What are the relationships between Neurodevelopmental and Psychiatric Disorders, Parkinson's Disease, Toxins, the Developmental Origins of Health and Disease and the Exposome over the Lifespan?

Lifespan Exposome of Brain Disorders

Brain disorders across the lifespan are linked by **developmental exposures** that interact with genes, biology, and later-life stressors rather than by a single shared cause (Dallere et al., 2025; Okubadejo et al., 2026). The literature splits into three connected facets: neurodevelopmental and psychiatric outcomes, Parkinson's disease, and the DOHaD/exposome frameworks that connect early-life toxic and social exposures to later vulnerability (Doi et al., 2022; Gutiérrez-Ortiz et al., 2025).

Brain-health exposome research has progressed from developmental-risk concepts to lifespan integration.

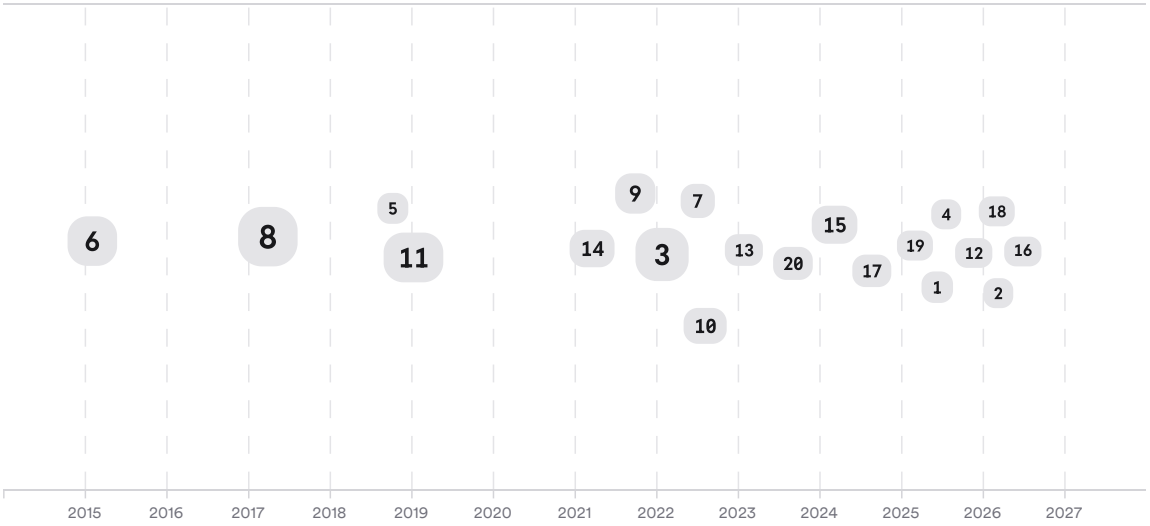

FIGURE 1 Timeline of lifespan exposome research in brain disorders

The timeline shows a shift from **early-life programming** models toward cumulative, multi-exposure, life-course models. Recent work increasingly treats psychiatric disorders and Parkinson's disease as outcomes of interacting exposures across sensitive developmental windows and aging (Fleiss et al., 2018; Okubadejo et al., 2026).

Neurodevelopmental And Psychiatric Disorders

Neurodevelopmental and psychiatric disorders are consistently framed as **multifactorial developmental outcomes** shaped by prenatal, childhood, and adolescent exposures (De Felice et al., 2015; Barzilay et al., 2022).

- Prenatal toxicants, including smoking, phthalates, BPA, persistent organic pollutants, and metals, are associated with ASD, ADHD, and broader altered child neurodevelopment (Tran & Miyake, 2017; Scattolin et al., 2021).
- Psychosis risk is particularly associated with household adversity, day-to-day adversity, and pregnancy or birth complications in adolescents (Pries et al., 2022).
- Early stressors can **prime the brain**, with later childhood or adult stress helping convert latent vulnerability into psychiatric disorder (Fleiss et al., 2018).

DOHaD And Mechanisms

The strongest unifying framework is DOHaD expanded by the **exposome**, where fetal and early-life environments shape later brain health through epigenetic and biological programming (Tran & Miyake, 2017; Grova et al., 2019).

Evidence Strength	Claim
Strong	Early-life environmental toxicants are strongly associated with later **neurodevelopmental disorders**, supported by many cohort and experimental studies (Tran & Miyake, 2017; Doi et al., 2022)
Moderate	Early-life exposome exposures can create a **latent phenotype** that remains susceptible to later environmental hits through epigenetic programming (Grova et al., 2019)
Moderate	Shared mechanisms across psychiatric, neurodevelopmental, and later neurodegenerative outcomes include neuroinflammation, oxidative stress, mitochondrial dysfunction, and altered synaptic pruning (Koppali et al., 2025; Fleiss et al., 2018)

FIGURE 2 Strength of evidence linking developmental exposures to brain outcomes

Mechanistically, the literature repeatedly points to **epigenetic change**, immune activation, mitochondrial dysfunction, oxidative stress, and altered synaptic pruning as bridges between exposure and later phenotype (Tran & Miyake, 2017; Anesti et al., 2023; Barzilay et al., 2022). Real-world exposome studies also show that toxic and social exposures act synergistically, not independently, and that diet and socioeconomic context can modify risk (Sarigiannis et al., 2021; Anesti et al., 2023).

Parkinson's Disease

Parkinson's disease is increasingly treated as a **lifelong trajectory** in which most cases reflect gene–environment interaction, with toxic exposures as important contributors (Sakowski et al., 2024; Ajibare et al., 2026).

- About **80% of PD** appears sporadic, and environmental exposures help explain risk beyond modest genetic effects (Sakowski et al., 2024).
- Pesticide exposure is one of the most identified PD risk factors, and greater occupational or longer exposure is associated with earlier onset and worse motor symptoms (Sakowski et al., 2024).
- Developmental toxicant studies, including dieldrin models, support persistent epigenetic disruption of dopaminergic neurodevelopmental pathways that can raise later PD susceptibility (Kochmanski et al., 2024).

Evidence for direct links from childhood neurodevelopmental disorders to later PD is **mixed rather than settled**. Some reviews describe higher later neurodegenerative risk and shared mechanisms such as oxidative stress, inflammation, and metabolic dysregulation, but they also stress that etiologic pathways remain poorly understood and human evidence is often indirect (Sian-Hulsmann et al., 2026; Ajibare et al., 2026).

Overall, neurodevelopmental disorders, psychiatric disorders, Parkinson's disease, toxins, DOHaD, and the exposome are related through a **life-course model of vulnerability and resilience**. Early toxic, psychosocial, nutritional, and immune exposures appear to shape later mental and neurological outcomes, but the clearest consensus is for developmental influence on risk, not for a single deterministic pathway from childhood disorder to Parkinson's disease.

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