

Editorial

CHILDHOOD EXPOSURES AND DEFICIENCIES AS DETERMINANTS OF CANCER RISK IN ADULTHOOD

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ABSTRACT:

Cancer development is increasingly recognized as a life-course process influenced by early-life exposures and nutritional status. During critical periods of growth and development particularly first 1000 days, biological systems are particularly susceptible to carcinogenic insults and epigenetic modifications that may predispose individuals to malignancy later in life. Deficiencies in key nutrients such as folate and vitamin D can impair DNA repair and immune function, while early exposure to environmental toxins and pollutants contributes to genomic instability and endocrine disruption. Additionally, persistent infections—including Hepatitis B (HBV), Human papillomavirus (HPV), and Helicobacter pylori infection—play a significant role in infection-related cancers, particularly in low- and middle-income countries. Socioeconomic disparities further exacerbate these risks by limiting access to nutrition, healthcare, and preventive interventions. Addressing these early determinants through integrated public health strategies—such as improved childhood nutrition, vaccination programs, and environmental regulation—offers a critical opportunity for cancer prevention. A shift toward early-life interventions is essential to reduce the long-term global cancer burden and advance equitable health outcomes.

Keywords: Early life exposures, life time cancer risk, environmental exposures, nutritional deficiencies

INTRODUCTION

Cancer is increasingly understood not merely as a disease of aging but as the cumulative outcome of life-course exposures, many of which originate in early development particularly first 1000 days of life. The paradigm linking early-life environment to adult disease—central to the Developmental Origins of Health and Disease (DOHaD) framework—has profound implications for oncology. Childhood represents a critical window of biological vulnerability during which environmental insults, nutritional deficiencies, and epigenetic modifications can predispose individuals to malignancy decades later.

Early age biological programming of cancer risk

Rapid cellular proliferation, organogenesis, and immune system maturation characterize childhood. During this period, exposure to carcinogens or deprivation of essential nutrients can induce long-lasting molecular alterations. Epigenetic reprogramming—through DNA methylation, histone modification, and microRNA expression—can dysregulate oncogenes and tumor suppressor genes without altering DNA sequence. These early changes may remain latent until triggered by additional exposures later in life, resulting in carcinogenesis. Moreover, immune surveillance mechanisms, which play a critical role in eliminating malignant cells, are shaped early in life. Nutritional deficiencies or chronic infections during childhood may impair immune competence, reducing the body's ability to prevent tumor initiation and progression. Childhood exposures are deeply embedded in socioeconomic context. Poverty influences nutrition, environmental safety, access to healthcare, and education—all of which modulate long-term cancer risk. Children growing up in disadvantaged settings are more likely to experience cumulative risk exposures, creating a trajectory of vulnerability that extends into adulthood.

Childhood nutritional deficiencies and cancer susceptibility

Malnutrition remains a persistent challenge in low- and middle-income countries (LMICs), including Pakistan. Deficiencies in micronutrients such as vitamins A, D, folate, and zinc have been implicated in increasing cancer susceptibility. Folate deficiency disrupts DNA synthesis and repair, increasing the likelihood of mutations. Vitamin D deficiency has been associated with higher risks of colorectal, breast, and prostate cancers due to its role in cell differentiation and apoptosis. Antioxidant deficiencies (vitamins C and E) reduce the capacity to neutralize oxidative stress, facilitating DNA damage. Chronic undernutrition may also lead to growth inhibition, which has

been linked with metabolic dysregulation and increased risk of certain adult malignancies. Thus, nutritional status specially micro-nutrients in childhood must be carefully watched and any malnutrition must be treated as the top priority.

Environmental and Chemical Exposures

Children in many regions are disproportionately exposed to environmental carcinogens including air pollution (particulate matter, polycyclic aromatic hydrocarbons), pesticides and agricultural chemicals, heavy metals such as arsenic and lead in drinking water and second-hand tobacco smoke. These exposures can induce DNA damage, oxidative stress, and endocrine disruption. For example, early exposure to endocrine-disrupting chemicals may alter hormonal pathways, increasing the risk of hormone-dependent cancers such as breast and prostate cancer in adulthood. In situations where there is high exposure of carcinogens and there is micronutrients deficiency and there is not enough anti-oxidants to neutralize oxidative stress and lack of vitamin to help in DNA repair the risk of cancer increases many folds.

Infectious Agents and Chronic Inflammation

Childhood infections are another critical yet under-recognized contributor to adult cancer risk. Persistent infections can lead to chronic inflammation and oncogenic transformation. Notable examples include: Hepatitis B and Hepatitis C infections acquired early in life, predisposing to hepatocellular carcinoma, Human papillomavirus, associated with cervical and other anogenital cancers, Helicobacter pylori infection, linked to gastric cancer, Epstein Bar virus to lymphoma. In LMICs, where vaccination and early treatment coverage may be suboptimal, the burden of infection-related cancers is particularly high.

Implications for Prevention and Policy

Recognizing cancer as a life-course disease necessitates a shift from reactive to preventive oncology. Interventions must begin early, especially considering first 1000 days of life:

1. Strengthening maternal and child nutrition programs
2. Expanding vaccination coverage (e.g., HBV, HPV)
3. Reducing environmental exposures through regulatory enforcement
4. Improving water, sanitation, and air quality
5. Integrating early-life risk awareness into public health strategies

Institutions such as World Health Organization have emphasized the importance of early prevention, yet implementation gaps remain substantial in many LMICs.

Research and Future Directions

There is a pressing need for longitudinal cohort studies in diverse populations to better quantify the relationship between childhood exposures and adult cancer outcomes. Biomarker discovery, epigenetic profiling, and life-course epidemiology should be prioritized to identify high-risk groups and inform targeted interventions. Academic institutions, particularly in LMICs, have a critical role to play in generating context-specific evidence and translating it into policy.

CONCLUSION

The development of cancer is a course of decades, establishing body environment suitable for cell proliferation requires multiple factors including weak immune system to kill cancer cells resulting from long term micronutrient deficiency, lack of anti-oxidant supply to balance oxidative stress. Addressing childhood exposures and deficiencies offers a strategic opportunity to reduce the global cancer burden. Thus shifting strategies from treatment prevention would be pragmatic approach.

Conflict of Interest

Author declare no conflict of interest.