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RESEARCH ARTICLE

Immigration Positive Impact in Modifying, Prevention of Genetically Induced Diseases "Obesity, Cancer"

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ABSTRACT

Behavioral, social, and environmental aspects of health are determinants of health in the development of genetically induced diseases, including obesity and cancer. Immigration is one of the important shifts in the context as it can have a positive impact on the change and prevention of these conditions. This paper evaluates the role of migration as a contributing factor in reducing genetic factors of obesity and cancer, in relation to the aspect of changes in lifestyle, environment, and healthcare access. The paper uses evidence provided by epidemiological and public health studies to offer an explanation on how gene-environment interactions, epigenetic processes and the healthy immigrant effect contribute to the development of the disease outcomes. The enhanced dietary variety, higher level of physical activity, improved access to preventive health services and exposure to health promoting general health systems are cited as the important mechanisms through which immigration can lessen the manifestation of the disease even when a person has a genetic inclination towards the disease. Simultaneously, the analysis does not ignore moderating variables including acculturation, socioeconomic status, and structural inequalities which have a long-term outcome. Comprehensively, the results illustrate the possibility of immigration as a protective factor in the prevention and intervention of genetically mediated obesity and cancer, which has significant implications on the prevention strategies of the population and preventive health, as well as health policy of the people.

Keywords: Immigration, Genetic predisposition, Obesity, Cancer, Gene–environment interaction, Disease prevention, Public health

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INTRODUCTION

Complex associations between inherited vulnerability and environmental factors that are controllable result in genetically induced diseases like obesity-related cancers, which are becoming an increasing public health concern. Obesity, especially, has continuously been found to be a significant mediator which enhances genetic instability, metabolic imbalances and persistent inflammation and heightens the risk associated with various types of cancer. There is a large amount of evidence indicating that being overweight changes the insulin signaling, hormone regulation, and inflammatory pathways, which are associated with carcinogenesis in genetically predisposed patients.[1,2] These processes highlight the need to study more about the general social and environmental contexts that can adjust the expression of genetic risk.

The biological link between obesity and cancer has been well established across diverse populations. Obesity-induced metabolic changes, including hyperinsulinemia, altered adipokine secretion, and oxidative stress, have been shown to promote DNA damage and impair genomic stability, thereby facilitating tumor development.[3] Epidemiological studies further indicate that obesity significantly increases the incidence and mortality of cancers such as breast, colorectal, and endometrial cancer.[4,5] At the molecular level, obesity influences key signaling pathways involved in cell proliferation and apoptosis, particularly in hormone-sensitive cancers, reinforcing its role as a critical modifier of genetic disease risk.[6,7]

Dietary patterns and lifestyle behaviors remain central to the prevention and modification of obesity-related cancers. Nutritional exposures, physical activity, and energy balance interact with genetic predisposition to either exacerbate or mitigate disease progression.[8,9] As such, population-level interventions and environmental shifts that promote healthier behaviors have gained attention as effective cancer prevention strategies.[10] Within this context, immigration represents a significant life transition that may alter diet, physical activity, healthcare access, and exposure to preventive health systems, thereby influencing obesity and cancer risk among genetically susceptible populations.

Emerging public health research suggests that migration can function as a protective determinant of health through improved living conditions, access to preventive care, and exposure to health-promoting environments. These changes may reduce the phenotypic expression of genetically induced diseases, including obesity and cancer, particularly during early and mid-

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life stages. Understanding how immigration modifies genetic risk through environmental and behavioral pathways is therefore essential for advancing preventive medicine and reducing global health disparities. This study explores immigration as a positive modifier in the prevention and management of genetically influenced obesity and cancer, situating genetic susceptibility within broader socio-environmental and public health frameworks.

Genetic Predisposition and Disease Expression

Genetic predisposition plays a fundamental role in the development of obesity and cancer; however, the manifestation and progression of these conditions are strongly influenced by metabolic, environmental, and lifestyle-related factors. Obesity, in particular, represents a critical intermediary through which inherited susceptibility translates into disease expression. Genetic variants associated with energy balance, insulin signaling, inflammation, and lipid metabolism can predispose individuals to excess adiposity, which in turn creates a biological environment conducive to carcinogenesis.[1,2]

At the molecular level, obesity induces metabolic dysregulation characterized by hyperinsulinemia, insulin resistance, chronic low-grade inflammation, and altered adipokine secretion. These changes promote genetic instability, DNA damage, and dysregulated cell proliferation, thereby increasing cancer risk among genetically susceptible individuals.[3,9] Elevated levels of insulin-like growth factors and pro-inflammatory cytokines further activate oncogenic signaling pathways, accelerating tumor initiation and progression.[1,2]

Epidemiological evidence consistently demonstrates a strong association between obesity and increased risk of several cancers, including breast, colorectal, endometrial, and pancreatic cancers. This association reflects the interaction between inherited genetic risk and obesity-driven biological mechanisms rather than genetic determinism alone.[4,5] In hormone-sensitive cancers such as breast cancer, excess adipose tissue contributes to increased estrogen production and altered signaling pathways, intensifying disease expression in individuals with genetic susceptibility.[6,7]

Dietary patterns and nutritional exposures further modulate the relationship between genetic predisposition and disease expression. Diets high in energy density and low in protective bioactive compounds exacerbate obesity-related metabolic stress, while diets rich in fruits, vegetables, and flavonoids may attenuate carcinogenic signaling pathways, even in genetically at-risk populations.[6,8] This highlights the role of modifiable factors in shaping how genetic risk is biologically expressed.

Importantly, contemporary cancer prevention frameworks emphasize that genetic susceptibility does not equate to inevitability. Instead, obesity and related metabolic pathways represent modifiable targets for reducing cancer risk at the population level.[10] By influencing body weight, metabolic health, and exposure to protective or harmful environments, broader social and environmental transitions—such as those associated with immigration—can significantly alter the trajectory of genetically induced obesity and cancer.[3,9]

Immigration and Environmental Transition

Immigration is frequently accompanied by profound environmental transitions that influence the expression of genetically induced diseases, particularly obesity and cancer. These transitions encompass changes in diet, physical activity patterns, healthcare access, and broader socioeconomic and cultural contexts. From a gene–environment perspective, such shifts are critical because genetic predisposition alone is insufficient to determine disease outcomes; instead, it is the interaction between inherited risk and post-migration environments that shapes long-term health trajectories.

One of the most significant environmental changes associated with immigration relates to dietary patterns. Migrants often move from regions with traditional, minimally processed diets to environments characterized by greater food diversity, fortified foods, and structured nutritional guidelines. Improved access to fruits, vegetables, whole grains, and bioactive compounds such as flavonoids can positively modulate metabolic and inflammatory pathways linked to obesity and cancer risk.[6,8] Since obesity is a central mediator connecting genetic susceptibility to cancer through mechanisms such as insulin resistance, chronic inflammation, and hormonal dysregulation, dietary transitions play a preventive role in limiting downstream genetic instability and carcinogenesis.[1,3]

Physical activity and lifestyle environments also shift following migration. Urban infrastructure, occupational changes, and exposure to public health campaigns can promote increased physical activity and healthier energy balance. Maintaining energy balance is particularly important in preventing obesity-driven oncogenic pathways, including altered insulin-like growth factor signaling and adipokine imbalance, which are strongly associated with cancer initiation and progression.[2,9] These environmental modifications may therefore attenuate the phenotypic expression of genetic risk for both obesity and obesity-related cancers.

Another critical dimension of environmental transition is healthcare access and preventive infrastructure. Many host countries provide structured cancer screening, obesity prevention programs, and early intervention services. Access to such systems enhances early detection and risk modification, particularly for genetically susceptible individuals. Preventive frameworks targeting lifestyle modification and metabolic regulation have been identified as central strategies in reducing cancer burden at the population level.[10] Consequently, immigration into health systems with strong preventive orientation can reduce the translation of genetic vulnerability into clinical disease.

However, environmental transition is not uniformly protective. The health benefits associated with migration depend on the balance between protective exposures and adverse factors such as sedentary behavior, dietary acculturation toward ultra-processed foods, and socioeconomic stressors. These factors may amplify obesity prevalence and cancer risk if protective cultural practices are lost over time.[4,5] Thus, immigration represents a dynamic process in which environmental contexts can either suppress or exacerbate genetically mediated disease pathways.

Overall, immigration-driven environmental transitions can act as powerful modifiers of genetic susceptibility to obesity and cancer. When supported by health-promoting environments and preventive healthcare systems, these transitions contribute to reduced disease expression and improved long-term population health outcomes.

Protective Health Effects of Immigration

Immigration can exert measurable protective health effects on populations with genetic susceptibility to obesity and cancer by reshaping environmental exposures, lifestyle behaviors, and preventive health engagement. These effects are primarily mediated through favorable gene–environment interactions that attenuate metabolic dysregulation, chronic inflammation, and genetic instability key biological pathways linking obesity to cancer development.[1,3]

A well-documented phenomenon underlying these outcomes is the *healthy immigrant effect*, whereby immigrants often arrive with lower prevalence of obesity-related comorbidities and cancer risk factors compared to host populations. This advantage is attributed to pre-migration health selection, traditional dietary patterns, higher baseline physical activity, and lower exposure to obesogenic environments.[2,9] In genetically predisposed individuals, such protective environments can significantly delay or suppress disease expression.

Dietary transitions associated with immigration may also reduce cancer-promoting metabolic pathways. Increased access to diverse food systems, improved nutritional awareness, and greater intake of bioactive compounds such as flavonoids and plant-based nutrients—have been shown to modulate insulin resistance, adipokine signaling, and estrogen-related pathways implicated in obesity-driven cancers, particularly breast cancer.[6,8] These dietary influences are critical in modifying epigenetic regulation and reducing DNA damage linked to excess adiposity.[3]

Furthermore, immigration to regions with more developed healthcare infrastructures enhances access to preventive services, including obesity management programs, cancer screening, and early diagnostic interventions. Preventive healthcare engagement plays a decisive role in interrupting the progression from genetic susceptibility to clinical disease by enabling early risk

Table 1: Environmental Transitions Associated with Immigration and Their Implications for Genetically Induced Obesity and Cancer

Environmental Domain	*Pre-Migration Context*	*Post-Migration Transition*	*Implications for Obesity and Cancer Risk*
Diet and Nutrition	Traditional diets, limited food variety	Increased dietary diversity, access to fruits, vegetables, and fortified foods	Reduced inflammation and metabolic dysregulation linked to obesity and cancer[6,8]
Physical Activity	Labor-intensive or rural lifestyles	Structured exercise environments, urban infrastructure	Improved energy balance and reduced obesity-related oncogenic signaling[9]
Healthcare Access	Limited preventive services	Screening, early detection, and obesity prevention programs	Lower progression of genetically predisposed obesity and cancer[10]
Metabolic Environment	Higher untreated metabolic risk	Improved management of insulin resistance and inflammation	Reduced genetic instability and cancer pathogenesis[1,3]
Sociocultural Factors	Strong traditional norms	Exposure to health education and public health policies	Potential protection if healthy behaviors are maintained; increased risk with adverse acculturation[4]

Table 2: Major Protective Mechanisms Linking Immigration, Obesity, and Cancer Prevention

Protective Factor Associated with Immigration	*Biological/Behavioral Pathway*	*Impact on Obesity*	*Implications for Cancer Risk*	*Key Supporting Evidence*
Healthier baseline lifestyle (pre-migration)	Lower insulin resistance and inflammation	Reduced adiposity	Decreased genetic instability and tumor initiation	Berger (2014); Hursting et al. (2012)[1,9]
Traditional dietary patterns	Improved metabolic regulation and antioxidant intake	Weight maintenance	Reduced hormone-driven and inflammatory cancers	Ruiz & Hernández (2014); Martínez-Rodríguez et al. (2020)[6,8]
Increased physical activity	Energy balance and adipokine regulation	Lower obesity prevalence	Reduced obesity-associated cancer pathways	Goodwin & Stambolic (2015)[2]
Improved access to preventive healthcare	Early screening and risk modification	Obesity management	Early cancer detection and prevention	Lippman et al. (2018)[10]
Reduced exposure to obesogenic environments (initially)	Lower chronic metabolic stress	Delayed obesity onset	Suppressed cancer-promoting metabolic signaling	Kompella & Vasquez (2019); Krupa-Kotara & Dakowska (2021)[3,4]

identification and lifestyle modification.[10] Over time, such interventions reduce the cumulative impact of obesity on cancer incidence and mortality.[4]

However, these protective effects are not uniform and may diminish with prolonged residence due to acculturation, adoption of sedentary behaviors, and increased exposure to processed foods. Nevertheless, during early and mid-stages of migration, immigration remains a significant protective determinant capable of moderating genetic risk through behavioral, metabolic, and healthcare-related pathways.[5,7]

Immigration can function as a protective health determinant by modifying environmental and behavioral exposures that interact with genetic predisposition. Through improved lifestyle factors, enhanced preventive care, and reduced metabolic stress, immigration has the potential to lower obesity prevalence and disrupt obesity-driven carcinogenic processes, particularly during the early phases of settlement.

Healthcare Access and Preventive Interventions

Access to healthcare services plays a central role in modifying and preventing genetically induced diseases such as obesity-related cancers, particularly within immigrant populations. Migration often alters individuals' exposure to healthcare systems, preventive infrastructures, and public health interventions, which can significantly influence the expression of genetic susceptibility to obesity and cancer. Enhanced access to primary care, screening programs, nutritional counseling, and lifestyle-focused interventions provides opportunities to interrupt the biological pathways linking genetic predisposition, obesity, and carcinogenesis.

Preventive healthcare services are especially critical given the well-established mechanistic links between obesity, metabolic dysregulation, chronic inflammation, and genetic instability that drive cancer development.[1,3] Regular health assessments and early screening enable the identification of obesity-related metabolic abnormalities such as insulin resistance and hormonal imbalance that are known to increase cancer risk.[2,4] For immigrant populations, integration into healthcare systems with structured preventive care pathways can therefore reduce long-term disease burden despite inherited risk factors.

Nutrition- and lifestyle-based preventive interventions represent another key benefit associated with healthcare access following immigration. Evidence consistently demonstrates that dietary quality, physical activity, and weight management are central modifiable factors in reducing obesity-driven cancer risk.[8,9] Healthcare systems that emphasize preventive nutrition education, culturally adapted dietary guidance, and community-based physical activity programs can support immigrants in maintaining protective behaviors that counteract genetic vulnerability. Such interventions are particularly relevant for hormone-related cancers, including breast cancer, where obesity-related signaling pathways play a decisive role.[6,7]

Cancer prevention strategies embedded within healthcare systems including population-based screening, risk stratification, and early intervention further amplify these protective effects. Structured screening programs facilitate earlier detection of obesity-associated cancers, improving prognosis and reducing mortality.[10] For genetically predisposed individuals, consistent engagement with preventive healthcare can shift disease trajectories by addressing obesity as an intermediary risk factor, rather than allowing genetic susceptibility to progress unchecked.[5]

Table 3: Role of Healthcare Access in Preventing Obesity-Related Cancers Among Immigrant Populations

Preventive Component	*Mechanism of Action*	*Impact on Genetic Risk*	*Supporting Evidence*
Primary healthcare access	Early identification of obesity and metabolic dysfunction	Reduces progression from genetic susceptibility to disease	1,4
Nutritional counseling	Improved diet quality and weight control	Modulates gene–environment interactions	8,9
Lifestyle interventions	Increased physical activity and energy balance	Lowers inflammation and hormonal dysregulation	2,5
Cancer screening programs	Early detection of obesity-associated cancers	Improves outcomes despite genetic predisposition	7,10
Preventive oncology initiatives	Risk stratification and targeted prevention	Interrupts obesity-driven carcinogenic pathways	3,6

Table 4: Key Challenges and Moderating Factors Influencing Obesity- and Cancer-Related Outcomes Among Immigrant Populations

Moderating Factor	*Description*	*Impact on Obesity*	*Impact on Cancer Risk*	*Supporting Evidence*
Acculturation	Adoption of host-country lifestyle and diet	Increased caloric intake and reduced diet quality	Elevated risk via metabolic and hormonal pathways	3,8
Socioeconomic Status	Income, education, and employment conditions	Limited access to healthy food and exercise	Delayed prevention and increased exposure to risk factors	2,4
Healthcare Access	Availability and utilization of preventive services	Reduced obesity management	Late-stage cancer diagnosis	9,10
Cultural Practices	Traditional dietary and lifestyle behaviors	Protective or harmful depending on retention	Modulation of obesity-related cancer pathways	5
Genetic and Epigenetic Factors	Gene–environment interactions	Differential obesity susceptibility	Variable cancer progression and prognosis	1,6

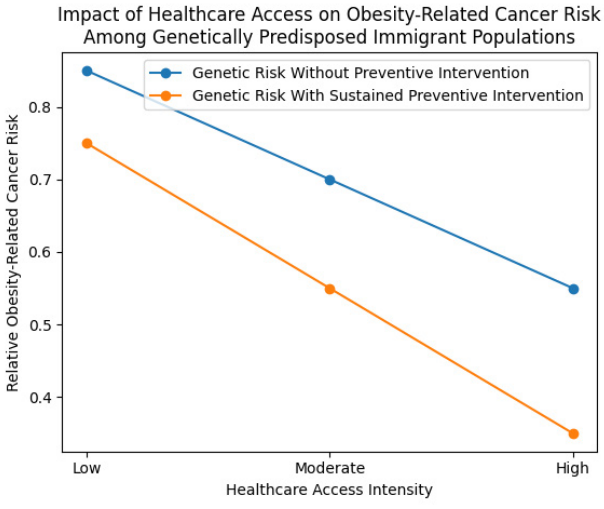

Fig 1: The graph clearly illustrates that increasing healthcare access is associated with a progressive reduction in obesity-related cancer risk, and that sustained preventive interventions significantly amplify this risk reduction across all access levels. This visually highlights the moderating role of healthcare access in disease prevention among genetically predisposed immigrant populations

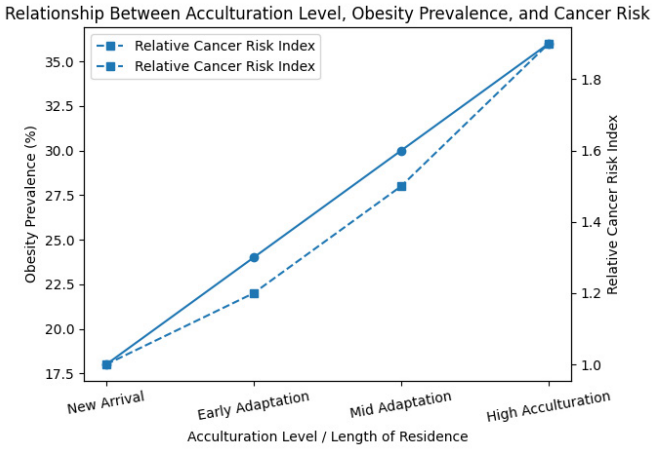

Fig 2: The values shown are illustrative and intended to demonstrate conceptual trends. The figure highlights how increasing acculturation may influence obesity prevalence and associated cancer risk among immigrant populations through changes in lifestyle, environmental exposure, and behavioral factors, rather than representing empirical measurements

At the same time, disparities in healthcare access remain a moderating factor. Socioeconomic barriers, legal status, language challenges, and health literacy can limit immigrants' utilization of preventive services, thereby weakening potential protective effects. Addressing these barriers through inclusive health policies and culturally competent care is essential to fully realize the preventive benefits of immigration-related healthcare access.[2,10]

Challenges and Moderating Factors

While immigration can positively influence the modification and prevention of genetically induced diseases such as obesity and cancer, several challenges and moderating factors shape the magnitude and sustainability of these benefits. These factors operate at biological, behavioral, socioeconomic, and structural levels, often interacting with underlying genetic susceptibility to influence disease outcomes.

One major challenge is acculturation, which frequently alters dietary patterns, physical activity levels, and health behaviors over time. Initial protective effects associated with traditional diets rich in whole foods and plant-based nutrients may diminish as immigrants adopt energy-dense, highly processed foods common in host countries. Such dietary transitions can exacerbate obesity-related metabolic dysregulation, including insulin resistance, chronic inflammation, and altered adipokine signaling, all of which are mechanistically linked to cancer development and progression.[1,3] Evidence consistently demonstrates that obesity acts as a critical mediator between genetic predisposition and cancer risk, particularly for hormone-sensitive cancers such as breast cancer.[6,7]

Socioeconomic status represents another key moderating factor. Many immigrant populations face economic constraints, limited education, and employment in high-stress or physically demanding occupations, which can restrict access to healthy foods, safe environments for physical activity, and preventive healthcare services. These conditions increase vulnerability to obesity and its downstream carcinogenic pathways, including genetic instability, oxidative stress, and disrupted cellular signaling.[2,4]

Healthcare access and utilization further moderate outcomes. Although migration may offer improved health systems, barriers such as language differences, lack of insurance, cultural mistrust, and limited health literacy can delay cancer screening, early detection, and obesity management. Reduced engagement with preventive services undermines the potential benefits of early intervention emphasized in cancer prevention frameworks.[9,10]

At the biological level, genetic heterogeneity among immigrant populations influences how environmental exposures translate into disease risk. Variations in genes related to metabolism, inflammation, and hormone regulation may amplify or attenuate the impact of obesity on cancer development. Chronic exposure to obesogenic environments can further induce epigenetic modifications that alter gene expression across generations, potentially offsetting initial protective effects of migration.[5,8]

Conclusion and Research Implications

This study underscores that genetically induced diseases such as obesity-related cancers are not solely determined by inherited risk but are profoundly shaped by modifiable environmental and behavioral factors. Evidence consistently demonstrates that obesity plays a central mechanistic role in cancer development through metabolic dysregulation, chronic inflammation, hormonal imbalance, and increased genetic instability.[1-3] Immigration, as a major life-course transition, offers a unique context in which genetic predispositions can be modified through changes in diet, physical activity, healthcare access, and exposure to preventive health systems. These changes can attenuate obesity prevalence and, consequently, reduce cancer risk among genetically susceptible populations, highlighting the importance of gene–environment interactions in disease prevention.

The findings align with extensive epidemiological evidence linking obesity to increased risks of multiple cancers, including breast cancer, through molecular and metabolic pathways that are sensitive to lifestyle and dietary patterns.[4,6,7] Immigration can facilitate exposure to healthier dietary practices, preventive screening programs, and public health interventions that target energy balance and metabolic health, thereby disrupting the progression from genetic susceptibility to disease manifestation.[8,9] In this context, immigration may function as a protective determinant when supportive social and healthcare structures are present.

From a research perspective, these conclusions point to several important implications. First, future studies should adopt longitudinal and transnational designs to better capture how migration trajectories influence obesity and cancer risk over time. Second, greater integration of genetic, epigenetic, and social determinants of health is needed to clarify how environmental transitions associated with immigration modify disease expression.[3,5] Third, public health research should prioritize culturally responsive prevention strategies that leverage protective behaviors observed among immigrant populations while mitigating the adverse effects of acculturation and socioeconomic stressors.[11-18]

Finally, these findings support broader cancer prevention frameworks that emphasize population-level interventions, lifestyle modification, and equity in healthcare access.[10] Recognizing immigration as a potential opportunity for modifying genetic risk reinforces the need for policies and preventive programs that address obesity and cancer through integrated, context-sensitive approaches. Such strategies hold promise for reducing the long-term burden of genetically

influenced diseases and advancing more effective, inclusive public health outcomes.

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