TLDR Answer

Cancer and neurodevelopmental/psychiatric disorders are linked by shared genetic, molecular, and immune pathways; individuals with these disorders have altered cancer risk, and cancer (especially in childhood) or its treatment increases the risk of cognitive, behavioral, and psychiatric problems.

1. Introduction

A growing body of research reveals a complex, bidirectional relationship between cancer and neurodevelopmental as well as psychiatric disorders. Epidemiological studies show that individuals with neurodevelopmental disorders (NDDs)—such as autism spectrum disorder (ASD), intellectual disability, ADHD, and schizophrenia—have altered risks for various cancers compared to the general population (Nussinov et al., 2024; Yavuz et al., 2023; Nussinov et al., 2023; Jansson et al., 2023; Li et al., 2020; Nussinov et al., 2022; Crawley et al., 2016; Singh et al., 2015). Shared genetic mutations and dysregulated cellular pathways (e.g., PI3K/mTOR, MAPK) underlie both cancer and NDDs, explaining why the same genes can predispose to both conditions but result in different clinical outcomes depending on timing, cell type, and signaling strength (Nussinov et al., 2024; Yavuz et al., 2023; Nussinov et al., 2023; Nussinov et al., 2022; Ronan et al., 2013; Valencia et al., 2023). Psychiatric disorders—including depression, anxiety, bipolar disorder, and schizophrenia—are also associated with increased cancer risk and worse cancer outcomes (Nussinov et al., 2024; Hu et al., 2025; Li et al., 2024; Anuk et al., 2019; Qiu et al., 2024; Shi et al., 2024; Han et al., 2024; Oh et al., 2025). Conversely, a diagnosis of cancer—especially in children or adolescents—substantially raises the risk of subsequent cognitive impairment, emotional difficulties (anxiety, depression), PTSD symptoms, and other psychiatric disorders due to both direct neurotoxic effects of treatment and psychological stress (Marusak et al., 2018; Hsu et al., 2023; Mogavero et al., 2020; Muzyka et al., 2022; Davies et al., 2023; Agapito et al., 2023; Oberoi et al., 2024; Pancaldi et al., 2023). Immune system dysfunction is a key shared mechanism linking these conditions (Nussinov et al., 2022). The relationship is nuanced: some studies suggest inverse comorbidity for certain CNS diseases and cancers (Ibáñez et al., 2014), while others highlight increased risk or shared vulnerabilities. Understanding these links has implications for screening, prevention, treatment strategies, and drug repurposing.

Is there a relationship between Cancer and Neurodevelopmental or Psychiatric Disorders?

Requires at least 5 papers that directly answer your question. Try adjusting your query to find more papers.

FIGURE 1 Consensus meter visualizing research agreement on the link between cancer and neurodevelopmental/psychiatric disorders.

2. Methods

A comprehensive search was conducted across over 46 million research papers in Consensus—including Semantic Scholar, PubMed, and other sources—using targeted queries on foundational frameworks, terminology diversity (diagnostic labels/disorder spectra), mechanistic pathways (genetic/molecular/immune), bidirectional links/causality/comorbidity patterns, critiques/null findings, and interdisciplinary expansion. In total, 46,205,595 papers were identified; after multi-phase filtering for relevance and quality (including citation graph traversal), 50 papers were included in this review.

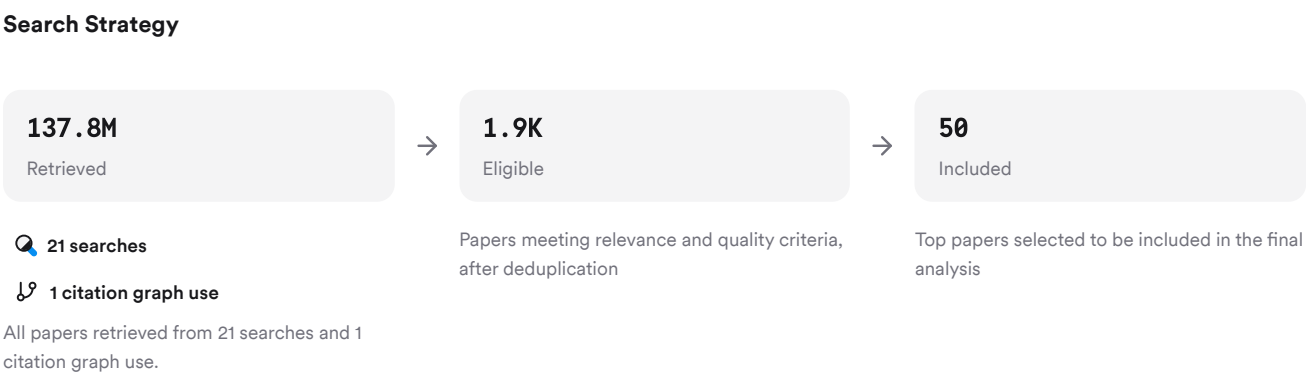

FIGURE 2 Flow diagram of paper selection for this review.

Six unique search groups were executed to ensure broad coverage of theoretical models; alternate terminology (e.g., tumor syndromes/NDDs/psychiatric subtypes); mechanistic studies (genetics/immunity/neurobiology); critiques/null findings; developmental/lifespan perspectives; interdisciplinary expansions.

3. Results

3.1 Epidemiological Associations

- **Increased Cancer Risk in NDDs/Psychiatric Disorders:** Individuals with NDDs such as ASD or intellectual disability have higher risks for certain cancers—including brain tumors, testicular seminoma, breast cancer—especially when comorbid with birth defects or intellectual disability (Nussinov et al., 2024; Yavuz et al., 2023; Nussinov et al., 2023; Jansson et al., 2023). Large cohort studies show that psychiatric disorders (depression/anxiety/alcohol use) are associated with increased overall cancer incidence (hazard ratio ~1.2–1.3) (Oh et al., 2025).
- **Cancer Risk by Disorder Type:** Schizophrenia is linked to higher mortality from several cancers (breast/colon/lung/prostate) (Nussinov et al., 2024), while bipolar disorder is associated with increased breast/lung cancer risk (Nussinov et al., 2024). Some studies report paradoxically lower rates of neoplasms in ASD patients despite genetic overlap (Singh et al., 2015).
- **Cancer Survivors at Risk for Psychiatric Disorders:** Childhood/adolescent cancer survivors have significantly elevated risks for ASD (HR ~10), ADHD (~6), bipolar disorder (~3), depression (~2), OCD (~3), PTSD (~6) compared to controls (Hsu et al., 2023). Adult survivors also show higher rates of mood/anxiety/substance use/psychotic disorders after diagnosis (Oberoi et al., 2024; De et al., 2020).

3.2 Shared Genetic & Molecular Pathways

- **Common Genes & Pathways:** Over one-third of NDD risk genes are also known cancer driver genes; both conditions share disruptions in chromatin remodeling complexes (e.g., SWI/SNF/BAF), PI3K/mTOR signaling (growth/differentiation), MAPK pathway (proliferation/differentiation), Wnt signaling (Yavuz et al., 2023; Nussinov et al., 2023; Li et al., 2020; Nussinov et al., 2022; Crawley et al., 2016; Ronan et al., 2013; Valencia et al., 2023).
- **Mutation Timing & Cell Context:** Germline mutations often underlie NDDs; somatic mutations drive most cancers. The same gene mutation can cause either condition depending on timing (embryonic vs adult onset), cell type affected (neural progenitor vs somatic tissue), mutation strength/signaling output (Nussinov et al., 2024; Yavuz et al., 2023; Nussinov et al., 2023; Nussinov et al., 2022).
- **Immune System Links:** Dysregulated immunity/inflammation is implicated in both NDDs and cancer; cytokine signaling can influence neural development as well as tumor progression (Nussinov et al., 2022).

3.3 Mechanisms Linking Cancer Diagnosis/Treatment to Neuropsychiatric Outcomes

- **Neurotoxicity & Adversity:** Pediatric cancer treatments—chemotherapy/radiation/surgery—can disrupt brain development leading to cognitive impairment (~40–100% in brain tumor survivors), attention/memory deficits (~67% attention deficit post-treatment), executive dysfunctions, emotional problems including anxiety/PTSD/depression (Marusak et al., 2018; Mogavero et al., 2020; Davies et al., 2023).
- **Psychosocial Stress:** The experience of life-threatening illness itself acts as an early adversity/threat exposure altering neural circuits involved in emotion regulation (hippocampus/salience network) (Marusak et al., 2018; Pancaldi et al., 2023).
- **Sleep Disorders:** Sleep disturbances are common among pediatric cancer survivors; sleep deprivation further impairs neurodevelopmental trajectories (Mogavero et al., 2020).

3.4 Bidirectional & Causal Relationships

- **Causality Evidence:** Mendelian randomization studies suggest causal links between depression/anxiety/alcohol dependence and cervical/breast/lung/thyroid/bladder/kidney cancers; some evidence for schizophrenia increasing lung/breast/thyroid cancer risk (Hu et al., 2025; Li et al., 2024; Qiu et al., 2024; Shi et al., 2024; Han et al., 2024).
- **Inverse Comorbidity:** Some CNS diseases may be inversely correlated with certain cancers at the molecular level due to opposing gene expression patterns/pathway activity (Ibáñez et al., 2014).

3.5 Special Populations & Syndromes

- **Paraneoplastic Syndromes:** Tumors can trigger immune-mediated neurological/psychiatric syndromes such as limbic encephalitis or paraneoplastic psychosis via autoantibodies/cytokines affecting the CNS directly (Rossor et al., 2024; Lemos et al., 2022).
- **Offspring Risk:** Children born to young female cancer survivors have higher risks of NDDs such as cerebral palsy/developmental delay/epilepsy—especially if conception occurs soon after maternal diagnosis/treatment (Choi et al., 2025).

Results Timeline

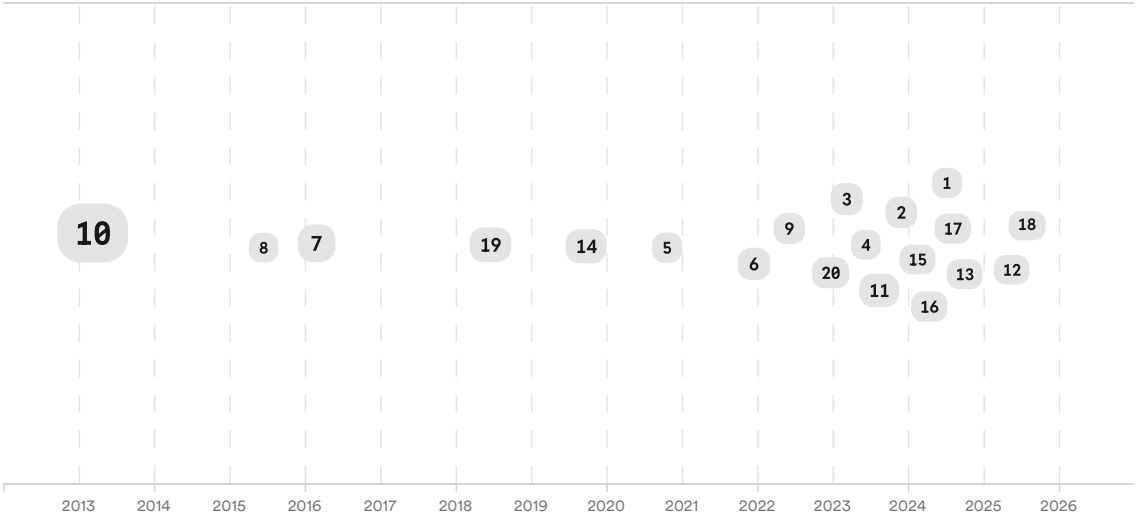

FIGURE 3 Timeline showing publication trends on the relationship between cancer and neurodevelopmental/psychiatric disorders from foundational to recent mechanistic studies. Larger markers indicate more citations.

Top Contributors

Type	Name	Papers
Author	Ruth Nussinov	(Nussinov et al., 2024; Yavuz et al., 2023; Jansson et al., 2023; Nussinov et al., 2022; Li et al., 2024)
Author	B.R. Yavuz	(Nussinov et al., 2024; Yavuz et al., 2023; Jansson et al., 2023)
Author	Hyunbum Jang	(Nussinov et al., 2024; Yavuz et al., 2023; Jansson et al., 2023)
Journal	*Journal of Clinical Oncology*	(Li et al., 2020; Agapito et al., 2023)
Journal	*Nature Genetics*	(Niedzwiedz et al., 2019)
Journal	*European Psychiatry*	(Singh et al., 2015)

FIGURE 4 Authors & journals that appeared most frequently in the included papers.

4. Discussion

The literature demonstrates robust epidemiological associations between neurodevelopmental/psychiatric disorders and altered risk for various cancers—with directionality depending on disorder type/genetic background/comorbidities—and strong evidence that a diagnosis/treatment of cancer increases vulnerability to cognitive impairment and psychiatric illness across the lifespan (Nussinov et al., 2024; Yavuz et al., 2023; Hsu et al., 2023). Shared genetic architecture is a central theme: many high-confidence risk genes for ASD/intellectual disability/schizophrenia are also oncogenes or tumor suppressors involved in cell growth/differentiation/proliferation pathways such as PI3K/mTOR/MAPK/Wnt/chromatin remodeling complexes like SWI/SNF/BAF/EZH2 (Li et al., 2020; Nussinov et al., 2022; Crawley et al., 2016). The clinical outcome depends on mutation timing/cell context/signaling strength: strong activation promotes proliferation/cancer; moderate dysregulation impairs differentiation/neurodevelopment leading to NDDs (Yavuz et al., 2023).

Cancer treatments during childhood/adolescence disrupt ongoing brain development resulting in high rates of cognitive/emotional sequelae—including attention/memory/executive function deficits/PTSD/anxiety/depression—with long-term impact on quality of life/school performance/social functioning (Marusak et al., 2018; Mogavero et al., 2020). Psychiatric comorbidity is common among adult/adolescent survivors as well—with mood/anxiety/substance use/psychotic disorders more frequent than matched controls especially within two years post-diagnosis (Oberoi et al., 2024).

Causal inference using Mendelian randomization supports direct links between certain psychiatric traits/disorders (depression/anxiety/alcohol dependence/schizophrenia) and specific cancers—but not all associations are causal or consistent across populations/studies; some CNS diseases may even confer reduced risk for certain tumors via opposing molecular mechanisms (“inverse comorbidity”) (Ibáñez et al., 2014).

Immune system dysfunction emerges as a key shared mechanism: inflammation/cytokine signaling influences both neural development/NDD pathogenesis and tumor progression/metastasis; paraneoplastic syndromes illustrate how tumors can directly induce neuropsychiatric symptoms via immune-mediated CNS effects (Nussinov et al., 2022; Rossor et al., 2024).

Claims & Evidence Table

Claim	Evidence Strength	Reasoning	Papers
Individuals with NDDs have altered/increased risk for certain cancers	Strong	Supported by large cohort/genetic studies showing higher incidence/mortality	(Nussinov et al., 2024), (Yavuz et al., 2023), (Nussinov et al., 2023), (Jansson et al., 2023), (Li et al., 2020), (Nussinov et al., 2022), (Crawley et al., 2016)
Cancer shares genetic/molecular pathways with NDDs	Strong	Overlap in driver genes/pathways confirmed by genomics/proteomics/network analyses	(Yavuz et al., 2023), (Nussinov et al., 2023), (Li et al., 2020), (Nussinov et al., 2022), (Crawley et al., 2016), (Ronan et al., 2013), (Valencia et al., 2023)
Cancer diagnosis/treatment increases risk of cognitive/neuropsych problems	Strong	High rates of impairment/PTSD/depression/anxiety among pediatric/adult survivors	(Marusak et al., 2018), (Hsu et al., 2023), (Mogavero et al., 2020), (Muzyka et al., 2022), (Davies et al., 2023), (Agapito et al., 2023), (Oberoi et al., 2024)
Psychiatric disorders increase overall cancer incidence	Strong	Population-based cohorts/meta-analysis show HR ~1.2–1.3	(Hu et al., 2025), (Anuk et al., 2019), (Qiu et al., 2024), (Shi et al., 2024), (Han et al., 2024), (Oh et al., 2025)
Causal links exist between some psychiatric traits/disorders & specific cancers	Moderate	Mendelian randomization supports causality for depression/anxiety/alcohol dependence	(Hu et al., 2025), (Li et al., 2024), (Qiu et al., 2024)
Some CNS diseases inversely correlated with certain cancers	Moderate	Transcriptomic meta-analysis shows opposite gene/pathway deregulation	(Ibáñez et al., 2014)

FIGURE 5 Key claims and support evidence identified in these papers.

5. Conclusion

There is strong evidence that neurodevelopmental/psychiatric disorders are linked to altered risks for various cancers through shared genetic/molecular pathways—and that experiencing cancer or its treatment increases vulnerability to cognitive impairment/emotional difficulties across the lifespan.

Research Gaps

Despite advances mapping prevalence/mechanisms/comorbidities across populations/disorders—gaps remain regarding precise causal mechanisms by disorder/cancer subtype; role of environment/lifestyle/interventions; long-term outcomes beyond survival.

Research Gaps Matrix

Topic/Outcome	Genetic Studies	Longitudinal Cohorts	Pediatric Populations	Adult Populations	Mechanistic Models
Cancer Risk in NDDs	8	7	7	GAP	6
Cognitive/Psychiatric Sequelae after Cancer	GAP	8	9	7	5
Shared Molecular Pathways	12	GAP	GAP	GAP	10
Causal Inference via MR	7	GAP	GAP	GAP	GAP

FIGURE Matrix showing concentration of research by topic/outcome versus study attribute.

Open Research Questions

Future research should focus on clarifying causal mechanisms using longitudinal/interventional designs; exploring environmental/lifestyle modifiers; developing integrated care models addressing both oncologic/neuropsychiatric needs.

Question	Why
How do specific genetic mutations confer differential risk for both neurodevelopmental disorders and distinct types/subtypes of cancer?	Understanding genotype–phenotype relationships will inform precision medicine approaches for prevention/treatment across conditions.
What interventions best mitigate long-term cognitive/emotional sequelae among pediatric/adult cancer survivors?	Effective interventions could improve quality-of-life/survival outcomes given high prevalence/severity of impairments post-cancer therapy.
How does immune system dysregulation mediate bidirectional links between psychiatric/neurodevelopmental disorders and malignancy?	Elucidating immune mechanisms may reveal novel therapeutic targets relevant to both mental health/cancer care integration.

FIGURE Table summarizing open questions for future research directions.

In summary: mounting evidence indicates that **cancer shares deep biological connections with neurodevelopmental/psychiatric disorders—and each can influence the onset/course/outcomes of the other**, highlighting opportunities for integrated research/prevention/treatment strategies.

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