nucleus — reduced cell size and maturation delay indices, sometimes combined with neuronal deficit alongside marked hypoplasia.[352] These data emphasize that infants with SIDS exhibit impaired formation and maturation of key brainstem cardiorespiratory centers, which may reduce the efficiency of automatic regulation of respiration and heart rhythm during sleep.

[348] *Matturri, L., Mauri, M., Ferrero, M. E., et al. Unexpected Perinatal Loss versus Sids-a Common Neuropathologic Entity. TONEUJ 02, 45–50 (2008). https://doi.org/10.2174/1874205X00802010045*

[349] *Matturri, L., Biondo, B., Mercurio, P., et al. Severe hypoplasia of medullary arcuate nucleus: quantitative analysis in sudden infant death syndrome. Acta Neuropathol 99, 371–375 (2000). https://doi.org/10.1007/s004010051138*

[350] *Giustina, E. D., Salviato, T., Fabbiani, L., et al. Severe Hypoplasia/Aplasia of the Medullary Arcuate Nucleus in a 4-Month-Old Patient with Sudden Infant Death Syndrome. Journal of Pediatric Neurology (2024). https://doi.org/10.1055/s-0044-1787856*

[351] *Matturri, L., Biondo, B., Suárez-Mier, M., et al. Brain stem lesions in the sudden infant death syndrome: variability in the hypoplasia of the arcuate nucleus. Acta Neuropathol 104, 12–20 (2002). https://doi.org/10.1007/s00401-001-0511-7*

[352] *Biondo, B., Lavezzi, A., Tosi, D., et al. Delayed neuronal maturation of the medullary arcuate nucleus in sudden infant death syndrome. Acta Neuropathol 106, 545–551 (2003). https://doi.org/10.1007/s00401-003-0757-3*

ABNORMALITIES TO THE SEROTONERGIC (5-HT) SYSTEM

In a number of studies, abnormalities to the serotonergic (5-HT) system of the medulla oblongata — the part of the brainstem regulating breathing, heartbeat, blood pressure, and responses to changes in oxygen and CO_2 levels — has been described in SIDS.[353] These alterations include changes in the quantity and distribution of serotonin and its receptors in brainstem nuclei; anomalies in structures involved in respiratory automaticity; as well as evidence that in some infants with SIDS, these deviations may have been congenital, that is present even before birth. Such changes are considered a manifestation of an anomaly of the brainstem serotonergic system, one of the leading hypotheses explaining increased vulnerability in SIDS.[353]

The identified anomalies are believed to arise in the perinatal period or even before birth, and may weaken the infant's automatic protective responses to hypoxia and hypercapnia during sleep.[353] These data support the hypothesis that intrauterine nanoplastic exposure may increase fetal vulnerability to impairments in the automatic regulation of respiration and heart rhythm, potentiall analogous to the morphofunctional anomalies identified in infants with SIDS.

POTENTIAL ROLE OF MITOCHONDRIA DYSFUNCTION IN DEVELOPMENT OF SIDS

Mitochondria represent one of the primary cellular targets of nanoplastic-induced toxicity.[354]

Their dysfunction lead to cellular energy deficit, potentially affecting the structural and functional development of organs and contributing to persistent disturbances in respiration, cardiac rhythm, and neural regulation of vital functions. Mitochondria are also highly responsive to cellular stress: under conditions such as hypoxia or inflammation, cells may increase mitochondrial DNA (mtDNA) copy number as a compensatory mechanism to maintain energy homeostasis.

Studies have shown that in children who died from SIDS, an elevated mtDNA copy number was detected in the cerebral cortex compared to the control group. This finding is interpreted as a compensatory response to chronic cellular stress and hypoxia, reflecting an increased demand for energy production.[355] These observations highlight a potential role of mitochondrial dysfunction and altered energy metabolism in the pathophysiology of SIDS and suggest increased mtDNA copy number as a candidate biomarker for further investigation.

[353] *Kinney, H. C. & Haynes, R. L. The Serotonin Brainstem Hypothesis for the Sudden Infant Death Syndrome. J Neuropathol Exp Neurol 78, 765–779 (2019).* https://doi.org/10.1093/jnen/nlz062
[354] *Liu, H., Li, H., Yao, X., et al. Environmental nanoplastics induce mitochondrial dysfunction: A review of cellular mechanisms and associated diseases. Environmental Pollution 382, 126695 (2025).* https://doi.org/10.1016/j.envpol.2025.126695
[355] *Danusso, R., Alfonsi, G., Ferrero, S., et al. Mitochondrial DNA content: a new potential biomarker for Sudden Infant Death Syndrome. Pediatr Res 92, 1282–1287 (2022).* https://doi.org/10.1038/s41390-021-01901-z

Taken together, experimental, morphological, and epidemiological data suggest that micro- and nanoplastics should be considered not merely as ubiquitous environmental contaminants, but as potentially significant biological factors capable of interfering with critical stages of early human development.

Their effects may occur at multiple levels — from placental dysfunction and impaired organogenesis to mitochondrial stress, neuroinflammation, and disturbances of autonomic regulation in the postnatal period.

Of particular concern is the vulnerability of developing systems — including the nervous, cardiovascular, immune, and endocrine systems — during periods of structural and functional immaturity.

In the context of the global accumulation of plastic particles in the biosphere, the impact of micro- and nanoplastics on intrauterine and early postnatal development extends beyond toxicology and acquires broader interdisciplinary relevance, encompassing neonatology, pediatrics, neurobiology, and public health.

These findings provide a scientific basis for further fundamental research, the development of early biomarkers of damage, and the refinement of prevention strategies aimed at protecting the most vulnerable population — children.

PART 12.

MICRO- AND NANOPLASTICS AS A POTENTIAL RISK FACTOR **FOR CARDIOVASCULAR DISEASES**

Cardiovascular diseases (CVDs) remain the leading cause of mortality worldwide.[356] Despite significant achievements in diagnostics, prevention, and therapy, the global burden of these pathologies continues to grow. Between 2025 and 2050 the prevalence of CVD is projected to increase by 90.0%, crude mortality by 73.4%, and the total DALY (disability-adjusted life years) metric by 54.7%. By 2050, the number of deaths from cardiovascular diseases will reach 35.6 million, compared to 20.5 million in 2025.[357]

These trends may suggest that, in addition to established determinants, other environmental factors could contribute to cardiovascular risk, although their role remains insufficiently characterized.

As outlined in this report, micro- and nanoplastics (MNPs), which may carry surface electrostatic charge, represent a potential emerging factor that could interfere with cardiovascular system function. Experimental evidence indicates that their effects may involve multiple interconnected mechanisms, including mitochondrial dysfunction, vascular injury, and prothrombotic alterations.

MITOCHONDRIAL DYSFUNCTION AS A MECHANISM OF CARDIAC DAMAGE

One of the key mechanisms of the adverse impact of MNPs is the impairment of mitochondrial function (Fig. 12.1).[358,359,360,361,362] Mitochondria provide cells with energy and play a central role in maintaining their physiological activity. Mitochondrial dysfunction leads to decreased ATP synthesis and disruption of tissue energy balance.

[356] *Naghavi, M., Ong, K. L., Aali, A., et al. Global burden of 288 causes of death and life expectancy decomposition in 204 countries and territories and 811 subnational locations, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. The Lancet 403, 2100–2132 (2024). https://doi.org/10.1016/S0140-6736(24)00367-2*

[357] *Chong, B., Jayabaskaran, J., Jauhari, S. M., et al. Global burden of cardiovascular diseases: projections from 2025 to 2050. European Journal of Preventive Cardiology 32, 1001–1015 (2025). https://doi.org/10.1093/eurjpc/zwae281*

[358] *Sun, Z., Wen, Y., Zhang, F., et al. Exposure to nanoplastics induces mitochondrial impairment and cytomembrane destruction in Leydig cells. Ecotoxicology and Environmental Safety 255, 114796 (2023). https://doi.org/10.1016/j.ecoenv.2023.114796*

[359] *Lin, S., Zhang, H., Wang, C., et al. Metabolomics Reveal Nanoplastic-Induced Mitochondrial Damage in Human Liver and Lung Cells. Environ. Sci. Technol. 56, 12483–12493 (2022). https://doi.org/10.1021/acs.est.2c03980*

[360] *Liu, H., Li, H., Yao, X., et al. Environmental nanoplastics induce mitochondrial dysfunction: A review of cellular mechanisms and associated diseases. Environmental Pollution 382, 126695 (2025). https://doi.org/10.1016/j.envpol.2025.126695*

[361] *Bang, E., Hwangbo, H., Lee, H., et al. Exposure to polystyrene nanoplastics promotes premature cellular senescence through mitochondrial ROS production and dysfunction in pre-differentiated skeletal myoblasts. Toxicology 510, 154002 (2025). https://doi.org/10.1016/j.tox.2024.154002*

[362] *Kong, L., Li, S., Fu, Y., et al. Microplastics/nanoplastics contribute to aging and age-related diseases: Mitochondrial dysfunction as a crucial role. Food and Chemical Toxicology 199, 115355 (2025). https://doi.org/10.1016/j.fct.2025.115355*

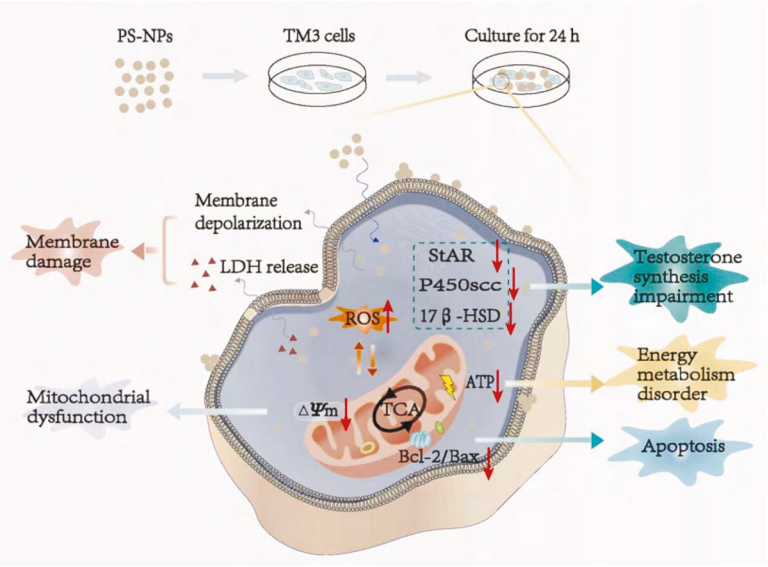


Figure 12.1.

Exposure to polystyrene nanoparticles (size 20 nm) in vitro produced cytotoxic effect on Leydig cells by inducing oxidative injury, mitochondrial impairment, apoptosis, and cytomembrane destruction.

Source: Sun, Z., Wen, Y., Zhang, F., et al. Exposure to nanoplastics induces mitochondrial impairment and cytomembrane destruction in Leydig cells. Ecotoxicology and Environmental Safety 255, 114796 (2023). https://doi.org/10.1016/j.ecoenv.2023.114796 License: CC BY-NC-ND 4.0.

The heart is among the most energy-demanding organs. Every myocardial contraction depends on continuous mitochondrial ATP production within cardiomyocytes. Impairment of mitochondrial function leads to energy insufficiency (Fig. 12.2), which compromises the force of cardiac contractions and reduces the organ's functional reserves.[363, 364] Thus, by interfering with cellular energy metabolism, MNPs may contribute to impaired myocardial contractile function and potentially increase the susceptibility of cardiac tissue to ischemic injury.

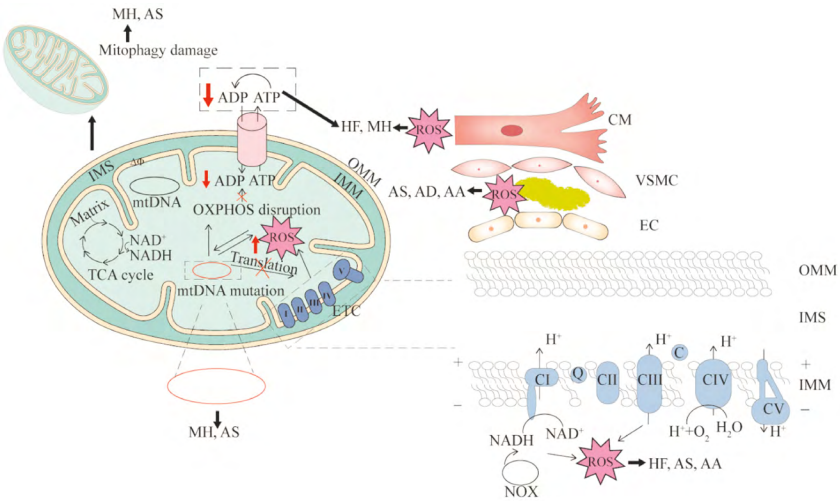


Figure 12.2.

The relationships between mitochondrial dysfunction and CVDs. Four features (mtDNA mutation, mitophagy damage, decreased oxidative phosphorylation - OXPHOS and increased reactive oxygen species - ROS) associated with mitochondrial dysfunction are demonstrated.

Source: Liu, Y., Huang, Y., Xu, C., et al. Mitochondrial Dysfunction and Therapeutic Perspectives in Cardiovascular Diseases. IJMS 23, 16053 (2022). https://doi.org/10.3390/ijms232416053

License: CC BY 4.0.

[363] Liu, Y., Huang, Y., Xu, C., et al. Mitochondrial Dysfunction and Therapeutic Perspectives in Cardiovascular Diseases. IJMS 23, 16053 (2022). https://doi.org/10.3390/ijms232416053

[364] Jahedi, F., Khaksar, M. A., Rashidi, P., et al. From pollution to palpitations: the heart's silent battle with microplastics. BMC Cardiovasc Disord 25, 837 (2025). https://doi.org/10.1186/s12872-025-05330-2

MICROPLASTICS IN ATHEROSCLEROTIC PLAQUES AND THE RISK OF VASCULAR EVENTS

Myocardial infarction and ischemic stroke remain the leading causes of death within the overall mortality structure.[356] In most cases, their pathogenetic basis is atherosclerosis—the formation of lipid-fibrous plaques within arterial walls. These formations can partially or completely occlude the vessel lumen. Particular danger is posed by plaque rupture or erosion with subsequent thrombosis, resulting in acute disruption of blood flow.

Data regarding the detection of micro- and nanoplastics within atherosclerotic plaques, including those of the carotid arteries, are of particular concern. It has been established that the presence of microplastics in the plaque is associated with a significantly increased risk of major cardiovascular events. Notably, when plastic particles are detected, the risk of myocardial infarction, stroke, or all-cause mortality increases 4.5-fold.[365]

DISRUPTION OF VASCULAR HOMEOSTASIS AND ENDOTHELIAL DYSFUNCTION

MNPs may also interfere with vascular homeostasis. Upon entering the systemic circulation, these particles can interact with blood components and the vascular wall. Owing in part to their surface physicochemical properties, including electrostatic charge, they are capable of adhering to the endothelium, adsorbing plasma proteins, and associating with circulating blood cells (Fig. 12.3, 12.4).[366]

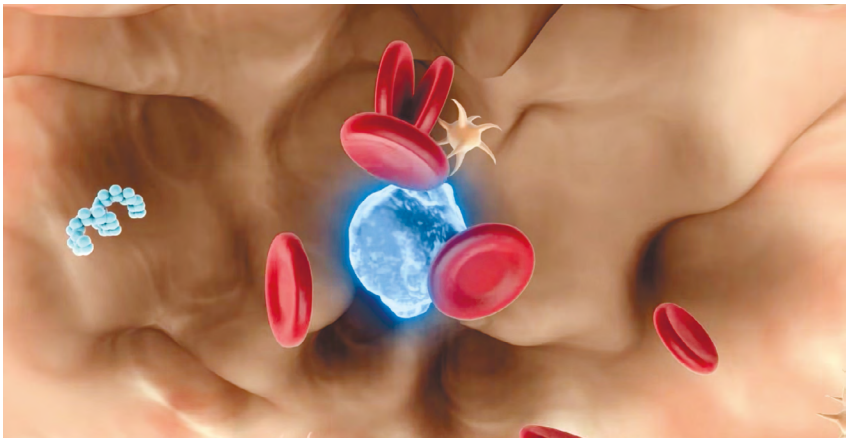


Figure 12.3.

Illustration of MNP interaction with the vascular endothelium. Due to their electrostatic charge, micro- and nanoplastic particles potentially adhere to blood vessel walls and attract circulating proteins and blood cells.

[365] *Marfella, R., Prattichizzo, F., Sardu, C., et al. Microplastics and Nanoplastics in Atheromas and Cardiovascular Events. N Engl J Med 390, 900–910 (2024). https://doi.org/10.1056/NEJMoa2309822*

[366] *Goldsworthy, A., O'Callaghan, L. A., Blum, C., et al. Micro-nanoplastic induced cardiovascular disease and dysfunction: a scoping review. J Expo Sci Environ Epidemiol 35, 746–769 (2025). https://doi.org/10.1038/s41370-025-00766-2*

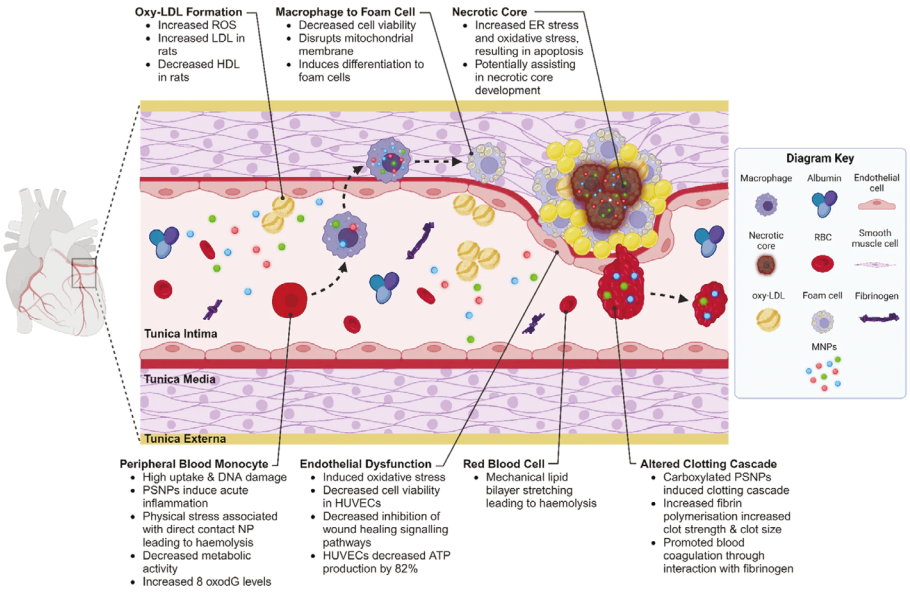


Figure 12.4.

Pathways involving MNPs in the etiology and pathophysiology of atherosclerosis.

Source: Goldsworthy, A., O'Callaghan, L. A., Blum, C., et al. Micro-nanoplastic induced cardiovascular disease and dysfunction: a scoping review. J Expo Sci Environ Epidemiol 35, 746–769 (2025). https://doi.org/10.1038/s41370-025-00766-2

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The resulting aggregates may exert adverse effect on the endothelium, which plays a key role in regulating vascular tone, maintaining hemostatic balance, and modulating inflammatory responses. Disruption of its structural integrity and functional activity may contribute to the development of endothelial dysfunction, promote vascular wall inflammation, and create conditions associated with increased thrombotic risk.[367]

IMPACT ON BLOOD CELLS AND THROMBOGENESIS PROCESSES

The increased risk of thrombosis is also attributed to the direct interaction of electrostatically charged microplastics with blood cells, specifically platelets and erythrocytes.[368]

Under physiological conditions, erythrocytes possess a negative surface charge (Fig. 12.5) that forms a negative zeta potential, ensuring electrostatic repulsion between blood cells. This mechanism reduces their aggregation tendency, maintains optimal blood rheology, and ensures the free passage of erythrocytes through the microvasculature.[369] Exposure to micro- and nanoplastics may disrupt these electrostatic properties through direct interactions with the erythrocyte membrane, leading to alterations in surface charge, oxidative damage, and structural impairment, which in turn promote erythrocyte aggregation and may compromise microcirculatory flow.

[367] *Sheng, C., Wang, G., Liu, Z., et al. Polystyrene nanoplastics enhance thrombosis through adsorption of plasma proteins. Journal of Hazardous Materials 480, 136168 (2024). https://doi.org/10.1016/j.jhazmat.2024.136168*

[368] *Rajendran, D. & Chandrasekaran, N. Journey of micronanoplastics with blood components. RSC Adv. 13, 31435–31459 (2023). https://doi.org/10.1039/D3RA05620A*

[369] *Funk, R. H. W. & Scholkmann, F. The significance of bioelectricity on all levels of organization of an organism. Part 1: From the subcellular level to cells. Progress in Biophysics and Molecular Biology 177, 185–201 (2023). https://doi.org/10.1016/j.pbiomolbio.2022.12.002*

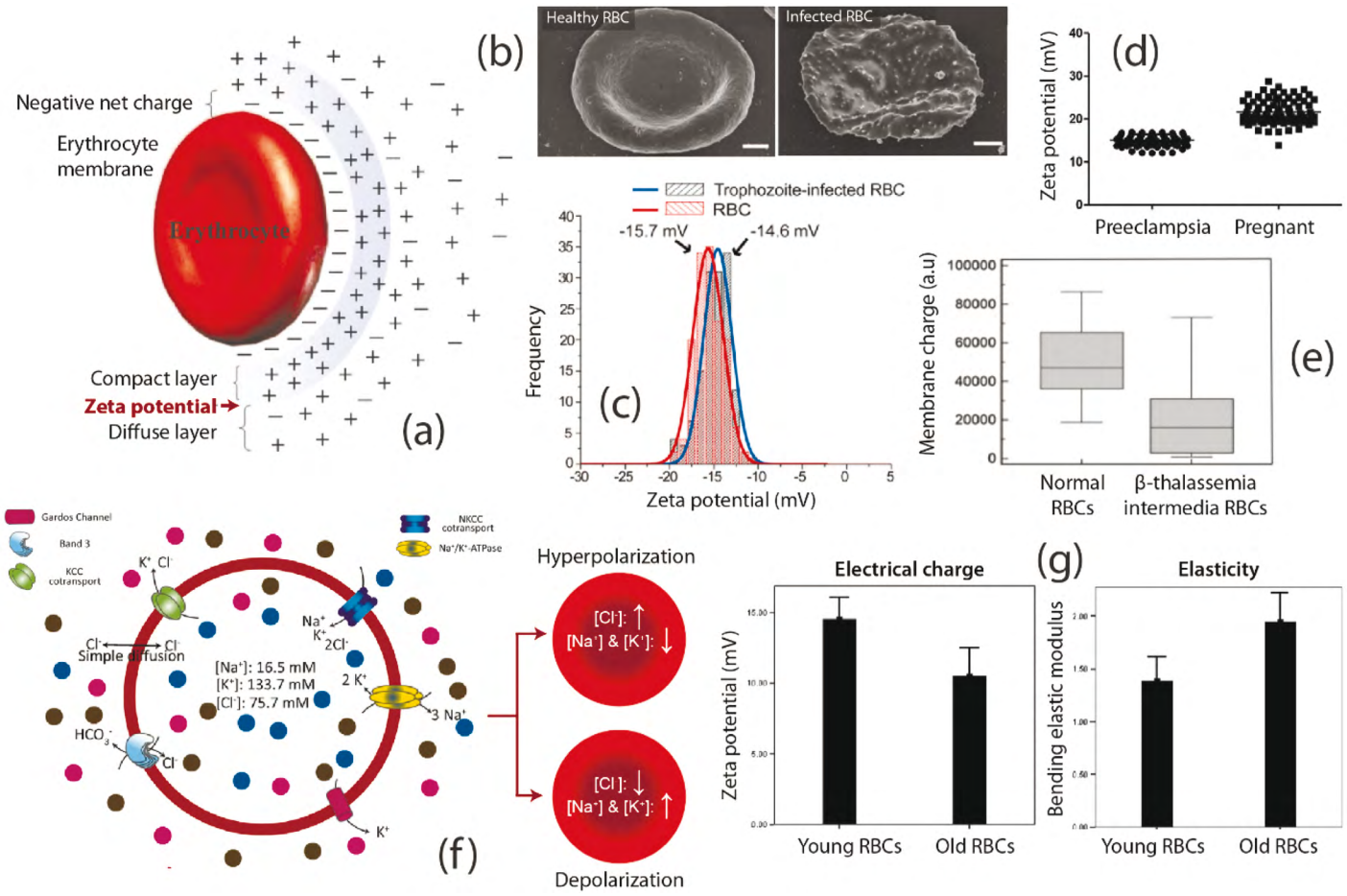


Figure 12.5.

Electrical properties of red blood cells (RBCs). **(a)** Charge distribution around a RBC with the zeta potential and the two ionic layers (compact layer and diffuse layer). The compact layer comprises cations and the cloud-like diffused layer of a mixture of anions and cations. **(b, c)** The effect of Plasmodium falciparum infection on the electrical properties of RBCs. **(b)** Electron microscopy images of healthy and Plasmodium falciparum-infected RBCs. **(c)** Plasmodium falciparum-infected RBCs have a lower zeta potential compared to healthy RBCs. **(d)** RBCs of women with preeclampsia have a reduced zeta potential of RBCs compared to the RBCs of healthy pregnant women. **(e)** RBCs of patients with β-thalassemia have a reduced membrane charge. **(f)** Visualization of driving forces for RBC plasma membrane potential changes. **(g)** Older RBCs have a lower surface charge and are less elastic.

Source: Funk, R. H. W. & Scholkmann, F. The significance of bioelectricity on all levels of organization of an organism. Part 1: From the subcellular level to cells. Progress in Biophysics and Molecular Biology 177, 185–201 (2023). https://doi.org/10.1016/j.pbiomolbio.2022.12.002 License: CC BY 4.0.

Microplastic particles , owing to their specific physicochemical properties — including surface charge (Fig. 12.6) and the ability to adsorb biomolecules, — may alter erythrocyte membrane characteristics.

A reduction in the negative zeta potential weakens intercellular electrostatic repulsion, thereby promoting red blood cell aggregation. (Fig. 12.7). This process may contribute to increased blood viscosity and impaired microcirculation.

Conditions of reduced blood flow favor activation of the coagulation cascade and thrombus formation. Thus, the alteration in erythrocyte surface properties under the influence of microplastics may represent one of the mechanisms contributing to thrombogenesis and vascular complications.[370]

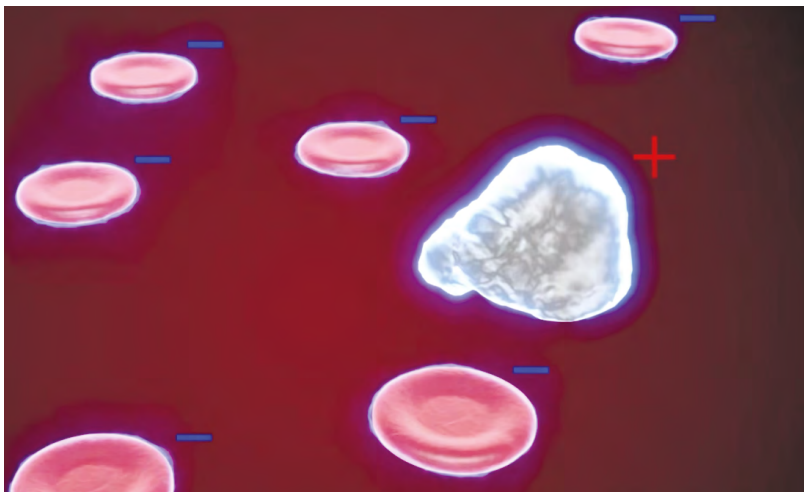


Figure 12.6.

Schematic representation of the surface charge on MNP particles and erythrocytes.

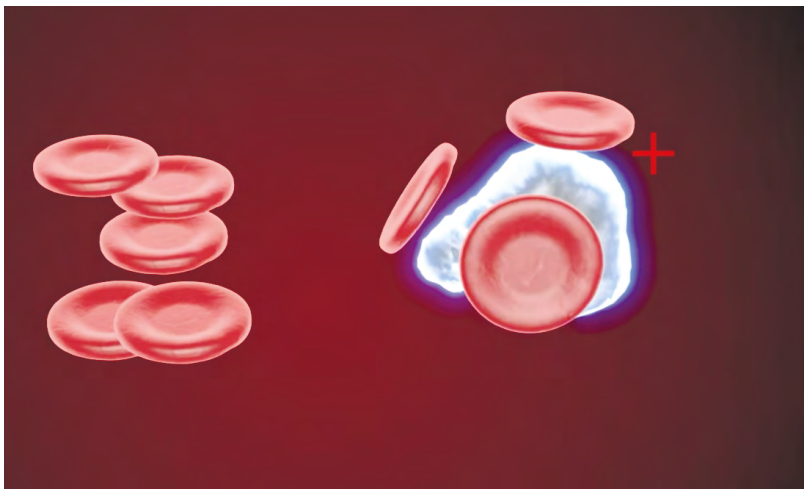


Figure 12.7.

Microplastic particles possessing specific physicochemical properties, including surface charge and the ability to adsorb biomolecules, can potentially lower the negative zeta potential of red blood cells, weaken electrostatic repulsion between them, and increase their propensity to aggregate.

[370] *Kim, E.-H., Choi, S., Kim, D., et al. Amine-modified nanoplastics promote the procoagulant activation of isolated human red blood cells and thrombus formation in rats. Part Fibre Toxicol 19, 60 (2022). https://doi.org/10.1186/s12989-022-00500-y*

These changes pose a particular threat to highly vascularized organs that are exceptionally sensitive to ischemia — the brain, heart, and retina. Resulting microcirculatory disturbances may contribute to tissue hypoxia and functional impairment, and in severe cases, may be involved in the development of acute vascular events, including myocardial infarction and ischemic stroke.

The totality of the available evidence supports the consideration of micro- and nanoplastics as a potentially significant factor capable of exacerbating the course of cardiovascular diseases.

Given the projected increase in the global burden of CVD by 2050, investigating the effects of micro- and nanoplastics represents an issue of substantial scientific, clinical, and public health significance.

MICRO- AND NANOPLASTICS AS A POTENTIAL RISK FACTOR FOR SUDDEN CARDIAC DEATH

Sudden cardiac death (SCD) remains a major unresolved clinical and public health problem. It is typically defined as an unexpected natural death of presumed cardiac origin, occurring within a short time period (generally within one hour of symptom onset), often in inividuals without previously recognized life-threatening conditions. The absence of prodromal symptoms in many cases significantly limits both timely prevention and accurate identification of underlying causes.

The global burden of SCD is substantial. It is estimated that up to approximately 4–5 million cases occur annually worldwide.[371] In high-income regions, including the United States and Western Europe, SCD accounts for roughly 15–20% of all natural deaths and up to 40–50% of cardiovascular mortality.[372,373]

Of particular concern is the observed increase in SCD incidence among younger populations. Epidemiological data from large nationwide studies in the United States indicate a marked rise in SCD cases among adults aged 25–44 years over the past two decades (Figure 12.8).[374] This trend cannot be fully explained by traditional cardiovascular risk factors alone and suggests the possible contribution of additional, previously underrecognized influences.

[371] *Marijon, E., Narayanan, K., Smith, K., et al. The Lancet Commission to reduce the global burden of sudden cardiac death: a call for multidisciplinary action. The Lancet 402, 883–936 (2023). https://doi.org/10.1016/S0140-6736(23)00875-9*

[372] *Zimmerman, D. S. & Tan, H. L. Epidemiology and risk factors of sudden cardiac arrest. Current Opinion in Critical Care 27, 613 (2021). https://doi.org/10.1097/MCC.0000000000000896*

[373] *Stecker, E. C., Reinier, K., Marijon, E., et al. Public Health Burden of Sudden Cardiac Death in the United States. Circ: Arrhythmia and Electrophysiology 7, 212–217 (2014). https://doi.org/10.1161/CIRCEP.113.001034*

[374] *Zuin, M., Mohanty, S., Aggarwal, R., et al. Trends in Sudden Cardiac Death Among Adults Aged 25 to 44 Years in the United States: An Analysis of 2 Large US Databases. JAHA 14, e035722 (2025). https://doi.org/10.1161/JAHA.124.035722*

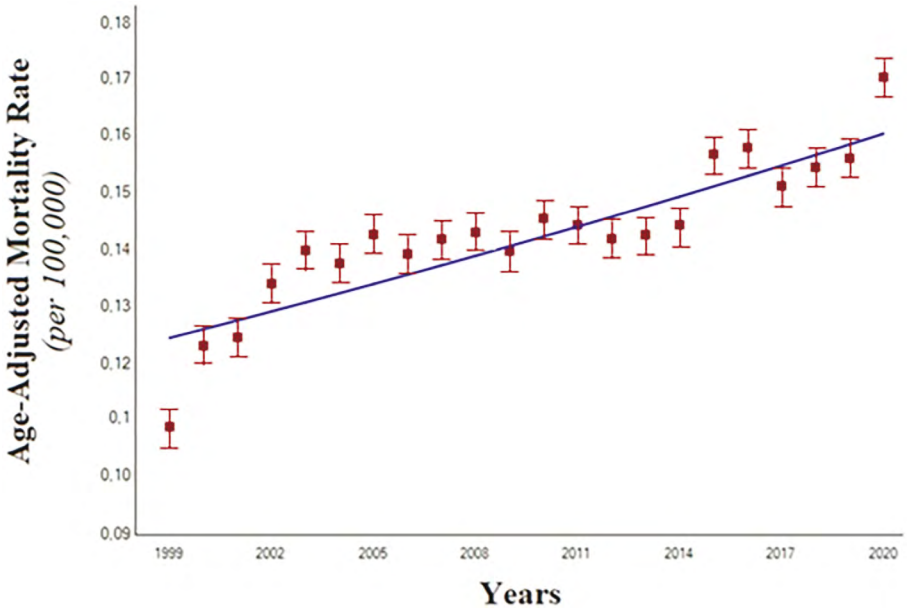


Figure 12.8.

Trends in age-adjusted mortality rates due to sudden cardiac death, with associated 95% CIs, in United States early adults, aged 25 to 44 years, 1999 to 2020.

Source: Zuin, M., Mohanty, S., Aggarwal, R., et al. Trends in Sudden Cardiac Death Among Adults Aged 25 to 44 Years in the United States: An Analysis of 2 Large US Databases. JAHA 14, e035722 (2025). https://doi.org/10.1161/JAHA.124.035722 License: CC BY-NC-ND 4.0.

Given its high incidence, frequent occurrence outside healthcare settings, and limited predictability, SCD represents a critical target for identifying novel risk modifiers and mechanistic pathways.

In the context of widespread environmental contamination, micro- and nanoplastics may be considered a novel and potentially significant factor contributing to the risk of sudden cardiac death.

The proposed impact of MNPs on SCD is based on two principal, potentially synergistic mechanisms:

1. Electrophysiological destabilization

MNPs may interfere with the generation and propagation of electrical signals in excitable tissues. This includes potential disruption of ion channel function, membrane properties, and intercellular electrical coupling in cardiomyocytes, as well as interference with neuronal signaling within central autonomic regulatory networks. Such alterations may reduce the stability and synchronization of cardiac electrical activity, thereby increasing susceptibility to malignant arrhythmias.

2. Mitochondrial dysfunction and energy deficit

As already widely discussed earlier in this report, MNP exposure can impair mitochondrial function, leading to decreased ATP production and increased oxidative stress.

In cardiomyocytes and neurons of autonomic control centers, high energy demand makes these cells particularly vulnerable to mitochondrial impairment. Resulting energy deficits may compromise cellular homeostasis and further destabilize electrophysiological processes.

The convergence of electrophysiological instability and impaired cellular energetics creates conditions conducive to the initiation of fatal cardiac arrhythmias. These events may rapidly progress to circulatory collapse and cessation of respiration, corresponding to the clinical manifestation of sudden cardiac death.

TRADITIONAL AND EMERGING PATHOGENETIC MODELS

Traditionally, sudden cardiac death is linked to undiagnosed cardiovascular pathologies: ischemic heart disease, life-threatening arrhythmias, and cardiomyopathies.

MNP particles are capable of penetrating the bloodstream[375] as well as accumulating in organs and tissues,[376] where they may provoke oxidative stress, inflammation and endothelial dysfunction (impairment of the normal function of the endothelium — the inner layer of blood vessels).[377,378,379,380,381,382] These processes underlie both the atherosclerotic damage to blood vessels (Fig. 12.4) and the disruption of myocardial electrical stability.[366] In turn, atherosclerosis creates conditions for ischemic injury to the myocardium, while electrical instability directly increases the risk of fatal cardiac arrhythmias.

THE ROLE OF MNPs IN THE PATHOGENESIS OF CARDIOVASCULAR DISORDERS

The damaging effects of nanoplastics, particularly their capacity to initiate multiple interacting cellular disturbances, have been associated with mechanisms that may contribute to the development of life-threatening arrhythmias and sudden cardiac death.

Electrophysiological processes in the myocardium and neurons are based on the tightly regulated transmembrane movement of ions (Na^+, K^+, Ca^{2+}) mediated by ion channels

[375] *Leslie, H. A., Van Velzen, M. J. M., Brandsma, S. H., et al. Discovery and quantification of plastic particle pollution in human blood. Environment International 163, 107199 (2022). https://doi.org/10.1016/j.envint.2022.107199*

[376] *Jenner, L. C., Rotchell, J. M., Bennett, R. T., et al. Detection of microplastics in human lung tissue using μFTIR spectroscopy. Science of The Total Environment 831, 154907 (2022). https://doi.org/10.1016/j.scitotenv.2022.154907*

[377] *Viana, M., Tonin, F. S. & Ladeira, C. Assessing the Impact of Nanoplastics in Biological Systems: Systematic Review of In Vitro Animal Studies. JoX 15, 75 (2025). https://doi.org/10.3390/jox15030075*

[378] *Zhao, B., Rehati, P., Yang, Z., et al. The potential toxicity of microplastics on human health. Science of The Total Environment 912, 168946 (2024). https://doi.org/10.1016/j.scitotenv.2023.168946*

[379] *Milillo, C., Aruffo, E., Di Carlo, P., et al. Polystyrene nanoplastics mediate oxidative stress, senescence, and apoptosis in a human alveolar epithelial cell line. Front. Public Health 12, 1385387 (2024). https://doi.org/10.3389/fpubh.2024.1385387*

[380] *Olatunji, G., Kokori, E., Ogieuhi, I. J., et al. Nanoplastics as emerging cardiovascular hazards: a narrative review of current evidence. Egypt J Intern Med 36, 62 (2024). https://doi.org/10.1186/s43162-024-00329-1*

[381] *Goldsworthy, A., O'Callaghan, L. A., Blum, C., et al. Micro-nanoplastic induced cardiovascular disease and dysfunction: a scoping review. J Expo Sci Environ Epidemiol 35, 746–769 (2025). https://doi.org/10.1038/s41370-025-00766-2*

[382] *Hu, M. & Palić, D. Micro- and nano-plastics activation of oxidative and inflammatory adverse outcome pathways. Redox Biology 37, 101620 (2020). https://doi.org/10.1016/j.redox.2020.101620*

and membrane pumps. These ionic currents underlie the generation of action potentials, determine cardiac rhythm, and enable the transmission of neural signals.

Experimental data indicate that micro- and nanoplastics can interact with biological membranes by embedding within the lipid bilayer or adsorbing onto the membrane surface, thereby altering the physicochemical properties of the membrane, including membrane fluidity, surface charge, and permeability.[383,384,385] Such alterations may disrupt the function of ion channels, affecting their gating kinetics and modifying the magnitude, direction, and timing of ionic currents—particularly those of Ca^{2+}, Na^{+}, and K^{+} (see Part 4).

As a consequence, membrane potential stability may be compromised, leading to reduced precision in electrical impulse generation and propagation. In neurons, this may manifest as impaired synaptic transmission and desynchronization of network activity.[386] In cardiomyocytes, these effects may result in alterations of action potential duration and morphology, slowed or heterogeneous conduction, and increased susceptibility to arrhythmogenesis.[387] The surface electrostatic charge of MNPs appears to play an important modulatory role in these processes. Experimental studies indicate that charged nanoparticles can directly influence cellular electrophysiology: positively charged particles have been shown to disrupt membrane integrity and induce cardiomyocyte damage, whereas negatively charged particles may alter membrane electrical properties and interfere with impulse conduction.[388,389]

A experimental study has demonstrated that charged nanoparticles are capable of directly affecting the electrical activity of cardiac cells in mice model. Positively charged nanoparticles were associated with impaired membrane integrity and reduced cardiomyocyte viability, whereas negatively charged nanoparticles were observed to alter membrane electrical properties and parameters of impulse conduction.[390]

On this basis, exposure to nanoplastics may contribute to functional instability of electrical activity in both the myocardium and neural centers involved in autonomic regulation. Such disturbances are recognized as established mechanisms underlying the development of life-threatening arrhythmias. However, the extent to which nanoplastics contribute to these processes in vivo, and their role in the pathogenesis of sudden cardiac death, remains to be determined.

[383] *Rossi, G., Barnoud, J. & Monticelli, L. Polystyrene Nanoparticles Perturb Lipid Membranes. J. Phys. Chem. Lett. 5, 241–246 (2014).* https://doi.org/10.1021/jz402234c

[384] *Warren, E. A. K. & Payne, C. K. Cellular binding of nanoparticles disrupts the membrane potential. RSC Adv. 5, 13660–13666 (2015).* https://doi.org/10.1039/C4RA15727C

[385] *Guan, R., Wang, Q., Li, D., et al. Polystyrene and polyethylene perturb the structure of membrane: An experimental and computational study. Environmental Pollution 375, 126317 (2025) .* https://doi.org/10.1016/j.envpol.2025.126317

[386] *Su, Z., Kong, R., Huang, C., et al. Exposure to polystyrene nanoplastics causes anxiety and depressive-like behavior and down-regulates EAAT2 expression in mice. Arch Toxicol 99, 2595–2609 (2025).* https://doi.org/10.1007/s00204-025-04002-6

[387] *Chia, S. P. S., Pang, J. K. S., Winanto, W., et al. Nanoplastics induces arrhythmia in human stem-cells derived cardiomyocytes. Ecotoxicology and Environmental Safety 289, 117657 (2025).* https://doi.org/10.1016/j.ecoenv.2024.117657

[388] *Perini, D. A., Parra-Ortiz, E., Varó, I., et al. Surface-Functionalized Polystyrene Nanoparticles Alter the Transmembrane Potential via Ion-Selective Pores Maintaining Global Bilayer Integrity. Langmuir 38, 14837–14849 (2022).* https://doi.org/10.1021/acs.langmuir.2c02487

[389] *Negoda, A., Kim, K.-J., Crandall, E. D., et al. Polystyrene nanoparticle exposure induces ion-selective pores in lipid bilayers. Biochimica et Biophysica Acta (BBA) - Biomembranes 1828, 2215–2222 (2013).* https://doi.org/10.1016/j.bbamem.2013.05.029

[390] *Miragoli, M., Novak, P., Ruenraroengsak, P., et al. Functional Interaction Between Charged Nanoparticles and Cardiac Tissue: A New Paradigm for Cardiac Arrhythmia? Nanomedicine 8, 725–737 (2013).* https://doi.org/10.2217/nnm.12.125

MITOCHONDRIAL DYSFUNCTION AS THE PATHOPHYSIOLOGICAL BASIS OF SCD

Mitochondrial dysfunction represents a critical pathological state for the heart[391] and brain,[392] as cardiomyocytes (Fig. 12.2) and neurons are cells with maximal energy demand.[393] Impairment of mitochondrial function is known to to result in three key consequences: reduced ATP synthesis, increased generation of reactive oxygen species, and disruption of calcium homeostasis.[392] This combination of factors creates conditions for electrical instability in both cardiac and neuronal cells.

Experimental studies, *in vitro* and *in vivo* show that micro- and nanoplastics can accumulate intracellularly, localize within mitochondria, where they are associated with features consistent with mitochondrial dysfunction.[394,395,396]

Reduction in ATP production directly impairs the operation of ion pumps in cardiac cells.[391] Without sufficient ATP, pumps (Na^+/K^+-ATPase and Ca^{2+}-ATPase) cease to effectively maintain ion balance. This leads to intracellular calcium accumulation, which increases the probability of early and late afterdepolarizations (spontaneous electrical impulses) and, ultimately, creates a substrate for the development of rhythm disturbances.

Mitochondrial dysfunction and the resulting cellular energy deficit are well-established contributors to the development of life-threatening arrhythmias. Their role in the pathophysiology of sudden cardiac death is also widely recognized, although typically in the context of defined cardiac disease.

In addition to cardiomyocytes, neurons within central autonomic regulatory centers—including the medulla oblongata, hypothalamus, and other brainstem structures—are highly dependent on mitochondrial function.[397] Energy deficit in these neurons may disrupt the balance between sympathetic (activating) and parasympathetic (inhibitory) influences, which is critical for stable cardiac rhythm regulation. Such dysregulation is associated with increased susceptibility to arrhythmia.

Taken together, mitochondrial impairment represents a biologically plausible pathway linking cellular stress to electrophysiological instability in both cardiac and neural tissues. Current evidence suggests that micro- and nanoplastics may contribute to such dysfunction under experimental conditions.

[391] *Liu, Y., Huang, Y., Xu, C., et al. Mitochondrial Dysfunction and Therapeutic Perspectives in Cardiovascular Diseases. IJMS 23, 16053 (2022). https://doi.org/10.3390/ijms232416053*

[392] *Exner, N., Lutz, A. K., Haass, C., et al. Mitochondrial dysfunction in Parkinson's disease: molecular mechanisms and pathophysiological consequences. EMBO J 31, 3038–3062 (2012). https://doi.org/10.1038/emboj.2012.170*

[393] *Maldonado, K. A. & Alsayouri, K. Physiology, Brain. in StatPearls [Internet] (StatPearls Publishing, 2025). https://www.ncbi.nlm.nih.gov/books/NBK551718/ (Accessed 20 March 2026)*

[394] *Jahedi, F., Khaksar, M. A., Rashidi, P., et al. From pollution to palpitations: the heart's silent battle with microplastics. BMC Cardiovasc Disord 25, 837 (2025). https://doi.org/10.1186/s12872-025-05330-2*

[395] *Araújo, A. M., Mota, C., Ramos, H., et al. The neurotoxic threat of micro- and nanoplastics: evidence from In Vitro and In Vivo models. Arch Toxicol 99, 3505–3525 (2025). https://doi.org/10.1007/s00204-025-04091-3*

[396] *Baroni, A., Moulton, C., Cristina, M., et al. Nano- and Microplastics in the Brain: An Emerging Threat to Neural Health. Nanomaterials 15, 1361 (2025). https://doi.org/10.3390/nano15171361*

[397] *Silvani, A., Calandra-Buonaura, G., Dampney, R. A. L., et al. Brain–heart interactions: physiology and clinical implications. Phil. Trans. R. Soc. A. 374, 20150181 (2016). https://doi.org/10.1098/rsta.2015.0181*

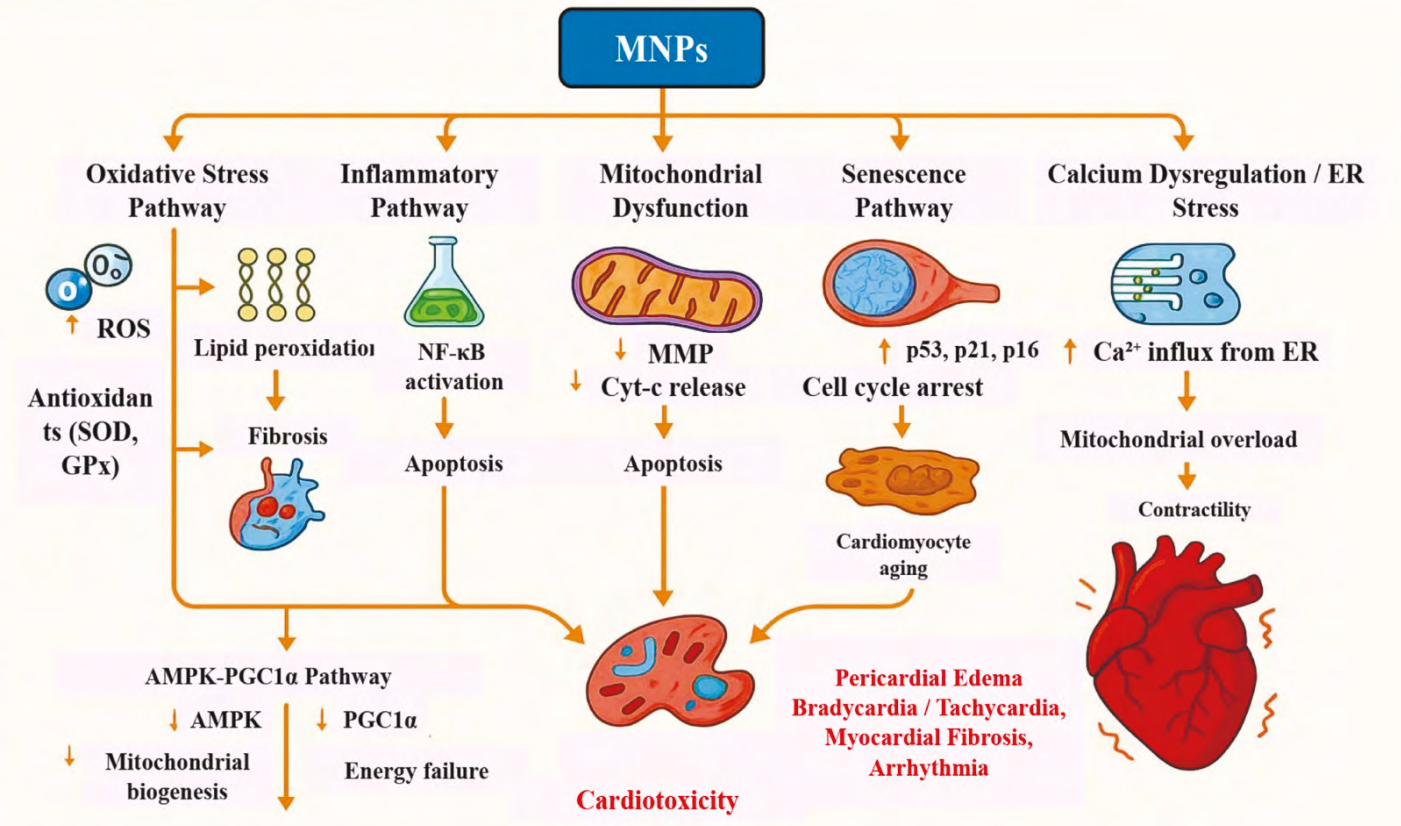


Figure 12.9.

Mechanistic pathways of MNP-Induced cardiotoxicity

Source: Jahedi, F., Khaksar, M. A., Rashidi, P., et al. From pollution to palpitations: the heart's silent battle with microplastics. BMC Cardiovasc Disord 25, 837 (2025). https://doi.org/10.1186/s12872-025-05330-2 License: CC BY-NC-ND 4.0.

Thus, the aggregate of contemporary data (Fig. 12.9) allows for the formulation of a biologically sound hypothesis: MNP exposure represents a genuine pathobiological mechanism capable not only of contributing to the pathogenesis of chronic diseases but also of elevating the risk of acute, fatal events, including sudden cardiac death. However, the full spectrum of the long-term effects of MNPs and their specific role in the epidemiology of SCD requires further in-depth interdisciplinary research.

PART 13.

MICRO- AND NANOPLASTICS AS A RISK FACTOR FOR **ONCOGENESIS: FROM CELLULAR MECHANISMS TO GLOBAL EPIDEMIOLOGY**

GLOBAL DYNAMICS AND PROGNOSIS OF ONCOLOGICAL MORBIDITY

Oncological diseases represent one of the most significant challenges in modern medicine. Over recent decades, their global burden has remained substantial, with many countries reporting increasing incidence rates, while mortality trends vary depending on cancer type, early detection, and advances in treatment (Fig. 13.1).[398,399]

According to data from the International Agency for Research on Cancer (IARC) of the World Health Organization (WHO), approximately 18.1 million[400] new cancer cases were registered globally in 2018, and by 2022, this figure approached 20 million,[401] corresponding to a 10.5% increase in just four years. Current prognostic models raise even more serious concerns:

"Cancer cases and deaths are expected to rise by 77% and 90% in 2050, respectively."[402]

The observed increase in oncological morbidity necessitates a broader analysis of potential anthropogenic risk factors. Within this context, micro- and nanoplastic particles have attracted particular attention as ubiquitous environmental pollutants of anthropogenic origin. Current evidence indicates that these particles are capable of crossing biological barriers and accumulating in tissues. This behavior is largely determined by their small size and surface physicochemical characteristics, including electrostatic charge (see Part 4).

In this section, micro- and nanoplastics are examined as a potential systemic ecological factor which, under chronic exposure, can disrupt fundamental mechanisms of cellular regulation. It is hypothesized that chronic contact with such particles is capable of amplifying oxidative stress, increasing genomic instability, and disrupting the regulatory mechanisms of cell division and apoptosis—processes inextricably linked to cancer development.

[398] *Our World in Data. Cancer Deaths by Type, World. https://ourworldindata.org/grapher/cancer-deaths-by-type-grouped (Accessed 20 March 2026)*
[399] *Berrington de Gonzalez, A., Brayley, M., Frost, R., et al. Trends in Cancer Incidence in Younger and Older Adults. Ann Intern Med 178, 1677–1687 (2025). https://doi.org/10.7326/ANNALS-24-02718*
[400] *International Agency for Research on Cancer (IARC). Latest global cancer data: Cancer burden rises to 18.1 million new cases and 9.6 million cancer deaths in 2018. (2018). https://www.iarc.who.int/featured-news/latest-global-cancer-data-cancer-burden-rises-to-18-1-million-new-cases-and-9-6-million-cancer-deaths-in-2018/ (Accessed 20 March 2026)*
[401] *World Health Organization (WHO). Global Cancer Burden Growing Amidst Mounting Need for Services. (2024). https://www.who.int/news/item/01-02-2024-global-cancer-burden-growing--amidst-mounting-need-for-services (Accessed 20 March 2026)*
[402] *Bizuayehu, H. M., Ahmed, K. Y., Kibret, G. D., et al. Global Disparities of Cancer and Its Projected Burden in 2050. JAMA Netw Open 7, e2443198 (2024). https://doi.org/10.1001/jamanetworkopen.2024.43198*

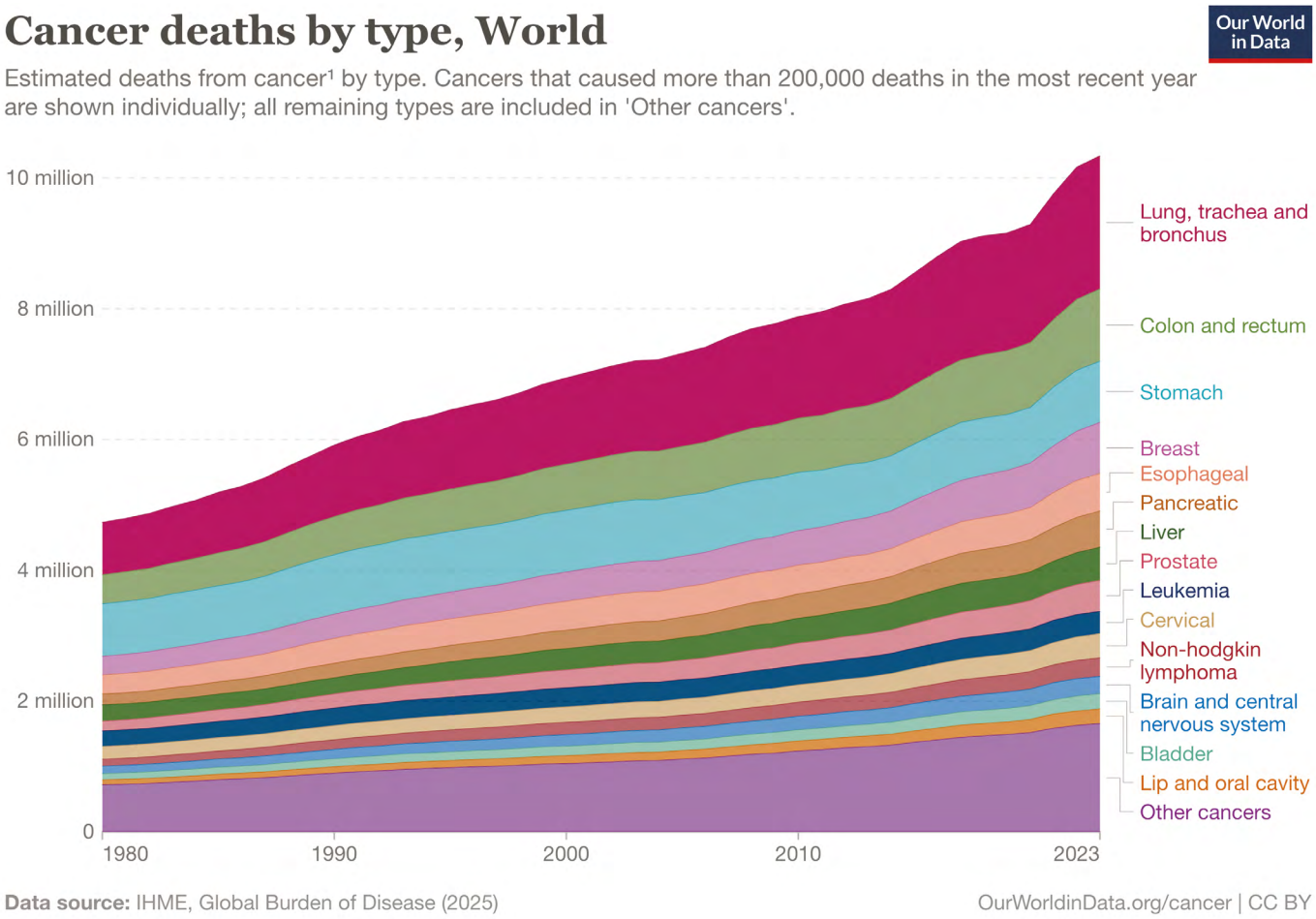


Figure 13.1.

Cancer deaths by type, World.

Source: Our World in Data. Cancer Deaths by Type, World. https://ourworldindata.org/grapher/cancer-deaths-by-type-grouped (Accessed 20 March 2026). License: CC BY 4.0.

EPIDEMIOLOGICAL SHIFT: THE PHENOMENON OF EARLY-ONSET ONCOGENESIS

For a long time, cancer was viewed predominantly as a disease of old age. This perspective was based on the cumulative nature of genetic and epigenetic alterations over time, the age-related decline in immune surveillance efficacy, endocrine alterations, dietary habits, and prolonged exposure to estabilished carcinogens.[403]

[403] *Montégut, L., López-Otín, C. & Kroemer, G. Aging and cancer. Mol Cancer 23, 106 (2024). https://doi.org/10.1186/s12943-024-02020-z*

Recent epidemiological analyses suggest a global shift toward increasing cancer incidence in younger populations.[404] Over approximately the past three decades, the number of new cancer cases in individuals under 50 years of age has increased substantially, with some studies estimating a rise of around 70–80% worldwide.[405,406]

The growing trend is documented across high-, middle-, and low-income countries.[406] The consistency of this pattern across diverse regions suggests the possible contribution of broadly distributed risk factors that extend beyond local socioeconomic conditions.

In the USA, 14 cancers had incidence rates that increased in at least one early-onset age group (i.e., 15–29-, 30–39-, and 40–49-year-olds)—9 of these also increased in at least one older-onset age group (i.e., 50–59, 60–69, and 70–79; i.e., female breast, colorectal, kidney, testicular, uterine and pancreatic cancers, and several lymphoid neoplasms) during the years 2010–2019.[407]

Global analyses show that the incidence of breast and colorectal cancer in individuals under 50 has risen substantially over recent decades.[406] The incidence of early-onset cancer in Brazil increased by 219% between 2000 and 2019, with breast cancer remaining the leading malignancy, marked by a pronounced rise in early-onset cases among women aged 19–44.[408] Data analyses for China also document an increasing frequency of early-onset breast and colorectal cancers in the <50 age cohort.[409,410]

In low-income countries, particularly in sub-Saharan Africa, breast cancer and cervical cancer remain among the leading causes of oncological morbidity and cancer mortality among women.[411,412] Global and regional data consistently show that these malignacies account for a substantial proportion of the overall cancer burden among women, including those of reproductive age, and the upward trend persists.[413]

Studies further indicate that the global cancer incidence among adolescents and young adults (10–24 years old) has also increased over the past three decades.[414] Within this age group, leukemia (especially acute lymphoblastic leukemia), brain cancer, and malignant tumors of bone and articular cartilage are currently the leading causes of mortality and disease burden. These findings point to the formation of a robust international trend of increasing oncological morbidity at a young age and highlight the need to analyze causes that extend beyond traditional risk factors.[399,404,415]

The timeline of global plastic pollution demonstrates a clear correlation with the epidemiological shift described above. According to an OECD report, global polymer production surged from 2 million tons in 1950 to 460 million tons in 2019. It is of fundamental importance that more than half of all the plastic ever created by humanity was produced within the last 20 years (Fig. 13.2).[416]

[404] *Lopes-Júnior, L. C. The Emerging Epidemic of Early-Onset Cancer: Global Patterns, Biological Complexity, and Urgent Calls for Action. Cancer Control 32, 10732748251386505 (2025). https://doi.org/10.1177/10732748251386505*

[405] *Ugai, T., Sasamoto, N., Lee, H.-Y., et al. Is early-onset cancer an emerging global epidemic? Current evidence and future implications. Nat Rev Clin Oncol 19, 656–673 (2022). https://doi.org/10.1038/s41571-022-00672-8*

[406] *Zhao, J., Xu, L., Sun, J., et al. Global trends in incidence, death, burden and risk factors of early-onset cancer from 1990 to 2019. BMJ Oncology 2, e000049 (2023). https://doi.org/10.1136/bmjonc-2023-000049*

[407] *Shiels, M. S., Haque, A. T., Berrington de González, A., et al. Trends in Cancer Incidence and Mortality Rates in Early-Onset and Older-Onset Age Groups in the United States, 2010–2019. Cancer Discov 15, 1363–1376 (2025) . https://doi.org/10.1158/2159-8290.CD-24-1678*

[408] *Grippa, W. R., Vasconcellos, V. F. de, Pessanha, R., et al. Early-onset (EO) cancer trends in Brazil: A comprehensive analysis of hospital-based cancer registry data (2000–2019). J Clin Oncol 43, 10552–10552 (2025). https://doi.org/10.1200/JCO.2025.43.16_suppl.10552*

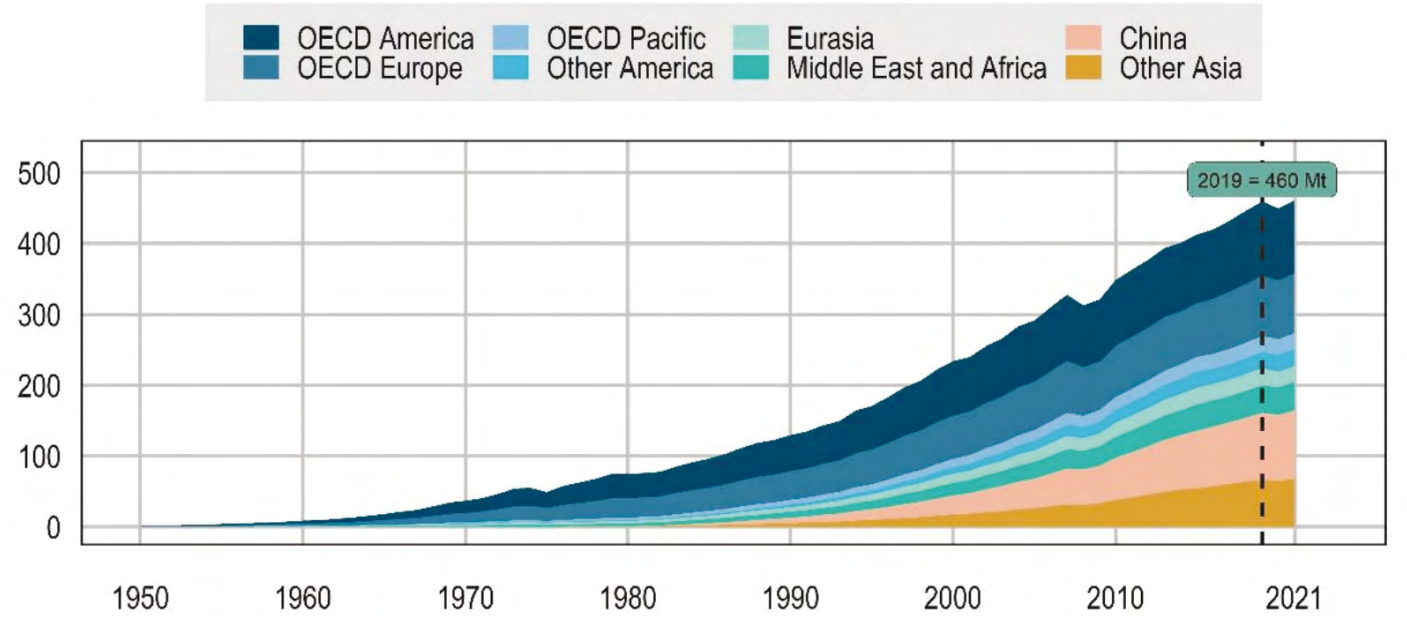


Figure 13.2.

Global plastics use (in million tonnes (Mt), 1950-2021) has quadrupled in 30 years, mainly driven by emerging economies.

Source: OECD. Global Plastics Outlook: Economic Drivers, Environmental Impacts and Policy Options. (OECD Publishing, Paris, 2022) https://doi.org/10.1787/de747aef-en License: OECD.

This exponential growth has shaped an unprecedented ecological background and led to the creation of a qualitatively new living environment for contemporary generations:

"Individuals born after the 1950s plastic boom have experienced continuous MP exposure beginning in utero, potentially predisposing them to carcinogenic pathways later in life."[417]

Thus, exposure is not episodic but chronic, starting even before birth and persisting throughout life. Such long-term exposure may contribute to cumulative biological effects; however, its specific role in carcinogenic processes remains under investigation.

[409] *Zhu, J., Li, T., Chen, J., et al. Trends in the disease burden of early-onset cancers in China from 1990 to 2021: a systematic analysis of the 2021 Global Burden of Disease study. BMC Cancer 25, 1168 (2025). https://doi.org/10.1186/s12885-025-14578-y*

[410] *Wang, X., Xia, C., Wang, Y., et al. Landscape of young breast cancer under 35 years in China over the past decades: a multicentre retrospective cohort study (YBCC-Catts study). eClinicalMedicine 64, 102243 (2023). https://doi.org/10.1016/j.eclinm.2023.102243*

[411] *Bray, F., Parkin, D. M., Gnangnon, F., et al. Cancer in sub-Saharan Africa in 2020: a review of current estimates of the national burden, data gaps, and future needs. The Lancet Oncology 23, 719–728 (2022). https://doi.org/10.1016/S1470-2045(22)00270-4*

[412] *Sung, H., Ferlay, J., Siegel, R. L., et al. Global Cancer Statistics 2020: GLOBOCAN Estimates of Incidence and Mortality Worldwide for 36 Cancers in 185 Countries. CA: A Cancer Journal for Clinicians 71, 209–249 (2021). https://doi.org/10.3322/caac.21660*

[413] *Jedy-Agba, E., Joko, W. Y., Liu, B., et al. Trends in cervical cancer incidence in sub-Saharan Africa. Br J Cancer 123, 148–154 (2020). https://doi.org/10.1038/s41416-020-0831-9*

[414] *Yang, J., Luo, G., Xiang, Q., et al. Global burden of cancer in adolescents and young adults aged 10–24 years: a trend analysis. Front. Oncol. 15, 1663523 (2025). https://doi.org/10.3389/fonc.2025.1663523*

[415] *Li, J. & Kuang, X. Global cancer statistics of young adults and its changes in the past decade: Incidence and mortality from GLOBOCAN 2022. Public Health 237, 336–343 (2024). https://doi.org/10.1016/j.puhe.2024.10.033*

[416] *OECD. Global Plastics Outlook: Economic Drivers, Environmental Impacts and Policy Options. (OECD Publishing, Paris, 2022) https://doi.org/10.1787/de747aef-en*

[417] *Bailey, A. M. & Hofseth, L. J. Microplastics in Early Onset Carcinogenesis. Carcinogenesis bgaf093 (2026). https://doi.org/10.1093/carcin/bgaf093*

CELLULAR MECHANISMS: OXIDATIVE STRESS AND DNA DAMAGE

Micro- and nanoplastics are considered a potentially significant factor of cellular stress (see Part 4), as experimental data point to their ability to induce oxidative stress and DNA damage. These effects align with the mechanisms well-described in oncology, through which a chronic elevation of reactive oxygen species can sustain a microenvironment conducive to carcinogenesis (Fig. 13.3).[418,419,420,421]

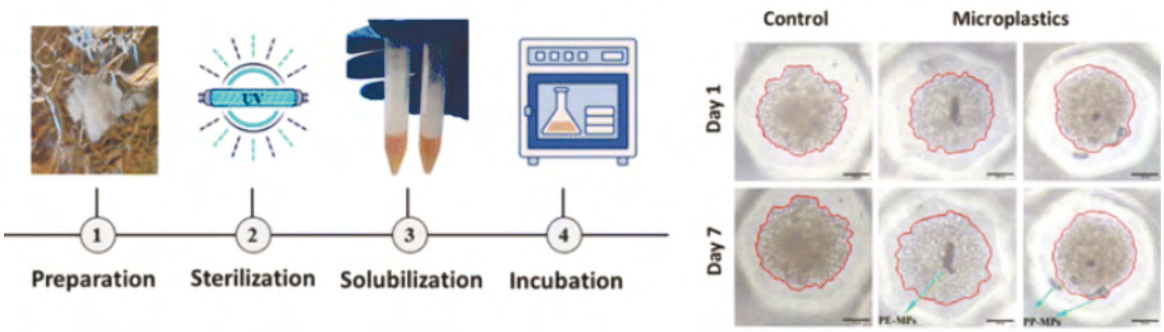


Figure 13.3.

Schematic representation of exposure of microplastics on cancer cells. Increased cancer cell proliferation in 2D models and enhanced growth of spheroids in 3D models exposed to microplastics, suggesting their potential role in tumor progression.

Source: Joseph, M. M., B. Nair, J. & Mary Joseph, A. Microscopic menace: exploring the link between microplastics and cancer pathogenesis. Environmental Science: Processes & Impacts 27, 1768–1795 (2025). https://doi.org/10.1039/D5EM00232J License: CC BY 3.0.

Modern oncology increasingly views tumor development as the result of a complex interplay of multiple mechanisms encompassing genetics,[422,423] epigenetics, intercellular communication, and environmental influences. A special role is assigned to complex disruptions in the cellular regulation system. In this context, reactive oxygen species (ROS) act as one of the pivotal factors. ROS perform physiological signaling functions and participate in regulating cellular proliferation, differentiation, and adaptive responses.[424,425]

[418] Jo, M., Nishikawa, T., Nakajima, T., et al. Oxidative stress is closely associated with tumor angiogenesis of hepatocellular carcinoma. J Gastroenterol 46, 809–821 (2011). https://doi.org/10.1007/s00535-011-0392-z
[419] Tertil, M., Jozkowicz, A. & Dulak, J. Oxidative stress in tumor angiogenesis - therapeutic targets. Curr Pharm Des 16, 3877–3894 (2010). https://doi.org/10.2174/138161210794454969
[420] Cirillo, F., Gianì, F., Sergi, V., et al. The hidden threat: Microplastics and cancer biology. Journal of Hazardous Materials 505, 141332 (2026). https://doi.org/10.1016/j.jhazmat.2026.141332
[421] Joseph, M. M., B. Nair, J. & Mary Joseph, A. Microscopic menace: exploring the link between microplastics and cancer pathogenesis. Environmental Science: Processes & Impacts 27, 1768–1795 (2025). https://doi.org/10.1039/D5EM00232J
[422] Swanton, C., Bernard, E., Abbosh, C., et al. Embracing cancer complexity: Hallmarks of systemic disease. Cell 187, 1589–1616 (2024). https://doi.org/10.1016/j.cell.2024.02.009
[423] Ciriello, G., Magnani, L., Aitken, S. J., et al. Cancer Evolution: A Multifaceted Affair. Cancer Discov 14, 36–48 (2024). https://doi.org/10.1158/2159-8290.CD-23-0530
[424] Reuter, S., Gupta, S. C., Chaturvedi, M. M., et al. Oxidative stress, inflammation, and cancer: How are they linked? Free Radic Biol Med 49, 1603–1616 (2010). https://doi.org/10.1016/j.freeradbiomed.2010.09.006
[425] Liang, X., Weng, J., You, Z., et al. Oxidative stress in cancer: from tumor and microenvironment remodeling to therapeutic frontiers. Mol Cancer 24, 219 (2025). https://doi.org/10.1186/s12943-025-02375-x

However, their chronic or excessive generation leads to the development of oxidative stress, accompanied by damage to DNA, proteins, and lipids, the disruption of signaling cascades, and the exacerbation of genetic instability.[426] A major review demonstrated that elevated ROS levels radically alter the behavior of tumor cells.[427] In such an environment, cells become more motile, divide more rapidly, penetrate adjacent tissues more readily, and are less susceptible to programmed cell death.[427] Simultaneously, ROS suppress the activity of immune cells that would normally recognize and eliminate atypical cells. As a result, a microenvironment is formed where the tumor secures multiple simultaneous advantages for growth and survival.[426,427,428]

Experimental data on animal models help to elucidate how prolonged "oxidative stress" can accelerate cancer development.[429] An elevated level of ROS can facilitate DNA damage and the more rapid accumulation of errors, from which the mutations that trigger tumor growth subsequently arise.[430] Consequently, tumors appear more frequently and in greater numbers. There are also data suggesting that enhanced ROS production can augment the proliferative capacity of tumor cells.[424] Therefore, reactive oxygen species should be viewed not solely as a marker of damage, but as a factor that can directly sustain conditions for the initiation and progression of a tumor.[425]

An additional mechanism linking ROS to tumor progression involves regulation of angiogenesis. Reactive oxygen species have shown to stimulate the growth of new blood vessels inside the tumor.[418,419] This stage is critical, as without the formation of a vascular network, the tumor's growth capacity is inherently restricted. ROS activate signaling pathways initiating the creation of this vascular network, which functionally provisions the tumor with resources and thereby supports its ongoing progression.

Collectively, the results presented in the cited sources form a coherent model: elevated ROS levels ensure that cells accumulate damage more easily, proliferate faster, survive more effectively, and successfully evade immune surveillance, while simultaneously securing support via angiogenesis. These effects must be viewed as interconnected components of a unified biological scenario explaining why chronic oxidative stress is considered a significant mechanism associated with carcinogenesis.

Thus, increased levels of reactive oxygen species (ROS) may represent one of the potential mechanistic links connecting exposure to micro- and nanoplastics with processes involved in carcinogenesis (Fig. 13.4).[431]

At the same time, it is essential to delineate the limits of the available evidence: the cited studies did not investigate nanoplastics directly. Their contribution to this rationale lies in demonstrating a fundamental principle: if a certain factor is capable of inducing

[426] *Hayes, J. D., Dinkova-Kostova, A. T. & Tew, K. D. Oxidative Stress in Cancer. Cancer Cell 38, 167–197 (2020). https://doi.org/10.1016/j.ccell.2020.06.001*

[427] *Kuo, C.-L., Ponneri Babuharisankar, A., Lin, Y.-C., et al. Mitochondrial oxidative stress in the tumor microenvironment and cancer immunoescape: foe or friend? J Biomed Sci 29, 74 (2022). https://doi.org/10.1186/s12929-022-00859-2*

[428] *Aboelella, N. S., Brandle, C., Kim, T., et al. Oxidative Stress in the Tumor Microenvironment and Its Relevance to Cancer Immunotherapy. Cancers (Basel) 13, 986 (2021). https://doi.org/10.3390/cancers13050986*

[429] *Ishikawa, K., Takenaga, K., Akimoto, M., et al. ROS-Generating Mitochondrial DNA Mutations Can Regulate Tumor Cell Metastasis. Science 320, 661–664 (2008). https://doi.org/10.1126/science.1156906*

[430] *Ohno, M., Takano, N., Hidaka, K., et al. Oxidative stress accelerates intestinal tumorigenesis by enhancing 8-oxoguanine-mediated mutagenesis in MUTYH-deficient mice. Genome Res 34, 47–56 (2024). https://doi.org/10.1101/gr.278326.123*

[431] *Ponce-Cusi, R., Barletta-Carrillo, C. & Pilco-Ferreto, N. Microplastics and Nanoplastics in Cancer Progression: Biology and Public Health. Biomedicines 14, 1 (2025). https://doi.org/10.3390/biomedicines14010001*

a sustained elevation of ROS and maintaining chronic oxidative stress in tissues, it can theoretically escalate carcinogenic risk, as it integrates into already established oncogenic pathways.

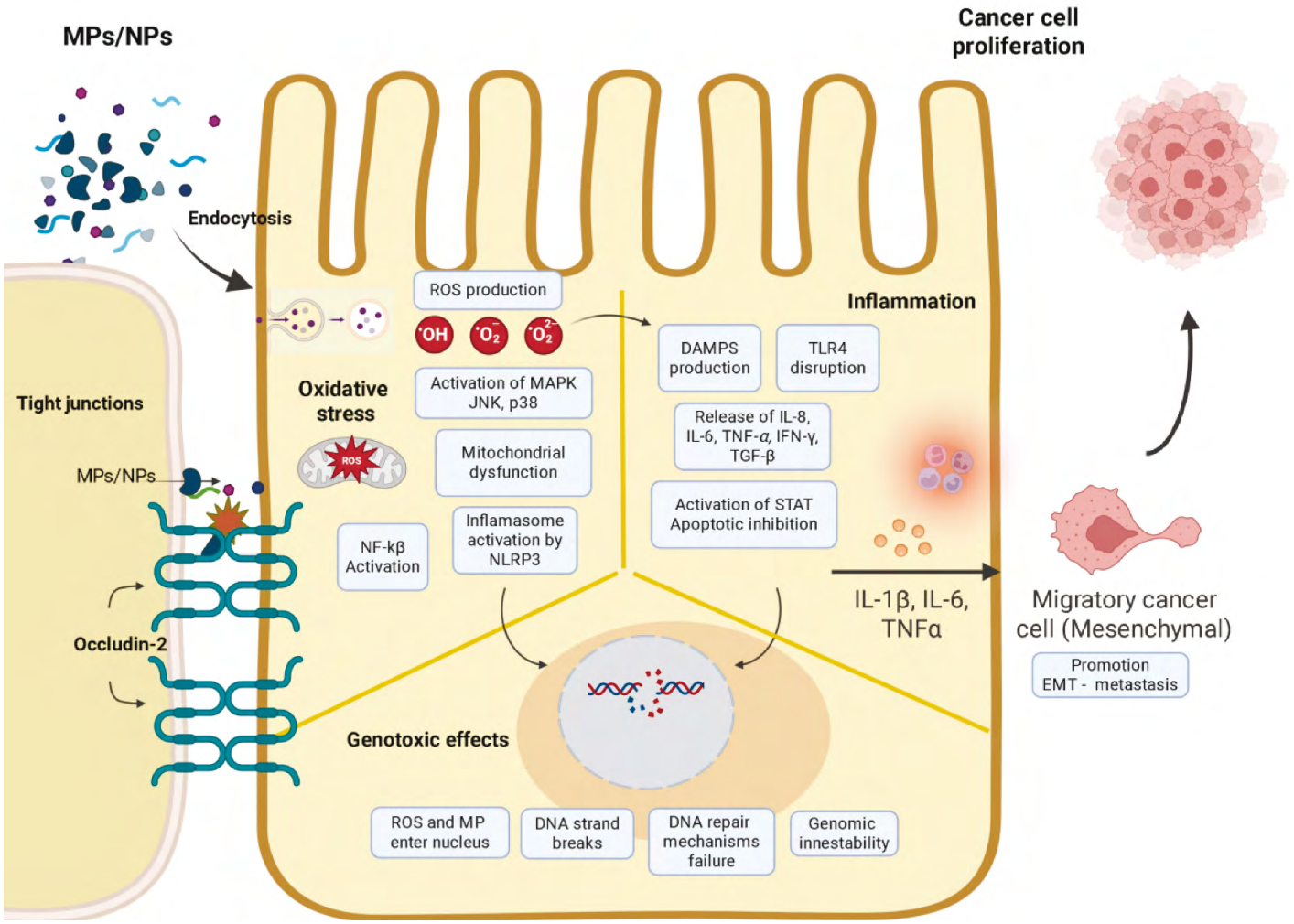


Figure 13.4.

Schematic model illustrating cellular mechanisms of MP/NP-induced carcinogenesis. MPs penetrate the epithelial barrier and are internalized by host cells, where they initiate a cascade of harmful events. In the oxidative stress pathway, MPs/NPs trigger excessive production of ROS, leading to mitochondrial dysfunction, NF-κB activation, and inflammasome stimulation via NLRP3. In parallel, chronic inflammation arises from damage-associated molecular patterns (DAMP) production and TLR4 disruption, promoting the release of proinflammatory cytokines (IL-8, IL-6, TNF-α, IFN-γ, TGF-β) and activation of the STAT pathway, which inhibits apoptosis and supports tumor-promoting signaling. Simultaneously, genotoxic effects emerge as MPs/NPs and ROS reach the nucleus, inducing DNA strand breaks, impairing repair mechanisms, and fostering genomic instability. These converging processes facilitate EMT, cancer cell migration, and ultimately, tumor proliferation.

Source: Ponce-Cusi, R., Barletta-Carrillo, C. & Pilco-Ferreto, N. Microplastics and Nanoplastics in Cancer Progression: Biology and Public Health. Biomedicines 14, 1 (2025). https://doi.org/10.3390/biomedicines14010001 License: CC BY 4.0.

In this context, micro- and nanoplastics cannot be considered a homogeneous class of particles. They form a complex array of entities whose impact on biological subjects depends on size, shape, charge, and surface chemistry.

> *"...surface charge properties of NPs play a critical role in their toxic effects and the mechanisms are generally attributed to their different accumulations and transmembrane potentials."* [432]

In evaluating biological activity, the ζ-potential of nanoparticles is used as a parameter reflecting their surface electrical properties and influence on particle-cell interactions. *In vitro* studies have shown that surface modification of particles and their charge are intrinsically linked to the level of intracellular ROS production and the onset of oxidative stress.[433] Specifically, in experiments on murine macrophage cultures, positively charged polystyrene PS-NH_2 nanoparticles induced approximately 23 times greater ROS generation than standard polystyrene particles without such surface modification at an equivalent concentration (100 µg/mL). Elevated ROS production is regarded as one of the mechanisms capable of driving the oxidative damage of biomolecules, including DNA.[433]

These processes are widely recognized as key mechanisms involved in carcinogenesis. Experimental studies indicat that the capacity of nanoplastics to induce DNA damage depends not merely on the presence of the nanoparticle, but on its physicochemical characteristics—primarily particle size and the specific type of surface functional groups.[433,435]

Consequently, oxidative stress and disruption of DNA integrity associated with nanoplastic exposure may represent plausible mechanistic pathways linked to carcinogenesis.

[432] *Wang, Y., Cong, J., Kong, X., et al. Positively Charged Nanoplastics Destruct the Structure of the PCK1 Enzyme, Promote the Aerobic Gycolysis Pathway, and Induce Hepatic Tumor Risks. Environ. Sci. Technol. 59, 3013–3023 (2025). https://doi.org/10.1021/acs.est.4c13165*

[433] *Płuciennik, K., Sicińska, P., Misztal, W., et al. Important Factors Affecting Induction of Cell Death, Oxidative Stress and DNA Damage by Nano- and Microplastic Particles In Vitro. Cells 13, 768 (2024). https://doi.org/10.3390/cells13090768*

[434] *Li, J. & Zhao, Y. Bioeffects of Nanoplastics: DNA Damage and Mechanism. Nano Lett. 25, 1660–1665 (2025). https://doi.org/10.1021/acs.nanolett.4c05794*

[435] *Feola, A., Madheswaran, M., Romano, G., et al. Polystyrene nanoparticles induce DNA damage and apoptosis in HeLa cells. Heliyon 11, e41298 (2025). https://doi.org/10.1016/j.heliyon.2024.e41298*

MITOCHONDRIAL DYSFUNCTION AS A MECHANISM OF THE ONCOGENIC IMPACT OF NANOPLASTICS

One of the key mechanisms through which nanoplastics can contribute to malignant cellular transformation is mitochondrial dysfunction, mitochondria orchestrate the cell's energy metabolism and participate in initiating programmed cell death (apoptosis); hence, their impairment is viewed as a critical step capable of elevating the probability of neoplastic development.

Review and experimental data indicate that nanoplastics can penetrate mitochondria and disrupt their operation: the accumulation of particles within the mitochondrial matrix, cristae deformation, fission/fusion imbalances, and mitochondrial membrane depolarization have all been documented.[436] As a direct consequence of impaired function of mitochondria, chronic oxidative stress ensues, which drives up the baseline of oxidative DNA damage and, under sustained exposure, amplifies the risk of genomic instability (Fig. 13.5).[437,438] Concurrently, stress and adaptive cellular mechanisms are activated, effectively sustaining the growth and survival of damaged cells.[439]

Moreover, excessive reactive oxygen species are capable of damaging both mitochondrial and nuclear DNA. Under scenarios of impaired repair mechanisms (restoration of damaged DNA), this can precipitate the accumulation of mutations alongside a concomitant decline in apoptotic efficacy. Under such conditions, there is an increased likelihood of preserving cells with genetic defects, whose division cycle should normally be blocked or terminated by apoptosis. This builds the requisite biological infrastructure for the subsequent malignant transformation of these cells.[440]

Experimental data show that when exposed to micro- and nanoplastics against a backdrop of mitochondrial stress and oxidative imbalance, a reconfiguration of energy metabolism can transpire. Specifically, glycolysis is intensified (the derivation of energy from glucose in the cytosol—the liquid matrix of the cell) alongside an increased production of lactate (lactic acid). Such metabolic shifts strongly mimic the adaptive metabolic strategies inherently characteristic of tumor cells.[441,442]

[436] Liu, H., Li, H., Yao, X., et al. Environmental nanoplastics induce mitochondrial dysfunction: A review of cellular mechanisms and associated diseases. Environmental Pollution 382, 126695 (2025). https://doi.org/10.1016/j.envpol.2025.126695

[437] Lin, S., Zhang, H., Wang, C., et al. Metabolomics Reveal Nanoplastic-Induced Mitochondrial Damage in Human Liver and Lung Cells. Environ. Sci. Technol. 56, 12483–12493 (2022). https://doi.org/10.1021/acs.est.2c03980

[438] Giri, S., Lamichhane, G., Pandey, J., et al. Micro(Nano)plastics in Human Carcinogenesis: Emerging Evidence and Mechanistic Insights. Microplastics 4, 78 (2025). https://doi.org/10.3390/microplastics4040078

[439] Chen, M., Linstra, R. & van Vugt, M. A. T. M. Genomic instability, inflammatory signaling and response to cancer immunotherapy. Biochimica et Biophysica Acta (BBA) - Reviews on Cancer 1877, 188661 (2022). https://doi.org/10.1016/j.bbcan.2021.188661

[440] Badrinath, N. & Yoo, S. Y. Mitochondria in cancer: in the aspects of tumorigenesis and targeted therapy. Carcinogenesis 39, 1419–1430 (2018). https://doi.org/10.1093/carcin/bgy148

[441] Bonanomi, M., Salmistraro, N., Porro, D., et al. Polystyrene micro and nano-particles induce metabolic rewiring in normal human colon cells: A risk factor for human health. Chemosphere 303, 134947 (2022). https://doi.org/10.1016/j.chemosphere.2022.134947

[442] Punnasseril, J. M. J., Auwal, A., Gopalan, V., et al. Metabolic Reprogramming of Cancer Cells and Therapeutics Targeting Cancer Metabolism. Cancer Medicine 14, e71244 (2025). https://doi.org/10.1002/cam4.71244

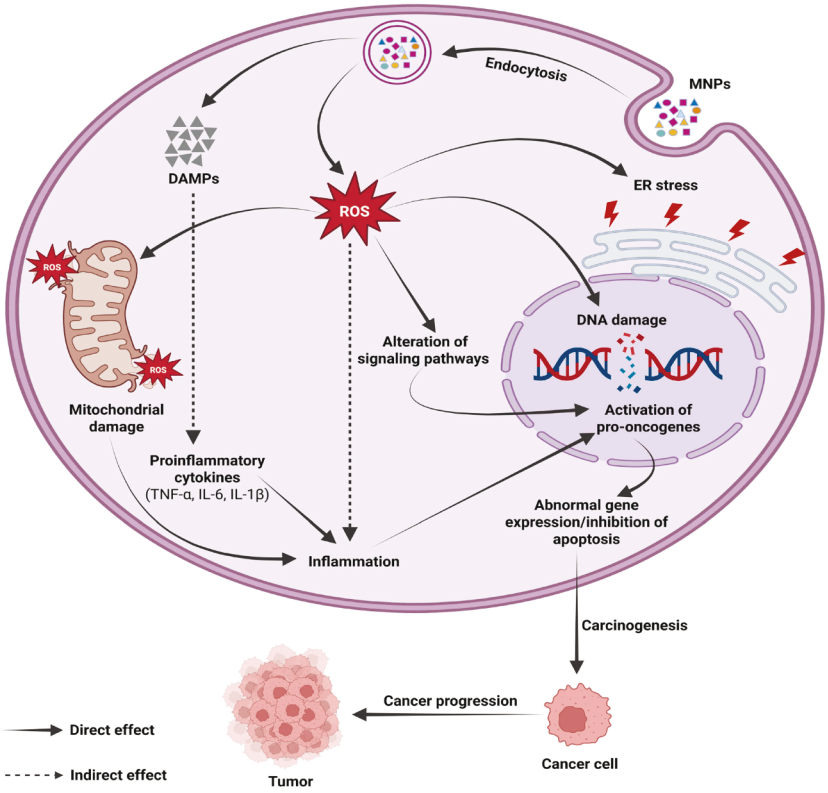


Figure 13.5.

Schematic illustration showing the mechanism by which micro(nano)plastics (MNPs) induce carcinogenesis and cancer progression. MNP particles enter the cell via clathrin- or caveolae-mediated endocytosis, macropinocytosis and elicit the generation of danger-associated membrane proteins (DAMPs). DAMPs evoke the production of pro-inflammatory cytokines such as TNF-α, IL-6, and IL-1β, leading to inflammation. Additionally, MNPs can also induce the generation of reactive oxygen species (ROS), which can lead to endoplasmic reticulum (ER) stress and mitochondrial damage, further leading to inflammation, oxidative DNA damage, and alteration in various signaling pathways like p53, MAPK (p38, JNK, ERK1/2), Nrf2, PI3K/Akt, and Wnt/β catenin. These processes can lead to a cascade of events, resulting in the activation of pro-oncogenes, abnormal gene expression, or inhibition of apoptosis, which results in carcinogenesis and tumor progression. Solid arrows represent direct effects/activation, dashed arrows indicate indirect or downstream effects.

Source: Giri, S., Lamichhane, G., Pandey, J., et al. Micro(Nano)plastics in Human Carcinogenesis: Emerging Evidence and Mechanistic Insights. Microplastics 4, 78 (2025). https://doi.org/10.3390/microplastics4040078 License: CC BY 4.0.

In tumor cells, enhanced glycolysis is not merely a consequence of a cellular defect, but a metabolic growth strategy.[443] It affords the tumor cell multiple distinct advantages:

- rapid energy acquisition, which is paramount for aggressively dividing cells.
- "Building materials" for growth—glycolytic intermediates are shunted not only for energy but also for the synthesis of nucleotides (for DNA), amino acids, and lipids (for membranes). Essentially, the cell secures raw materials for division.
- Oxygen independence. Tumors frequently grow in poorly vascularized zones. Glycolysis enables survival amidst oxygen deficiency. Acidification of the microenvironment (via lactate) suppresses immune cells, facilitating invasion and metastasis. Thus, it is a highly advantageous survival and aggressive growth strategy for the tumor.[443]

[443] *Hanahan, D. & Weinberg, R. A. Hallmarks of Cancer: The Next Generation. Cell 144, 646–674 (2011). https://doi.org/10.1016/j.cell.2011.02.013*

Therefore, factors capable of disrupting mitochondrial function, sustaining chronic oxidative stress, and attenuating apoptotic control can integrate directly into the multi-stage trajectory of carcinogenesis. In this context, nanoplastic particles possessing an electrostatic charge are considered a potential external agent which, by impacting mitochondria and cellular signaling pathways, is capable of elevating the likelihood of the retention and accumulation of DNA-damaged cells, thereby engineering conditions inherently favorable for malignant transformation and neoplasm progression.

MICRO- AND NANOPLASTICS AS A POTENTIAL FACTOR IN TUMOR METASTASIS

Micro- and nanoplastics must be evaluated not only as instigators of chronic inflammation but as a potential vector amplifying the aggressiveness of tumor cells, including their capacity to disseminate and form metastases. A possible mechanism may lie in the prolonged retention of nanoparticles within dividing cells, driving fundamental alterations in their functional activity.

Experimental studies indicate that micro- and nanoplastics (MNPs) can interact with proliferating cells, including malignant cell populations.[444] Exposure to these particles is correlated with shifts in the parameters of cell survival and migration—processes that are critical for metastasis. At the cellular level, such effects may contribute to conditions that favor tumor growth and dissemination. Study data confirm the enduring intracellular presence of nanoplastics:

> *"During cell division, particles were distributed between mother and daughter cells. Interestingly, we observed no signs of elimination from the cells."* [444]

This observation is of considerable significance as it challenges the assumption that rapidly proliferating cell populations can effectively eliminate intracellular particles through dilution during cell division. Instead, available data suggest that nanoplastics may be retained and redistributed between daughter cells across successive division cycles (Fig. 13.6).

[444] Brynzak-Schreiber, E., Schögl, E., Bapp, C., et al. Microplastics role in cell migration and distribution during cancer cell division. Chemosphere 353, 141463 (2024). https://doi.org/10.1016/j.chemosphere.2024.141463

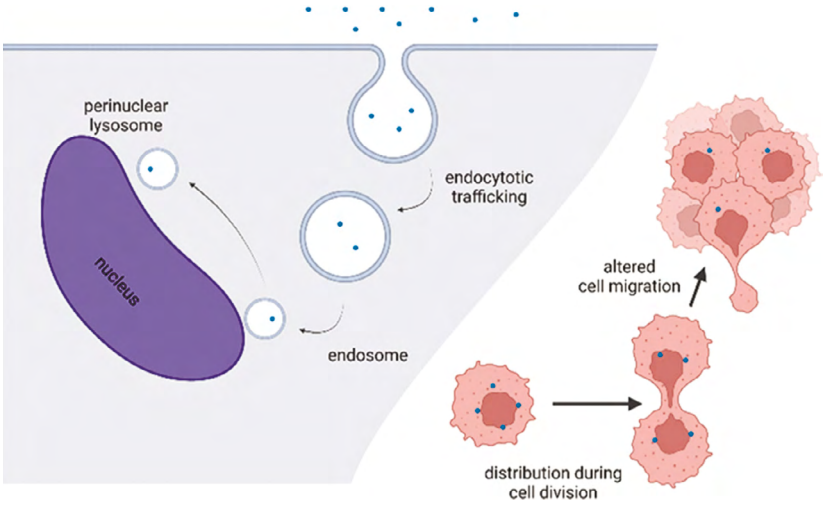


Figure 13.6.

The role of microplastics in cell migration and distribution during cancer cell division.

Source: Brynzak-Schreiber, E., Schögl, E., Bapp, C., et al. Microplastics role in cell migration and distribution during cancer cell division. Chemosphere 353, 141463 (2024). https://doi.org/10.1016/j.chemosphere.2024.141463

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Experiments demonstrated that the presence of MNPs is associated with increased cell migratory activity. Specifically, submicron polystyrene particles (~0.25 μm) augmented the motility of colorectal cancer cells in vitro.[444] Analogous effects were also documented in vivo, where enhanced cellular migration is evaluated as an explicit driver facilitating the invasion of surrounding tissues and the formation of metastases. Thus, nanoplastics may influence tumor biology not only through their persistence within cells but also by modulating cellular behaviors, including proliferation and migration, as suggested by experimental studies.[445]

THE PROBLEM OF RESISTANCE TO THERAPY

The impact of micro- and nanoplastics on cells may extend beyond tumor initiation and potentially influence the effectiveness of anticancer therapies.

Preclinical studies suggest that the presence of micro- and nanoplastics (MNPs) may attenuate the efficacy of radiation therapy. In lung cancer cell model experiments, polystyrene nanoparticles induced radioresistance via the activation of the NF-κB signaling pathway, which upregulates genes responsible for cell survival and represses programmed cell death.[446] As a result, cells may retain viability despite the presence of radiation-induced DNA damage.

[445] *Kim, Y., Park, S. Y., Jo, A., et al. Long-term low-dose exposure to polystyrene nanoplastics induces morphological and transcriptional reprogramming to enhance metastatic potential of colorectal cancer cells. Toxicology 520, 154356 (2026). https://doi.org/10.1016/j.tox.2025.154356*

[446] *Zhou, H., Liu, Y., Ren, Y., et al. Polystyrene Microplastics Induce Radiotherapy Resistance in Lung Cancer by Suppressing Ferroptosis Through NF-κB Activation. Antioxidants & Redox Signaling 15230864251389969 (2025). https://doi.org/10.1177/15230864251389969*

Additionally, MNPs may influence the efficacy of chemo- and immunotherapy.[447,448] Nanoparticles can depress the sensitivity of tumor cells to antineoplastic agents and alter the profile of the immune response, potentially shaping a microenvironment that favors the survival of cells with damaged DNA.[447,449]

Under conditions of chronic exposure, such effects could contribute to a reduced effectiveness of standard treatment strategies; however, the clinical relevance of these findings remains to be established.[449]

The aggregation of experimental and review data mandates that micro- and nanoplastics be viewed as potent potential modifiers of the therapeutic response of tumors.[450]

Taken together, available findings suggest that MNPs may act as a systemic environmental factor capable of influencing multiple stages of carcinogenesis under conditions of chronic exposure. Their effects have been described at cellular and tissue levels, including the induction of oxidative stress, DNA damage, and mitochondrial dysfunction—processes that may contribute to genomic instability and impaired apoptotic regulation. In addition, experimental data indicate potential roles in promoting a pro-inflammatory microenvironment, modulating angiogenesis, and altering cellular migration, all of which are associated with tumor progression and metastasis.

Another potentially relevant aspect is the influence of MNPs on treatment response. Preclinical studies suggest reduced efficacy of radiotherapy, chemotherapy, and immunotherapy, possibly mediated through activation of pro-survival signaling pathways in tumor cells. However, these observations require validation in clinical settings. The temporal overlap between the rapid increase in global plastic production and reported shifts in cancer epidemiology, including earlier onset in some populations, highlights the need for further investigation. Chronic exposure to micro- and nanoplastics—potentially beginning in utero—should therefore be considered a plausible environmental factor of interest, warranting systematic evaluation in the context of global oncological burden.

[447] *Pan, W., Han, Y., Zhang, M., et al. Effects of microplastics on chemo-resistance and tumorigenesis of colorectal cancer. Apoptosis 30, 1005–1020 (2025).* https://doi.org/10.1007/s10495-025-02085-1
[448] *Mishra, S. K., Sanyal, T., Kundu, P., et al. Microplastics as emerging carcinogens: from environmental pollutants to oncogenic drivers. Mol Cancer 24, 248 (2025).* https://doi.org/10.1186/s12943-025-02409-4
[449] *Hu, D., Liu, H., Guo, Y., et al. Microplastics promote chemoresistance by mediating lipid metabolism and suppressing pyroptosis in colorectal cancer. Apoptosis 30, 2287–2300 (2025).* https://doi.org/10.1007/s10495-025-02143-8
[450] *Deng, X., Gui, Y. & Zhao, L. The micro(nano)plastics perspective: exploring cancer development and therapy. Mol Cancer 24, 30 (2025).* https://doi.org/10.1186/s12943-025-02230-z

PART 14.

MICRO- AND NANOPLASTICS AS A FACTOR IN **ACCELERATED BIOLOGICAL AGING**

Micro- and nanoplastics should be regarded as a potential factor in accelerated biological aging, as their impact initiates and sustains the same cellular and molecular processes that underlie age-related tissue degradation and the development of chronic diseases.[451] Through mechanisms of oxidative stress, mitochondrial dysfunction, chronic inflammation, and genomic instability, micro- and nanoplastics may contribute to premature functional decline of the organism.[452,453]

POPULATION MANIFESTATIONS OF ACCELERATED BIOLOGICAL AGING

Despite advances in healthcare, some epidemiological data suggest less favorable health trajectories in more recent generations (Fig. 14.1). A cohort comparison in Scotland[454] — of individuals born between 1936–1945 and between 1951–1960 — revealed that the younger cohort is characterized by higher levels of multimorbidity, defined as the presence of two or more chronic diseases simultaneously, compared to their predecessors at the same age. These findings may indicate a shift of chronic diseases toward earlier onset, although the contribution of improved diagnostics and disease recognition must also be considered.

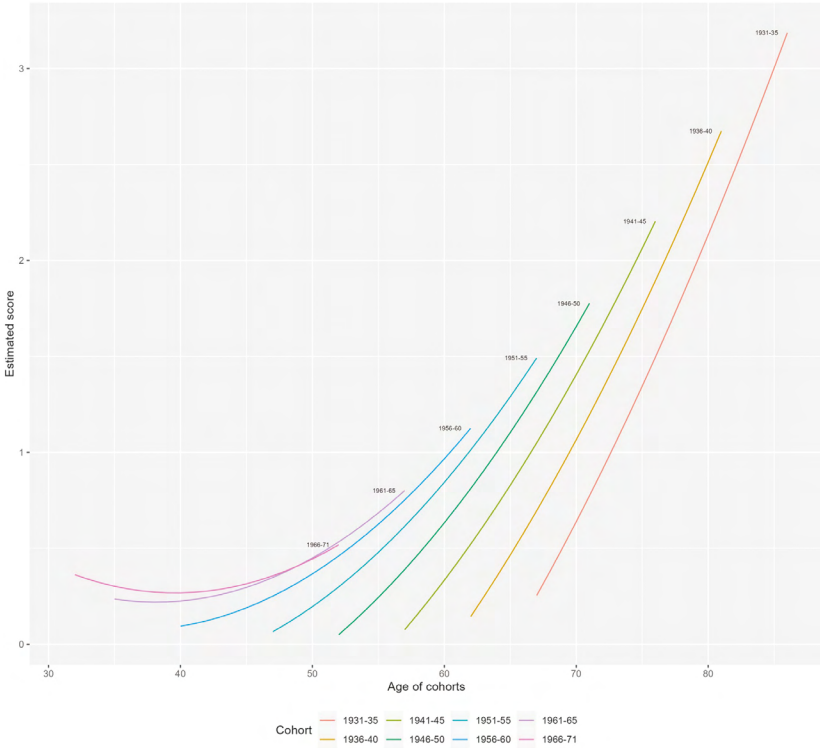


Figure 14.1.

Predicted multimorbidity scores by cohort and age.

Source: Ribe, E., Cezard, G. I., Marshall, A., et al. Younger but sicker? Cohort trends in disease accumulation among middle-aged and older adults in Scotland using health-linked data from the Scottish Longitudinal Study. European Journal of Public Health 34, 696–703 (2024). https://doi.org/10.1093/eurpub/ckae062

License: CC BY 4.0.

[451] *Bang, E., Hwangbo, H., Lee, H., et al. Exposure to polystyrene nanoplastics promotes premature cellular senescence through mitochondrial ROS production and dysfunction in pre-differentiated skeletal myoblasts. Toxicology 510, 154002 (2025). https://doi.org/10.1016/j.tox.2024.154002*

[452] *Kong, L., Li, S., Fu, Y., et al. Microplastics/nanoplastics contribute to aging and age-related diseases: Mitochondrial dysfunction as a crucial role. Food and Chemical Toxicology 199, 115355 (2025). https://doi.org/10.1016/j.fct.2025.115355*

[453] *Ali, N., Katsouli, J., Auyang, E., et al. Microplastic and nanoplastic pollution and associated potential disease risks. The Lancet Planetary Health 9, 101390 (2025). https://doi.org/10.1016/j.lanplh.2025.101390*

[454] *Ribe, E., Cezard, G. I., Marshall, A., et al. Younger but sicker? Cohort trends in disease accumulation among middle-aged and older adults in Scotland using health-linked data from the Scottish Longitudinal Study. European Journal of Public Health 34, 696–703 (2024). https://doi.org/10.1093/eurpub/ckae062*

Similar trends have been reported in several regions worldwide, although their magnitude varies across populations and disease categories. In recent decades, a growing body of epidemiological evidence indicates an earlier onset of selected non-communicable diseases (NCDs), particularly metabolic and cardiovascular conditions. Increasing prevalence of obesity, type 2 diabetes, and elevated blood pressure has been documented among adolescents and young adults. For instance, between 2014 and 2022, the proportion of adolescents with overweight and obesity increased on average from 22% to 25% among boys and from 1% to 16% among girls,[455] while the prevalence of childhood hypertension nearly doubled between 2000 and 2020.[456] A substantial increase is also noted in the incidence of type 2 diabetes in the 10–24 age group.[457]

Alongside established lifestyle determinants (poor diet quality, sedentary behavior, and chronic stress), increasing attention is being directed toward environmental exposures, including MNPs.

BIOLOGICAL AGING: WHAT OCCURS AT THE CELLULAR LEVEL

From a biological perspective, aging represents a progressive, multifactorial process that originates at the cellular and molecular levels and precedes overt clinical manifestations such as alterations in skin integrity, decreased physical performance, and hair depigmentation. It is characterized by the gradual accumulation of structural and functional alterations in cells, leading to impaired tissue homeostasis and declining organ function over time.

The main mechanisms of aging include oxidative stress,mitochondrial dysfunction,chronic inflammation, telomere shortening, accumulation of DNA damage,and epigenetic alterations.[458,459, 460,461,462] These processes are interconnected and form a unified pathogenetic complex underlying age-related tissue degradation.

[455] *OECD. Health at a Glance 2025: OECD Indicators. (OECD Publishing, 2025). https://doi.org/10.1787/8f9e3f98-en*

[456] *Zhou, J., Shan, S., Wu, J., et al. Global prevalence of hypertension among children and adolescents aged 19 years or younger: an updated systematic review and meta-analysis. The Lancet Child & Adolescent Health 10, 11–21 (2026). https://doi.org/10.1016/S2352-4642(25)00281-0*

[457] *Xu, S.-T., Sun, M. & Xiang, Y. Global, regional, and national trends in type 2 diabetes mellitus burden among adolescents and young adults aged 10–24 years from 1990 to 2021: a trend analysis from the Global Burden of Disease Study 2021. World J Pediatr 21, 73–89 (2025). https://doi.org/10.1007/s12519-024-00861-8*

[458] *Maldonado, E., Morales-Pison, S., Urbina, F., et al. Aging Hallmarks and the Role of Oxidative Stress. Antioxidants 12, 651 (2023). https://doi.org/10.3390/antiox12030651*

[459] *Al-Kufaishi, A. M. A. & Al-Musawi, N. J. T. Oxidative Stress and Related Diseases: A Comprehensive Review. Journal of Preventive, Diagnostic and Treatment Strategies in Medicine 4, 65–70 (2025). https://doi.org/10.4103/jpdtsm.jpdtsm_21_25*

[460] *Zhou, W., Qu, J., Xie, S., et al. Mitochondrial Dysfunction in Chronic Respiratory Diseases: Implications for the Pathogenesis and Potential Therapeutics. Oxidative Medicine and Cellular Longevity 2021, 5188306 (2021). https://doi.org/10.1155/2021/5188306*

[461] *Hruby, A. J. & Higuchi-Sanabria, R. Mitochondrial dysfunction in cellular senescence: a bridge to neurodegenerative disease. npj Aging 11, 99 (2025). https://doi.org/10.1038/s41514-025-00291-4*

[462] *López-Otín, C., Blasco, M. A., Partridge, L., et al. Hallmarks of aging: An expanding universe. Cell 186, 243–278 (2023). https://doi.org/10.1016/j.cell.2022.11.001*

Mitochondria are key organelles responsible for energy metabolism and the regulation of cellular functions. They play a central role in the mechanisms of cell survival and death. Their condition directly impacts disease resistance, the rate of aging, and both the length and quality of life. When mitochondrial function is impaired, the cell experiences an energy deficit and accumulates harmful metabolic byproducts, including reactive oxygen species that damage DNA and proteins. This disrupts cellular metabolism and triggers inflammatory processes.

Over time, damaged cells that no longer perform their functions accumulate in the tissues. Cells that have lost their ability to divide are not cleared away; instead, they build up in the tissues and secrete substances that sustain chronic inflammation. Simultaneously, the number of stem cells responsible for tissue renewal and repair decreases. As a result, organ function gradually declines, and the body loses its resilience to stress and disease — processes that are directly linked to the development of chronic illnesses and aging.

Functional status of mitochondria, essential organelles involved in cellular energy metabolism (Part 4), is closely associated with cellular resilience, aging-related processes, and susceptibility to disease. Impairment of mitochondrial function leads to reduced ATP production and increased generation of reactive oxygen species, resulting in oxidative damage to DNA, proteins, and lipids. These alterations disrupt cellular metabolic homeostasis and may activate pro-inflammatory signaling pathways, thereby contributing to tissue dysfunction.

With advancing age, cells exhibiting functional impairment accumulate within tissues. A subset of these cells enters a state of cellular senescence, characterized by a permanent loss of proliferative capacity and resistance to apoptosis. These senescent cells are not efficiently cleared and can accumulate over time, secreting a range of bioactive molecules collectively referred to as the senescence-associated secretory phenotype (SASP), which contributes to chronic low-grade inflammation.

Concurrently, the regenerative capacity of tissues declines, partly due to a reduction in the number and functional potential of stem and progenitor cells. These processes result in progressive impairment of tissue homeostasis, decreased organ function, and reduced resilience to physiological stressors.

Chronic conditions such as diabetes mellitus, cancer, myocardial infarction, and neurodegenerative disorders, including Alzheimer’s disease, exhibit distinct clinical manifestations but share common underlying biological mechanisms associated with aging, including cellular senescence, chronic inflammation, and impaired regenerative capacity.

ACTIVATION OF KEY BIOLOGICAL AGING MECHANISMS BY MICRO- AND NANOPLASTICS

Micro- and nanoplastics (MNPs) have been shown in experimental models to interact with biological pathways implicated in aging processes.[451,452,453]

Available in vitro and in vivo data indicate that exposure to MNPs may be associated with increased oxidative stress, DNA damage, mitochondrial dysfunction, pro-inflammatory responses, and alterations in epigenetic regulation. Some studies also suggest potential effects on telomere dynamics, although evidence remains limited (Part 4). Collectively, these findings indicate that MNPs may contribute to mechanisms of cellular damage and functional decline that are commonly associated with biological aging (Fig. 14.2).

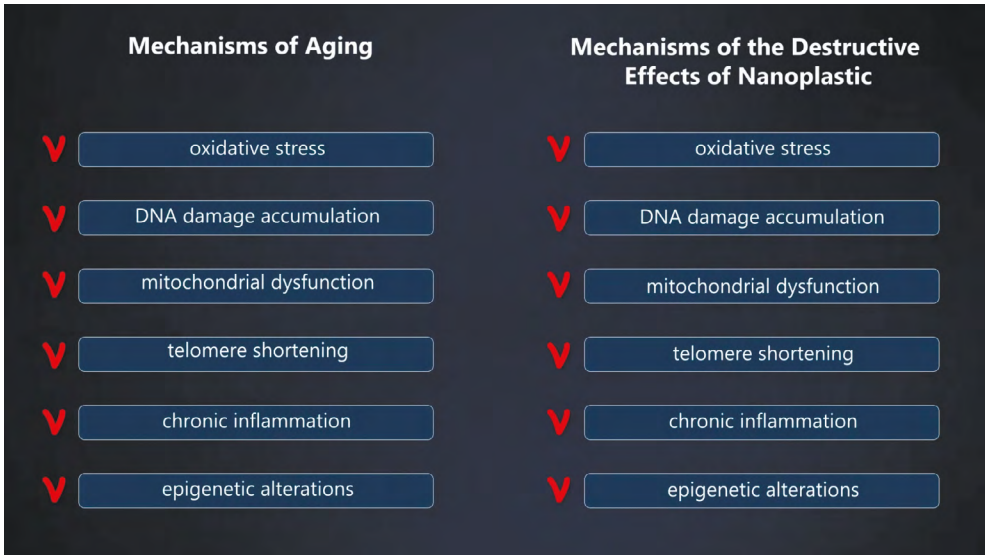


Figure 14.2.

Comparison of the key mechanisms of aging and the pathophysiological impacts of nanoplastics. Nanoplastics trigger and sustain the same processes that underlie biological aging.

Processes that typically develop over decades may, under conditions of chronic exposure to micro- and nanoplastics (MNPs), may proceed at an accelerated rate, as suggested by experimental data. This supports the consideration of MNPs as a factor potentially contributing to accelerated biological aging.

The totality of the presented data suggests a systemic link between the earlier onset of chronic non-communicable diseases and growing environmental pollution with micro- and nanoplastics. While this relationship does not establish causality, the convergence of mechanistic findings and epidemiological trends supports the consideration of MNP pollution as a potentially relevant contributing factor in the context of accelerated biological aging in contemporary populations.

PART 15.

THE INFLUENCE OF **MNPs** ON **CLIMATE PROCESSES**

Global climate change and environmental contamination by micro- and nanoplastics represent potentially interconnected processes. Due to their physicochemical, surface, and colloidal properties, MNPs may participate in the modification of heat and mass exchange processes within the ocean–atmosphere system. This allows them to be considered an additional anthropogenic factor capable of influencing the planet's thermal, hydrological, and energy balance.

THE ROLE OF OCEANS IN THE CLIMATE SYSTEM

The World Ocean is Earth's largest ecosystem and a key climate regulator. Its primary function is heat accumulation: current estimates suggest oceans absorb about 90% of the excess heat accumulating in the Earth system.[463]

The high specific heat capacity of water, combined with its role in global oceanic and atmospheric circulation, facilitates the redistribution of heat on a planetary scale. This helps to smooth out temperature extremes and maintain climatic conditions suitable for life.

The ocean also draws heat away from lithospheric plates and releases it into the atmosphere.[464] The ocean and atmosphere form a unified climate system, where seawater acts as the primary buffer mitigating sharp temperature fluctuations.

THE IMPACT OF MICRO- AND NANOPLASTICS

Since the second half of the 20th century, there has been a sharp, documented increase in the volume of plastic waste entering the global ocean.[465] This trend coincides with the period of intensive industrial development and mass production of polymeric materials (Fig. 15.1).

[463] *National Aeronautics and Space Administration (NASA). The Ocean and Climate Change. https://science.nasa.gov/earth/explore/the-ocean-and-climate-change/ (Accessed 20 March 2026).*
[464] *Schmeling, H., Marquart, G. & Nawa, V. The role of hydrothermal cooling of the oceanic lithosphere for ocean floor bathymetry and heat flow. JGR Solid Earth 122, 3934–3952 (2017). https://doi.org/10.1002/2016JB013881*
[465] *Ostle, C., Thompson, R. C., Broughton, D., et al. The rise in ocean plastics evidenced from a 60-year time series. Nat Commun 10, 1622 (2019). https://doi.org/10.1038/s41467-019-09506-1*

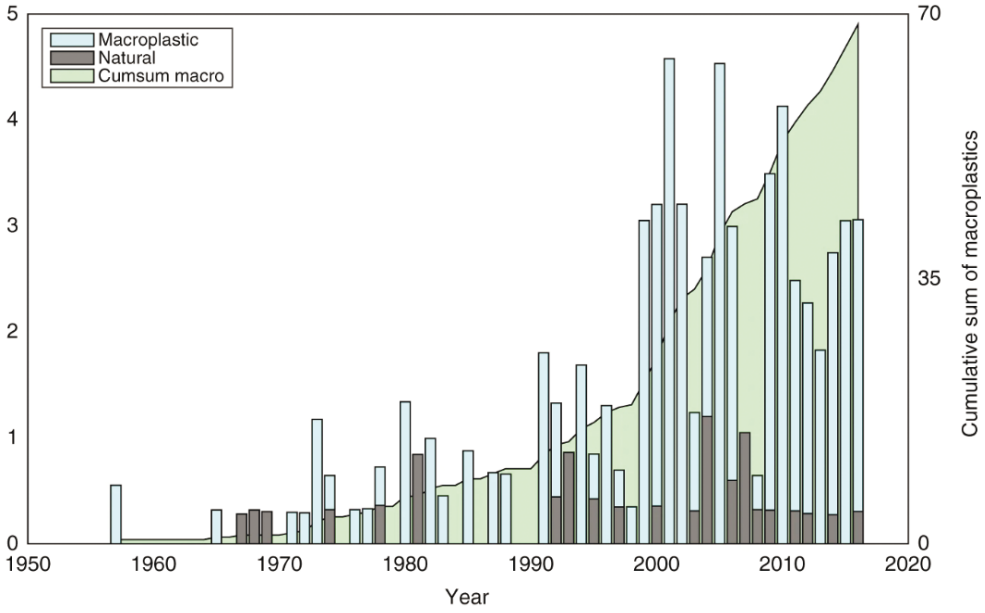


Figure 15.1.

Annual counts of macroplastic = light blue bars (significant trend = 0.057 ± 0.0066 (1S.D.)), and natural items = grey bars (non-significant trend = 0.0045 ± 0.0028 (1S.D.)). The cumulative sum of the total counts of macroplastic are plotted on the second y-axis in green.

Source: Ostle, C., Thompson, R. C., Broughton, D., et al. The rise in ocean plastics evidenced from a 60-year time series. Nat Commun 10, 1622 (2019). https://doi.org/10.1038/s41467-019-09506-1 License: CC BY 4.0.

During that same period (from 1960 to 2025), a pronounced change in the ocean's thermal state was observed (Fig. 15.2): monitoring data demonstrate a steady rise in mean ocean water temperatures and an increase in the total ocean heat content.[466]

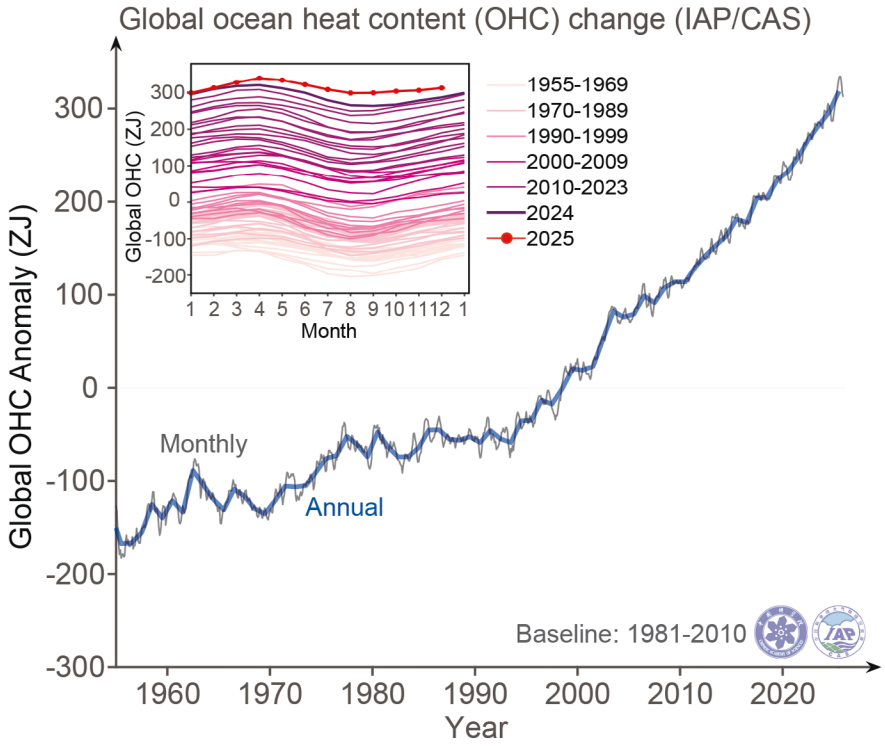


Figure 15.2.

Global upper 2000 m ocean heat content (OHC) changes from 1955 through 2025 (units: ZJ). The thick lines denote the annual values, while the thin lines represent the monthly values. The OHC anomalies are relative to the 1981–2010 baseline. The inner box shows the intra-annual variation of OHC, with 2025 values highlighted in red.

Source: Pan, Y., Cheng, L., Abraham, J., et al. Ocean Heat Content Sets Another Record in 2025. Adv. Atmos. Sci. (2026). https://doi.org/10.1007/s00376-026-5876-0

License: CC BY 4.0.

[466] Pan, Y., Cheng, L., Abraham, J., et al. Ocean Heat Content Sets Another Record in 2025. Adv. Atmos. Sci. (2026). https://doi.org/10.1007/s00376-026-5876-0

Comparison of these two time series reveals their parallel dynamics and suggests a potential correlation between increased plastic pollution and rising ocean temperatures.

Although this proposed correlation does not fully explain the unprecedented rate of ocean warming, it does underscore the need to view plastic not merely as an environmental pollutant but as a potential factor capable of amplifying thermal transformation processes in the oceanic environment.

This approach reframes the issue of plastic pollution within a broader interdisciplinary context that encompasses physicochemical and climatic interactions.

A more detailed analysis of the causes of ocean heating requires a comprehensive evaluation of the factors influencing Earth's energy balance, atmospheric dynamics, and the state of the ocean system. Furthermore, it is essential to account for the contribution of geodynamic processes, including geothermal heat flux and tectonic activity. These issues are examined in detail in the report, "ON THE PROGRESSION OF CLIMATE CATACLYSMS ON EARTH AND THEIR CATASTROPHIC CONSEQUENCES" (Fig. 15.3).[467]

Climate Report: **ON THE PROGRESSION OF CLIMATE CATACLYSMS ON EARTH AND THEIR CATASTROPHIC CONSEQUENCES**

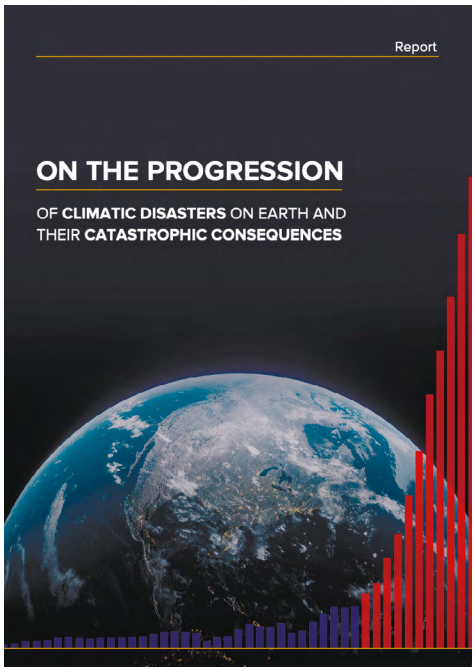


Figure 15.3.

Climate Report: On The Progression Of Climate Cataclysms On Earth And Their Catastrophic Consequences.

Source: ALLATRA International Public Movement. Climate Report. https://allatra.org/storage/app/media/reports/en/Climate_Report.pdf (Accessed 20 March 2026).

[467] *ALLATRA International Public Movement. Climate Report: On The Progression Of Climate Cataclysms On Earth And Their Catastrophic Consequences. (2024). https://allatra.org/storage/app/media/reports/en/Climate_Report.pdf (Accessed 20 March 2026)*

MECHANISMS OF MNP INTERACTION WITH THE AQUEOUS ENVIRONMENT

Large-scale ocean pollution by micro- and especially nanoplastics leads to interference in interfacial processes occurring in the aqueous environment.

Plastic nanoparticles can acquire a surface electric charge as the result of oxidation and the adsorption of seawater ions (Na^+, Mg^{2+}, Ca^{2+}, Cl^-, SO_4^{2-}, etc.). Charged particles form hydration shells around themselves—ordered structures of water molecules stabilized by hydrogen bonds (Fig. 15.4).[468]

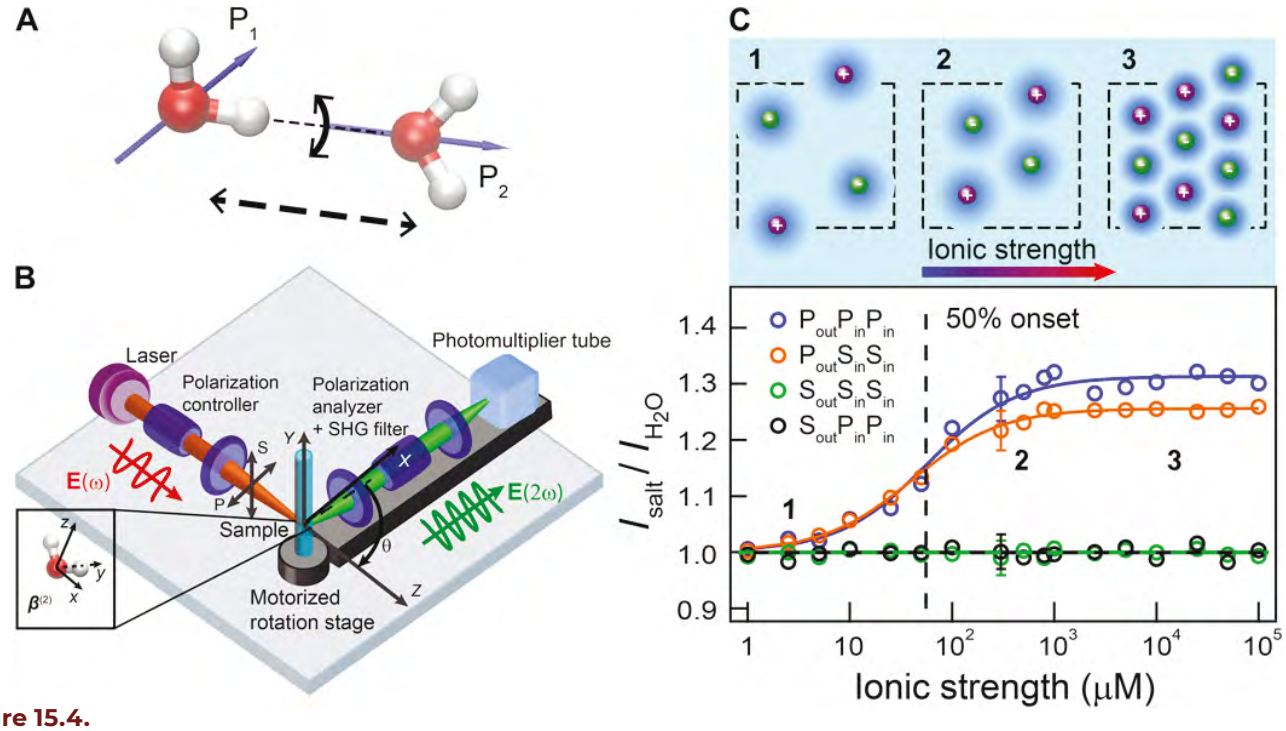


Figure 15.4.

Fig. 1 Snapshots of long-range perturbations in aqueous NaCl solutions. (A) Illustration of two H-bonded water molecules that are orientationally correlated. The black arrows represent different axes along which H-bonds can be broken. Femtosecond elastic second harmonic scattering (fs-ESHS) is only sensitive to the breaking of this H-bond via rotation (black curved arrow). Nuclear quantum effects predict that the H-bond bending mode is stronger, whereas the H-bond stretching mode is weaker, for D_2O compared to H_2O. (B) Sketch of the fs-ESHS experiment. fs-ESHS intensities are recorded at a scattering angle (θ) of 90°. P(S) refers to a polarization direction parallel (perpendicular) to the scattering plane. (C) Top: Illustration of the different regimes probed in the experiment. At low ionic strengths (1), each ion induces long-range structural water correlations in its vicinity, forming a water-ordered domain. At higher ionic strengths, more domains appear. These domains (2) start to overlap and (3) interfere with one another, leading to a saturation of the observed signal. Bottom: fs-ESHS intensities, relative to that of H_2O, measured from NaCl solutions. Four different polarization combinations were measured: $P_{out}P_{in}P_{in}$, $P_{out}S_{in}S_{in}$, $S_{out}P_{in}P_{in}$, and $S_{out}S_{in}S_{in}$. Only the PoutPinPin and PoutSinSin intensities change with increasing NaCl concentrations.

Source: Chen, Y., Okur, H. I., Gomopoulos, N., et al. Electrolytes induce long-range orientational order and free energy changes in the H-bond network of bulk water. Sci. Adv. 2, e1501891 (2016). https://doi.org/10.1126/sciadv.1501891 License: CC BY-NC 4.0.

[468] *Chen, Y., Okur, H. I., Gomopoulos, N., et al. Electrolytes induce long-range orientational order and free energy changes in the H-bond network of bulk water. Sci. Adv. 2, e1501891 (2016). https://doi.org/10.1126/sciadv.1501891*

Near the surface of polymers containing polar functional groups (such as nylon and PET), a localized restructuring of the hydrogen bond network occurs, highlighting the specific nature of their interfacial interactions.[469,470]

The formation of hydration shells alters the mobility of water molecules. Molecules incorporated into these structures are less effective at transferring heat, which locally reduces the effective specific heat capacity and thermal conductivity of the aqueous medium. In the presence of nanoplastics, water heats up faster and dissipates heat less effectively,[471] forming microzones of overheating. When scaled across vast volumes of water, this effect potentially creates the conditions for the accelerated accumulation of heat within the ocean.

Observed changes in the ocean's thermal state are consistent with the intensification of such processes.

Recent studies indicate that ocean warming is occurring not only at the surface but also in the deep ocean layers. Scientists at NOAA have determined that in the Argentine Basin, at depths exceeding 4,500 meters, a warming rate of 0.02°C per decade was recorded between 2009 and 2019.[472] Heating such massive volumes of deep water requires a colossal amount of energy.

A similar pattern is observed in waters south of the Antarctic Circle. German scientists have found that in the Weddell Sea off the coast of West Antarctica, the upper 700 meters experience almost no warming, whereas the average rate of warming below 2,000 meters is approximately five times greater than the global ocean average.[473]

This indicates that current warming trends are affecting not only the surface layers directly exposed to solar radiation, but also the deep ocean regions, where thermal changes are driven by the redistribution of energy within the oceanic system itself.

Even at low concentrations, nanoplastics can potentially impact vast volumes of water due to their surface area and electrostatic charge. Their interfaces readily adsorb and exchange ions, forming electrostatically active surfaces that influence the structure and dynamics of surrounding water. According to research, a single ion can influence approximately one million water molecules, and particles with high surface charge and adsorbed ions amplify this effect.[468] Therefore, the scale of impact is determined not only by the mass of pollution but also by the physicochemical activity of the particles.

[469] *Eslami, H. & Müller-Plathe, F. Molecular Dynamics Simulation of Water Influence on Local Structure of Nanoconfined Polyamide-6,6. J. Phys. Chem. B 115, 9720–9731 (2011).* https://doi.org/10.1021/jp204423z

[470] *Myalitsin, A., Ghosh, S., Urashima, S., et al. Structure of water and polymer at the buried polymer/water interface unveiled using heterodyne-detected vibrational sum frequency generation. Phys. Chem. Chem. Phys. 22, 16527–16531 (2020).* https://doi.org/10.1039/D0CP02618B

[471] *Kirthika, V., Galpaya, C., Induranga, A., et al. Influence of polyethylene microplastics on thermal properties of water and seawater: A novel detection method for microplastics and nanoplastics. Journal of Water Process Engineering 83, 109601 (2026).* https://doi.org/10.1016/j.jwpe.2026.109601

[472] *Meinen, C. S., Perez, R. C., Dong, S., et al. Observed Ocean Bottom Temperature Variability at Four Sites in the Northwestern Argentine Basin: Evidence of Decadal Deep/Abyssal Warming Amidst Hourly to Interannual Variability During 2009–2019. Geophysical Research Letters 47, e2020GL089093 (2020).* https://doi.org/10.1029/2020GL089093

[473] *Strass, V. H., Rohardt, G., Kanzow, T., et al. Multidecadal Warming and Density Loss in the Deep Weddell Sea, Antarctica. Journal of Climate 33, 9863–9881 (2020).* https://doi.org/10.1175/JCLI-D-20-0271.1

THE MEDITERRANEAN SEA AS A MODEL OF MNP CONCENTRATION INFLUENCE ON THE MARINE THERMAL REGIME

The large-scale presence of nanoplastic particles capable of carrying a surface charge may be considered a potential additional factor influencing interfacial processes in the "water–atmosphere" system. At high concentrations of charged MNP particles in confined marine basins, their participation in colloidal and aerosol interactions could theoretically reflect on local heat and mass exchange processes.

It is this mechanism that is viewed as a possible factor requiring further study of accelerated warming in specific regions.

The Mediterranean region is an illustrative example of how high micro- and nanoplastic concentrations may be linked to changes in the marine thermal regime. Beyond the 5 known oceanic accumulation zones, researchers identify a sixth "garbage patch"[474] located in the Mediterranean Sea (Fig. 15.5).[475]

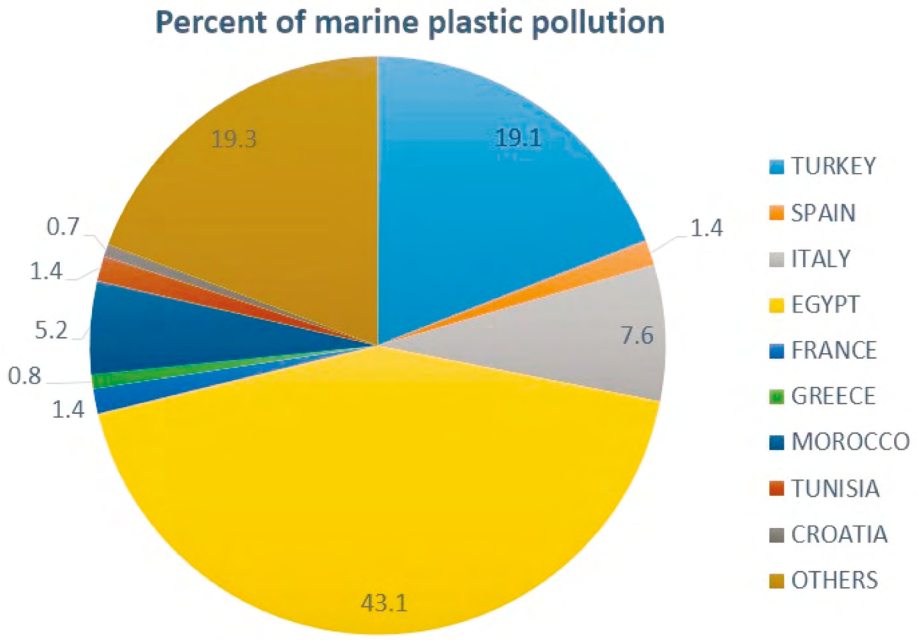


Figure 15.5.

Percentage of plastic leakage into the Mediterranean Sea.

Source: Cantasano, N. Marine Pollution by Microplastics in the Mediterranean Sea. JMSE 10, 858 (2022). https://doi.org/10.3390/jmse10070858

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It is termed "invisible" because the bulk of pollution consists not of large fragments but of micro- and nanoplastics.

According to studies, the concentration of MNPs in this region is approximately four times higher than in the North Pacific Ocean,[476] where the Great Pacific Garbage Patch is located. Thus, the Mediterranean basin is developing an environment with an exceptionally high density of fine plastic particles.[477]

[474] *Cantasano, N. Marine Pollution by Microplastics in the Mediterranean Sea. JMSE 10, 858 (2022). https://doi.org/10.3390/jmse10070858*

[475] *Bohdan, K. Estimating global marine surface microplastic abundance: systematic literature review. Science of The Total Environment 832, 155064 (2022). https://doi.org/10.1016/j.scitotenv.2022.155064*

[476] *Sharma, S., Sharma, V. & Chatterjee, S. Microplastics in the Mediterranean Sea: Sources, Pollution Intensity, Sea Health, and Regulatory Policies. Front. Mar. Sci. 8, 634934 (2021). https://doi.org/10.3389/fmars.2021.634934*

[477] *Liubartseva, S., Coppini, G., Lecci, R., et al. Tracking plastics in the Mediterranean: 2D Lagrangian model. Marine Pollution Bulletin 129, 151–162 (2018). https://doi.org/10.1016/j.marpolbul.2018.02.019*

Given that micro- and nanoparticles are capable of carrying a surface charge and participating in interfacial interactions, their possible influence on physicochemical processes in the "water–atmosphere" system requires further study.

Under conditions of accelerated regional warming, this mechanism may be considered a potential additional factor; however, its contribution to the energy balance requires quantitative verification.

> *"As a climate hotspot warming at a rate 20% faster than the global average, the Mediterranean faces unprecedented challenges that threaten its ecosystems, economies, and communities."* [478]

The accelerated warming of the region is already manifesting in an increase in climatic anomalies, more frequent extreme weather events, and a higher risk of natural disasters.

Meanwhile, the MNP content in the environment continues to increase.

GLOBAL DISTRIBUTION OF MNPs

The spatial distribution of MNPs exhibits a systemic nature. Ocean currents facilitate the global transport of these particles (Fig. 15.6):[479] lighter fractions remain suspended in the surface layers, while denser ones settle on the bottom or accumulate in thermocline zones,[480] where sharp temperature and density gradients form stable layers with elevated MNP concentrations.

[478] *Union for the Mediterranean (UfM). Scientific reports on climate and environmental change in the Mediterranean. https://ufmsecretariat.org/what-we-do/energy-and-climate-action/medecc-scientific-reports-on-climate-and-environmental-change-in-the-mediterranean (Accessed 20 March 2026)*

[479] *Zhao, S., Kvale, K. F., Zhu, L., et al. The distribution of subsurface microplastics in the ocean. Nature 641, 51–61 (2025). https://doi.org/10.1038/s41586-025-08818-1*

[480] *Uurasjärvi, E., Pääkkönen, M., Setälä, O., et al. Microplastics accumulate to thin layers in the stratified Baltic Sea. Environmental Pollution 268, 115700 (2021). https://doi.org/10.1016/j.envpol.2020.115700*

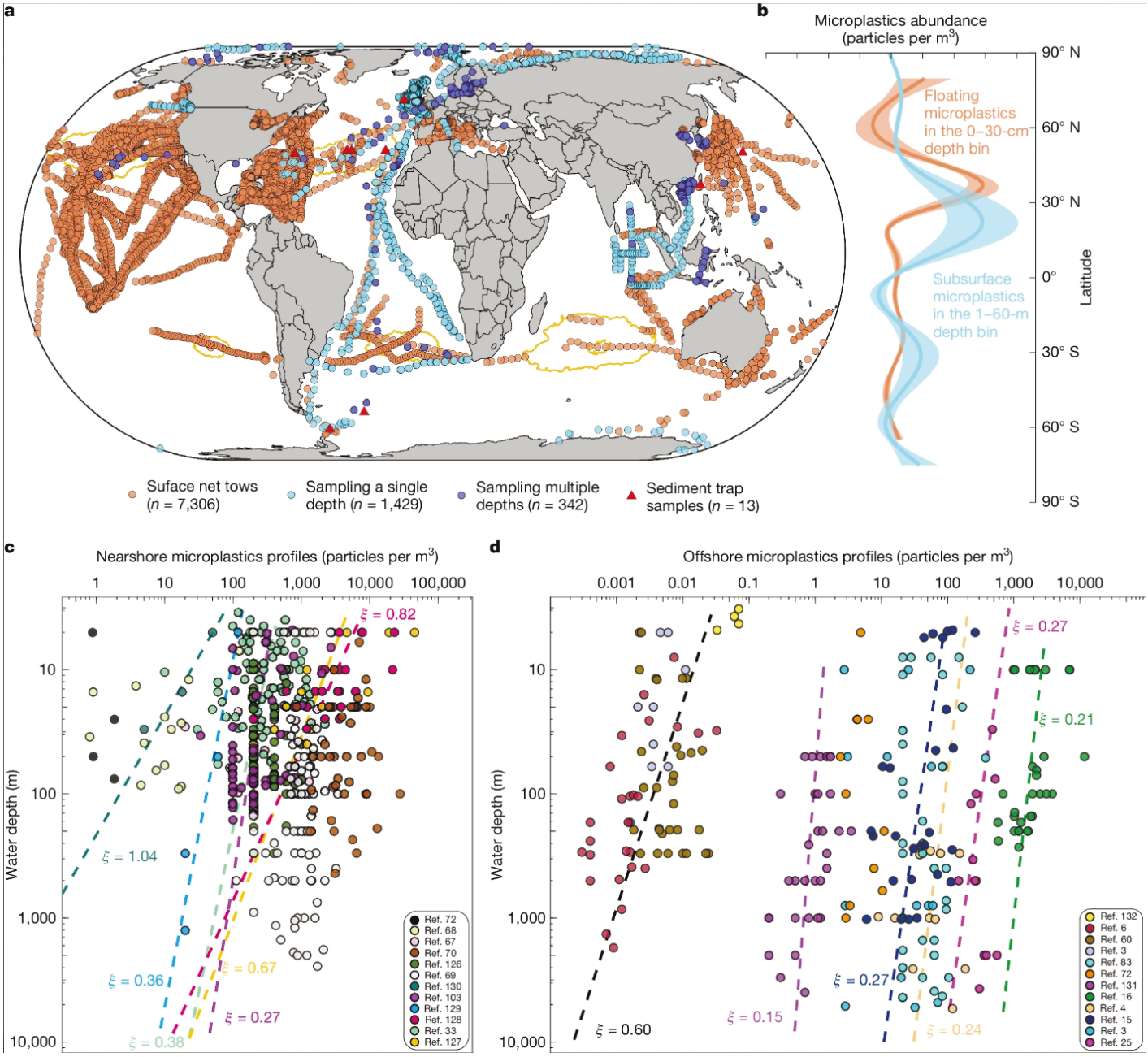


Figure 15.6.

a, Global observation stations of marine microplastics collected through surface net tows (orange dots), at a single depth beneath the surface using various techniques (light blue dots), from multiple water depths (dark blue dots), and from sediment traps (red triangles). The solid yellow lines represent the predicted boundaries of the offshore convergence zones, where floating plastic debris accumulates. **b,** Latitudinal trends in the adjusted large microplastics, residing within the 1–60-m depth bin (light blue line, subsurface microplastic data collected at a single depth) and those floating microplastics at the sea surface (0–50 cm, orange line). Only the trends of coloured lines are considered here. The coloured shaded areas denote ±1 s.d. from 1,000 Monte Carlo runs that used varying predictors for the Generalized Additive Models (GAMs). **c,d,** Depth profiles of microplastic abundances (particles per m^3) observed in nearshore **(c)** and offshore **(d)** waters with the log–log regression fits (dashed lines) between microplastics abundances and water depth of individual studies. The exponent (ξ), also referred to as the slope, provides information on the rate of abundance change along the depth gradients.

Source: Zhao, S., Kvale, K. F., Zhu, L., et al. The distribution of subsurface microplastics in the ocean. Nature 641, 51–61 (2025). https://doi.org/10.1038/s41586-025-08818-1 License: CC BY-NC-ND 4.0.

THE INFLUENCE OF MICRO- AND NANOPLASTICS ON OCEAN ACIDIFICATION

Furthermore, the leaching of plastics can alter seawater biogeochemistry through the release of dissolved organic matter and other compounds. The abiotic plastic degradation processes under solar radiation lead to the formation of weakly acidic products and CO_2, which may contribute to a local reduction in seawater pH. This effect is particularly pronounced in plastics that have already undergone degradation and structural changes in the ocean environment, as these aged fragments possess a larger specific surface area and elevated physicochemical reactivity.[481]

A decrease in pH in aquatic environments is frequently accompanied by an increase in dissolved organic carbon, driven by the leaching of organic compounds from plastic particles. The more dissolution and leaching products are generated, the more pronounced the acidification processes become, as dissolved organic acids and CO_2 in the aquatic environment mutually reinforce their impact on acid-base balance. Thus, plastic in the marine environment may not only serve as a pollution vector but also actively participate in altering the water's chemical composition and biogeochemical parameters, including acidity (Fig. 15.7).[481]

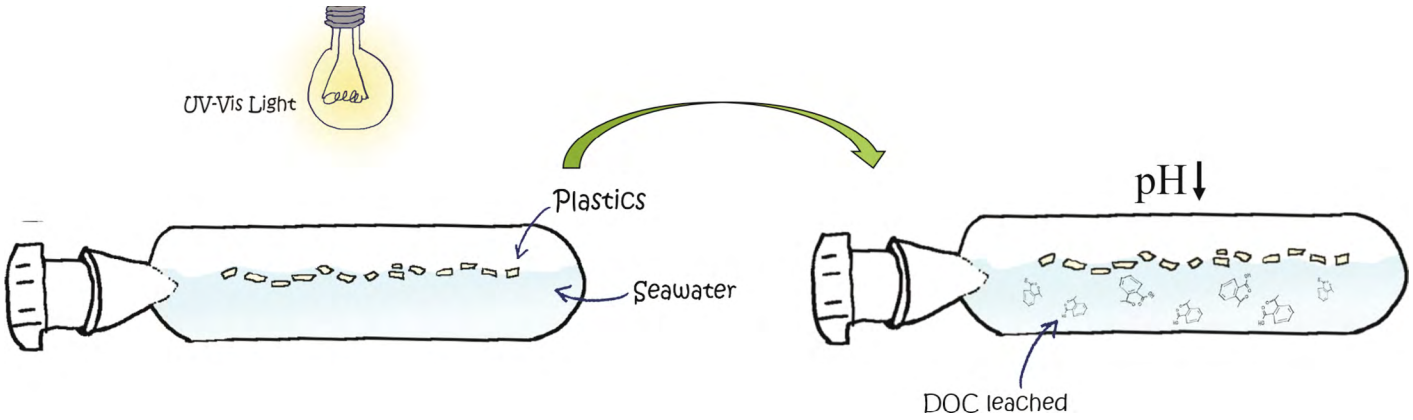


Figure 15.7.

Plastics leaching can lead to a decrease in seawater pH by up to 0.5 units.

Source: Romera-Castillo, C., Lucas, A., Mallenco-Fornies, R., et al. Abiotic plastic leaching contributes to ocean acidification. Science of The Total Environment 854, 158683 (2023). https://doi.org/10.1016/j.scitotenv.2022.158683

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[481] *Romera-Castillo, C., Lucas, A., Mallenco-Fornies, R., et al. Abiotic plastic leaching contributes to ocean acidification. Science of The Total Environment 854, 158683 (2023). https://doi.org/10.1016/j.scitotenv.2022.158683*

Microplastics can disrupt the function of the biological carbon pump,leading to reduced phytoplankton photosynthesis, altered zooplankton feeding, and a slowdown in the sinking of carbon into the deep ocean.[482]

Additionally, microplastic particles can affect albedo: darker MNP fragments can absorb more solar radiation, potentially causing a localized increase in temperature.

Experiments simulating beach conditions have shown that microplastics can alter the temperature profile of the sand, increasing the temperature by approximately 0.017°C per 1% v/v microplastic added (Fig. 15.8).[483]

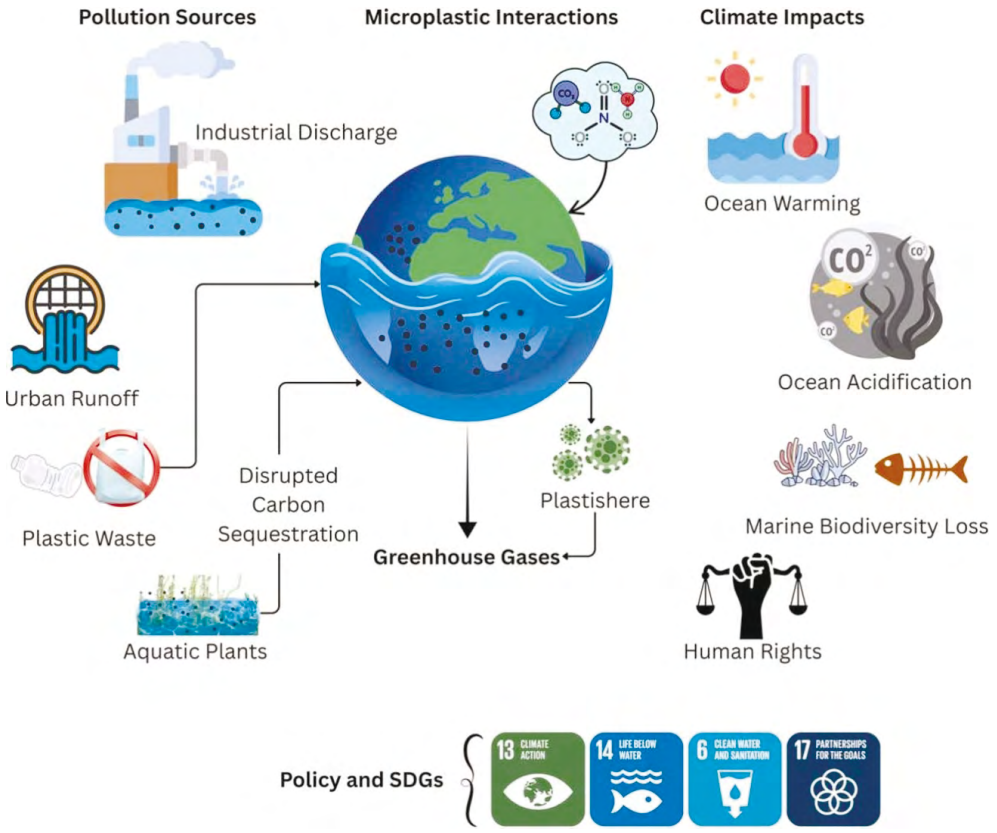


Figure 15.8.

Schematic representation of the impact of microplastics on climatic and biogeochemical processes in the ocean. Microplastic particles can exacerbate global warming and ocean acidification, as well as contribute to a decline in marine biodiversity. MNP pollution hinders progress toward multiple United Nations Sustainable Development Goal (UN SDG) targets.

Source: Nawab, A., Khan, M. T., Ihsanullah, I., et al. From pollution to ocean warming: The climate impacts of marine microplastics. Journal of Hazardous Materials: Plastics 2, 100032 (2026). https://doi.org/10.1016/j.hazmp.2025.100032

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At the same time, since the mid-20th century, the ocean has lost about 1–2% of its dissolved oxygen,[484] and approximately 700 "dead zones"[485] have formed in marine ecosystems worldwide.

This indicates a disruption of gas exchange and biogeochemical cycles accompanying the thermal and chemical transformation of the marine environment.

[482] *Shen, M., Ye, S., Zeng, G., et al. Can microplastics pose a threat to ocean carbon sequestration? Marine Pollution Bulletin 150, 110712 (2020). https://doi.org/10.1016/j.marpolbul.2019.110712*
[483] *Nawab, A., Khan, M. T., Ihsanullah, I., et al. From pollution to ocean warming: The climate impacts of marine microplastics. Journal of Hazardous Materials: Plastics 2, 100032 (2026). https://doi.org/10.1016/j.hazmp.2025.100032*
[484] *Stramma, L. & Schmidtko, S. Spatial and Temporal Variability of Oceanic Oxygen Changes and Underlying Trends. Atmosphere-Ocean 59, 122–132 (2021). https://doi.org/10.1080/07055900.2021.1905601*
[485] *Limburg, K. E., Breitburg, D., Swaney, D. P., et al. Ocean Deoxygenation: A Primer. One Earth 2, 24–29 (2020). https://doi.org/10.1016/j.oneear.2020.01.001*

INFLUENCE OF MICRO- AND NANOPLASTICS ON THE ATMOSPHERE

Ocean heating intensifies evaporation and increases atmospheric humidity, which, in turn, amplifies evaporation and disrupts atmospheric circulation. Consequently, atmospheric temperature and humidity rise. For every 1°C increase in temperature, the atmosphere can hold approximately 7% more water vapor.[486] Along with evaporating moisture, aerosol particles, including MNPs, rise into the atmosphere (Fig. 16.9).[487,488] Once in the atmosphere, these particles can serve as highly effective condensation nuclei, around which water droplets, ice crystals, and hail form more rapidly.[489,490]

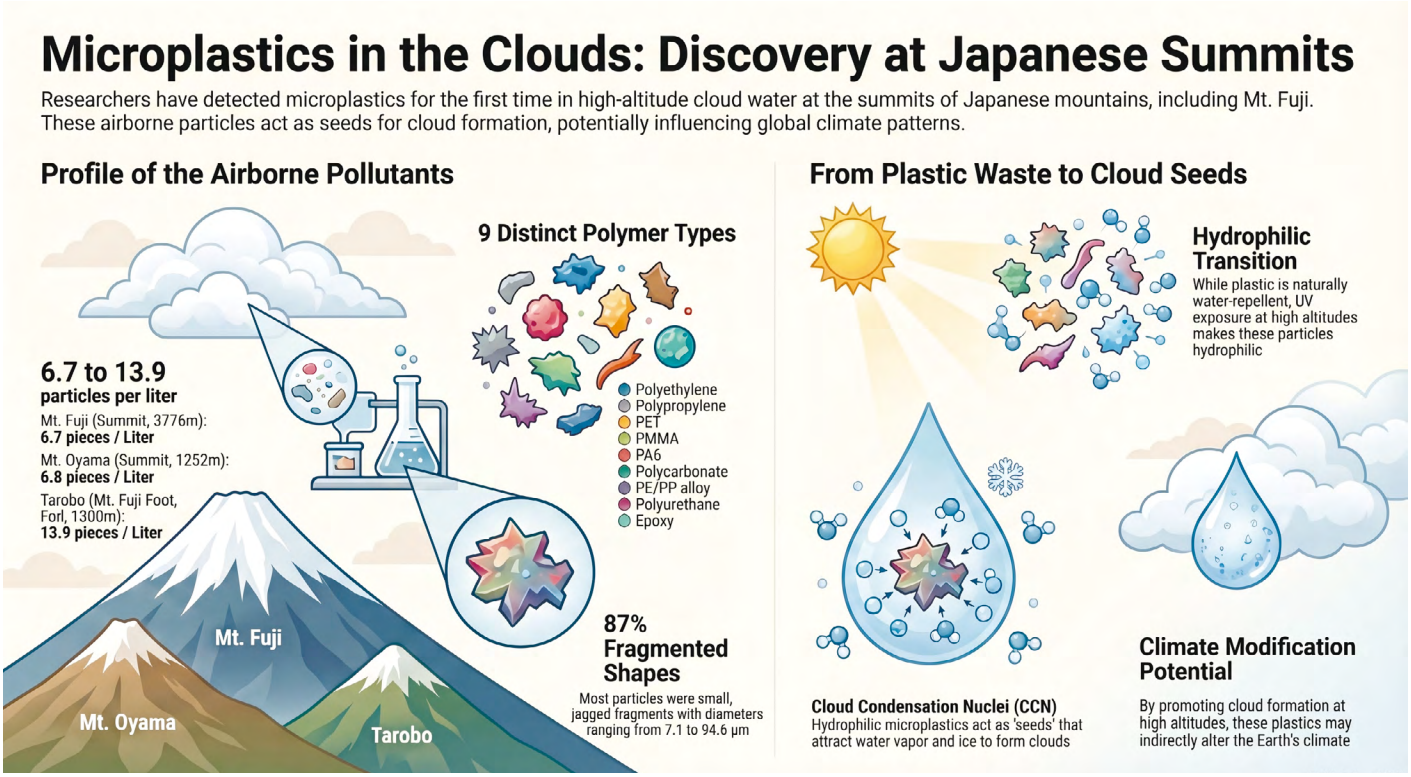


Figure 15.9.

Airborne hydrophilic microplastics in cloud water at high altitudes and their role in cloud formation.

Data source: Wang, Y., Okochi, H., Tani, Y., et al. Airborne hydrophilic microplastics in cloud water at high altitudes and their role in cloud formation. Environ Chem Lett 21, 3055–3062 (2023). https://doi.org/10.1007/s10311-023-01626-x

[486] *National Aeronautics and Space Administration (NASA). Steamy Relationships: How Atmospheric Water Vapor Amplifies Earth's Greenhouse Effect. https://science.nasa.gov/earth/climate-change/steamy-relationships-how-atmospheric-water-vapor-amplifies-earths-greenhouse-effect/ (Accessed 20 March 2026)*

[487] *Waseda University. Plastic Cloud: New Study Analyzes Airborne Microplastics in Clouds. (2023). https://www.waseda.jp/top/en/news/78501 (Accessed 20 March 2026)*

[488] *Wang, Y., Okochi, H., Tani, Y., et al. Airborne hydrophilic microplastics in cloud water at high altitudes and their role in cloud formation. Environ Chem Lett 21, 3055–3062 (2023). https://doi.org/10.1007/s10311-023-01626-x*

[489] *Giles, R. H. Atmospheric Electricity and Cloud Microphysics (2002)*

[490] *Aeschlimann, M., Li, G., Kanji, Z. A., et al. Potential impacts of atmospheric microplastics and nanoplastics on cloud formation processes. Nat. Geosci. 15, 967–975 (2022). https://doi.org/10.1038/s41561-022-01051-9*

As a result, the freezing of supercooled droplets can initiate at higher temperatures (4–10°C higher compared to homogeneous ice nucleation) and, consequently, occur in the lower layers of the atmosphere.[491]

> *"Throughout the past two decades of research into microplastics, scientists have been finding that they're everywhere, so this is another piece of that puzzle," "It's now clear that we need to have a better understanding of how they're interacting with our climate system, because we've been able to show that the process of cloud formation can be triggered by microplastics."* [492] *Miriam Freedman, Professor of Chemistry at Pennsylvania State University.*

Therefore, it is reasonable to hypothesize that nanoplastic particles carrying a surface electrical charge can participate in the microphysics of clouds and potentially influence precipitation patterns.

ENERGY BALANCE AND THE CLIMATE CRISIS

According to observations, warming increases not only air humidity but also lightning frequency, by approximately 12%.[493] The growth of heat, moisture, and aerosol concentrations leads to energy accumulation in the atmosphere–ocean system and the intensification of climatic anomalies.

An additional aspect of the problem is related to the planet's energy balance. Geoff Smith, Emeritus Professor in Applied Physics at the University of Technology Sydney (UTS), noted that data accumulated over 70 years indicate an acceleration in the rate of ocean heating and an increase in the volume of energy stored within it.

[491] *Busse, H. L., Ariyasena, D. D., Orris, J., et al. Pristine and Aged Microplastics Can Nucleate Ice through Immersion Freezing. ACS EST Air 1, 1579–1588 (2024). https://doi.org/10.1021/acsestair.4c00146*
[492] *Pennsylvania State University (Penn State). Microplastics impact cloud formation, likely affecting weather and climate. (2024). https://www.psu.edu/news/research/story/microplastics-impact-cloud-formation-likely-affecting-weather-and-climate (Accessed 20 March 2026)*
[493] *Romps, D. M., Seeley, J. T., Vollaro, D., et al. Projected increase in lightning strikes in the United States due to global warming. Science 346, 851–854 (2014). https://doi.org/10.1126/science.1259100*

According to Professor Smith, ***"Current scientific models in the presence of ongoing rises in atmospheric greenhouse gases do not predict this threatening acceleration."*** [494]

Previously, excess heat was partially dissipated through the ocean and atmosphere into space. Changes in the physicochemical properties of water and the disruption of heat exchange under the influence of micro- and nanoplastics may reduce the efficiency of these energy dissipation pathways.

A complete solution to the climate problem is impossible without a systemic accounting of MNPs as a new physicochemical environmental factor. Plastic has already integrated into the global water cycle and Earth's energy balance, influencing processes in the ocean, atmosphere, and biosphere. The surface electrical charge of the particles plays a substantial role: as plastic fragments down to the micro- and nanoscale, it begins to function as colloidal, electrophysically active matter.

Charged particles alter the nature of interactions in the aquatic environment, participate in atmospheric processes as aerosols, and actively interact with biological structures. Thus, we are no longer speaking simply of pollution, but of a new physicochemical factor of planetary scale.

[494] *Phys.org. Quantum mechanism identified as a key to accelerating ocean temperatures. (2024). https://phys.org/news/2024-11-quantum-mechanism-key-ocean-temperatures.html (Accessed 20 March 2026)*

PART 16.

THE ILLUSORY NATURE OF EXISTING TECHNOLOGICAL SOLUTIONS TO GLOBAL PLASTIC POLLUTION

Existing strategies for combating plastic pollution[495,496]—ranging from mechanical cleanup of the ocean to thermal disposal and recycling—are illusory in nature. Contrary to popular belief, these methods do not eliminate the problem but merely transform it, shifting pollution from a macro- to the micro- and nanoscale,[497] or transferring it between different natural environments (from the hydrosphere to the atmosphere and lithosphere). Even assuming a hypothetical immediate cessation of plastic production and the removal of all visible items, polymers will persist in the oceans for at least 400 years.[498,499] Moreover, scientific review data indicate that depending on chemical composition, environmental conditions (UV radiation, temperature, depth), and the rate of natural degradation, plastics can persist for hundreds to thousands of years.[498] Throughout this period, inevitable fragmentation of large objects will sustain the circulation of micro- and nanoparticles within the biosphere.

LIMITATIONS OF MECHANICAL CLEANUP: SCALE AND TIME

Empirical data demonstrate a critical gap between the rate of plastic entry into the environment and the capabilities of current removal technologies. Currently, approximately 200 million tons of plastic waste are concentrated in the World Ocean. Meanwhile, the annual volume of recovered debris amounts to only about 20,000 tons, which corresponds to less than 0.01% of the total mass (Fig. 16.1).

[495] *Parker-Jurd, F. N. F., Smith, N. S., Gibson, L., et al. Corrigendum to "Evaluating the performance of the 'Seabin' – A fixed point mechanical litter removal device for sheltered waters" [Mar. Pollut. Bull. 184 (2022) 114199]. Marine Pollution Bulletin 194, 115437 (2023).* https://doi.org/10.1016/j.marpolbul.2023.115437

[496] *Pan, Y., Gao, S.-H., Ge, C., et al. Removing microplastics from aquatic environments: A critical review. Environmental Science and Ecotechnology 13, 100222 (2023).* https://doi.org/10.1016/j.ese.2022.100222

[497] *Teng, Y., Zheng, Y., Wang, Z., et al. Mechanical recycling and upcycling of marine macro- and micro- plastics: technologies, challenges, and future directions. Front. Mar. Sci. 12, 1625561 (2025).* https://doi.org/10.3389/fmars.2025.1625561

[498] *Chamas, A., Moon, H., Zheng, J., et al. Degradation Rates of Plastics in the Environment. ACS Sustainable Chem. Eng. 8, 3494–3511 (2020).* https://doi.org/10.1021/acssuschemeng.9b06635

[499] *McCarthy, N. Infographic: Plastic Can Take 500 Years To Bio-Degrade In The Ocean. Statista Daily Data (2018)* https://www.statista.com/chart/15905/the-estimated-number-of-years-for-selected-items-to-bio-degrade *(Accessed 20 March 2026)*

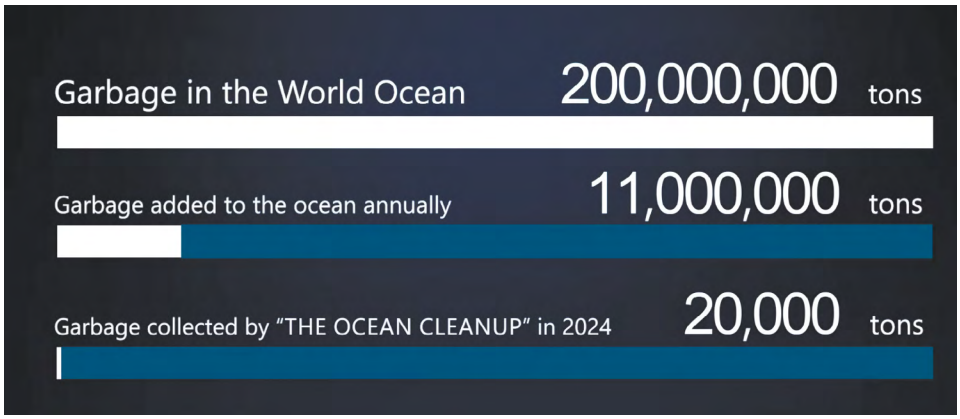


Figure 16.1.

Comparison of the volumes of accumulated, annually entering, and removed waste in the global ocean.

Calculations show that if current extraction rates are maintained, complete cleanup of the ocean would require approximately 10,000 years, even assuming a total halt to new plastic release. Even under a hypothetical scenario of radical technological progress and massive investment capable of increasing cleanup efficiency fivefold, eliminating accumulated plastic would take approximately 2,000 years. This confirms that mechanical extraction cannot be considered a sufficient solution: existing methods are physically incapable of eliminating annual waste flows, let alone multi-year accumulation.

ECOLOGICAL RISKS OF ECOSYSTEM INTERVENTION

Attempts at mechanical plastic removal, aside from low efficiency, are associated with serious ecological risks. Organizations[500] focusing on waste collection in "garbage patches" also inevitably capture neuston[501]—a community of microorganisms and small animals inhabiting the upper 15 cm of the water column. Primary observations *in situ* have shown that plastic debris actively integrates into the biocenosis.[501] On aged PET and expanded polystyrene particles, the processes of surface adhesion, absorption, and colonization of macroinvertebrates (ascidians, mollusks, crustaceans) were noted, while microorganisms, diatoms, and tube worms colonized various polymer types. Living organisms use plastic for nesting, hunting, and oviposition. Consequently, the extraction of such plastic causes direct damage to established ecosystems.

[500] *The Ocean Cleanup. Research. https://theoceancleanup.com/research/ (Accessed 20 March 2026)*

[501] *Maddison, C., Sathish, C. I., Lakshmi, D., et al. An advanced analytical approach to assess the long-term degradation of microplastics in the marine environment. npj Mater Degrad 7, 59 (2023). https://doi.org/10.1038/s41529-023-00377-y*

RISKS OF TRADITIONAL LANDFILLING AND WASTE MIGRATION

A significant portion of waste is directed to traditional disposal systems, including landfill sites.[502] However, this method does not ensure the isolation of pollutants. In the case of burying plastic underground, there remains a high threat of its further migration into the environment.[503] Within the soil layers, gradual degradation of material into micro- and nanoparticles creates sources of secondary contamination of soils and groundwater.[504,505]

Furthermore, the practice of illegal dumps, where disposal occurs in violation of environmental regulations, is widespread globally. Consequently, rather than serving as a final stage of waste management, landfill sites turn into focal points of long-term ecological stress.

THE DISPOSAL PROBLEM AND POLLUTION TRANSFER TO THE ATMOSPHERE

Even assuming the complete technical success of extracting plastic from the hydrosphere, the key problem of its subsequent disposal remains unsolved.[506,507,508] In many countries, unauthorized dumping[509,510] and open waste burning[511,512] are widespread, especially in resource-limited nations. Estimates suggest that in 2020, approximately 30 million tons of plastic[513] were burned without any control measures, leading to the uncontrolled release of toxic substances (Fig. 16.2).[514,515,516]

[502] Wojnowska-Baryła, I., Bernat, K. & Zaborowska, M. Plastic Waste Degradation in Landfill Conditions: The Problem with Microplastics, and Their Direct and Indirect Environmental Effects. IJERPH 19, 13223 (2022). https://doi.org/10.3390/ijerph192013223

[503] Shen, M., Xiong, W., Song, B., et al. Microplastics in landfill and leachate: Occurrence, environmental behavior and removal strategies. Chemosphere 305, 135325 (2022). https://doi.org/10.1016/j.chemosphere.2022.135325

[504] Pratiwi, O. A., Achmadi, U. F. & Kurniawan, R. Microplastic pollution in landfill soil: Emerging threats the environmental and public health. Environ Anal Health Toxicol 39, e2024009 (2024). https://doi.org/10.5620/eaht.2024009

[505] Kabir, M. S., Wang, H., Luster-Teasley, S., et al. Microplastics in landfill leachate: Sources, detection, occurrence, and removal. Environmental Science and Ecotechnology 16, 100256 (2023). https://doi.org/10.1016/j.ese.2023.100256

[506] Tursi, A., Baratta, M., Easton, T., et al. Microplastics in aquatic systems, a comprehensive review: origination, accumulation, impact, and removal technologies. RSC Adv. 12, 28318–28340 (2022). https://doi.org/10.1039/D2RA04713F

[507] Shukla, S., Khan, R. & Roccaro, P. Plastic Waste Recycling is Insufficient to Mitigate Plastic Pollution: the Need for a Paradigm Shift. Curr Pollution Rep 11, 62 (2025). https://doi.org/10.1007/s40726-025-00392-4

[508] Falk-Andersson, J., Rognerud, I., De Frond, H., et al. Cleaning Up without Messing Up: Maximizing the Benefits of Plastic Clean-Up Technologies through New Regulatory Approaches. Environ. Sci. Technol. 57, 13304–13312 (2023). https://doi.org/10.1021/acs.est.3c01885

[509] Vaverková, M. D., Maxianová, A., Winkler, J., et al. Environmental consequences and the role of illegal waste dumps and their impact on land degradation. Land Use Policy 89, 104234 (2019). https://doi.org/10.1016/j.landusepol.2019.104234

[510] Malizia, A., Galindo-Cardona, A., Matias, E., et al. Mapping open dumps and waste dynamics in a subtropical ecoregion of Argentina. Sci Rep 15, 17981 (2025). https://doi.org/10.1038/s41598-025-02653-0

[511] Cruz, M. B., Saikawa, E., Hengstermann, M., et al. Plastic waste generation and emissions from the domestic open burning of plastic waste in Guatemala. Environmental Science: Atmospheres 3, 156–167 (2023). https://doi.org/10.1039/D2EA00082B

[512] Velis, C. A. & Cook, E. Mismanagement of Plastic Waste through Open Burning with Emphasis on the Global South: A Systematic Review of Risks to Occupational and Public Health. Environ Sci Technol 55, 7186–7207 (2021). https://doi.org/10.1021/acs.est.0c08536

[513] Cottom, J. W., Cook, E. & Velis, C. A. A local-to-global emissions inventory of macroplastic pollution. Nature 633, 101–108 (2024). https://doi.org/10.1038/s41586-024-07758-6

[514] Wang, X., Firouzkouhi, H., Chow, J. C., et al. Characterization of gas and particle emissions from open burning of household solid waste from South Africa. Atmospheric Chemistry and Physics 23, 8921–8937 (2023). https://doi.org/10.5194/acp-23-8921-2023

[515] Rogers, K., WaMaina, E., Barber, A., et al. Emissions from plastic incineration induce inflammation, oxidative stress, and impaired bioenergetics in primary human respiratory epithelial cells. Toxicological Sciences 199, 301–315 (2024). https://doi.org/10.1093/toxsci/kfae038

[516] Salim, R., Kapur, S., Schervish, M., et al. Plastic burning particulate matter as a source of environmentally persistent free radicals and reactive oxygen and chlorine species. npj Clean Air 1, 14 (2025). https://doi.org/10.1038/s44407-025-00015-8

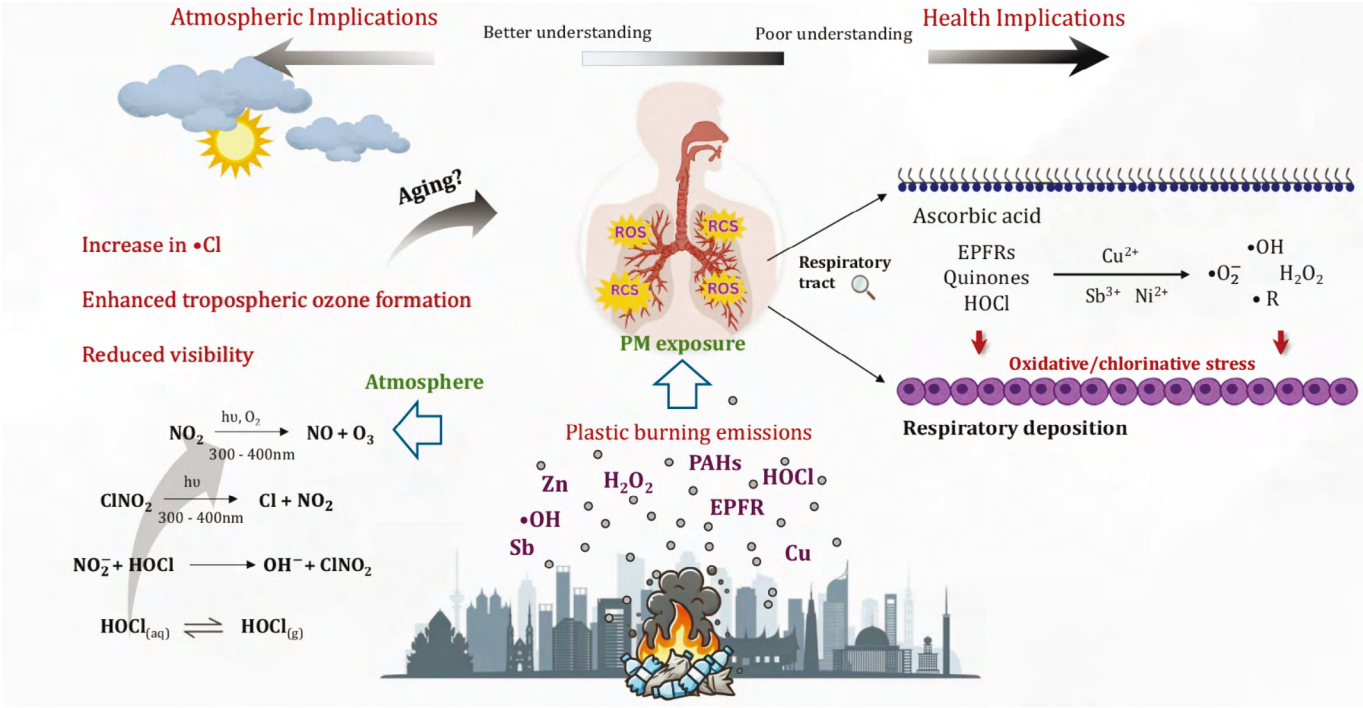


Figure 16.2.

Schematic diagram showing the emissions of various pollutants from the plastic burning including polycyclic aromatic hydrocarbons (PAHs), transition metals, and environmentally persistent free radicals (EPFRs). Respiratory deposition of particulate matter (PM) can lead to the release of reactive oxygen species (ROS) and reactive chlorine species (RCS) in lung lining fluid to cause oxidative and chlorinative stress, possibly causing serious adverse health effects. Additionally, resulting environmental impacts due to chlorine emissions from plastic burning in the form of hypochlorous acid (HOCl) influencing the ozone chemistry. HOCl can significantly affect the visibility and air quality through chlorine and nitrogen oxides (NOx) cycling.

Source: Salim, R., Kapur, S., Schervish, M., et al. Plastic burning particulate matter as a source of environmentally persistent free radicals and reactive oxygen and chlorine species. npj Clean Air 1, 14 (2025). https://doi.org/10.1038/s44407-025-00015-8 License: CC BY 4.0.

Industrial incineration, accounting for about 19% of all plastic waste (more than 72 million tons annually), is also not a safe alternative.[517] Despite the presence of filtration systems, the efficiency of which varies, incineration can still result in the emission of particulate matter, volatile organic compounds, microplastic particles (Fig. 16.3, 16.4), carbon monoxide, and metalloids.[518,519,520] Persistent organic pollutants (POPs),[518] such as dioxins, per- and polyfluoroalkyl substances (PFAS), and polycyclic aromatic hydrocarbons (PAHs), pose a particular danger, contributing to the development of respiratory, cardiovascular, and endocrine diseases.

[517] Rogers, K. & Jaspers, I. ToxPoint: Waste incineration management of plastic materials—an issue of increasing global public health importance. Toxicol Sci 206, 230–232 (2025). https://doi.org/10.1093/toxsci/kfae111
[518] Liu, J., Zhao, D., Wu, F., et al. Impact of Air Pollution Control Devices on VOC Profiles and Emissions from Municipal Waste Incineration Plant. Toxics 13, 1067 (2025). https://doi.org/10.3390/toxics13121067
[519] Lin, X., Ma, Y., Chen, Z., et al. Effect of different air pollution control devices on the gas/solid-phase distribution of PCDD/F in a full-scale municipal solid waste incinerator. Environmental Pollution 265, 114888 (2020). https://doi.org/10.1016/j.envpol.2020.114888
[520] Su, T., Wang, H., Gu, X., et al. Atmospheric microplastics emission from municipal solid waste incineration power plant: Field evidence and characterizations. Journal of Hazardous Materials Letters 6, 100149 (2025). https://doi.org/10.1016/j.hazl.2025.100149

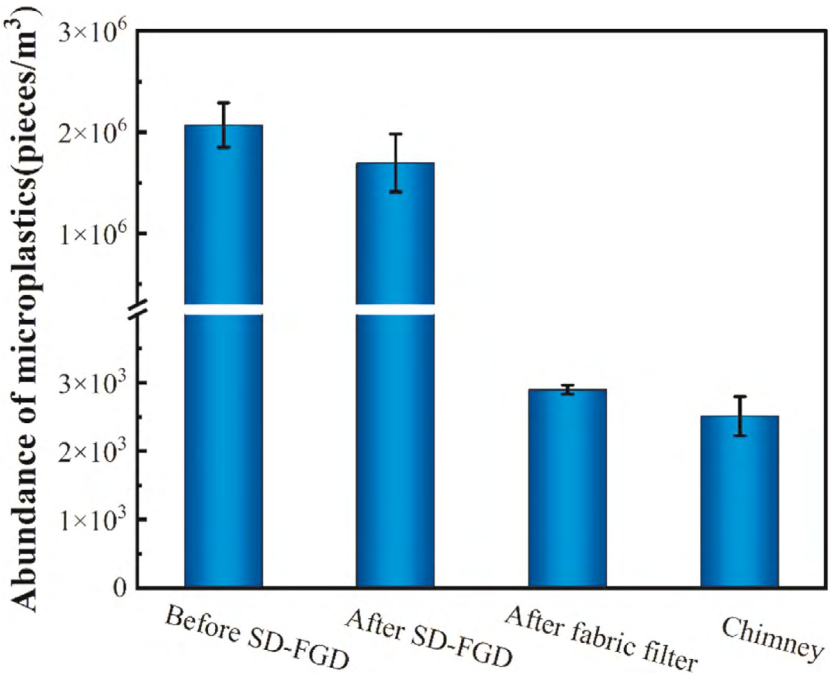


Figure 16.3.

Abundance of microplastics in the flue gas of a solid waste incinerator. The microplastic abundance in the chimney remained around 2.5×10^3 pieces/m³.

Considering the flue gas emission rate (1.14×10^5 m³/h), the annual microplastic emission of this incinerator is estimated at 2.1×10^{12} pieces. SD-FGD means semi-dry flue gas deacidification.

Source: Su, T., Wang, H., Gu, X., et al. Atmospheric microplastics emission from municipal solid waste incineration power plant: Field evidence and characterizations. Journal of Hazardous Materials Letters 6, 100149 (2025). https://doi.org/10.1016/j.hazl.2025.100149

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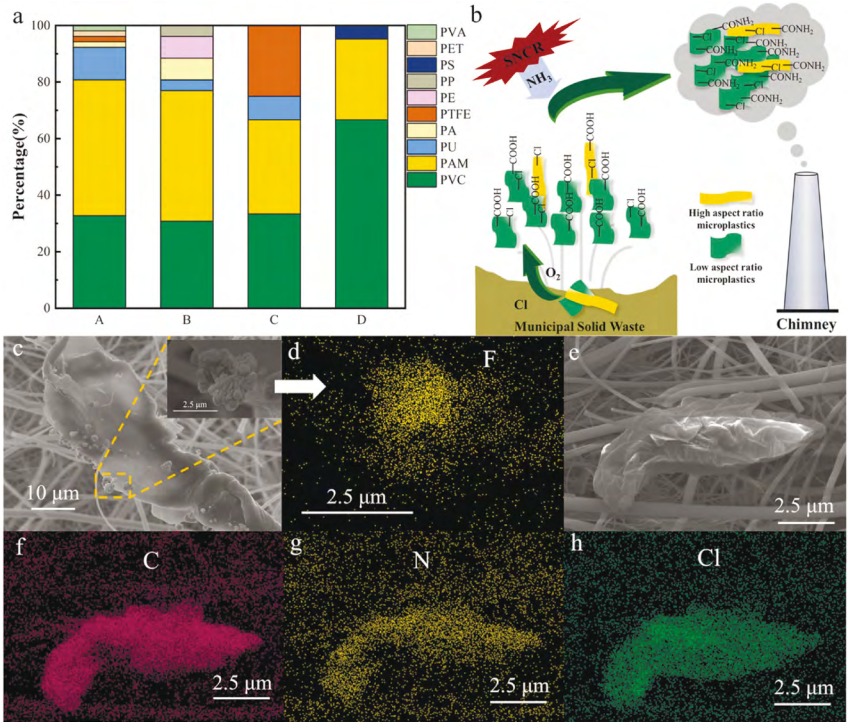


Figure 16.4.

(a) Proportion of various polymer types of microplastics in the flue gas. (b) Surface chemical structure change mechanism of microplastics. SEM images of (c) fiber shape microplastic with particles and (e) fragment shape microplastic in the chimney. (d) F element mapping of the particle. (f) C, (g) N, and (h) Cl element mapping of the fragment shape microplastic.

Source: Su, T., Wang, H., Gu, X., et al. Atmospheric microplastics emission from municipal solid waste incineration power plant: Field evidence and characterizations. Journal of Hazardous Materials Letters 6, 100149 (2025). https://doi.org/10.1016/j.hazl.2025.100149

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THERMAL DEGRADATION AS A SOURCE OF MICROPOLLUTION

It is critically important to note that the incineration of plastic—from bonfires to industrial facilities—creates an illusion of the material's "disappearance".[511,521] In reality, thermal degradation occurs, resulting in the formation of airborne particulate matter (PM), including micro- and nanoparticles,[516,522,523,524,525] which are a factor in premature mortality. These fragments settle on surfaces and return to the soil and water with precipitation.[526,527,528]

Pyrolysis–the heating of waste in an oxygen-free environment at temperatures ranging from 300 to 1300°C[529,530]–also carries several risks. Although this method allows for obtaining up to 50–90% liquid fuel at low and medium temperatures, and up to 90% gas during high-temperature pyrolysis, the process is accompanied by the formation of solid residue and toxic substances (Fig. 16.5).[531,532,533,534]

> *"Even if pollutant emissions from pyrolysis plants are generally lower and of different composition compared to those exiting from controlled combustion, the environmental problems linked to dioxins and other pollutants released into the atmosphere by pyrolysis plants are still significant."* [535]

[521] Wu, D., Li, Q., Shang, X., et al. Commodity plastic burning as a source of inhaled toxic aerosols. Journal of Hazardous Materials 416, 125820 (2021). https://doi.org/10.1016/j.jhazmat.2021.125820
[522] Yang, Z., Lü, F., Zhang, H., et al. Is incineration the terminator of plastics and microplastics? Journal of Hazardous Materials 401, 123429 (2021). https://doi.org/10.1016/j.jhazmat.2020.123429
[523] Hubai, K., Kováts, N., Sainnokhoi, T.-A., et al. Phytotoxicity of particulate matter from controlled burning of different plastic waste types. Bull Environ Contam Toxicol 109, 852–858 (2022). https://doi.org/10.1007/s00128-022-03581-9
[524] Chen, Y., Niu, J., Xu, D., et al. Wet Deposition of Globally Transportable Microplastics (<25 μm) Hovering over the Megacity of Beijing. Environ. Sci. Technol. 57, 11152–11162 (2023). https://doi.org/10.1021/acs.est.3c03474
[525] Hu, L., Fu, J., Wang, S., et al. Microplastics generated under simulated fire scenarios: Characteristics, antimony leaching, and toxicity. Environmental Pollution 269, 115905 (2021). https://doi.org/10.1016/j.envpol.2020.115905
[526] Huang, Y., He, T., Yan, M., et al. Atmospheric transport and deposition of microplastics in a subtropical urban environment. Journal of Hazardous Materials 416, 126168 (2021). https://doi.org/10.1016/j.jhazmat.2021.126168
[527] Weber, C. J. & Bigalke, M. Forest soils accumulate microplastics through atmospheric deposition. Commun Earth Environ 6, 702 (2025). https://doi.org/10.1038/s43247-025-02712-4
[528] Sun, J., Peng, Z., Zhu, Z.-R., et al. The atmospheric microplastics deposition contributes to microplastic pollution in urban waters. Water Research 225, 119116 (2022). https://doi.org/10.1016/j.watres.2022.119116
[529] Mishra, M. K. & Duan, B. The Essential Handbook of Polymer Terms and Attributes. (CRC Press, Boca Raton, 2024). https://doi.org/10.1201/9781003161318
[530] Hashemi, M., Nguyen, K. T. Q., Robert, D. J., et al. Advanced pyrolysis methods for mixed plastic waste management: Enhanced char and gas yields. Journal of Analytical and Applied Pyrolysis 193, 107360 (2026). https://doi.org/10.1016/j.jaap.2025.107360
[531] Pivato, A., Gohar, H., Antille, D. L., et al. Air-Polluting Emissions from Pyrolysis Plants: A Systematic Mapping. Environments 11, 149 (2024). https://doi.org/10.3390/environments11070149
[532] Tang, Y., Tao, D., Li, G., et al. Formation behavior of PCDD/Fs during waste pyrolysis and incineration: Effect of temperature, calcium oxide addition, and redox atmosphere. Environmental Pollution 350, 124011 (2024). https://doi.org/10.1016/j.envpol.2024.124011
[533] Oravová, L., Snow, J., Tolaszová, J., et al. Polycyclic Aromatic Hydrocarbons Formed During the Pyrolysis Process of Plastics – Characterization, Quantification and Risk Assessment. Ecol. Eng. Environ. Technol. 25, 256–264 (2024). https://doi.org/10.12912/27197050/186124
[534] Liang, J., Wang, R., Liu, H., et al. Unintentional formation of mixed chloro-bromo diphenyl ethers (PBCDEs), dibenzo-p-dioxins and dibenzofurans (PBCDD/Fs) from pyrolysis of polybrominated diphenyl ethers (PBDEs). Chemosphere 308, 136246 (2022). https://doi.org/10.1016/j.chemosphere.2022.136246
[535] Moranda, A. & Paladino, O. Controlled Combustion and Pyrolysis of Waste Plastics: A Comparison Based on Human Health Risk Assessment. Recycling 8, 38 (2023). https://doi.org/10.3390/recycling8020038

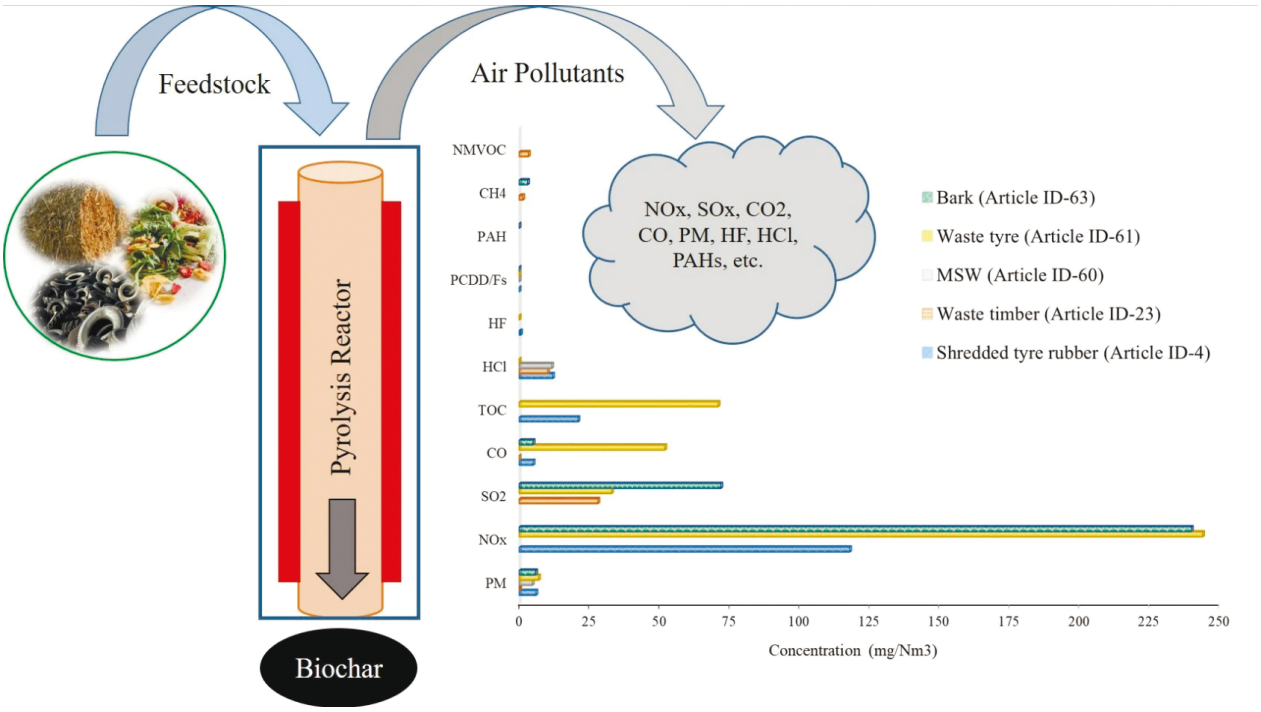


Figure 16.5.

Schematic representation of emissions from waste pyrolysis. During the processing of various feedstocks, toxic and carcinogenic compounds can be generated, including PAHs and polychlorinated dibenzo-p-dioxins/furans (PCDD/Fs), as well as fine particulate matter, which pose the greatest risk to human health and the environment.

Source: Pivato, A., Gohar, H., Antille, D. L., et al. Air-Polluting Emissions from Pyrolysis Plants: A Systematic Mapping. Environments 11, 149 (2024). https://doi.org/10.3390/environments11070149 License: CC BY 4.0.

The sanitary and environmental price of obtaining energy in this way remains extremely high.

THE RECYCLING PARADOX

Mechanical recycling can also contribute to secondary contamination.[525] At the stages of shredding and intensive washing, plastic loses mass due to the formation of microparticles that are carried away into wastewater (Fig. 16.6, 16.7).[536] According to studies, "traditional water treatment plants are not designed to remove MP and they have been globally identified as a major source of MP to the environment."[537] Thus, recycling infrastructure may unintentionally intensify the infiltration of microplastics into water systems.

[536] *Brown, E., MacDonald, A., Allen, S., et al. The potential for a plastic recycling facility to release microplastic pollution and possible filtration remediation effectiveness. Journal of Hazardous Materials Advances 10, 100309 (2023). https://doi.org/10.1016/j.hazadv.2023.100309*

[537] *Tursi, A., Baratta, M., Easton, T., et al. Microplastics in aquatic systems, a comprehensive review: origination, accumulation, impact, and removal technologies. RSC Adv. 12, 28318–28340 (2022). https://doi.org/10.1039/D2RA04713F*

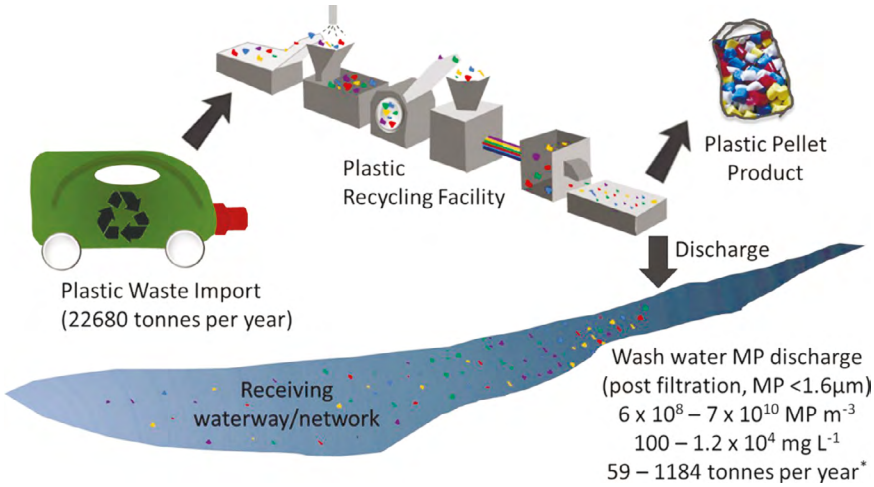


Figure 16.6.

Schematic representation of microplastic generation and release at plastic recycling facilities. During the recycling process, microplastic particles are generated and introduced into the wash water. Despite the application of filtration systems, predominantly coarse particles are removed, while fine fractions (<5–10 µm) remain suspended in the effluent and are ultimately discharged into the surrounding environment.

Source: Brown, E., MacDonald, A., Allen, S., et al. The potential for a plastic recycling facility to release microplastic pollution and possible filtration remediation effectiveness. Journal of Hazardous Materials Advances 10, 100309 (2023). https://doi.org/10.1016/j.hazadv.2023.100309 License: CC BY 4.0.

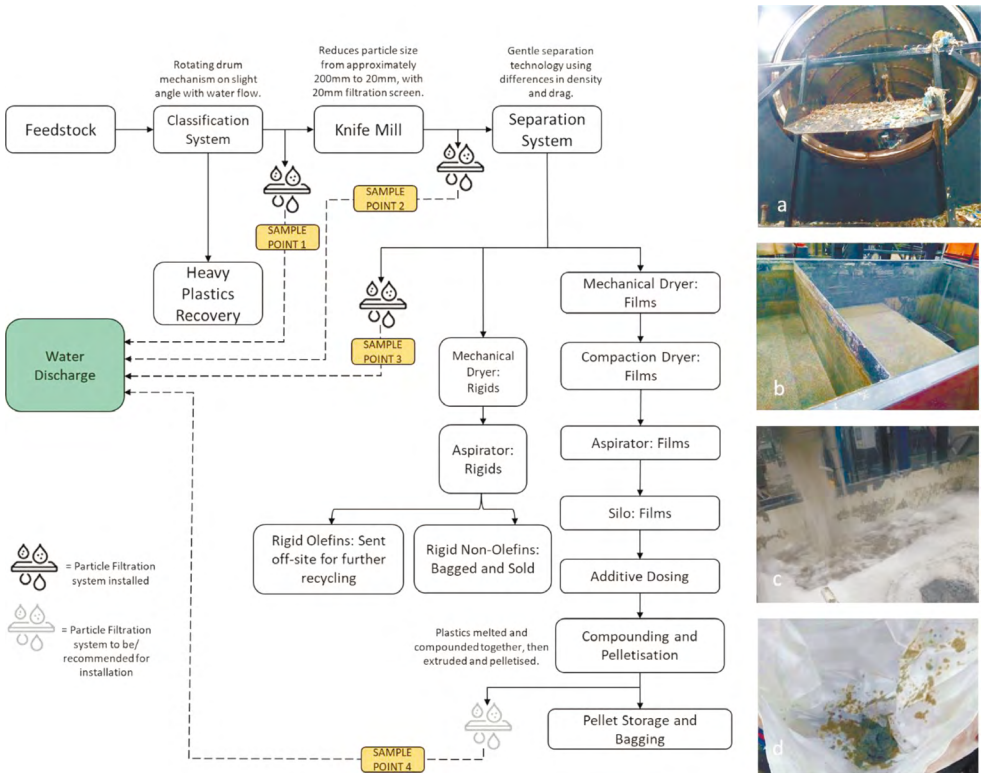


Figure 16.7.

Block flow diagram of plastic recycling facilities showing plastic flow through processes and cumulative water discharge from each point.

Source: Brown, E., MacDonald, A., Allen, S., et al. The potential for a plastic recycling facility to release microplastic pollution and possible filtration remediation effectiveness. Journal of Hazardous Materials Advances 10, 100309 (2023). https://doi.org/10.1016/j.hazadv.2023.100309

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MICRO- AND NANOPLASTICS: IRREVERSIBILITY OF POLLUTION AND BIOLOGICAL INTEGRATION

From the moment it enters the environment, plastic becomes an almost irremovable component of ecosystems, engaging in global biogeochemical cycles. Under the influence of physical and chemical factors, large fragments degrade into micro- and nanoparticles (Fig. 16.8, 16.9).[538,539,540,541] This fragmentation process is cumulative: experimental data show that a single microplastic particle is capable of breaking down into a vast quantity of nanoparticles, which accumulate in the water column and become inaccessible for extraction. [542,543,544]

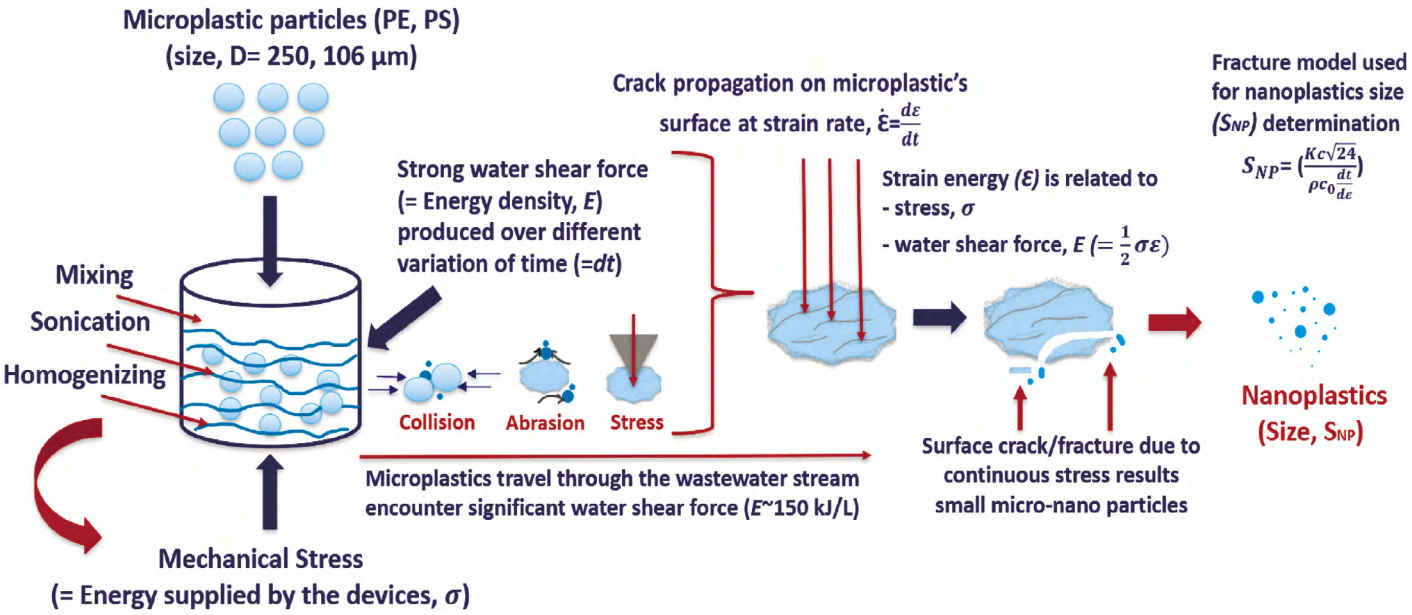


Figure 16.8.

Mechanism of microplastic fragmentation into nanoplastics during the common mixing process used in the primary treatment stage of the wastewater treatment plants.

Source: Monira, S., Roychand, R., Hai, F. I., et al. Microplastic fragmentation into nanoplastics by water shear forces during wastewater treatment: Mechanical insights and theoretical analysis. Environmental Pollution 364, 125310 (2025). https://doi.org/10.1016/j.envpol.2024.125310 License: CC BY 4.0.

[538] *Maddison, C., Sathish, C. I., Lakshmi, D., et al. An advanced analytical approach to assess the long-term degradation of microplastics in the marine environment. npj Mater Degrad 7, 59 (2023). https://doi.org/10.1038/s41529-023-00377-y*

[539] *Song, Y. K., Hong, S. H., Eo, S., et al. Fragmentation of nano- and microplastics from virgin- and additive-containing polypropylene by accelerated photooxidation. Environmental Pollution 327, 121590 (2023). https://doi.org/10.1016/j.envpol.2023.121590*

[540] *Pfohl, P., Santizo, K., Sipe, J., et al. Environmental degradation and fragmentation of microplastics: dependence on polymer type, humidity, UV dose and temperature. Micropl.&Nanopl. 5, 7 (2025). https://doi.org/10.1186/s43591-025-00118-9*

[541] *Monira, S., Roychand, R., Hai, F. I., et al. Microplastic fragmentation into nanoplastics by water shear forces during wastewater treatment: Mechanical insights and theoretical analysis. Environmental Pollution 364, 125310 (2025). https://doi.org/10.1016/j.envpol.2024.125310*

[542] *Pan, Y., Gao, S.-H., Ge, C., et al. Removing microplastics from aquatic environments: A critical review. Environmental Science and Ecotechnology 13, 100222 (2023). https://doi.org/10.1016/j.ese.2022.100222*

[543] *Parker-Jurd, F. N. F., Smith, N. S., Gibson, L., et al. Evaluating the performance of the 'Seabin' - A fixed point mechanical litter removal device for sheltered waters. Mar Pollut Bull 184, 114199 (2022). https://doi.org/10.1016/j.marpolbul.2022.114199*

[544] *Richon, C., Kvale, K., Lebreton, L., et al. Legacy oceanic plastic pollution must be addressed to mitigate possible long-term ecological impacts. Micropl.&Nanopl. 3, 25 (2023). https://doi.org/10.1186/s43591-023-00074-2*

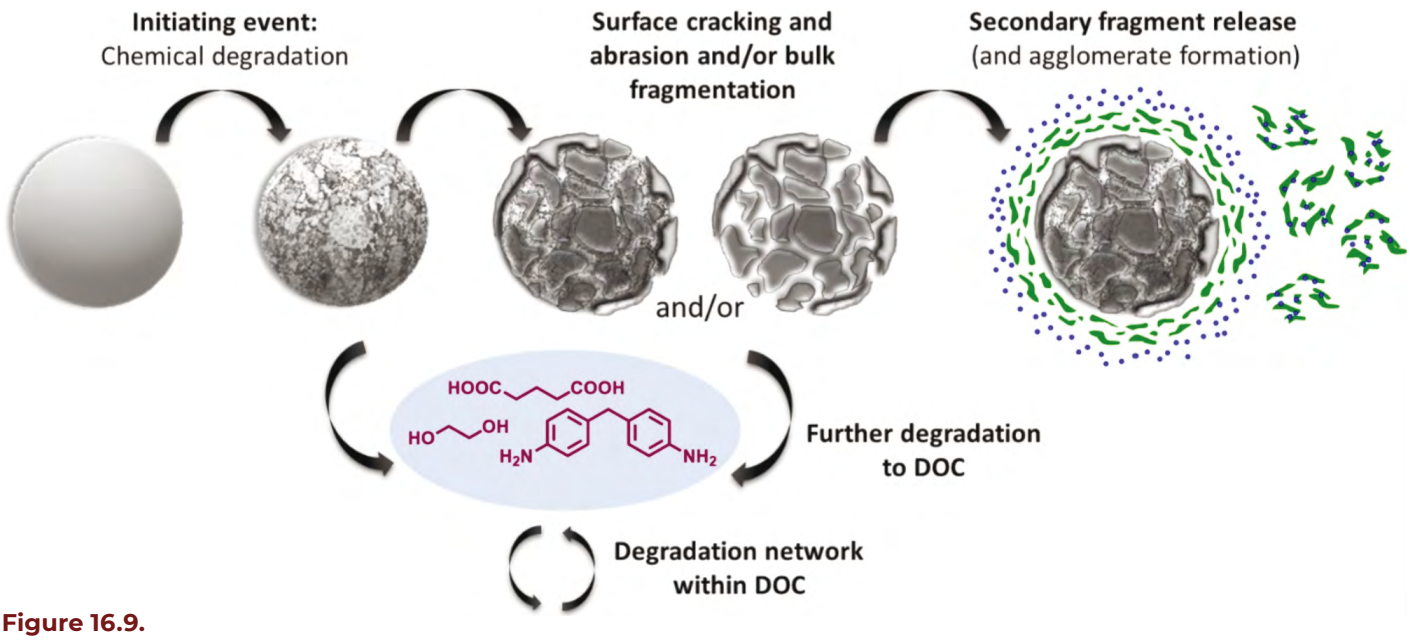


Figure 16.9.

Schematic representation of fragmentation and degradation steps during the aging of microplastics in the environment. Due to chemical degradation, microplastic particles are more prone to mechanical stress, leading to surface abrasion or bulk fragmentation and secondary micro- and nanoplastic release, which might tend to form agglomerates.

Source: Pfohl, P., Santizo, K., Sipe, J., et al. Environmental degradation and fragmentation of microplastics: dependence on polymer type, humidity, UV dose and temperature. Micropl.&Nanopl. 5, 7 (2025). https://doi.org/10.1186/s43591-025-00118-9 License: CC BY 4.0.

This transition of matter to the nanoscale fundamentally changes the nature of the threat, transforming the problem from a purely ecological one into a biomedical one.

The combination of micro- and nano size and electric charge allows MNP particles to overcome natural biological barriers,[545] which are evolutionarily not adapted for protection against synthetic polymers. As a result, particles penetrate into cells[546] and tissues of organisms, integrating into physiological processes. Thus, nanoplastic may pose a risk to human health and the biological integrity of the biosphere precisely due to its ability to interact with living matter at the cellular level.

Analysis reveals a current absence of effective and environmentally safe methods of eliminating plastic pollution, particularly for pollution located at depth or present in micro-scale fractions.[544,547,548] Existing cleanup, incineration, and recycling technologies are often characterized by limited efficiency, high costs, and the generation of secondary ecological risks.[549]

[545] *Bai, C.-L., Wang, D., Luan, Y.-L., et al. A review on micro- and nanoplastics in humans: Implication for their translocation of barriers and potential health effects. Chemosphere 361, 142424 (2024). https://doi.org/10.1016/j.chemosphere.2024.142424*

[546] *Araújo, A. M., Mota, C., Ramos, H., et al. The neurotoxic threat of micro- and nanoplastics: evidence from In Vitro and In Vivo models. Arch Toxicol 99, 3505–3525 (2025). https://doi.org/10.1007/s00204-025-04091-3*

[547] *Tursi, A., Baratta, M., Easton, T., et al. Microplastics in aquatic systems, a comprehensive review: origination, accumulation, impact, and removal technologies. RSC Adv. 12, 28318–28340 (2022). https://doi.org/10.1039/D2RA04713F*

[548] *Wu, N., Grieve, S., Manning, A., et al. Coupling fragmentation to a size-selective sedimentation model can quantify the long-term fate of buoyant plastics in the ocean. Philos Trans A Math Phys Eng Sci 383, 20240445 (2025). https://doi.org/10.1098/rsta.2024.0445*

[549] *Teng, Y., Zheng, Y., Wang, Z., et al. Mechanical recycling and upcycling of marine macro- and micro- plastics: technologies, challenges, and future directions. Front. Mar. Sci. 12, 1625561 (2025). https://doi.org/10.3389/fmars.2025.1625561*

"There is no out-right preferred technology from the environmental point of view for either treating or recycling MPW (mixed plastic waste)." [550]

Thus, the plastic waste management strategy must shift focus from attempts at technical "cleanup" (which often only modifies the form of pollution) to preventing synthetic polymers from entering the environment.[551,552] Any regulatory measures must rely on a comprehensive cost-benefit analysis, comparing the damage from plastic pollution with the negative consequences of the cleanup measures themselves and long-term socio-economic scenarios. Without solutions for safe disposal and a radical revision of the material life cycle, plastic accumulation will continue to represent a long-term, progressive threat to planetary ecosystems.[544,551]

[550] *Jeswani, H., Krüger, C., Russ, M., et al. Life cycle environmental impacts of chemical recycling via pyrolysis of mixed plastic waste in comparison with mechanical recycling and energy recovery. Science of The Total Environment 769, 144483 (2021). https://doi.org/10.1016/j.scitotenv.2020.144483*

[551] *Ghosh, S., Sinha, J. K., Ghosh, S., et al. Microplastics as an Emerging Threat to the Global Environment and Human Health. Sustainability 15, 10821 (2023). https://doi.org/10.3390/su151410821*

[552] *Lalrinfela, P., Vanlalsangi, R., Lalrinzuali, K., et al. Microplastics: Their effects on the environment, human health, and plant ecosystems. Environmental Pollution and Management 1, 248–259 (2024). https://doi.org/10.1016/j.epm.2024.11.004*

PART 17.

FROM THE **ILLUSION OF "PURIFICATION"** TO STRATEGIES FOR **MNP NEUTRALIZATION**

Today, humanity faces a complex challenge: widespread environmental pollution and, as a result, the contamination by micro- and nanoplastics of people themselves. This raises the question regarding the feasibility of removing particles that have already entered the organism. However, the existing methods positioned as "purification" modalities are incapable of resolving the fundamental problem of systemic MNP distribution and tissue accumulation. Moreover, the medical environment may serve as a source of direct particle introduction during these procedures. Consequently, a fundamentally different approach is required to reduce the biological hazard of MNPs: specifically, the development of strategies aimed at neutralizing their key damaging properties—primarily their surface electrostatic charge.

LIMITED EFFICACY OF EXISTING "PURIFICATION" METHODS

Currently, various methodologies are being considered as means of "purifying the organism" from micro- and nanoplastics, ranging from hardware-based techniques (plasmapheresis and hemoadsorption) to the application of adsorbents. These methods are based on the classical principle of toxin elimination. However, these approaches encounter an insurmountable limitation. Experimental data derived from animal studies and corroborated by research on human tissues unequivocally demonstrate that particles, particularly nanoplastics, are capable of crossing biological barriers and accumulating in tissues and organs (see Parts 5,6,8).[553,554,555,556,557]

Thus, procedures designed to clear the bloodstream can only affect the fraction circulating within the blood, leaving the bulk of MNP particles that have already penetrated tissues and cells intact. Consequently, such procedures are fundamentally incapable of ensuring complete detoxification.

[553] *Nihart, A. J., Garcia, M. A., El Hayek, E., et al. Bioaccumulation of microplastics in decedent human brains. Nat Med 31, 1114–1119 (2025). https://doi.org/10.1038/s41591-024-03453-1*

[554] *Ganan-Calvo, A. M. Analytical review of nanoplastic bioaccumulation data and a unified toxicokinetic model: from teleosts to human brain. Preprint (2026). https://doi.org/10.48550/arXiv.2512.22593*

[555] *Ragusa, A., Svelato, A., Santacroce, C., et al. Plasticenta: First evidence of microplastics in human placenta. Environment International 146, 106274 (2021). https://doi.org/10.1016/j.envint.2020.106274*

[556] *Shan, S., Zhang, Y., Zhao, H., et al. Polystyrene nanoplastics penetrate across the blood-brain barrier and induce activation of microglia in the brain of mice. Chemosphere 298, 134261 (2022). https://doi.org/10.1016/j.chemosphere.2022.134261*

[557] *Deng, Y., Zhang, Y., Lemos, B., et al. Tissue accumulation of microplastics in mice and biomarker responses suggest widespread health risks of exposure. Sci Rep 7, 46687 (2017). https://doi.org/10.1038/srep46687*

THE MEDICAL PARADOX: THE TREATMENT SYSTEM AS A SOURCE OF CONTAMINATION

Paradoxically, the very medical environment intended for performing such procedures serves as a source of MNP entry into the organism. A significant part of medical devices and hospital equipment is manufactured from plastics, ranging from gloves to medical equipment casings.[558,559]

Plastic packaging and infusion delivery systems represent a proven source of microplastics that enter directly into the patient's systemic circulation.[560,561,562] Experimental data indicate that prepared solutions from such infusion sets can contain up to approximately 7,500 MP/L (Fig. 17.1),and thousands of microplastic particles may be released during the infusion process.[563,564,565] This creates a situation of direct particle introduction into the patient's organism during treatment, which casts doubt upon the very logic of hardware-based detoxification.

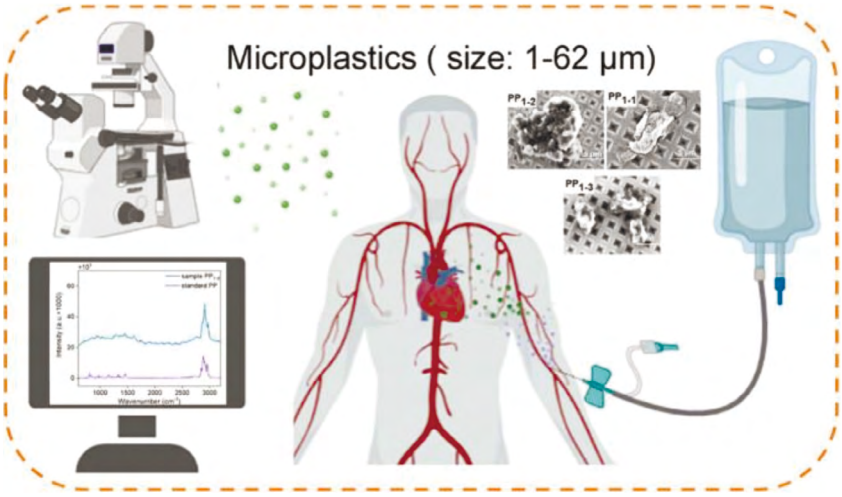


Figure 17.1.

Schematic representation of detected polypropylene microplastics (particles 1–62 μm, approximately 7,500 particles/L) in infusion solutions.

Source: Huang, T., Liu, Y., Wang, L., et al. MPs Entering Human Circulation through Infusions: A Significant Pathway and Health Concern. Environ. Health 3, 551–559 (2025). https://doi.org/10.1021/envhealth.4c00210. License: CC BY-NC-ND 4.0.

[558] Rizan, C., Mortimer, F., Stancliffe, R., et al. Plastics in healthcare: time for a re-evaluation. J R Soc Med 113, 49–53 (2020). https://doi.org/10.1177/0141076819890554

[559] Ivanović, T., Meisel, H.-J., Som, C., et al. Material flow analysis of single-use plastics in healthcare: A case study of a surgical hospital in Germany. Resources, Conservation and Recycling 185, 106425 (2022). https://doi.org/10.1016/j.resconrec.2022.106425

[560] Casella, C., Cornelli, U., Zanoni, G., et al. Health Risks from Microplastics in Intravenous Infusions: Evidence from Italy, Spain, and Ecuador. Toxics 13, 597 (2025). https://doi.org/10.3390/toxics13070597

[561] Tarafdar, A., Xie, J., Gowen, A., et al. Advanced optical photothermal infrared spectroscopy for comprehensive characterization of microplastics from intravenous fluid delivery systems. Science of The Total Environment 929, 172648 (2024). https://doi.org/10.1016/j.scitotenv.2024.172648

[562] Gopinath, P. M., Parvathi, V. D., Yoghalakshmi, N., et al. Plastic particles in medicine: A systematic review of exposure and effects to human health. Chemosphere 303, 135227 (2022). https://doi.org/10.1016/j.chemosphere.2022.135227

[563] Zhang, X., Li, N., Li, X., et al. Reactive oxygen species drive aging-associated microplastic release in diverse infusion ingredients. Journal of Hazardous Materials 490, 137728 (2025). https://doi.org/10.1016/j.jhazmat.2025.137728

[564] Huang, T., Liu, Y., Wang, L., et al. MPs Entering Human Circulation through Infusions: A Significant Pathway and Health Concern. Environ. Health 3, 551–559 (2025). https://doi.org/10.1021/envhealth.4c00210

[565] Zheng, X., Feng, Q. & Guo, L. Quantitative analysis of microplastics and nanoplastics released from disposable PVC infusion tubes. Journal of Hazardous Materials 465, 133246 (2024). https://doi.org/10.1016/j.jhazmat.2023.133246

INABILITY OF CONTEMPORARY APPROACHES TO NEUTRALIZE THE BIOLOGICAL THREAT

Even if one abstracts from the risk of MNP re-contamination during the procedure itself and assumes that such methods are capable of reducing the total toxic load, they fundamentally fail to address key pathogenetic tasks, as they are unable to:

- remove nanoplastics from the intracellular space;
- restore disrupted cellular signaling pathways;
- ensure specific protection of primary cellular targets, including vital organelles, primarily mitochondria and the genetic apparatus (DNA).

At the same time, the problem is exacerbated by the fact that new micro- and nanoplastic particles enter the organism daily with air, food, water, and even the skin.[566,567]

Given that the quantity of MNPs in the environment is constantly rising, the probability of their ingestion increases proportionally.

THE CRITICAL ROLE OF MICRO- AND NANOPLASTICS: APPROACHES TO RISK MANAGEMENT

In the context of the information presented, the most obvious solution would be to completely abandon the use of plastics. However, this approach is unrealistic and inexpedient. Synthetic polymers form the basis of modern industry, healthcare, the energy sector, transportation, electronics, and construction. Their unique properties—versatility, manufacturability, and compatibility with other materials—render them indispensable for a wide spectrum of critical applications: from flexible electronics to aerospace structures.

It is important to note that the primary danger is posed not by polymers in their macroscopic form, but by their micro- and nanoparticles (see Part 2). The high biological activity of these particles is determined by their size, mobility, and, most crucially, their capacity to carry a surface electrostatic charge, which potentiates their interaction with cells, cellular membranes, and macromolecules (see Part 4). This leads to their damage, functional disruption, and, ultimately, cell death.

[566] *Ma, Q., Lei, J., Pang, Y., et al. Neurotoxicity of Micro- and Nanoplastics: A Comprehensive Review of Central Nervous System Impacts. Environ. Health 4, 4–24 (2026). https://doi.org/10.1021/envhealth.5c00087*
[567] *Menichetti, A., Mordini, D. & Montalti, M. Penetration of Microplastics and Nanoparticles Through Skin: Effects of Size, Shape, and Surface Chemistry. Journal of Xenobiotics 15, 6 (2024). https://doi.org/10.3390/jox15010006*

Consequently, the key objective lies not in the rejection of plastics as a class of materials, but in the minimization of biological effects specifically associated with their micro- and nanosized fractions. This necessitates a shift in focus: from attempts at particle extraction to the neutralization of their damaging effects directly within tissues. Precisely this approach, based on managing the biological effects of MNPs, could form the basis for fundamentally new preventive and therapeutic solutions. Achieving this goal requires moving beyond traditional disciplines and consolidating the efforts of materials scientists, toxicologists, biophysicists, and clinicians on a unified research platform.

PART 18.

DISCUSSION ON THE RISKS OF MICRO- AND NANOPLASTICS **TO LIVING ORGANISMS**

An active and legitimate discussion is currently underway within the scientific community and broader society at large regarding the degree of riskt posed by micro- and nanoplastics to biological systems. Micro- and nanoplastics represent a fundamentally new type of ecological and biological stressor. Their unique physicochemical and chemical properties, capacity to bypass biological barriers, and ability to transport toxic substances necessitate that this issue to be examined beyond the traditional paradigms of particulate matter or background pollution. The following section provides an analysis of the most common questions and debated aspects concerning the impact of MNPs on living organisms in the context of modern scientific evidence.

COMPARISON WITH NATURAL PARTICLES: WHAT MAKES PLASTIC UNIQUE?

Thesis for discussion: It is frequently suggested that micro- and nanoplastics do not fundamentally differ from other fine particles of natural origin—such as sand, clay, or volcanic ash—with which living organisms have coexisted for millions of years.

To evaluate the validity of this assertion, it is necessary to examine the physicochemical properties of micro- and nanoplastics and compare them with the characteristics of natural particles.

1. ABSENCE OF EVOLUTIONARY ADAPTATION MECHANISMS TO MNPS

For millions of years, living organisms have interacted with natural aerosol particles—mineral dust, volcanic ash, and organic particulates. As a result, physiological defense mechanisms have evolved, including the self-cleaning function of the respiratory tract, the phagocytic activity of macrophages, and the operation of excretory systems that ensure the detoxification and elimination of many organic compounds, as well as the clearance of a range of inorganic particles of natural origin.

However, micro- and nanoplastics represent a new type of particle that only appeared massively in the environment in the second half of the 20th century. Consequently, biological systems lack mechanisms developed through evolution for the effective removal of such particles, particularly in the case of fine microplastics and nanoplastics.[568]

[568] *PlasticsEurope. History of Plastics. https://plasticseurope.org/plastics-explained/history-of-plastics/ (Accessed 20 March 2026).*

2. SURFACE CHARGE AND THE ABILITY TO BYPASS BIOLOGICAL BARRIERS

Micro- and nanoplastics possess pronounced surface physicochemical properties, including the ability to accumulate and retain an electrical charge over extended periods, as well as to recharge in various media.

Due to their small size and surface charge, these particles can effectively interact with cellular membranes and biological barriers (see Part 4). In particular, micro- and nanoplastic particles are capable of bypassing the epithelial barriers of the intestine[569] and respiratory tract,[570,571] penetrating the systemic circulation,[572] and potentially crossing specialized biological barriers, such as the blood-brain[573] and placental barriers.[574]

3. RESISTANCE TO BIODEGRADATION

Unlike many organic particles of natural origin, MNPs are characterized by high chemical stability. They are practically not degraded by the enzymatic systems of the body, which facilitates their long-term persistence in tissues and the environment.[575]

4. THE TROJAN HORSE EFFECT

Plastic particles exhibit pronounced hydrophobicity, due to which persistent organic pollutants (POPs) and heavy metals from the environment actively adsorb onto their surface. Upon entering the body, such particles can transport and release these adsorbed contaminants within biological media, creating an additional toxic burden.[576,577,578]

[569] *Liang, B., Zhong, Y., Huang, Y., et al. Underestimated health risks: polystyrene micro- and nanoplastics jointly induce intestinal barrier dysfunction by ROS-mediated epithelial cell apoptosis. Part Fibre Toxicol 18, 20 (2021). https://doi.org/10.1186/s12989-021-00414-1*

[570] *Jenner, L. C., Rotchell, J. M., Bennett, R. T., et al. Detection of microplastics in human lung tissue using μFTIR spectroscopy. Science of The Total Environment 831, 154907 (2022). https://doi.org/10.1016/j.scitotenv.2022.154907*

[571] *Tokito, T., Kido, T., Nagafuchi, O., et al. Microplastics in Human Bronchoalveolar Lavage Fluid. Respirology 30, 1141–1152 (2025). https://doi.org/10.1111/resp.70107*

[572] *Rajendran, D. & Chandrasekaran, N. Journey of micronanoplastics with blood components. RSC Adv. 13, 31435–31459 (2023). https://doi.org/10.1039/D3RA05620A*

[573] *Kopatz, V., Wen, K., Kovács, T., et al. Micro- and Nanoplastics Breach the Blood–Brain Barrier (BBB): Biomolecular Corona's Role Revealed. Nanomaterials 13, 1404 (2023). https://doi.org/10.3390/nano13081404*

[574] *Ragusa, A., Svelato, A., Santacroce, C., et al. Plasticenta: First evidence of microplastics in human placenta. Environment International 146, 106274 (2021). https://doi.org/10.1016/j.envint.2020.106274*

[575] *Bacha, A.-U.-R., Nabi, I., Zaheer, M., et al. Biodegradation of macro- and micro-plastics in environment: A review on mechanism, toxicity, and future perspectives. Science of The Total Environment 858, 160108 (2023). https://doi.org/10.1016/j.scitotenv.2022.160108*

[576] *Ateia, M., Zheng, T., Calace, S., et al. Sorption behavior of real microplastics (MPs): Insights for organic micropollutants adsorption on a large set of well-characterized MPs. Science of The Total Environment 720, 137634 (2020). https://doi.org/10.1016/j.scitotenv.2020.137634*

[577] *Ramsperger, A. F. R. M., Narayana, V. K. B., Gross, W., et al. Environmental exposure enhances the internalization of microplastic particles into cells. Science Advances 6, eabd1211 (2020). https://doi.org/10.1126/sciadv.abd1211*

[578] *Hildebrandt, L., Nack, F. L., Zimmermann, T., et al. Microplastics as a Trojan horse for trace metals. Journal of Hazardous Materials Letters 2, 100035 (2021). https://doi.org/10.1016/j.hazl.2021.100035*

5. CHEMICAL ADDITIVES

Plastics contain various processing additives, including phthalates, bisphenols, flame retardants, and stabilizers. When macroplastics fragment down to micro- and nanoscales, the surface area of the particles increases significantly, which can lead to a more intensive leaching of these substances. Many of these belong to compounds with proven endocrine-disrupting properties.[579]

Thus, micro- and nanoplastics cannot be considered a mere analogue of natural aerosol particles. The combination of their physicochemical properties (stability, surface activity, the ability to transport pollutants, and the presence of chemical additives) constitutes a qualitatively new type of interaction with biological systems.

EXTRAPOLATION OF DATA: FROM IN VITRO AND ANIMAL MODELS TO HUMANS

Thesis for discussion: A significant portion of research is based on in vitro experiments (on cell cultures) or on animal models (in vivo). There is active discussion within the scientific community regarding the extent to which these results can be extrapolated to the human body.

The degree of applicability of such results to humans and their scientific significance are supported by the following points.

1. The use of cell cultures and animal models is a generally accepted method in toxicology. Direct experiments with potentially hazardous substances on humans are impossible for ethical reasons; therefore, preliminary risk assessment is conducted under laboratory conditions.

2. Cell-based studies allow the identification of mechanisms by which substances affect biological structures. Such experiments can reveal, for example, disruption of cellular membrane integrity, increased oxidative stress, and other indicators of cellular damage. These fundamental processes are conserved across the cells of most mammals, including humans, making such data critical for assessing potential risks.[580]

3. The very fact that these particles possess the capacity to penetrate human tissues necessitates an assessment of their potential impact on the body. Additional support for this is provided by laboratory data showing that exposure to micro- and nanoplastics can lead to cell death and disruption of hormonal regulation in human tissues (see Part 4).

[579] *Ullah, S., Ahmad, S., Guo, X., et al. A review of the endocrine disrupting effects of micro and nano plastic and their associated chemicals in mammals. Front. Endocrinol. 13, 1084236 (2023). https://doi.org/10.3389/fendo.2022.1084236*

[580] *Araújo, A. M., Mota, C., Ramos, H., et al. The neurotoxic threat of micro- and nanoplastics: evidence from In Vitro and In Vivo models. Arch Toxicol 99, 3505–3525 (2025). https://doi.org/10.1007/s00204-025-04091-3*

Consequently, despite the limitations of laboratory models, the totality of data from cellular studies and animal experiments serves as a vital foundation for evaluating potential risks to human health. Ignoring **such** data would contradict the fundamental principle of medical safety.

COMPARABILITY OF LABORATORY MNP DOSES WITH THE ACTUAL LEVEL OF HUMAN EXPOSURE

Thesis for discussion: Experimental studies demonstrating toxicity often utilize extremely high doses of plastic that a person will never encounter in real life. Consequently, the actual threat is exaggerated.

To correctly evaluate this thesis, several important factors must be considered.

Laboratory experiments typically span days or weeks. To capture potential biological effects over such a limited timeframe, researchers frequently employ elevated particle concentrations. This approach enables the identification of cellular and tissue responses that would likely manifest significantly more slowly at lower exposure levels.

In real-world conditions, exposure follows a different pattern. Humans encounter micro- and nanoplastics daily—through water, food, and air—and this exposure continues for decades. Estimates from atmospheric pollution studies suggest that the annual intake of microplastic particles from outdoor air inhalation can reach up to 1 million particles, while indoor concentrations are often even higher.[581] Modern analytical methods have detected between 110,000 and 370,000 plastic particles, predominantly nanoplastics, in 1 liter of bottled water.[582] A substantial number of plastic particles is also ingested with food.

In this context, even relatively small doses of micro- and nanoplastics, when subjected to prolonged and continuous intake, can lead to a substantial cumulative impact on the body.

Since synthetic polymers are largely resistant to degradation by the body's enzymatic systems, a fraction of the particles can persist in various tissues, including the liver, kidneys, spleen, and brain.[583,584] This elevates the significance of even low-intensity, yet prolonged, exposure. Thus, laboratory experiments simulate an accelerated version of processes that occur slowly but continuously in real life.

[581] *Liao, Z., Ji, X., Ma, Y., et al. Airborne microplastics in indoor and outdoor environments of a coastal city in Eastern China. Journal of Hazardous Materials 417, 126007 (2021). https://doi.org/10.1016/j.jhazmat.2021.126007*

[582] *Qian, N., Gao, X., Lang, X., et al. Rapid single-particle chemical imaging of nanoplastics by SRS microscopy. Proceedings of the National Academy of Sciences 121, e2300582121 (2024). https://doi.org/10.1073/pnas.2300582121*

[583] *Bai, C.-L., Wang, D., Luan, Y.-L., et al. A review on micro- and nanoplastics in humans: Implication for their translocation of barriers and potential health effects. Chemosphere 361, 142424 (2024). https://doi.org/10.1016/j.chemosphere.2024.142424*

[584] *Thapliyal, C., Negi, S., Nagarkoti, S., et al. Mechanistic insight into potential toxic effects of microplastics and nanoplastics on human health. Discov Appl Sci 7, 645 (2025). https://doi.org/10.1007/s42452-025-07214-8*

THE PROBLEM OF MEASUREMENT ACCURACY AND THE ASSESSMENT OF ACTUAL CONCENTRATIONS

Thesis for discussion: Methods for detecting MNPs, especially nanoplastics, are still being perfected. The argument is raised that it is impossible to speak of a proven hazard until we know the exact quantity of particles in the body and the environment.

Scientific inquiry is a process of continuous refinement and development of research methods. Indeed, just a few years ago, the applied filtration and analysis methods did not allow for the detection of particles smaller than 1 µm, that is, nanoplastics.

The emergence of more sensitive analytical techniques, including stimulated Raman spectroscopy, has revealed that the quantity of plastic particles in water and food products can exceed previous estimates by orders of magnitude, as nanoparticles previously remained virtually "invisible" to the instruments used.

It is precisely nanoplastics—particles smaller than 1 µm—that cause particular scientific concern, because due to their size and physicochemical properties, they are capable of bypassing biological barriers, including the blood-brain and placental barriers (see Parts 5, 11).

Thus, methodological limitations in the field of micro- and nanoplastic detection do genuinely exist. However, accumulated data demonstrate that the imperfection of methods has, for a long time, led not to an overestimation, but rather to a systematic underestimation of the scale of nanoplastic proliferation.

From a toxicological perspective, the uncertainty in exact calculations of the global volume does not negate the already established fact: these particles are present in blood, placenta, breast milk, and brain tissues. The precautionary principle operates in science: the difficulty of precise quantitative assessment must not serve as an obstacle to recognizing a qualitative risk.

MNP CONCENTRATIONS AGAINST THE BACKDROP OF OTHER TOXINS

Thesis for discussion: The fraction of microplastics entering the body is negligibly small compared to the volumes of inhaled exhaust gases, heavy metals, and other industrial toxins.

Toxicology outlines the concept of a non-linear dose-response relationship, which is particularly characteristic of substances affecting the endocrine system. Plastic rarely consists of just the polymer: it contains various chemical additives—plasticizers, stabilizers, and

flame retardants—which can constitute a significant portion of its mass, sometimes up to several tens of percent.

Many of these substances are not covalently bound to the polymer and are capable of gradually migrating from the material into the environment and body tissues. Some of these additives (for example, certain phthalates or bisphenol A) belong to the class of endocrine disruptors—substances capable of interfering with the hormonal system. For such substances, the "the dose makes the poison" rule does not strictly apply. They operate along non-monotonic (U-shaped) dose-response curves, mimicking the action of hormones.

This means that even picomolar and nanomolar concentrations (extremely small quantities) can induce serious disruptions in cellular signaling[585] and reproductive function. Furthermore, MNPs do not negate the impact of other toxins, but act in concert with them. As previously mentioned, plastic acts as a transport vehicle, concentrating other pollutants on its surface and delivering them directly inside cells.

Studies on cellular and animal models show that micro- and nanoplastics can trigger a series of biological responses at the cellular level, including oxidative stress, inflammation, metabolic disruptions, and DNA damage (see Part 5).

The history of public healthcare knows of examples (such as asbestos or lead) where the demand for definitive proof of direct harm to humans led to delays in preventive and regulatory measures for decades.

Meanwhile, chronic exposure to low doses of pollutants can have a cumulative nature. Therefore, waiting for conclusive epidemiological data while experimental risk signals are present is viewed within the scientific and medical community as a factor that may impede the timely assessment of potential threats to public health.

PUBLICATION OF STUDIES

Statement for discussion: Critical publications sometimes present the argument that a publication bias may exist in the scientific literature, skewing toward studies that demonstrate the negative effects of plastic.

In micro- and nanoplastic research, the correlation of data from disparate scientific disciplines plays a crucial role. Ecologists, marine biologists, toxicologists, cardiologists, and reproductive health specialists utilize various methods and study different biological systems; however, numerous works observe convergent results indicating cellular toxicity and the capacity of MNPs to induce inflammatory responses. When data obtained by independent research vectors point to the identical biological processes, it enhances the reliability of the conclusions and reduces the probability that the observed effects are incidental.

[585] *Hua, X., Zhang, L. & Wang, D. Nanoplastic at environmentally relevant concentrations activates a germline mir-240-rab-5 signaling cascade to affect the secreted ligands associated with transgenerational toxicity induction in C. elegans. Environ. Sci.: Nano 11, 3615–3624 (2024). https://doi.org/10.1039/D4EN00309H*

In conclusion, it must be emphasized that micro- and nanoplastics constitute a fundamentally new class of anthropogenic pollutants with which living systems have never evolutionarily interacted. Summarizing the analysis of the presented counterarguments, it is necessary to note that doubts regarding the hazard of micro- and nanoplastics can often be based on outdated paradigms of materials physics and the inappropriate extrapolation of macro-object properties to the nanoscale. Modern research dictates that micro- and nanoplastics should not be viewed as inert particulate matter. Owing to their physicochemical properties, MNPs exhibit high capacity for biological penetration and can act as systemic stressors affecting both biological and ecological processes.

The body's defense systems, which efficiently manage natural agents, prove powerless against synthetic nanoparticles acting as carriers of chemical and biological hazards. Recognizing this danger based on the existing body of robust data is a necessary step toward scientific pragmatism.

The transition from macroscopic, visible plastic pollution to the invisible infiltration of nanoparticles at the cellular level demands a paradigm shift in risk assessment. Continued investigation of this issue is critically vital for developing scientifically grounded strategies to counter the impact of MNPs on living systems.

PART 19.

RESEARCH PERSPECTIVE: CAN SUBSURFACE ELECTRICAL ORGANIZATION OF NANOPLASTICS CONTRIBUTE TO THEIR BEHAVIOR?

Numerous studies indicate that the electrical properties of nanoplastics are biologically and ecologically significant: they influence particle stability in the environment, aggregation, interactions with cells, and a range of toxicologically relevant effects. In most research, the electrical properties of nanoplastics are described through a single primary parameter: the zeta (ζ) potential. However, it is important to understand that the ζ-potential is not a direct measurement of a particle's "true charge." It is an indirect value determined by how a particle moves within an electric field. This raises a fundamental question: **is this single value sufficient to describe the electrical nature of nanoplastics, or does it reflect only a part of a more complex reality?**

1. WHY THE ζ-POTENTIAL ALONE MAY BE INSUFFICIENT

In their study *Electrophoresis Characterization of Nanoplastic Particle Surface Charge in Dilute Aqueous Electrolytes,* Lin et al. demonstrates that determining the ζ-potential of nanoplastic particles is not a direct measurement of surface charge, but rather the result of interpreting electrophoretic data through a set of models and assumptions. This work emphasizes that the ζ-potential refers not to the particle surface itself, but to the **shear plane (or slipping plane)** — a region near the surface of the nanoplastic particle where the surrounding liquid begins to move relative to the particle. For the particles investigated in their study, this plane was estimated to be located approximately 0.4–2.1 nm away from the physical surface.[586]

It means that the measured value applies not to the surface itself, but to the interfacial layer between the particle and the surrounding medium. To transition from measured electrophoretic mobility to ζ-potential values, multiple variables must be accounted for: particle size, concentration, the ionic strength of the solution, the type of electrolyte, and the selected electrokinetic model.

The authors further show that the exact same electrophoretic mobility can yield different ζ-potential values depending on the conversion model utilized. Moreover, the validity of these measurements is confined to a specific range of particle concentrations: if the size is too small, Brownian motion plays a prominent role, and at high concentrations, interparticle interactions and overlapping of the electric double layers occur.

This concept is further supported in the study by Varenne et al., *Evaluation of zeta potential of nanomaterials by electrophoretic light scattering: Fast field reversal versus Slow field reversal modes*, which demonstrates that even the measurement mode can influence both the results and their subsequent interpretation.[587]

Additional clarification is provided by Předota et al. in Molecular Origins of the Zeta

[586] *Lin, J.-Y., Lee, I., Feng, C., et al. Electrophoresis Characterization of Nanoplastic Particle Surface Charge in Dilute Aqueous Electrolytes. Environmental Engineering Science 41, 467–476 (2024). https://doi.org/10.1089/ees.2024.0076*

[587] *Varenne, F., Coty, J.-B., Botton, J., et al. Evaluation of zeta potential of nanomaterials by electrophoretic light scattering: Fast field reversal versus Slow field reversal modes. Talanta 205, 120062 (2019). https://doi.org/10.1016/j.talanta.2019.06.062*

Potential. The authors reveal that, at the molecular level, the ζ-potential forms within a narrow interfacial zone and depends not only on the particle material itself but also on water structure, ion sorption, local electrostatics, and hydrodynamics.[588]

This leads to a critical conclusion: the ζ-potential is a useful measurable parameter describing the electrical behavior of a particle at the interface with its medium; however, it does not provide an exhaustive description of the particle's overall electrical architecture. Consequently, relying solely on the ζ-potential may be insufficient for a complete characterization of the electrical nature of nanoplastics.

2. WHY THE "HARD PARTICLE" MODEL MAY BE TOO CRUDE

If the ζ-potential primarily describes particle behavior at the environmental interface, the logical question arises: how accurate is it to model nanoplastics as hard particles with charge exclusively concentrated on their outer surface?

The works by Ohshima *(Theory of electrostatics and electrokinetics of soft particles)*,[589] and *Gopmandal & Duval (Electrostatics and electrophoresis of engineered nanoparticles and particulate environmental contaminants: Beyond zeta potential-based formulation)*,[590] are central to this discussion.

These studies argue that for polymer particles, particularly in the nanoscale range, the simplified "hard colloid" model may be insufficient. For such systems, it is often more appropriate to employ the "soft particle" concept. In this context, "soft" does not refer to the mechanical plasticity of the material. Rather, it indicates that the surface layer of such a particle may be partially permeable to water and ions, and electrically active groups may be situated not only on the surface itself, but also within the near-surface region. In other words, the particle's electrical behavior is driven not only by its strict outer boundary but also by a part of its internal region.[590]

Ohshima emphasizes that for *soft particles*, the concept of ζ-potential — in the sense traditionally applied to hard particles — loses its physical meaning.[589] The review by **Gopmandal and Duval** expands on this approach, noting that standard interpretations of electrophoretic behavior still frequently rely on the "hard colloid with 2D surface charge distribution" model, which treats a particle as a hard body with charge concentrated on its outer surface. However, for nanoplastics, such a description may be too crude.[590]

[588] *Předota, M., Machesky, M. L. & Wesolowski, D. J. Molecular Origins of the Zeta Potential. Langmuir 32, 10189–10198 (2016). https://doi.org/10.1021/acs.langmuir.6b02493*

[589] *Ohshima, H. Theory of electrostatics and electrokinetics of soft particles. Science and Technology of Advanced Materials 10, 063001 (2009). https://doi.org/10.1088/1468-6996/10/6/063001*

[590] *Gopmandal, P. P. & Duval, J. F. L. Electrostatics and electrophoresis of engineered nanoparticles and particulate environmental contaminants: Beyond zeta potential-based formulation. Current Opinion in Colloid & Interface Science 60, 101605 (2022). https://doi.org/10.1016/j.cocis.2022.101605*

Unlike a classic hard particle, a nanoplastic is not a perfectly solid sphere. Instead, it consists of entangled polymer chains bearing functional groups distributed throughout its three-dimensional volume, and it possesses a degree of permeability to ions and electroosmotic flow. Therefore, the authors suggest that a "soft particle" model is more appropriate for nanoplastics. This framework necessitates accounting not only for surface charge but also for volumic charge, the intraparticulate electrokinetically active region, and the influence of the particle's internal structure on its behavior within an electric field.[590]

Significantly, this research discusses a diagnostic marker for such behavior: if the electrophoretic mobility of a particle does not trend toward zero as the salinity of the surrounding medium increases (as would be expected for an ideal hard particle) but instead reaches a non-zero plateau, this suggests that a permeable, internal region of the particle is actively participating in its electrokinetics. This demonstrates that for certain polymer particles, the simple "surface charge + hard particle" scheme may be incomplete.[590]

3. INSIGHTS FROM POLYMER PHYSICS

Polymer physics provides further physical justification for questioning the completeness of current descriptions regarding the electrical nature of nanoplastics. In materials science, it has long been established that polymer dielectrics can retain trapped charges and maintain polarized states within their polymer matrix.

This is specifically demonstrated in the work of Hedir et al., *Investigation of electrical ageing effects on the structural and electrical properties of polystyrene by charging and discharging current method*. The authors investigate the electrical behavior of polystyrene, one of the most common materials used in model nanoplastic research. The study analyzes charging and discharging currents in polystyrene using the charging–discharging current method, meaning it measures the material's response to the application and subsequent removal of electrical voltage.[591]

The paper describes what is termed "anomalous discharge current": after the external voltage is removed, the current does not flow merely as a mirror image of the charging current, but exhibits more complex behavior. This phenomenon is interpreted as an indication of space charge accumulation within the material. The authors attribute this to the presence of bulk and surface charge traps, structural defects, impurities, and other inhomogeneities capable of trapping charge within the polymer matrix.[591]

These data indicate that the polymer matrix itself is essentially capable of retaining electrically active states.

[591] *Hedir, A., Slimani, F., Moudoud, M., et al. Investigation of electrical ageing effects on the structural and electrical properties of polystyrene by charging and discharging current method. Phys. Scr. 99, 055028 (2024). https://doi.org/10.1088/1402-4896/ad3bee*

4. FINDINGS FROM ELECTROPHORETIC STUDIES OF NANOPLASTICS

An additional argument favoring a more complex description of the electrical state of nanoplastics stems from electrophoretic studies of the nanoplastic particles themselves. The research article *"Capillary Electrophoresis as a Complementary Analytical Tool for the Separation and Detection of Nanoplastic Particles"* highlights that during the capillary electrophoresis of polystyrene nanoparticles in the approximately 30–300 nm range, as well as several other nanoplastics, separation is driven not only by linear but also by non-linear electrophoretic effects.[592]

Throughout the study, the authors observed how nanoplastics move within a controlled aqueous system under an electric field.

They discovered that the measured behavior of the particles could not be reduced to a straightforward dependence on a single theoretical "surface parameter." The absolute value of the effective electrophoretic mobility increased alongside particle size, whereas the calculated surface charge density, conversely, decreased.[592] In other words, the electrophoretic response of the nanoplastics proved more complex than what a simplified framework—where the entire electrical nature of the particle is dictated exclusively by the charge of its external boundary—would predict.

Thus, these results demonstrate that at the experimental level, the behavior of nanoplastics does not always fully align with the simple "hard particle + surface charge" model.

5. THE RESULTING QUESTION FOR FURTHER RESEARCH

If the surface charge of nanoplastics is determined only indirectly — via the particle's electrophoretic behavior, the position of the shear plane, and the electrical double layer model — and if experiments observe effects that cannot always be satisfactorily described by the ζ-potential alone, a logical question arises: does the standard measurement of the ζ-potential fully reflect the electrical nature of a nanoplastic particle?

Alternatively, do the observed ζ-potential values represent only the external manifestation of a more complex electrical state? Such a state, in addition to the interfacial surface layer, may include subsurface and internal charge distributions that this method cannot directly distinguish.

[592] *Adelantado, C., Lapizco-Encinas, B. H., Jordens, J., et al. Capillary Electrophoresis as a Complementary Analytical Tool for the Separation and Detection of Nanoplastic Particles. Anal. Chem. 96, 7706–7713 (2024).* https://doi.org/10.1021/acs.analchem.4c00822

WORKING HYPOTHESIS

Based on the data presented, this paper proposes a working hypothesis: for a portion of nanoplastic particles composed of common polymers — such as polyethylene, polystyrene, polypropylene, polyethylene terephthalate, and others — the observed electrical behavior under certain environmental conditions and following material aging may be determined not only by the properties of the external surface itself, but also by additional subsurface or internal electrically active states of the polymer matrix.

Within the framework of this hypothesis, such states encompass a wide range of possible factors: bulk-distributed charged groups in a permeable near-surface layer, localized traps for captured charge, partially stabilized dipole configurations, or, in the strongest version of the hypothesis, electret-like states. It is important to emphasize that this is not yet a proven mechanism, but rather a hypothesis that requires targeted experimental validation.

In simplified terms, this potential contribution may henceforth be referred to as the "internal charge" of the nanoplastic. This denotes not a single point charge within the particle, but a more complex intraparticle electrical architecture that could potentially influence electrophoretic mobility, aggregation, sorption properties, and interactions with protein coronas, cell membranes, biofilms, and aerosol systems.

It is crucial to reiterate: this hypothesis does not negate the established mechanisms of nanoplastic activity, nor does it claim to be an exhaustive explanation for all observed effects. Its purpose is to offer an additional level of physicochemical explanation where existing models may prove insufficient.

This hypothesis requires direct experimental verification; therefore, it is fundamentally important that it be independently confirmed or refuted through specifically designed experimental studies.

THE SCIENTIFIC VALUE OF THIS RESEARCH DIRECTION

The value of this research direction lies in its addressing a genuine gap in knowledge regarding nanoplastics and their behavior. Currently, the electrical behavior of nanoplastics is frequently described using the ζ-potential and external surface characteristics. However, the collective body of cited studies indicates that such a description may be incomplete in all instances.

If subsurface or internal electrical states indeed play a role in driving nanoplastic behavior, this could help explain a number of observations that have not yet been fully interpreted. For instance: why particles with similar external parameters behave differently in solution, why their aggregation and cellular interactions are not always predicted by surface chemistry alone, and why the electrophoretic response of certain systems proves more complex than classical models would predict.

Therefore, this hypothesis may deepen the scientific understanding of the issue and point toward a domain where new mechanisms essential for explaining nanoplastic behavior may reside.

PRACTICAL IMPORTANCE

The practical value of such a direction extends beyond merely improving risk assessments. If research indicates that ζ-potential measurements alone are insufficient to predict the behavior of nanoplastics in water, air, and biological media, it could impact several fields simultaneously: analytical methods, particle migration modeling, toxicological assessment, and the search for new approaches to harm reduction.

This presents a particularly compelling prospect. If a portion of the biological activity of nanoplastics is indeed determined not only by surface chemistry, but also by the state of its charge architecture, then the subject of research could shift from mere diagnostics to the controlled physical modification of this state.

In other words, a deeper study of the charging properties of nanoplastics could potentially lead to the development of approaches that use external physical stimuli to transition existing nanoplastics into a state of reduced electrostatically mediated bioreactivity. This refers to mitigating the portion of a particle's biological activity that is driven by its electrical properties, such as its tendency to adhere to cell membranes, penetrate biological barriers, persist in tissues, or interact actively with proteins and other components of the biological environment.

Put simply, the ultimate goal of this direction is to determine whether it is possible to make existing nanoplastics less biologically active and less hazardous, without transforming them into a new chemical material and without causing unacceptable collateral harm to living systems or the environment.

At this stage, this is, naturally, not a finalized technology, but rather a long-term research goal. However, this framing makes the direction particularly relevant: it elevates the issue of nanoplastic charge from a narrow physicochemical metric to a matter of potential practical risk management.

Thus, investigating the subsurface and internal electrical organization of nanoplastics holds value far beyond its status as a fundamental task. If this path proves viable, it could serve as the foundation for future strategies aimed at the physical "deactivation" of the nanoplastics already present in the biosphere, specifically, reducing their ability to penetrate cells and tissues, persist in the body long-term, and induce adverse biological effects.

PART 20.

CONCLUSION

The analysis conducted in this report regarding the multi-level impact of polymer micro- and nanoparticles on biological and ecological processes clearly demonstrates that micro- and nanoplastics (MNPs) represent a fundamentally new class of man-made particles requiring rigorous interdisciplinary scientific analysis. As stated at the outset of this report, the physicochemical properties of micro- and especially nanoplastics dictate their behavior in the environment and within living systems. Due to their size, high specific surface area, and the ability to retain surface electrostatic charge, these particles actively interfere with fundamental processes, ranging from surface interactions and cellular effects to tissue-level and systemic consequences, which could potentially affect the biosphere and human health.

Throughout this work, particular attention was paid to the mechanisms of impact on living organisms already supported by modern data: the development of mitochondrial dysfunction, the induction of oxidative stress, the crossing of the blood-brain and placental barriers, and systemic effects on the reproductive, cardiovascular, and nervous systems. At the same time, several issues were identified that remain the subject of scientific debate, specifically, the influence of the internal charge architecture of nanoparticles on their toxicity and long-term epidemiological consequences. This balanced approach allows us to view micro- and nanoplastics not merely as a static element of global pollution, but as a dynamically active agent potentially altering the electrophysiological and biochemical parameters of the biosphere.

Today, the problem of micro- and nanoplastic pollution is obvious and cannot be solved solely by mechanical methods of cleaning the environment, which often create only the illusion of a solution. Since it is physically impossible to extract already fragmented plastic from the biosphere, it is critical to refocus efforts toward supporting scientific research and experts specifically studying the effects of nanoplastics on living systems. The fundamental task of the coming decades is to answer the question: can we find mechanisms to neutralize or mitigate the negative impact of the micro- and nanoplastics that already exist in our biosphere?

Because this involves a potential threat to the basic functioning of nature and human health, fragmented laboratory studies are no longer sufficient. Achieving this goal clearly requires coordinated, large-scale international cooperation that in its scope, funding, and level of interdisciplinary organization could potentially be comparable to the greatest scientific initiatives of the past, such as the Human Genome Project. This global problem is unprecedented and, therefore, demands an equally large-scale, consolidated, and strictly scientific solution.

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