

What are the relationships among Neurodevelopmental and Psychiatric Disorders,

Parkinson's Disease, Toxins, the Developmental Origins of Health and Disease and the Exposome over the Lifespan?

Lifespan Links Across Brain Disorders

These relationships center on **developmental timing**, **environmental exposures**, and shared biology across mental, neurodevelopmental, and neurodegenerative disorders. The literature connects neurodevelopmental and psychiatric disorders with Parkinson's disease through a DOHaD-and-exposome model in which early-life and cumulative exposures shape later vulnerability (Sakowski et al., 2024; Heindel & Vandenberg, 2015).

Neurodevelopmental and psychiatric disorders, Parkinson's disease, and toxins are linked through three facets: developmental programming, shared mechanisms, and lifelong cumulative exposure.

Research has progressed from early developmental programming to a lifespan exposome model.

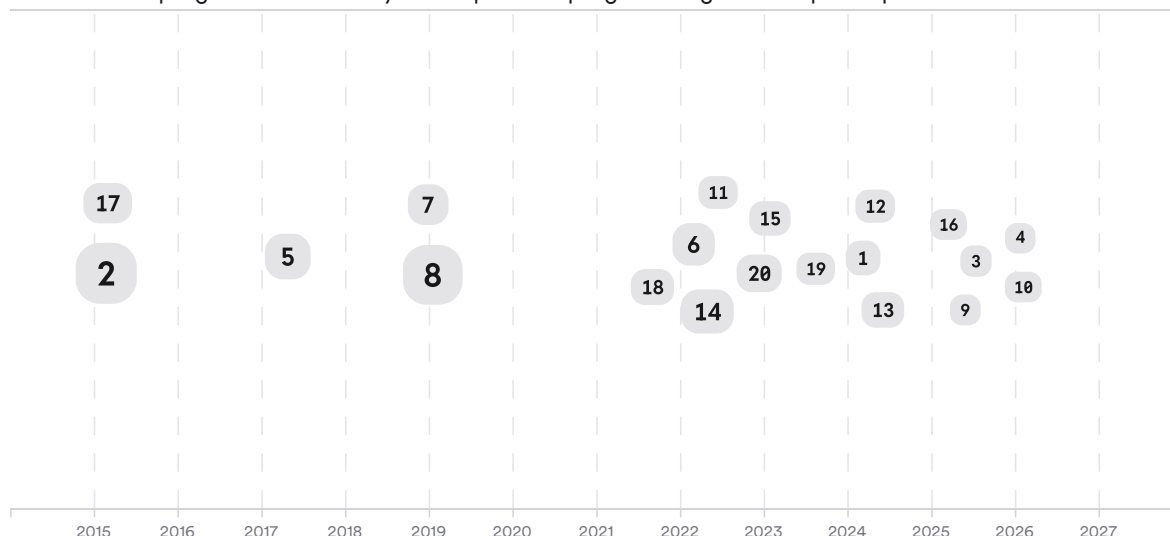


FIGURE 1 Timeline of developmental and exposome brain disorder research

The arc moves from DOHaD, which focused on early sensitive windows, to exposome frameworks that track interacting exposures from conception through aging. More recent reviews explicitly connect neurodevelopment, psychiatric trajectories, and neurodegeneration within one life-course model (Dallere et al., 2025; Koppali et al., 2025).

Developmental Programming

DOHaD holds that fetal and childhood environments influence disease risk across the lifespan, including neurodevelopmental disorders and later neurological disease (Tran & Miyake, 2017; Doi et al., 2022). This framework has been extended to include pollutants, with early-life exposome factors interacting with genetic susceptibility to create a latent phenotype that can be unmasked by later-life exposures (Grova et al., 2019).

- Prenatal toxicant exposure is associated with **ASD and ADHD risk** in cohort and epidemiologic studies (Tran & Miyake, 2017).
- Maternal distress is associated with **higher offspring psychopathology**, including about a twofold increase in probable mental disorder in some studies (Monk et al., 2019).
- Developmental risks begin **before conception** and include parental and early-childhood exposures, not only direct gestational exposures (Tsuji & Molnár, 2025).

Shared Mechanisms

Across neurodevelopmental, psychiatric, and Parkinsonian outcomes, the recurring mechanisms are epigenetic alteration, immune dysregulation, oxidative stress, mitochondrial dysfunction, and abnormal synaptic development or pruning (Tran & Miyake, 2017; Koppali et al., 2025). Evidence linking neurodevelopmental disorders directly to later neurodegeneration is compelling in some studies but remains mixed overall (Sian-Hulsmann et al., 2026).

- Environmental toxicants can alter **DNA methylation**, which is a leading mechanistic bridge between exposure and altered neurodevelopment (Tran & Miyake, 2017).
- Psychosis risk is tied to **sensitive periods** in pregnancy, childhood, and adolescence, with disrupted synaptic pruning and immune activation as candidate mechanisms (Barzilay et al., 2022).
- Parkinson's disease shares **oxidative stress, neuroinflammation, and metabolic dysregulation** with neurodevelopmental conditions in current models (Sian-Hulsmann et al., 2026).

Toxins and Parkinson's Disease

Parkinson's disease appears strongly linked to environmental toxicants, especially in mostly sporadic cases where genes explain only part of risk (Sakowski et al., 2024).

Exposure Type	Relation to PD	Notes
Pesticides	Associated with higher risk	Longer and closer exposure relates to earlier onset and worse motor features (Sakowski et al., 2024)
Industrial toxins	Highest odds in meta-analysis	Occupational industrial exposures had OR 10.74 (Aravindan et al., 2024)
Solvents	Contribute to disease development	Trichloroethylene is specifically implicated (Lefèvre-Arbogast et al., 2024)
Metals	Repeatedly implicated	Mn, Hg, Pb, Cd, As and others are linked in reviews (Nabi & Tabassum, 2022)
Air pollution / PM	Implicated across lifespan models	Included among neurodevelopmental and PD-relevant toxicants (Wyllie & Short, 2023; Aravindan et al., 2024)

FIGURE 2 Environmental toxicant categories associated with Parkinson disease risk

The key implication is that **brain disorders form a lifespan continuum**, not isolated childhood versus old-age diseases. Across the 20 papers, the strongest synthesis is that cumulative and time-specific exposures within the exposome interact with genes and social context to shape risk for neurodevelopmental disorders, psychiatric illness, and Parkinson's disease over decades.

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