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Review Article

The Link Between Childhood Poverty and Metabolic Syndrome: An Indian Scenario

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Abstract

Acute poverty has a severe impact on children in India, where 30% of all children living in extreme poverty worldwide are born. The truth is that 36% of the world's poorest children reside in South Asia, with India hosting 84 percent of this population. Besides, more than 45 million children in India are affected by the COVID-19 pandemic's extreme poverty, which accounts for 30% of all children worldwide. Childhood poverty, which is frequently associated with accelerated aging, may have a significant impact on immune system function, which may lead to dysregulation of inflammatory processes in response to foreign substances and a change to unfavorable proinflammatory states. The term "Metabolic Syndrome" (MetS) describes a group of disorders, such as high blood pressure, high blood sugar, insulin resistance (IR) and elevated adiposity, that frequently co-occur and increase the risk of stroke, type 2 diabetes (T2DM), and cardiovascular diseases. An extensive incidence of IR among children exhibiting MetS was found in an Indian cross-sectional investigation. Over time, the scientific community has become more cognizant of the critical role the immune system plays in maintaining systemic metabolic homeostasis. The maintenance of excellent "metabolic health" over the course of a person's life depends critically on this interaction between the immune and metabolic systems. Two major stress-signaling pathways that contribute to immunological dysregulation in children during poverty are the Autonomic Nervous System (ANS) and the Hypothalamic-Pituitary-Adrenal (HPA) axis. Prolonged HPA axis activation brought on by poverty-induced stress can directly contribute to the pathophysiology of T2DM. Early traumatic events and lifestyle modifications induced by poverty may also have an impact on how quickly telomeres shorten throughout the course of a person's lifespan. Telomere shortening brought on by immune system aging slows down T- and B-cell population renewal and clonal proliferation, aggravating MetS. Early-life nutrition results in long-lasting alterations in DNA methylation that have an effect on a person's health and aging-related disorders throughout their lifetime. In order to further validate the causal relationship between these crucial intersecting events that the article seeks to capture during poverty, additional research will be needed to collect data on the prevalence of MetS, immunological parameters, including retrospective and prospective longitudinal studies in larger Indian cohorts.

Introduction

Understanding poverty in India

With roughly 1.4 billion people, India is one of the most populous nations in the world. More than 17% of the world's population resides in India alone [1]. With such a large population, there aren't enough resources to maintain the majority of residents' livelihoods and standards of living. India has a long history of poverty, with 63.1% of its people subsisting on less than $1.90 a day in 1977 [2,3]. This percentage has since sharply declined to 22.5 percent in 2011; nonetheless, this still equates to an alarming 296 million people living in extreme poverty. More specifically, children in India bear a heavy burden of acute poverty. 30% of all children in extreme poverty worldwide are born in India [4,5]. In reality, South Asia is home to 36% of the world's poorest children, while India alone makes up 84 percent of this population [2]. In a recent research titled "Ending Extreme Poverty: A Focus on Children," the World Bank Group and UNICEF (United Nations Children's Fund) discovered that children are disproportionately impacted by extreme poverty [6]. It's interesting to note that children made up half of the extremely poor despite making up only a third of the population under

study. Children therefore have a higher likelihood of living in extreme poverty than adults. It's significant to note that from the start of the COVID-19 pandemic, 150 million more children around the world have ended up living in poverty [7]. More than 45 million children in India are affected by the COVID-19 pandemic's extreme poverty, which accounts for 30% of all children worldwide [8].

The potential association between childhood poverty and metabolic syndrome

Aging often causes a progressive loss in immune system function, among other things, which raises the risk of a number of illnesses such infections and cancer [9,10]. This aging-related immunological dysfunction is specifically referred to as "immune-senescence," which describes changes in the organizational and functional characteristics of various immune components, including the innate immune system, as well as the loss of diversity in adaptive immunity [11,12]. Childhood poverty, which is typically linked to accelerated aging [13] may have a major effect on immune system function, which can cause dysregulation of inflammatory processes in response to foreign substances and a shift to unfavorable proinflammatory states [14,15]. For instance, a recent study [16] on 342 African American teenagers from the Southeast of the United States examined the potential link between familial poverty throughout adolescence (years 11 to 18) and Insulin Resistance (IR) in young adulthood (ages 25 to 29). The participants were tracked for nearly two decades (2001-2019). The findings suggested that adolescent family hardship might have contributed to both rapid immune cell aging and greater levels of IR in young adults. The researchers also discovered that the longer subjects lived in poverty throughout youth, the higher their chance of developing diabetes and insulin resistance as adults, suggesting a potential connection between poverty and accelerated immunological aging. Importantly, this study also emphasizes the importance of taking a life course perspective when examining social differences throughout time. This is partly because, when compared to researching the population at younger ages, examining the older population at a certain age range will leave out other significant health differences [17].

The term "metabolic syndrome" (MetS) refers to a group of disorders, including elevated blood pressure, high blood sugar, and elevated adiposity, frequently co-occur and raise the risk of stroke, Type 2 Diabetes (T2DM) and cardiovascular disease [18-20]. Also closely related to IR is the metabolic syndrome [21,22]. For instance, a cross-sectional study conducted in India discovered a substantial prevalence of IR among schoolchildren producing MetS . In total, 21.8 percent of these kids had MetS. A HOMA (Homeostatic Model Assessment)-IR of 2.5 was present in almost 55% of the children [23]. As a result, the purpose of this research is to investigate the relationship between metabolic syndrome, immunological aging, and childhood poverty in India. MetS affects about 25% of the world's population, and it is more common in people with low socioeconomic status (SES) [24,25]. Low early-life SES was linked to an 83 percent higher risk of MetS in later life, according to a study comparing the relative contributions of early-life SES and current SES in determining MetS risk [24,26]. This further demonstrates that implementing targeted interventions in childhood may lessen the prevalence of MetS among the poor. Low SES during pregnancy and gestation causes pregnant women to lack certain macronutrients like protein and carbs [27,28], which leads to reduced child birth weight, a surrogate marker for fetal growth, and later insulin resistance, glucose intolerance, hypertension, and obesity in adults. Furthermore, famine is also a natural paradigm for examining how undernutrition in adolescence affects persons later in life [29]. Following the end of World War II, the Dutch winter famine (1944-1945) was one of the most Well-known famines in history, characterized by a 5-month period of extreme undernourishment in the western urban region of the country. Lumey [30] investigated a birth cohort of 3307 singletons born between 1945 and 1946. A study [31] found a greater prevalence of T2DM and dyslipidemia in adult offspring aged 59 following maternal undernutrition as a result of famine during pregnancy. Moreover, the Dutch Famine Birth Cohort Study carried out by Ravelli et al. [32]. showed that exposure to a famine during pregnancy resulted in considerable glucose intolerance and insulin resistance in offspring at ages 50 and 58 [32].

Case studies in India of malnutrition and the metabolic syndrome

Additionally, postnatal malnutrition can result in hyperinsulinemia, impaired glucose tolerance, and an increased risk of diabetes in children [33,34]. Previous research has shown a connection between T2DM and low SES [35,36]. Additionally, more research tend to point to the crucial connection between childhood undernutrition and a higher risk of acquiring T2DM [37,38]. A 10-year follow-up study conducted in an urban South Indian population found a significant association between SES gradient and prevalence of diabetes and CV risk factors, with a higher concentration among those in the middle and lower-income categories [39,40]. Another serial epidemiological study from Jaipur [41], India found that the prevalence of smoking, diabetes and dyslipidemia rose greater in those with lower educational status compared to those with higher education. Individuals in the low SES group had a higher overall cardiovascular risk based on the widely used worldwide cardiovascular risk assessment methodology. As a result, this may help to explain why, despite a very low prevalence of obesity in Indians compared to Americans, diabetes is at least twice as common in Indians. This is likely because more people in India belong to low SES groups [42]. In addition, one in two persons in the age range of 25 to 64 in India's tribal (Aboriginal) population has hypertension either reported or determined to exist [43]. Despite a constant increase in the GDP over the previous 10 years, India's average inflation rate, particularly food inflation [44], has remained high. Since the poor have a harder time affording the healthier options due to the high inflation rate, the risk of developing metabolic syndrome is enhanced [45].

The symphony of immunological dysfunction, metabolic syndrome and undernourishment

An exhaustive assessment of the literature looked at malnutrition and compromised immune function [46,47]. A literature review [48] that included 3402 articles published

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between 1970 and 1990 and 33 articles after 2003, of which 245 met the inclusion criteria, found that malnutrition was associated with impaired gut-barrier function, decreased exocrine secretion of protective substances, low plasma complement levels along with atrophic thymus, and significant reductions in antibody levels in severely malnourished children after vaccination, as opposed to no such change. Cytokine patterns were skewed towards an anti-inflammatory The 2-response. The study's observational nature and cross-sectional analysis approach, however, may be seen as potential weaknesses [49]. The immunological priming of Dendritic Cells (DC) and monocytes, as well as the activity of effector memory T cells, are both known to be compromised by starvation [50,51]. Furthermore, it's likely that poverty-related hardships prevent parents from having kind and considerate relationships with their kids, which could potentially negate the impact of SES on the inflammatory process in kids [52,53]. Children from low SES backgrounds typically have lower levels of education as adults, which keep them from acquiring healthy dietary habits [54] and behavior patterns to reduce chronic inflammatory processes. Early childhood poverty has been associated with poor adult mental health [55,56]. There is evidence that people with mental health disorders experience persistent inflammation [57,58].

Along with its better-known functions of providing defense against external infections and preventing the growth of tumors, the immune system also plays a critical role in the control of systemic metabolic homeostasis, which has gained widespread recognition over time [59,60]. The maintenance of excellent "Metabolic Health" over the course of a person's life depends critically on this interaction between the immune and metabolic systems [61,62]. Any disturbances in this complex immune-metabolic cross talk have the potential to cause MetS, which will most likely lead to T2DM and Cardiovascular Illnesses (CVDs) [63-66]. Moreover, researchers looked at the relationship between early childhood income and disease states that occur as adults and have a strong correlation with immune system malfunction and immunological-mediated pathogenic processes [14,67]. This study employed annual family income reports obtained between the prenatal year and age 15 years, in contrast to numerous epidemiological studies that only used retrospective data of childhood SES. It followed participants from birth to adulthood. To eliminate the possibility of any other confounding variables, the study also included wealthy controls for conditions governed by income. This result therefore confirmed earlier findings from a few other research that a steady income stream is essential for a metabolically healthy adult life, particularly for young people at the low end of the income distribution. The absence of direct evaluations of immune parameters, which was a major flaw in this study's design based on the basic immunological premise, was identified.

The Autonomic Nervous System (ANS) and the Hypothalamic-Pituitary-Adrenal (HPA) axis are two primary stress-signaling pathways that contribute to immune dysregulation [68,69]. When the brain perceives a stressful situation, such as poverty, it activates the HPA axis [70] and the Sympathetic-Adrenal Medullary Axis (SAM), resulting in the release of hormones that are known to modulate immune cell functions, including Adrenocorticotropic Hormone (ACTH), cortisol, growth hormone, prolactin, epinephrine and norepinephrine. Additionally, prolonged HPA axis activation brought on by stress can directly contribute to the pathophysiology of T2DM [71,72]. Additionally, pro-inflammatory indicators like CRP (C-reactive protein), IL-1, and IL-6 are driven by cytokines called adipokines that are generated from visceral adipose tissue, supporting the hypothesis that stress and inflammation are linked and result in T2DM and other metabolic illnesses [73-75]. Visceral obesity frequently co-occurs in people with T2DM, produced by stress-induced elevated cortisol levels [76,77]. Furthermore, unfavorable experiences in life and changes in lifestyle due to poverty may also have an impact on the pace of telomere shortening over the course of a person's lifespan [78,79]. These results led Kiecolt et al. [80] to demonstrate that childhood adversities have detrimental impacts on cell aging in later life, as seen by the existence of shortened telomeres, demonstrating the lasting effects of childhood adversity throughout the life. Telomere shortening, linked to immune system aging, slows down cell renewal and clonal expansion of T- and B-cell populations [81,82].

Importantly, it should be noted that immunological dysfunction and starvation have a somewhat "chicken-and-egg" relationship, with each condition both causing and resulting from the other. Immune dysfunction brought on by poverty can leave permanent epigenetic marks on DNA that can be passed on to offspring, resulting in children inheriting a compromised immune system that can even be passed down multiple generations [83,84]. Children who have a nutritious diet may nonetheless experience the effects of malnutrition, such as MetS, due to their altered immune systems. Early-life nutrition causes long-lasting alterations in DNA methylation that have an effect on a person's health and aging-related disorders throughout their lifetime [83]. Inhibiting epigenetic enzymes like DNMT (DNA methyl transferase), HDAC (Histone deacetylase), or HAT (Histone acetyl transferase) or modifying the availability of substrate required for those enzymatic activities are two ways that nutrients can act. The expression of key genes is subsequently altered, which has an effect on our longevity and general health. By regulating the activity and function of microRNAs (miRNAs), nutrition may also be able to influence gene expression in a variety of biological processes, including development, differentiation, cell proliferation, metabolism and inflammation, as well as in a number of pathological ones [85,86]. Recent research suggests that dietary factors have important roles in the development of CVDs, T2DM, and other conditions through modulation of miRNA expression. Given the interactions between DNA methylation, miRNAs, and post-translational modification of histones (PTMs), it is likely that nutrients change the nature of PTMs in addition to altering the DNA methylation pattern to regulate gene expression in a variety of tissues, including immune cells [87-89].

Conclusion

When considered as a whole, it is not unexpected that India, where more than 40% of children under the age of 5 are malnourished, has also earned the title of "diabetes capital" of the world, with an estimated 65 million diabetic patients aged 20-

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79 years in 2013 and a high prevalence of MetS. The Well-known "thrifty genotype" hypothesis proposed that population metabolic differences have arisen through different ancestral exposure to "feast and famine" cycles, causing a large Indian population to live with MetS and then develop full-blown metabolic diseases like T2DM and CVDs, among others, along with diverse immunological dysfunction that in turn can further exacerbate the severity of MetS, thereby establishing a vicious cycle of interconnected events. As a result, holding that there is a causal link between immunological aging and/or malfunction and MetS may not be incorrect. Designing an effective public strategy to reduce poverty-related problems while focusing on early health-related interventions in the Indian population is crucial since this nexus might have long-term detrimental impacts on a person's metabolic health. The proper prenatal and postnatal nutritional interventions among pregnant women and their offspring must be emphasized with particular attention in a policy-directed way, especially to the people who belong to low SES. More studies will also be required to accumulate information on the prevalence of MetS, immunological parameters, including retrospective and prospective longitudinal studies in larger Indian cohorts, in order to further validate the causal relationship among these intersecting events the article seeks to capture.

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Conflict of Interest

The authors have no conflict of interests to report.

Ethical Statement

The research has been conducted ethically.

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