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Review Article

Brain Syndemics: Cognitive Deficit, Pathways of Interaction, and the Biology of Inequality

Merrill Singer*

Department of Anthropology, University of Connecticut, Storrs, Connecticut, USA

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ABSTRACT

Children born into and raised in disadvantaged families tend to experience poorer health and more developmental delays, lower achievement, and a greater number of behavioural and emotional problems than children from wealthier homes. There is growing evidence that poverty and social inequality leave their imprint on brain structure as well. The brain exhibits considerable plasticity, one expression of which is shaped by the biology of inequality. A specific consequence is cognitive deficit found among children raised in poverty and subject to social discrimination. This paper argues that several pathways impacted by poverty, including chronic stress, malnutrition, exposure to heightened levels of air pollution, and other toxin exposures, syndemically link social inequality to underlying neural mechanisms and to suboptimal brain development and structure. These deficits need not be permanent and are reversible through urgently needed structural, socio-economic intervention.

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Introduction

Children born into and raised in disadvantaged families tend to experience poorer health and more developmental delays, lower achievement, and a greater number of behavioural and emotional problems than children from wealthier homes. There is growing evidence that poverty leaves its imprint on brain structure as well [1, 2]. The brain exhibits considerable plasticity, one expression of which is shaped by the biology of inequality. A specific consequence is cognitive deficit found among children raised in poverty [3, 4]. Several pathways impacted by poverty, including chronic stress, malnutrition, air pollution, and other toxin exposures, link social inequality to underlying neural mechanisms and to suboptimal brain development and structure. The continual interactions of these kinds of biological and social processes underlie what Lock and Nguyen call “biosocial differentiation” [5]. This body of research contributes to our understanding of the embodiment of social inequality and to recognition that brain development is a biosocial process [6-8].

The human brain has a protracted period of development that begins during pregnancy. Fetal programming is the process whereby the fetus

engages and is acted on by the intrauterine environment. During sensitive periods of development, fetal programming can produce structural and functional changes in cells, tissues, organs, and organ systems independently, or through interactions with the external environment. Neural development continues after birth as structural and functional changes are impacted by the child’s physical and social environment.

Analysis of a large dataset showed that changes in behavioural traits can be predicted from brain structure [9]. Specifically, research shows relationships between: i) poverty and brain surface area, most prominently in regions supporting language, reading, executive functions and spatial skill; ii) growing up in poverty and the experience of greater degrees of chronic stress, malnutrition, and environmental toxins; and iii) level of exposure to these environmental threats to health early in life and both poorer cognitive development and school performance on average. While these demonstrated relationships do not prove causality, they are highly suggestive. Socio-economic disparities in the brain’s neurocognitive systems are seen as early as the second year of life and persist throughout the lifespan [10]. Considering this research, the question is raised: under conditions of experiencing childhood

*Correspondence to: Merrill Singer, Ph.D., Emeritus Professor, Department of Anthropology, University of Connecticut, Storrs, 06269, Connecticut, USA; Tel: 8605935349; E-mail: Merrill.Singer@Uconn.edu

poverty, is there a tripartite brain syndemic composed of stress (e.g., as seen in cortisol levels), malnutrition, and environmental toxins that lead to lowered cognitive development without intervention?

Without a doubt, consideration of these issues is fraught as it risks tumbling into “blame the victim” assertions. As Lende underlines, a primary risk is the tendency to “conceal the social forces – both the actual poverty suffered by people and the systemic effects and politics of inequality – from view” by stressing failures of parental care and limitation on cognitive stimulation in poor homes [11]. Such concealment is avoided here by addressing the structural causes of poverty and its embedment in multiple structurally imposed burdens (e.g., discrimination, stigmatization) [12]. Focus on structural vulnerability is a key component of syndemic theory [13, 14]. With regard to social structure and brain health, Lende observes, “The embodied brain is not just a fundamental place of suffering, it is a fundamental cause of social suffering [11]. People suffer through their embodied brains, through despair and toxic stress, and destructive behaviour. The brain suffers poverty.” Poverty, in turn, is a social product. It reflects the distribution of power and wealth in society and, it must be stressed, provides benefits to the non-poor, especially the wealthier sectors of society [15]. Stinchcombe contended that there are “two broad approaches to studying inequality” [16].

The first explains socioeconomic differences based on individual characteristics, such as ethnicity, gender, character, personality, or education. The second emerges from “a theory that people at the top try to keep people at the bottom unequal, for their own advantage.” As Brady, Biradavolu, and Blankenship state, “Rather than simply saying [the poor] are oppressed or disadvantaged, exploitation means there is an identifiable actor receiving disproportionate rewards” [17]. Flowing from this relational understanding, I suggest there is a causal “power/poverty/brain connection” that should not be ignored in assessing the health and developmental costs of inequality. Some parts of the structure of inequality are intentional (e.g., redlining in housing) and some not seemingly intentional (e.g., aspects of the tax code) but all of which constitutes structural violence suffered by the poor [18, 19].

In this paper, I review the relevant bodies of research on embodied inequality and its effects on the brain; propose and support the assertion that there is a potential adverse syndemic brain interaction involving biological and social conditions, discuss the implications for children and society, and assert the need for additional research on this possible syndemic.

Syndemics, Health and Experience

Syndemic theory addresses complex biosocial relationships, namely interactions among multiple diseases or health conditions and their socio-political or ecological contexts [20]. Syndemics scholarship rests on the understanding that health conditions are a corollary of social context and that social conditions contribute significantly to biosocial negative feedback loops resulting in heightened rates of morbidity and mortality. Central to the formulation of the syndemics approach is a focus on the vertical linkages among behaviour patterns, social groups (e.g., classes, ethnicities), population health patterns, and encompassing political and economic structures. Syndemic theory is used to investigate

the health and social consequences of “the biology of poverty and the consequences of discrimination, the role of power, control, oppression, and social inequality in the making of health and disease, and forms of resistance to these forces” [13]. Specifically, the term syndemics labels the consequential interaction of two or more diseases in a population and non-disease factors like social inequality in promoting disease interface. While syndemics adversely affect the body, health, and life experience, the reverse may also be true; that is, life experience affects the shape and function of the body and its constituent tissues. Syndemics have been described involving various body organs, including the lungs, kidneys, liver, and heart [21-23]. While syndemics have been documented that involve human mental health, previous research has not explored the possibility of syndemics involving brain structure and its connection to life experience.

An exemplary of the biosocial interaction in these kinds of epidemiological events is the diabetes/depression syndemics described by Mendenhall. In her initial research, Mendenhall examined the interactions that occur among violence, immigration, diabetes, depression, and abuse in a sample of Mexican immigrant women in Chicago [24]. A primary finding of this research was that among these women, their dominant sickness experience was not diabetes per se but rather their personal histories of exposure to violence, fear attached to harsh immigration policy, missing sisters and mothers in Mexico, anxiety about financial issues, and untreated depression. Additionally, she found that the participants in her study felt social pressure to adhere to traditional ‘women’s activities,’ such as cooking and cleaning, despite being told by their doctors to change their diets and increase physical activity. Change was resisted by their husbands, who adhered to traditional norms leading to conflict and depression that interacted adversely with their diabetes. Subsequent research investigated how a combination of global and differing local factors modulate the ways diabetes is perceived, experienced, and embodied among women in varying contexts [25-27]. Using life history analysis, she described the diverse social, political, and cultural factors that produce syndemic diabetes differently between countries and across income groups. Adds Kline, in syndemics, social factors often interact to create complex forms of social subjugation. Consequently, it is important to examine intersecting inequalities that produce the social contexts in which syndemics occur [28].

As this discussion suggests, syndemics are local in their configuration and reflect local realities that result in unique co-morbidity clusters that are promoted by various configurations of social, economic, and political forces.

Brain Structure and Poverty

For some time, neuroscientists have known that rather than being fully hard-wired, the brain is both malleable and shaped by life experience [29, 30]. A child’s experience, in turn, varies based on family circumstances, meaning that experience imprints on brain development [31]. One such influence appears to be family socio-economic status. Socio-economic status is a “complex construct” that reflects “household income, material resources, education, and occupation, as well as related neighbourhood and family characteristics” [32]. A substantial body of literature shows an association between family poverty and poor school

performance. Socio-economic differences are found across multiple cognitive and achievement measures, including IQ (IQ tests are said to index the quality of cognitive functioning in certain domains; they have been critiqued for including cultural bias and for inflating their predictive value), reading ability, verbal ability, achievement test scores, motivation to learn, and high school graduation rates. Coming from a poor family is especially associated with lower reading comprehension [33].

Children who experience poverty early in life (preschool and early school years) tend to have lower rates of school completion than children who only experience poverty later on [34, 35]. Even as early as 48 months of age, many children from poor households have a lower ability to understand written language than their middle socio-economic counterparts [36]. A number of studies have found that socio-economic disparities in a number of abilities, including in language and memory, can be detected by the second year of life [37-39]. These findings suggest that the impacts of poverty begin early in a child's life and have prolonged consequences. This supports the idea that intervention should begin early in child development. Unclear from this educational and social science research, however, are the precise factors associated with poverty and poor outcomes for children, as discussed in the next section.

In recent years, using noninvasive neuroimaging technologies, neuroscientists have made important contributions to the literature on the effects of poverty on children, specifically on brain structure. In a study of over 1,000 children and adolescents recruited from socio-economically different homes located in ten sites across the United States, Noble and colleagues assessed the cerebral cortex, the outer layer of brain cells [39]. These cells, commonly called grey matter, are distributed in a thin coat covering the brain. Importantly, this layer consists of folded ridges (called gyri) that produce gullies (called sulci). These features allow a larger cortical surface area than would otherwise be possible within the confines of the skull. The result is an expanded brain surface area. This is important as the cortex is primarily responsible for thinking and information processing, as well as producing and understanding language. Moreover, brain surface area is greater in more intelligent (measured by IQ) children by age 10 [40]. In their multi-site pediatric imaging, neurocognition and genetics study, Noble and co-workers found that two social factors were associated with differences in the surface area of children's brains: parental educational attainment and family income. These associations were found across most sectors of the brain, but especially in the bilateral inferior temporal, insula and inferior frontal gyrus, and in the right occipital and medial prefrontal cortex, regions involved in language, executive functions, spatial skills, and self-regulation. These capacities have been shown in multiple studies to vary by parental socio-economic status [41-43].

This study also addresses three questions raised in the literature. First, can the differences in brain structure be explained by genetic differences among children or in terms of so-called racial groups? As they note, in the U.S. "tests regularly show sizable gaps in school readiness between young white children and young black and Hispanic children...", but these patterns may occur because ethnicity and socio-economic status in the U.S. are entwined and complex: racism promotes poverty among disparaged ethnic minorities while the high poverty levels among ethnic minorities is used to reinforce racism [35, 44, 45]. Consequently, it is

important to adequately distinguish socio-economic status from ethnic background. Second, do all children raised in poverty have comparatively smaller cortical surface areas and, vice versa, do all children raised in wealthier homes have comparatively larger cortical surface areas? In other words, do the risk factors of poverty adversely affect all poor children [46]? Third, are differences in family income proportionately associated with greater differences in brain structure among children from the most disadvantaged families? In short, is there a traceable linear link between income and brain surface area?

Noble and her colleagues found: i) contra Trzaskowski *et al.*, socio-economic differences in brain structure were independent of genetically linked ethnic groups (or what are still problematically labeled as "races"); ii) outcomes could not be individually predicted based on family income; many children from disadvantaged homes had larger cortical surface areas, and the reverse was true of many children from advantaged homes who had comparatively smaller surface areas; and iii) family income was logarithmically associated with surface area, which means that incremental increases in family income are reflected in increases in children's brain surface area, especially at the lower end of the family income continuum [47].

Other studies by Noble's group; and by other researchers, have also affirmed the relationships identified in Noble *et al.* [48, 49]. In a longitudinal MRI study of over 600 children and adolescents, McDermott *et al.* reported finding a positive association between socio-economic status and cortical surface area in the frontal lobe, temporal lobe, and hippocampus [50]. Specifically, the study found higher family socio-economic status was associated with expansion of lateral prefrontal, anterior cingulate, lateral temporal, and superior parietal cortices and ventrolateral thalamic, and medial amygdalo-hippocampal, subregions of the brain involved in learning and memory. Differences in brain structure may account for between 15 and 44 percent of family income-related school achievement differences during adolescence [51, 52]. There are still many areas of uncertainty in linking brain structure to socio-economic status, especially on the issue of cortical thickness, and not all studies have supported this relationship [53]. A confounding factor may be that socio-economic association with cortical thickness may vary with age or cognitive ability [54].

Poverty, Chronic Stress, Malnutrition, and Air Pollution

Which features of growing up in poverty might leave a traceable imprint on brain structure? In other words, how does life experience shape brain biology? Three health-related environmental stimuli appear to be particularly important in this biosocial process: enduring chronic stress, suffering malnutrition, and exposure to environmental toxins like air pollution. These are pathways through which inequality can get under the skin and impact neural development [55-57].

I Chronic Stress

Early life stress involves the exposure during childhood to single or multiple events that challenge emotional and physical well-being and development and can overwhelm the ability to cope [58, 59]. While short-term adversity during childhood can activate the body's adaptive stress response system facilitating successful coping, prolonged or

chronic stress has biological costs [59]. Most research on early life stress addresses its influences on mental health; less attention has been devoted to structural effects including impacts on grey matter development [60]. Available evidence is beginning to suggest, however, that early life stress may interfere with typical brain development through several processes including accelerating synaptic pruning (a process that at normal rates removes less used and “redundant” synapses that are overproduced in the early years of life) and increases in myelination (the insulation of nerve fibers with a fatty sheath) [61, 62]. These are believed to be key components of brain plasticity, allowing the brain to re-organize as it adapts to the environment and recovers from injury during development. Animal studies indicate that adverse childhood experiences are among the most prominent risk factors for lasting alterations in neuronal circuits, neurotransmitter systems, neuronal architecture and plasticity, effects that are associated with emotional and cognitive information processing [63].

In humans, early life stress correlates with low socio-economic status. As Blair and Raver states, “It has become increasingly clear that one of the mechanisms through which poverty affects the health and well-being of children and adults is through the toxic effects of stress on the brain” [64]. As they point out, “in addition to reduced opportunity for types of stimulation that positively affect development, poverty is also characterized by an overabundance of types of stimulation that negatively affect development” [64]. Among these adverse stimuli are substandard housing, loud background noise, household turmoil and interpersonal conflict, distress suffered by family members (linked to financial pressures, stigma, discrimination, unsafe physical and social environments, lack of consonance with societal ideals, social alienation), and the impacts of these stressors on adult caregiving [65-69]. Of note, parental job loss has been shown to negatively affect both children’s health and school performance [70-72]. Moreover, Charles and Stephens documented a rise in divorce following job layoffs, while Lindner and Peters found negative effects of job loss on family stability and hence on the well-being of children [73, 74].

As this discussion suggests, poverty does not arise in a vacuum but is entwined with and magnified by other forms of social disadvantage that are embedded in a broader political economy of injustice, including institutional racism, factory closure, and the ethnic control functions of the carceral system [75-77]. Together, these factors produce an array of toxic biological and psychosocial stressors [78]. The accumulated burden induced by chronic stress is referred to as the allostatic load. When environmental challenges exceed an individual’s ability to cope, a state of allostatic overload ensues which can produce pathophysiological consequences [79]. Notably, high levels of allostatic load are found among people living in impoverished and ethnically segregated minority neighbourhoods [80, 81].

Of course, children from wealthier families may experience high levels of stress, and even with far fewer resources, poorer families find ways to buffer socio-economic pressures. A qualitative study of twelve low-income families, for example, found that although many reported they sometimes did not have enough to eat, they engaged in various strategies to limit food insecurity including seeking help from friends and kin, rationing, and selective purchasing [82]. In other research, parents, particularly mothers, report eating less so their children would have more

to eat [83]. As a result of successful coping and other factors, many poor children “beat the odds” and succeed at school and beyond. Still, there is abundant evidence that the cumulative stressor exposure among the poor is higher than for their wealthier counterparts, as reflected in various measures including self-reported psychological distress by children and parents [84]. Moreover, the confluence of poverty, inequality, and discrimination on average takes an enduring toll on parents and children alike [85, 86].

Among the mechanisms mediating stressor impact on brain development are stress-related hormones like cortisol [87]. Cortisol is produced by the hypothalamic-pituitary-adrenocortical system. Stress hormone levels modulate synaptic activity in the prefrontal cortex, the seat of executive function. Short-term exposure to cortisol mobilizes energy availability, enhances memory, and supports the immune system. It is secreted into the bloodstream in larger quantities during stressful events that trigger the flight-or-fight adaptive response. Chronic stress unleashes a constant flow of stress hormones pushing the body’s immune system into suboptimal functioning [88]. Longer-term or chronic exposure produces a range of adverse responses in the brain and elsewhere in the body [78]. Longitudinal research on a predominantly low-income population-based sample of over 1200 children followed from birth by Blair *et al.* found a higher level of salivary cortisol in chronically stressed individuals at ages 7, 15, and 24 months [89]. At age 3 years, higher cortisol levels were associated with lower executive function ability and, to a lesser extent, lower IQ. Similarly, in another longitudinal study, Sour *et al.*, found that an elevated pattern of cortisol was associated with the lowest mean level of cognitive functioning at age four [90].

II Malnutrition

Healthy brain development, especially during late gestation and early infancy, is dependent on adequate nutrient intake [91, 92]. It is well documented that malnutrition can affect brain development and structure. Various micronutrients, for example, vitamin B12, vitamin A, folate, retinoic acid, omega-3 fatty acids, zinc, copper, and iron play an epigenetic role in regulating gene expression that has consequences for both shaping brain development and playing a role in specific neurologic processes, such as modifying neuroplasticity, dendritic branching, synapse formation (especially the exuberant synaptogenesis of early brain development), neuron proliferation, and myelination [93]. Further, nutrient deficiencies (e.g., iron) “may contribute to the cycle of poverty in the inner city by limiting the ability of women to work, earn money, and afford iron-rich sources of food” for their children [94]. Research affirms that food insecure children are significantly more likely to suffer iron deficiency anemia compared to food secure children [95]. Undernutrition influences physical growth, activity, and motor development in children that may, in turn, modulate brain development. Two pathways are likely. Undernutrition may diminish both a child’s level of exploration of the environment due to illness or irritability and to a lower level of initiation of caregiver interactions. Either of these could lead to poor brain development [96].

Socio-economic factors impact childhood nutrition in several ways. Research shows that it is expensive to be poor. First, low-income Americans are forced to spend a significantly larger proportion of their income on housing than high-income Americans, which in turn, leaves

less money for food. Most poor families live in rental housing and must pay at least half of their available income for housing, while a quarter spend over 70% on housing [97]. Defining “rental exploitation” as the ratio of annual rents from all rental units over property value, Desmond and Wilmers show that rental apartments in neighbourhoods with less than 15% poverty rates have exploitation rates of about 10% [98]. By contrast, in neighbourhoods with high levels of poverty (50%-60%), exploitation more than doubles. This is especially evident in comparisons between African American and non-African American neighbourhoods. The substantial rent burden of low-income families directly contributes to them experiencing economic scarcity and inability to meet other basic needs, including food [99, 100].

Second, undernutrition occurs primarily when parents lack enough total income to spend a sufficient amount on food. Poor families must make difficult expenditure decisions in an environment of limited resources. According to the US Energy Information Administration, almost one third of American households have difficulty paying their utility bills [101]. About 20% of households reported inability to fully pay for food or medication after they pay their energy bills. This has been phrased as the “heat or eat” dilemma that includes ability to cook, boil water, and have lighting as well. Frank *et al.* tested this idea by evaluating the association between a family's enrollment in a low-income home energy assistance programme and the anthropometric status and health of their young children [102]. They found that in enrolled families there was less evidence of undernutrition, no evidence of increased overweight, and lowered odds of acute hospitalization from an emergency department visit compared with children in comparable households that were not enrolled. Simply put, reducing the need for an impoverished family to pay for heat leads to increased spending on food and the health benefits this entails. Associated research shows that as household energy insecurity (inability to adequately meet basic household energy needs) increases, infants and toddlers experience heightened odds of food insecurity as well as poorer health, increased hospitalizations, and various developmental costs [103].

Moreover, the poor often live in what have been called food deserts, a concept developed in Scotland in the early 1990s that refers to geographic areas in which access to food outlets that stock fresh, affordable, and healthy food options are few or nonexistent. Originators of the concept argue that without the ability to gain access to healthy foods, people are forced to buy less healthy food from available retailers who typically sell food of lower nutrient value at comparatively higher prices [45]. Beyond actual geographic availability, low-income shopper behaviour is also influenced by knowledge about food outlet locations, time availability, travel duration, and their economic accessibility and cultural appropriateness, including accommodation (how well shoppers are accepted) and acceptability (shopper attitudes about the characteristics of food outlets and their staffs) [104, 105]. Research on food deserts in Seattle-King County, Washington found that only 34% of low-income vulnerable populations could walk to a supermarket, and as few as 3% could walk to a low-cost supermarket [106]. A study of two urban and two rural communities in Minnesota with higher than state average poverty rates found available foods were costly, of fair or poor quality, and limited in both in number and type in stock. Focus groups and survey data show that study participants were concerned that locally available healthy foods were not affordable and believed that people in

their community suffered from food insecurity. The researchers conclude that the “absence of quality, affordable food for low-income residents in these four Minnesota communities prevents or diminishes their ability to choose foods that help maintain a healthy lifestyle” [107].

Third, and finally, there is the issue of structurally incurred debt. Of note is the often-hidden financial obligations associated with involvement with the criminal justice system. The United States incarcerates more people than any other country in the world and millions, especially the poor and people of colour people, are under correctional supervision in some form. Incarceration exacerbates social and ethnic inequalities as people who are or have been incarcerated and their families face serious financial challenges that impact food intake [108]. One form of debt in such families stems from the significant costs incurred as a direct result of criminal justice system involvement [109-111]. These costs, which have been dramatically rising in recent years and are now charged in most states, including both cash bail and legal financial obligations, including victim restitution, criminal fines and surcharges added to fines to fund a particular government function or a general fund, court fees, extradition costs, incarceration charges, and probation fees and costs of post-release supervision [112].

In some carceral settings, inmates are charged for talking to family members on the phone or for emails sent through a prison's email system. They also may be charged booking and release fees. Additionally, inmates may be expected to pay for health care and personal hygiene items like soap. In many facilities, basic necessities are sold by private vendors, often with high markups or added service fees. On-going post-release fees may be assessed to cover drug testing and counseling. According to Hadar Aviram, a professor at the University of California, Hastings College of the Law, “Public prisons are public only by name... These days, you pay for everything in prison” [113]. Many current and formerly incarcerated individuals and their families have been forced into what has been called a “debt-enforcement regime” [114]. Contrary to the belief that imprisonment for debt was long ago abolished, failure to pay criminal justice debt can lead to re-incarceration in several states. As a result, inmates leave behind prison bars only to be shackled to debt.

Criminal justice debt has become a taxing burden on low-income families that functions to trap people in poverty. Survey research in Alabama found 83% of respondents with criminal justice debts avoid payments for necessities like rent to pay the government for the money they find they owe [115]. As a result, “Incarceration increases the risk of hunger, food insecurity, and nutritional deficiencies for individuals, families, and communities” [116].

III Air Pollution and Other Environmental Toxins

Children from poorer families are more likely than non-poor children to be exposed to a varied array of environmental toxins, including air pollution, polychlorinated biphenyls (PCBs), and heavy metals like mercury and lead [117]. Moreover, notes Perera, poor children face a disproportionate burden of disease from the interaction of environmental pollution and climate change [118]. Emissions produced by the burning of fossil fuels consist of fine particulate matter (PM), black carbon, polycyclic aromatic hydrocarbons (PAHs), mercury, nitrogen dioxide

(NO₂), sulfur dioxide (SO₂), and carbon monoxide (CO). Each of these pollutants has been linked to various negative health impacts on children [119]. Kravitz-Wirtz *et al.* investigated the interaction between early-life exposures to air pollution (ambient NO₂ and PM) and neighbourhood poverty using data from the Environmental Protection Agency's Air Quality System [120]. They found that residents of poor neighbourhoods may be more vulnerable to the health burdens of pollution due to compromised health status and a lack of prenatal health care access. This research confirms a link between ethnic/income residential segregation and differential levels of pollutant exposure as well as to community stress and diminished access to community resources [121].

Ozone (O₃) is created by photochemical reactions that occur in chemical pollutants in the presence of sunlight and are accelerated at the higher temperatures being ushered in by climate change. Bell, Zanobetti and Dominici carried out a systematic review of epidemiological studies on mortality and hospital admission following short-term ozone exposure for the years 1988-2013 [122]. They found that these ozone-related health indicators were associated with unemployment and lower occupational status. PCBs, a group of 209 manmade neurotoxic chemicals that were used widely in electrical equipment like capacitors and transformers, are no longer produced in the United States but they persist in the environment. PCBs, as a result, continue to be found in food due to improper waste disposal and weather impacts on disposed chemicals. People living in urban impoverished areas face greater PCB exposure than people living in wealthier or rural areas [123]. Lead exposure during childhood is significantly associated with socio-economic status [124]. Not only are poor children more likely to be exposed to lead, but there is also a greater adverse association between living in a high-lead-risk census tract among poor children than among children from higher-income families [87]. In the body, lead alters the sending of electrical signals in the brain, causing changes in brain plasticity and synaptic communication [125].

The developing fetus and young child are particularly vulnerable to the many adverse effects of toxic air and other pollutants. Inhalation of air pollutants triggers the induction of oxidative stress and inflammation as the body tries to detoxify. Exposures early in life to "PM_{2.5}, PAHs, and O₃ are associated with a multiplicity of effects on the developing fetus and child, which can have long-term consequences for child health" [118]. Low-income communities and communities of colour have disproportionately high exposure to toxic air pollution produced by vehicles, manufacturing, coal-fired power, and hazardous waste dumps [126]. A National Institute of Environmental Health Sciences-funded review of existing literature found that most North American studies show that "areas where low-socio-economic-status (SES) communities dwell experience higher concentrations of ... air pollutants" [126]. PCBs have been linked to numerous health issues including neurodevelopmental disorders. Guo *et al.*, for example, found that prenatal exposure to PCBs was linked to low birth weight, delayed developmental milestones, and lower IQ [127]. In addition, environmental factors associated with poverty may magnify the effect of some toxins. Even low levels of lead are related to worse performance on cognitive tasks and reduced auditory recognition ability. Similarly, environmental tobacco smoke has greater effects on cognitive outcomes among children from lower SES backgrounds relative to the higher SES children.

While limited, various studies have found evidence tying air pollutants to neurodevelopmental disorders in children. Perera *et al.* report an association between prenatal exposure to PAHs and developmental delay, reduced IQ, symptoms of anxiety, depression, and inattention, while Peterson *et al.* found an association between PAH exposure and reduction in brain white matter surface in children from inner city families [128, 129]. The latter study, the authors submit, suggests that prenatal exposure to air pollutants contributes to slower brain processing speed, attention-deficit/hyperactivity disorder symptoms, and externalizing problems among urban youth. They link these problems to disruptions in the development of left hemisphere white matter.

Research has shown a relationship exists between roadway proximity and decreased cognitive function [130]. Based on animal studies, the major suspected source of this connection is very small ambient particulate matter that has an aerodynamic diameter of <100 nm [131]. Small particle size facilitates penetration and diffusion in the respiratory tract and subsequent transmission to the brain. This research is supported by child autopsy studies in highly polluted areas of Mexico City and by emerging epidemiological research [132, 133]. The latter is based on neuropsychological tests of several cognitive areas of the brain, including IQ, language development, executive function, and motor development. Findings indicate that reduced cognitive development emerged by three years of age in pollution-exposed children [134, 135].

This discussion raises issues about environmental inequality and environmental justice and the disproportionate health burdens from anthropogenic environmental toxins endured by low-income and (especially) communities of colour. According to the Environmental Protection Agency, environmental justice refers to "fair treatment and meaningful involvement of all people regardless of race, colour, national origin, or income with respect to the development, implementation, and enforcement of environmental laws, regulations, and policies" [136]. Environmental injustice, like the other forms of oppression discussed above, reflects the unequal distribution of power in society [137].

Are there Brain Syndemics?

As the discussion thus far indicates, available research affirms that children living in poverty face multiple structurally rooted challenges that can have impacts on brain structure. Specifically, poor children face a triple jeopardy health crisis involving stress, malnutrition, and environmental toxins. Cumulative risk assessment refers to the investigation of how multiple agents work together to produce disease [138]. The hallmark of syndemics is interaction. In this instance, is there adverse interplay in early childhood among stress, malnutrition, and environmental toxins promoted by perilous socio-economic factors?

I Interaction of Malnutrition and Stress

Hoeijmakers, Lucassen, and Korosi hypothesize that exposure to adversity early in life (namely stress, undernutrition, and infection) leads to life-long alterations in cognitive functions, at least in part due to changes in hippocampal neurogenesis [139]. Moreover, based on a review of the literature, they argue that these three elements do not act alone but rather there is a synergistic action among them that shapes cognition throughout life. This view is supported by Lindsay *et al.*, who

assert that the available evidence suggests that “the effects of maternal nutrition and stress are context dependent and may be moderated by one another” [140]. This interaction appears to be bidirectional. These conclusions are based on a review of available animal and human research. They found that despite variation in study design and methodology, two broad patterns of results emerge: i) diets that are higher in fat or that include increased intake and/or status of specific dietary fats and ii) increases in dietary intake or supplementation of targeted nutrients mitigate adverse outcomes. These patterns are seen in animal studies showing that early micronutrient supplementation protects against early stress-induced cognitive impairments [141]. While there are many unanswered questions in understanding the nutrition-stress connection and its role in brain development, there is reason to believe synergistic interaction is occurring.

II Interaction of Malnutrition and Toxins

Miller and Rayalam, observed, “The well-studied, deleterious health effects attributed to environmental pollutants and poor nutrition may act in combination to produce more severe adverse health outcomes than any one factor alone [142]. Deficiencies in specific nutrients render the body more susceptible to injury which may influence the pathways that serve as the mechanistic responses to ambient air pollutants.” Bharatraj and Yathapu, added, “Biototoxicity due to interaction between pollutants (heavy metals) and nutritional status may affect health of neonates, growing children, pregnant women and occupationally exposed population” [143]. One effect of malnutrition is that dietary deficiencies can lead to higher toxin absorption. For example, as noted, poor children are at higher risk of iron deficiency anemia. Low iron levels, in turn, contribute to an increase in the body’s absorption of lead, one of the most well-documented neurotoxins [125].

In a study of 60 children, Hegazy and colleagues found blood levels of iron and ferritin (a blood protein that contains iron) to be significantly lower in individuals with high blood lead level than in those with a low blood lead level [144]. Exposure occurred through the use of lead plumbing and consequent drinking water contamination, as occurred in the infamous Flint, Michigan case where as many as 12,000 children were exposed to drinking water with high levels of lead. Flint is a majority African American city where over 40 percent of residents are impoverished [145]. Secondly, there is some evidence that a poor-quality diet, one low in polyunsaturated fatty acids, antioxidants, fiber, and protein but high in fat is associated with reduced protection against the adverse effects of pollution possibly through inflammatory and oxidative stress pathways [146]. This connection is also indicated by research showing a reduction of some of the harmful effects of air pollution is achieved through increasing intake of essential micronutrients and long-chain polyunsaturated fatty acids [147, 148]. Thirdly, inadequate vitamin intake is associated with greater consequences of exposure to polluted air. Noting that pollution is a primary factor limiting the amount of solar UVB rays that reach Earth, Feizabad *et al.* compared more and less polluted parts of a city and found atmospheric pollution appears to play a significant role in the development of vitamin D deficiency [149]. This body of research provides support for the potential occurrence of syndemic interaction between malnutrition and exposure to environmental toxins that could impact neurologic development.

III Interaction of Stress and Toxins

There is growing interest in the role chronic psychological stress may be playing in enhancing children’s vulnerability to toxic exposures. Because stress is a nonspecific constellation of physiological effects, it is unlikely that there is a single pathway for stress influences on a child’s pollution susceptibility. However, there is evidence that stress may influence the same physiological pathways as certain chemical toxicants (e.g., pollutants, tobacco smoke). For example, air pollutants may have impacts through oxidative stress pathways similar to psychological stressors. Consequently, it is “plausible that the biologically compromised system(s) related to early life stress may be more vulnerable to subsequent environmental toxins and vice versa” [150]. Moreover, “those living in disadvantaged social circumstances may be most at risk for synergistic effects” [150].

In the first animal study to illustrate the role of stress in amplifying respiratory response to air pollution, Clougherty and colleagues found that the respiratory effects of exposure in rats were exacerbated by stress [151]. Subsequent epidemiological research affirms the connection between stress, pollutants and toxicants, and disease in humans [152, 153]. While thus far the focus of synergy research has been on respiratory and emotional effects, this research affirms that stress and pollutant exposure are interactive and adverse.

As a body, existing research supports the idea that there is a potential structural brain syndemic involving the continuous and harmful interaction in early childhood among stress, malnutrition, and environmental toxins that is promoted by socio-economic factors. Given the serious health and social consequences, this candidate syndemic merits closer examination, including identification of methods of mitigation.

Conclusion

The syndemics orientation has the potential to improve health and health-related policy by drawing attention to how socio-economic factors affect disease patterns and both physical and social development. Given brain plasticity, an emergent concern in syndemics research is the investigation of the social role interventions can play. In limiting harm by creating new social environments and experiences for children born into poverty. Of special interest are innovative structural interventions that directly address poverty rather than the conventional approaches which address child learning Goldin *et al.*, 2014), parent-child relations, and increased investments in the early childhood school education environment (although all of these approaches may be combined in comprehensive intervention) [154-157].

As noted, in their 2015 study, Noble and colleagues found that small increases in family income had a significantly greater impact on the brains of the poorest children than similar income increases among wealthier children [39]. This suggested to Noble that assisting families to move out of poverty might be an effective intervention to bring children closer to cognitive developmental norms. Consequently, Noble and co-workers developed a four-city study (New York City, New Orleans, the Twin Cities and Omaha) called Baby’s First Years of 1,000 low-income mothers randomized to receive either a larger (\$333) or

smaller (\$20) monthly income supplement during the first three years of their children's lives. Families are free to use the money as they wish. During the study period (which began in 2018), assessments (using a mobile EEG to measure brain wave patterns) and other methods allow researchers to estimate the impact of supplemental payments on children's cognitive, emotional and brain development, and on family functioning. Because of the COVID-19 pandemic, researchers were forced to extend the study to age 4 and will not collect the second set of brain data until 2022 [158].

In an Op-Ed in the Washington Post, Noble noted that her study would take at least five years to complete, which, she argued, is "far too long for young children living in poverty today [159]. We should not wait until then to push for policies that can help inoculate young children's pliable brains against the ravages of poverty." In 2021, President Biden's expanded child tax credit constitutes a structural intervention along the same lines as Noble's study. The credit raises the maximum annual benefit level for many families to \$3,600 (\$300/month) per child under age six beginning in July 2021. The change will lift millions of children above the poverty line [160]. It constitutes a natural experiment that could be studied for its effects on brain development among children. Beyond cognitive changes, such a study could investigate the impact on the syndemic factors (especially stress and nutrition, and, to the degree that it allows families to move, pollution exposure as well). Assessment of this sort is critically needed in light of the short- and long-term health and social costs of poverty.

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