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Research Paper

Neurodegenerative Diseases, Psychiatric Conditions, and Cancer: Interconnections and Shared Pathways

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ABSTRACT

This article explores the complex interaction and shared pathways between neurodegenerative diseases, psychiatric conditions, and cancer. Alzheimer's disease (AD) and cancer share mechanisms such as apoptosis and oxidative stress, showing an inverse correlation, with AD patients having a reduced cancer risk. Parkinson's disease (PD) and cancer demonstrate complex interplays, with PD patients showing reduced risks for certain cancers but increased risks for melanoma and brain cancer. Schizophrenia (SZ) is linked to breast cancer through genetic and immunological pathways, while epilepsy often co-occurs with brain tumors, with seizures arising from neoplasms or cancer treatments. Anxiety and depression are prevalent in cancer patients, affecting their prognosis and quality of life. Insomnia significantly worsens cancer patients' quality of life, exacerbated by treatments and stress. Lastly, while bipolar disorder (BD) and cancer connections are influenced by lifestyle factors and medication effects, recent studies show no increased cancer risk in BD patients.

Keywords: *Neurodegenerative Diseases, Psychiatric Conditions, Cancer, Interconnections, Shared Pathways*

ALZHEIMER'S

Cancer and Alzheimer's disease (AD) are complex conditions with multifactorial origins, and while their pathophysiological mechanisms are not fully understood, researchers have identified several commonalities and overlapping risk factors between the two diseases. The bidirectional relationship between cancer and Alzheimer's disease (AD) remains an area of active research. Pathological processes such as apoptosis, synaptic loss, neuronal dysfunction, and oxidative stress intricately interact and exacerbate each other, ultimately culminate in neuronal loss, which is a hallmark feature of advanced AD pathology (Lugue-Contreras D, Carvajal K et al. 2014; Huang WJ, Zhang X and Chen WW. 2016; Giulia N, Simona S et al. 2017). Cancer is indeed characterized by uncontrolled and excessive cell growth. This uncontrolled growth typically results from genetic mutations or alterations that disrupt the normal regulatory mechanisms governing cell division and proliferation. These mutations can affect genes involved in cell cycle regulation, apoptosis (programmed cell

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death), DNA repair, and signaling pathways that control cell growth and differentiation. While cancer involves uncontrolled cell growth, AD involves the opposite—neuronal death and loss of brain tissue (Sethi G, Shanmugam MK et al. 2017; Zhang Y, Kong W et al.2017). Aging can have negative effects on the immune system, as well as on various metabolic processes. Age-related changes can lead to dysregulation in pathways related to bioenergetics, inflammation, DNA repair, oxidative stress, and cell cycle regulation, which may contribute to the development of neurodegenerative diseases and cancer (Ganguli M.2015; Sanabria-Castro A, Alvarado-Echeverria et al 2017; Sethi G, Shanmugam MK et al. 2017). The observation that cancer and Alzheimer's disease (AD) rarely occur together in the same patients, despite both being associated with aging. The hypothesis that obesity-related mechanisms could offer new avenues for prevention and treatment for both diseases is interesting. Obesity is known to be a risk factor for certain types of cancer and has also been linked to an increased risk of developing AD (Lahiri DK. 2017). The opposite relationship between the epidemiology of cancer and Alzheimer's disease (AD) adds to the complexity of understanding their pathogenesis. Both diseases involve interplays of genetic, environmental, and lifestyle factors. The contrasting epidemiological patterns may stem from differences in the underlying mechanisms driving each disease, such as cellular proliferation and DNA damage in cancer versus neurodegeneration and protein misfolding in AD. Further research is needed to elucidate these mechanisms and uncover potential links between cancer and AD. The observation that many existing oncology drugs have shown favorable effects on Alzheimer's disease (AD) suggests a potential biological inverse correlation between cancer and AD. For example, some oncology drugs target pathways involved in inflammation, cell cycle regulation, and protein misfolding, which are also relevant in AD pathology. This supports the idea that understanding the interconnectedness of these diseases at molecular levels (Ancidoni et al., 2021; Araki, 2013; Monacelli et al., 2017). The observed inverse correlation between the risk of developing cancer and Alzheimer's disease (AD), where patients with AD show a 61% decreased risk of cancer incidence compared to reference subjects, suggests a complex interplay between the underlying mechanisms of these diseases. This negative association hints at the possibility that susceptibility to one disease may confer protection against the other. Understanding the biological pathways and factors contributing to this inverse correlation could offer valuable insights into the pathogenesis of both diseases and potentially lead to the development of novel preventive and therapeutic strategies**.** A much better understanding of the underlying mechanisms linking cancer and Alzheimer's disease (AD) holds great potential for not only developing new strategies for prevention but also for therapy. By understanding the intricate connections between these diseases at the molecular, cellular, and systemic levels, researchers can identify common pathways and targets that may be amenable to therapeutic intervention. This holistic approach may lead to the discovery of novel treatments that simultaneously address aspects of both diseases, ultimately improving outcomes for patients affected by either condition (Zabłocka A, et al. 2021).

PARKINSON'S DISEASE

Parkinson's disease (PD) is a complex and intriguing phenomenon that has garnered increasing attention in recent years. Numerous epidemiological studies have revealed a multifaceted relationship between cancer and PD, with both diseases demonstrating intricate interconnections at molecular, cellular, and clinical levels. The convergence of evidence from various studies highlights distinct patterns in cancer occurrence among PD patients. A comprehensive meta-analysis, encompassing diverse study designs such as case control, nested case control, cohort, and cross-sectional studies, elucidated contrasting risks of

specific cancer types in PD cohorts compared to controls. Notably, PD patients exhibited lower risks of developing lung, genitourinary, gastrointestinal, and haematological cancers, while experiencing elevated risks of melanoma and brain cancer. These findings underscore the intricate interplay between neurodegenerative processes in PD and cancer pathogenesis (Leong et al., 2021). Further corroborating these observations, large observational studies have provided compelling evidence of a reduced overall cancer risk in PD patients, with particularly pronounced risk reductions observed for smoking-related cancers. The association between PD and hematological malignancies, characterized by a significantly decreased risk, highlights potential shared etiological factors or pathways underlying both diseases. However, due to small sample sizes, statistical significance was not attained for certain cancer entities, warranting continued investigation into their relationships with PD (Beckar et al., 2010). Exploring the underlying mechanisms, molecular pathways implicated in both PD and cancer pathogenesis emerge as key focal points. Common biological pathways, including αα-synuclein aggregation, mutations in genes such as PINK1, PARKIN, and DJ-1, mitochondrial dysfunction, and oxidative stress, provide mechanistic links between these seemingly disparate conditions. αα-synuclein, a central player in PD pathology, exhibits dual roles in cancer, with its dysregulation implicated in some neoplastic formations while being downregulated in others. Similarly, genes associated with PD pathogenesis, such as PINK1 and PARKIN, demonstrate complex interactions within cancer biology, acting as either oncogenic or tumor suppressor factors depending on the context (Ejma et al., 2020). Clinical observations further support the intricate relationship between PD and cancer, with distinct patterns of cancer occurrence identified in PD cohorts. Skin cancers, including nonmelanoma skin cancer and melanoma, exhibit differential frequencies in PD patients compared to controls, suggesting potential protective effects of certain cancers on PD risk (Tacik et al., 2016). Cancer-induced Parkinson's disease represents a fascinating intersection of neurodegeneration and oncogenesis, underscored by epidemiological, molecular, and clinical evidence (West et al., 2005). Continued research efforts aimed at unraveling the complex interplay between PD and cancer hold promise for elucidating novel therapeutic targets and strategies for both diseases.

SCHIZOPHRENIA

The investigation into the relationship between schizophrenia (SZ) and cancer reveals intriguing connections at biological, genetic, and immunological levels. One study identified common biological pathways and responses to antipsychotics in SZ and cancer, highlighting six categories of antipsychotic anti-cancer effects. However, the utility of miRNAs in predicting cancer risk in SZ patients is limited due to the complex nature of both conditions and the lack of a specific genetic profile for SZ. Variability in miRNA measurement methods and tissue origin further complicates research efforts. Despite these challenges, future research should aim for more targeted comparisons with larger sample sizes to better understand the molecular basis of phenotypic correlations (Brown 2022). Recent epidemiological and genetic studies have also uncovered links between SZ and breast cancer, potentially driven by immunological mechanisms involving molecules like interleukin-33 (IL-33) and its receptor ST2. The IL-33/ST2 axis may serve as a crossroad in SZ-breast cancer comorbidity, suggesting the potential therapeutic utility of selective estrogen receptor modulators like raloxifene and tamoxifen. Moreover, evidence suggests a balance of morphisms in SZ that results in above-normal exposure to catecholamine derivatives, which may explain the inverse relationship between SZ and cancer. Abnormally high exposure to catecholamines simultaneously increases susceptibility to SZ while reducing the likelihood of developing cancer (Rizos et al., 2016). However, the utility of

miRNAs in predicting cancer risk in SZ patients is limited due to the complex nature of both conditions and the lack of a specific genetic profile for SZ. Variability in miRNA measurement methods and tissue origin further complicates research efforts. Despite these challenges, future research should aim for more targeted comparisons with larger sample sizes to better understand the molecular basis of phenotypic correlations (Borovcanin et al., 2021). These findings have significant implications for the treatment of both illnesses, pointing towards potential therapeutic strategies that leverage the immune system and hormonal pathways. Integrating knowledge about immunity and catecholamine exposure into somatic and psychiatric therapeutic protocols could lead to more effective treatments for both SZ and cancer, offering new avenues for exploration in future research endeavors (Foster et al., 2004).

EPILEPSY

The recognition of seizures as a manifestation of cerebral neoplasms dates back to the latter part of the 19th century (Jackson JH 1882). In a particular series of patients at a tertiary cancer center, 13% of individuals diagnosed with systemic cancer were referred for neurologic issues. Among them, 5% were identified as having seizures, with half of those patients showing signs of cerebral metastasis (Clouston PD et al, 1992). Seizures are commonly a notable sign of brain tumors, often appearing as the first symptom, especially in cases involving low-grade tumors (Cascino GD 1990). Within the cancer population, seizures may arise either due to an intracranial neoplasm or as a consequence of systemic cancer treatment. In terms of occurrence rates, patients experiencing seizures can be categorized into two cohorts: those whose seizures stem from a primary or metastatic brain tumor and those who encounter seizures due to other factors, notably their cancer therapy (Grewal J et al, 2008). The epilepsy associated with brain tumors presents a complex amalgamation of challenges, encompassing therapeutic, support, and psychosocial dimensions. This condition reflects the intricate interplay between two significant pathologies: the presence of a brain tumor and the concurrent occurrence of epilepsy (Maschio M 2012). Seizures caused by brain tumors commonly present as focal seizures, which may or may not progress to secondary generalized seizures. Nearly one-third of patients with these seizures do not respond well to antiepileptic medications (You G et al, 2012). The connection between tumorigenesis (tumor formation) and epileptogenesis (development of epilepsy) likely involves similar genetic, molecular, and cellular processes (Aronica E et al, 2023). In this paradigm, anti-seizure medications (ASMs) stand out as intriguing contenders. Capitalizing on their well-documented pharmacological attributes, encompassing blood-brain barrier penetrance and proven efficacy in seizure amelioration, ASMs emerge as plausible candidates for harboring antineoplastic properties (J. Stritzelberger, et al 2021, R.J. Slegers, et al 2020). Effective seizure management can markedly enhance the patient's psychological and social well-being, positively impacting their personal, professional, and interpersonal relationships (Maschio M 2012). While a multitude of studies, including meta-analyses, have been conducted on epileptic patients, the practical application of their findings in clinical settings often proves challenging (Klein, M et al 2003, Beghi, E 2004] French, J.A 2004, Zaccara, G 2006, Cramer, J.A 2001).

ANXIETY AND DEPRESSION

The exploration of risk perception and cancer-related worry in psychology research is crucial. However, the precise mechanisms and interplay between emotional and cognitive factors remain unclear when understanding the relationship between cancer leading to depression and anxiety. The existing literature is relatively sparse and outdated, providing

limited insight into the directionality of associations between these variables. Depression significantly affects both risk perception and cancer-related worry, with higher levels correlating with increased intensity and frequency of worry. Anxiety also influences risk perception and cancer-related worry, with health care-related fears mediating the association between anxiety and worry. The findings align with Leventhal's self-regulation model, emphasizing the impact of individuals' perception of illness on their responses. Anxiety emerges as a stronger predictor of cancer-related worry and risk perception compared to depression, with direct effects on risk perception not mediated by health care fears. Family history of cancer impacts risk perception, with the number of affected family members increasing perceived risk. Both personal and family experiences with cancer heighten worry about developing cancer. Understanding these complex relationships can permit new interventions to be developed so as to improve psychological well-being in at-risk populations (Caruso A, et al 2023). There are studies that highlight the significant impact of anxiety and depression on breast cancer patients, affecting their prognosis, treatment adherence, and quality of life. It identifies genetic factors like PER2 and COMT variants associated with depression and anxiety, respectively, and clinical factors such as Fos aprepitant use during chemotherapy and social support from marriage linked to reduced depression symptoms. Cognitive impairments and sleep disturbances worsen anxiety and depression, suggesting a need for early interventions. The research reveals a positive association between depression and breast cancer (BC) risk, implicating shared genetic regions like 9q31.2 and 6p22.1 in common biological pathways. Mendelian randomization analyses suggest a possible causal role of depression in BC development among European populations. Despite limitations such as small sample size and reliance on self-report measures, the study underscores the need for further research to validate findings and explore therapeutic implications for reducing BC risk (Hajj A, et al.2021, Wu X, Zhang W, et al. 2023, Almeida SS, et al. 2023)

INSOMNIA

Insomnia significantly worsens the quality of life for cancer patients, with numerous studies documenting its high prevalence. Nearly half of the patients with a recent cancer diagnosis experience insomnia symptom (Pinucci, I et al, 2023). Studies objectively measuring sleep quality in cancer patients are relatively limited. However, actigraphic data reveal that sleep disturbances significantly worsen as cancer patients progress through their ongoing chemotherapy regimen (Madsen et al, 2015). A recent study reported that 64% of cancer patients experience clinically significant sleep difficulties. However, only a small portion of these patients mentioned sleep disorders as their primary concern during their integrative oncology consultation (Narayanan, S et al 2022).

Stress-related cancer diagnosis (Distress) is linked to hyperarousal, characterized by heightened somatic, cortical, and cognitive activation. Research indicates elevated levels of cortisol, increased body temperature, heightened 24-hour metabolic rate, and accelerated heart rate in cancer patients experiencing symptoms of insomnia (Pinucci, I et al, 2023). Moreover, pain caused by the disease, surgical procedures, or pharmacological treatments can significantly affect the quality of sleep (Büttner-Teleaga, A et al, 2021, Theobald, D.E 2004). Moreover, similar to treatments prescribed for non-cancer patients, anticancer drugs can also induce sleep disturbances. This can be due to emotional distress triggered by treatment or directly from their side effects. Breast cancer patients and survivors are particularly susceptible to insomnia caused by hot flashes as well as the effects of chemotherapy, radiotherapy, and hormone therapy (Yaremchuk, K 2018, Savard, J. et al,

2001, Savard, J. et al, 2004, Fiorentino, L.; Ancoli-Israel, S. 2007, Costa, A.R et al, 2014, Pinucci, I et al, 2023).

BIPOLAR DISORDER

Over the past decade, epidemiologists and clinicians have increasingly focused on exploring the potential association between bipolar disorder (BD) and cancer. Proposed biological reasons for this connection include lifestyle factors such as obesity, heavy smoking, and poor health behaviors, as well as metabolic and molecular consequences of prolonged use of mood-stabilizing or antipsychotic medications (McIntyre et al., 2006, Waxmonsky et al.,2005, Kilian et al., 2006, Newcomer, 2006).

This study is the first to investigate the potential link between cancer and bipolar disorder (BD) within a national managed care population, using data from a single health service provider in Israel. Unlike previous studies, our research includes a diverse group of BD patients at all disease stages and across all age groups. This heterogeneity may explain why our findings do not support previously reported associations between BD and an increased cancer risk (Kahan NR et al, 2018).

CONCLUSION

This article highlights the intricate relationships between neurodegenerative diseases, psychiatric conditions, and cancer, emphasizing shared pathways and bidirectional interactions. The inverse correlation between Alzheimer's disease (AD) and cancer suggests that mechanisms like apoptosis and oxidative stress may have protective effects. Parkinson's disease (PD) shows varied cancer risks, with lower risks for certain types but higher risks for melanoma and brain cancer, indicating complex interplays. Schizophrenia's (SZ) link to breast cancer underscores genetic and immunological intersections. Epilepsy's co-occurrence with brain tumors, particularly through seizures from neoplasms or cancer treatments, highlights the intertwined nature of these conditions. Anxiety and depression significantly impact cancer patients' prognosis and quality of life, stressing the need for comprehensive psychological support. Insomnia exacerbates cancer patients' quality of life, often worsened by treatments and stress. Despite lifestyle and medication influences, recent studies show no increased cancer risk in bipolar disorder (BD) patients. Understanding these connections is crucial for developing integrated therapeutic strategies.

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