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REVIEW
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The Role of Social Determinants of Health in Childhood Epilepsy

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ABSTRACT

Social determinants of health (SDHs) are significant and potentially modifiable drivers of neurologic diseases, including childhood epilepsy. Social determinants of health greatly influence the epidemiology, management, and outcomes associated with these conditions. Social determinants of health affect every aspect of a family's journey with epilepsy—from initial diagnosis to accessing effective treatments and ongoing care. Despite notable advancements in understanding the genetic and molecular underpinnings of pediatric epilepsies, there remains a relative lack of knowledge about the nature and impact of SDHs on these disorders. Epilepsy is a symptom of much more profound underlying determinants of health. Addressing the broader context of epilepsy can transform health outcomes. This narrative review appraises some available evidence and explores possible solutions.

Keywords: Childhood epilepsy, global health, healthcare access, social determinants of health, socioeconomic determinants of health

INTRODUCTION

Social determinants of health are "the conditions in which people are born, grow, live, work, and age," grouped into 5 domains: (1) economic stability, (2) education access and quality, (3) health care access and quality, (4) neighborhood and built environment, and (5) social and community context.[1] These domains affect health through various intermediate and proximate determinants in complex ways. It is necessary to characterize and understand each SDH's mechanisms, effect sizes, and interactions to address health inequalities. In addition, we must identify groups that are more vulnerable to specific SDH and devise new, scalable, and sustainable solutions.

The epidemiological landscape of epilepsy is evolving, and historically, we have often been slow to respond to contextual changes affecting medical conditions. For instance, hippocampal sclerosis—a condition once commonly associated with drug-resistant epilepsy—has become significantly less prevalent in high-income countries in recent decades.[2] This decrease may signal unanticipated shifts in the natural history of the syndrome, potentially influenced by SDH factors. Rising socioeconomic inequality and increasing air pollution, exacerbated by climate change, are poised to affect the epidemiology and outcomes of pediatric epilepsies significantly.[3]

EPIDEMIOLOGY

Social determinants of health considerably influence several established risk factors for childhood epilepsy, including hypoxic-ischemic encephalopathy (HIE), prematurity, febrile convulsions, neonatal seizures, hydrocephalus, head trauma, and certain infections associated with epilepsy (Table 1). Emerging evidence suggests that environmental pollutants, such as air and microplastics, can disrupt normal cortical development, potentially leading

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Table 1. Risk Factors of Childhood Epilepsy and SDH

Risk Factor	Role of Social Determinants of Health
HIE	• Increased risk with lower socioeconomic status (SES), lower education level, and reduced access and utilisation of prenatal and perinatal maternal healthcare. • Lower SES is associated with poorer maternal nutrition, maternal anemia and suboptimally treated maternal infections. • Rural location, lack of transport, language barriers reduce access to maternal healthcare.
Prematurity	• Increased risk with lower SES.
Febrile Convulsion	• Increased risk with lower SES, lower education level, food insecurity, childhood nutritional deficiencies (e.g. iron and zinc). • Lower SES is a risk factor for complex febrile convulsion and recurrent febrile convulsion.
Neonatal Seizure	• Increased risk with lower socioeconomic status, poor maternal care.
Hydrocephalus	• Increased risk of congenital hydrocephalus with lower SES, lack of prenatal vitamins, and poorer maternal care. • Lower SES is associated with delays in access to hydrocephalus management and higher rates of shunt failure outside hospital.
Head trauma	• Increased risk with lower SES. • Lower SES is associated with poorer outcomes after pediatric traumatic brain injury.
Childhood infections that affect CNS	• Bacterial meningitis risk increases with low SES, maternal education, smoking, reduced access to vaccines, poor household ventilation, and overcrowding. • Atypical bacterial meningitis presentations, e.g., otitis media-associated bacterial meningitis, are more common in lower SES. • Suboptimal access to antiretroviral therapy is associated with higher rates of new-onset seizure in children in HIV. • Suboptimal access to onchocerciasis elimination programs is associated with higher rates of onchocerci asis-associated epilepsy.
Malformations of Cortical Development (MCD)	• Increased risk with reduced access and utilisation of prenatal and perinatal maternal healthcare. • Increased risk with lack of prenatal vitamin. • Exposure to environmental toxins that potentially disrupt normal cortical development is more common in lower SES.

to cortical malformations like heterotopias and focal cortical dysplasia.[4] The roles of other SDHs in these risk factors remain underexplored. Epilepsy is a symptom of much more profound underlying determinants of health.[5] In the symptom-component cause model, various factors act alone or in combination with other component causes to produce epilepsy (Figure 1).

Economic Instability

There is a well-established association between poverty and higher epilepsy incidence and prevalence globally.[6-12] Approximately 80% of the 70 million people with epilepsy reside in low- and middle-income countries (LMICs). Within countries, individuals of lower socioeconomic status (SES)—measured by income, education, occupation, and other indicators—exhibit higher epilepsy rates. Meta-analysis shows that epilepsy is 1.38 times more prevalent in children of low SES.[8] Evidence suggests that lower SES is a risk factor for developing epilepsy rather than a consequence of it. Low parental income is associated with an increased risk of HIE,[13] prematurity,[14] febrile convulsion,[15,16] neonatal seizures,[17,18] hydrocephalus,[19] head trauma[20-22] and childhood infection.[23] Food insecurity is prevalent and an independent risk factor for poor maternal health and febrile convulsion.[24] Poverty is also linked to higher rates of harmful behavior, e.g., maternal smoking, and exposure to toxins, e.g., air pollution.[25] Surgical treatment access is delayed in poorer individuals with hydrocephalus,[26,27] with higher chances of shunt failure rates outside the hospital.[28] Recovery from pediatric Traumatic brain injury (TBI) is often more protracted or even poorer.[29]

Education

Parental education is closely related but distinct from parental income. Lower general education and health illiteracy are consistently associated with reduced healthcare utilization, poorer management of potentially treatable medical conditions, and increased rates of other risk factors of childhood epilepsy, e.g., hydrocephalus,[27] meningitis,[23] febrile convulsion.[15,30] For example, 1.4% of parents in an Australian study[31] and 3.5% in a Moroccan study correctly evaluated and managed their febrile children.[32] In another study, only 13% knew that febrile convulsions could be prevented by reducing the child's temperature.[33] In a Nigerian study, the use of harmful pre-hospital interventions (e.g., herbal preparations) is associated with lower SES.[34]

Health Access and Quality

Good maternal health and adequate healthcare access and quality are central to normal fetal cortical development. Antenatal care (ANC) visits are crucial for maintaining maternal health, preventing and detecting potential causes of obstetric complications, and averting newborn death, stillbirth, and long-term neurological disability. Maternal infections, which can be mitigated through proper ANC, are known risk factors for HIE and childhood epilepsy.[35] Women in the poorest quintiles and countries often receive substantially fewer visits and lower-quality care.[36] Their nutrition is inadequate. Maternal anemia is more common.[37] Exposure and consumption of ultra-processed food, associated with higher inflammation and oxidative stress, is higher.[38] Lack of prenatal vitamins, such as folic acid, increases the risk of congenital malformations

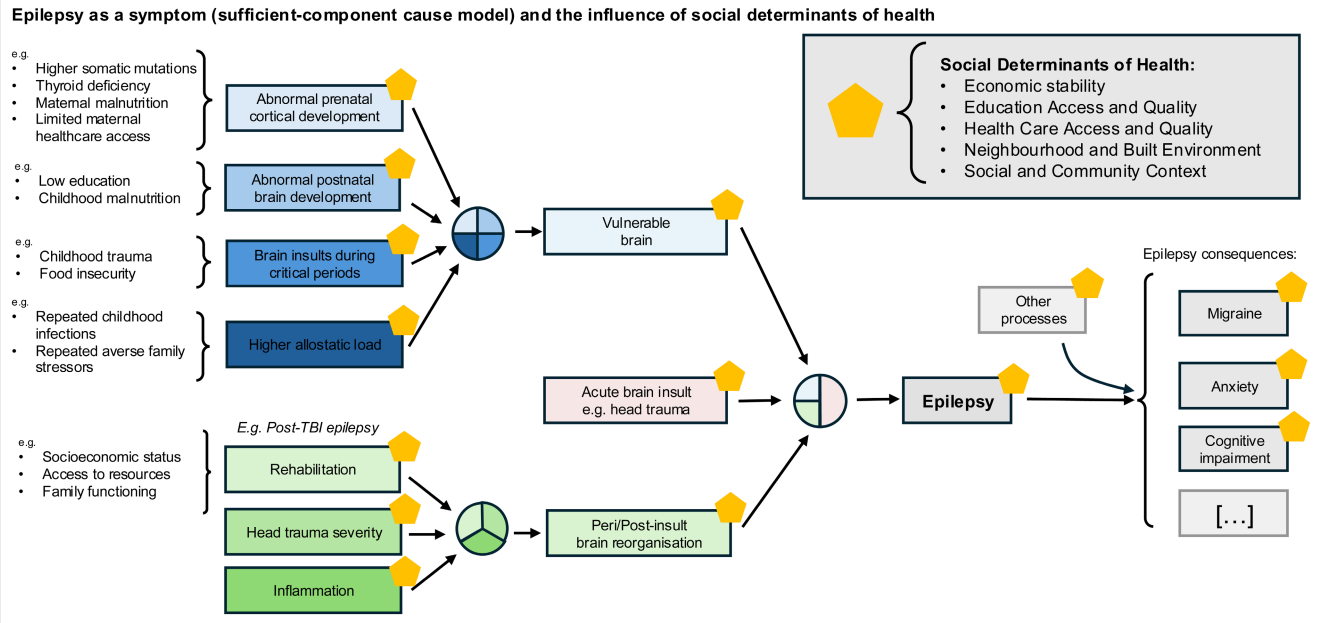

Figure 1. Epilepsy as a symptom (sufficient-component cause model) and the influence of social determinants of health. Epilepsy is usefully conceptualized as a symptom of 1 or more pathogenic processes.[5] Using the sufficient-component cause model, these processes represent component causes which alone or in combination with other component causes can form a sufficient cause to produce epilepsy. Epilepsy itself also has consequences such as migraine, anxiety, and cognitive impairment, which involve other processes in their generation. Social determinants of health can potentially influence the development of epilepsy at any or many of the component causes in the hierarchical causal tree. This figure illustrates 1 example causal pie where the necessary cause is an acute brain insult (red), e.g., head trauma. Other important component causes in the development of post-insult epilepsy are the baseline vulnerable brain state and peri-/post-insult brain organization processes. A vulnerable brain itself can be caused by multiple processes, including abnormal prenatal cortical development (that can lead to malformation of cortical developments), abnormal postnatal brain development, brain insults during critical developmental periods, and a state of high allostatic load. Social determinants of health (e.g., poverty, low education, limited maternal healthcare access, air pollution) are drivers of these component causes. Many factors influence peri/post-insult brain reorganization, including parental socioeconomic status, access to rehabilitation resources, and family functioning.

and hydrocephalus.[39] Suboptimal intrapartum management of diabetes is linked to neonatal seizures.[40] Access to skilled birth attendance and comprehensive emergency obstetric care is inequitable because of affordability, logistics, and linguistic constraints.

Globally, there is inequality of access to vaccines against meningitis, a preventable risk factor for childhood epilepsy. Health access inequality may also explain higher rates of more uncommon causes of bacterial meningitis, e.g., the higher rates of otitis media-associated bacterial meningitis in lower SES.[41] In some geographical regions, treatable risk factors for epilepsy are left untreated. For example, high rates of new-onset epilepsy in children with HIV in Zambia reflect the underutilization of ART (Antiretroviral therapy). In onchocerciasis-endemic areas, onchocerciasis-associated epilepsy (peak age of 8-11 years) is treatable. Ivermectin and vector control reduce the incidence of epilepsy. Despite this, elimination of transmission is only achieved in 9% of endemic areas in sub-Saharan Africa.[42]

Neighbourhood and Built Environment

Studies globally show that many poorer neighborhoods are exposed to higher pollutant levels. Ultrafine particles, microplastics (MP), associated heavy metals, and persistent organic pollutants (POPs) can cross the placental and immature blood-brain barriers.[43-46] Prenatal exposure to diesel exhaust disrupts normal reelin[47] and heat shock pathways, which are critical for neuronal migration.[48] Volatile organic compounds, polyaromatic hydrocarbons, particulate matter, and airborne MPs destabilize microtubule networks. Heavy metals (e.g., Co, Hg, Cd) and POPs directly impair neuronal migration.[49-54] Air pollutants elevate maternal and fetal inflammation. A Mexican study, particularly relevant to epilepsy, found higher titres of anti-GAD65 antibodies in children exposed to high PM2.5 levels in metropolitan cities.[55,56] Lower thyroxine levels in first-trimester pregnant women are associated with air pollution exposure.[57] Transient modest maternal thyroid deficiency may lead to brain heterotopias.[58] Numerous meta-analyses have linked air pollution with disorders of impaired placentation: pre-eclampsia, gestational hypertension, and intrauterine growth restriction.[59] Impaired placentation, the most common cause of chronic fetal hypoxia, can lead to polymicrogyria and heterotopia.[60,61]

Social and Community Context

Social barriers to optimal care drive considerable disparities in health outcomes in people with epilepsy. Of the SDH domains, this is the least assessed concerning risk factors of childhood epilepsy. Social support networks, social exclusion, and social isolation independently predict poor maternal mental health and prenatal healthcare utilization, which likely affects the child's cortical development.[62-64] Pre-clinical studies show the detrimental effects of maternal separation on HIE outcomes.[65] Social isolation in early life alters neurotransmission and neurocircuitry and the development of glial cells in specific brain regions.[63] Early life social isolation in humans and animal models increases rates of anxiety and depression, common comorbidities of epilepsy.[63]

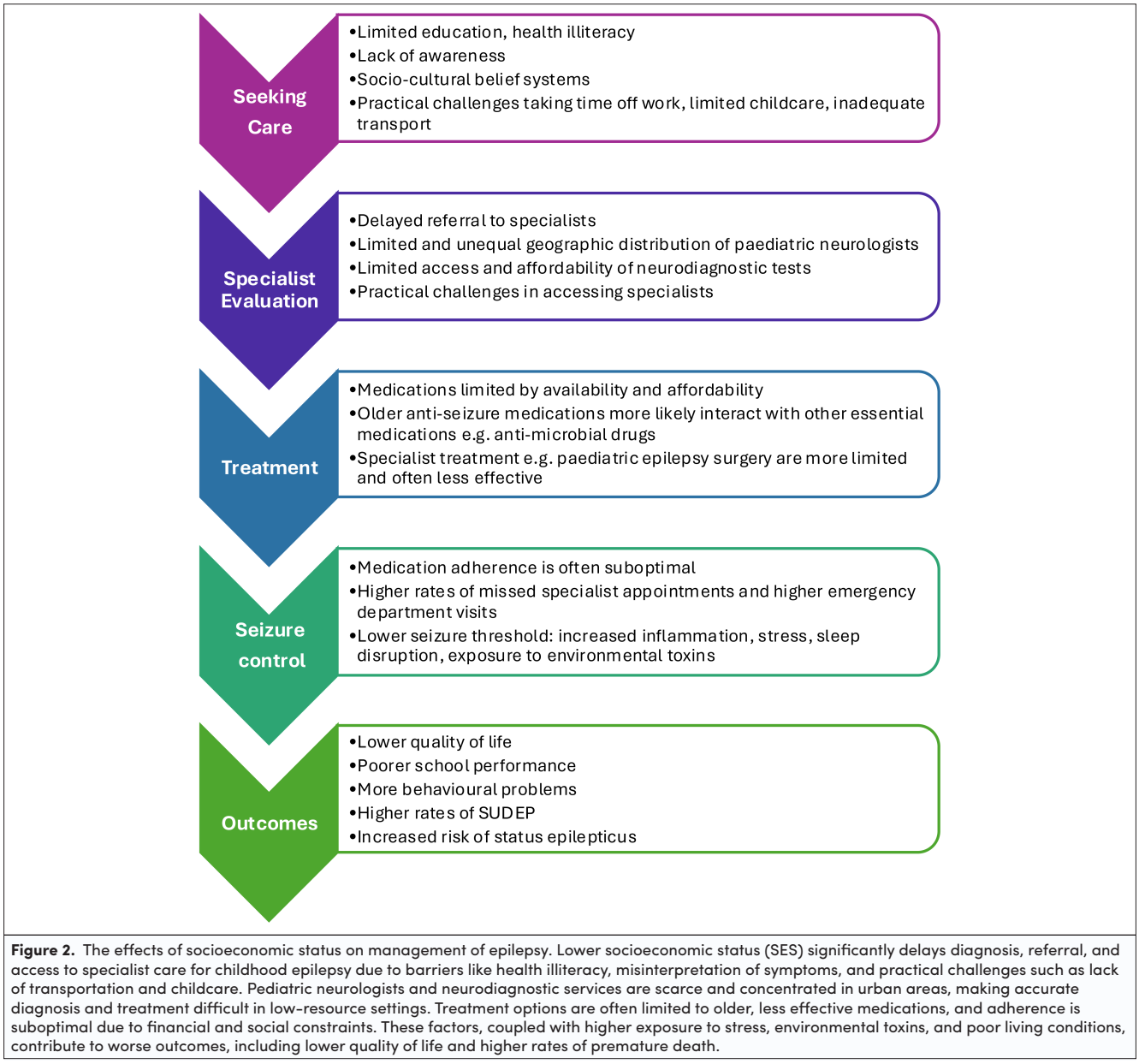

Figure 2. The effects of socioeconomic status on management of epilepsy. Lower socioeconomic status (SES) significantly delays diagnosis, referral, and access to specialist care for childhood epilepsy due to barriers like health illiteracy, misinterpretation of symptoms, and practical challenges such as lack of transportation and childcare. Pediatric neurologists and neurodiagnostic services are scarce and concentrated in urban areas, making accurate diagnosis and treatment difficult in low-resource settings. Treatment options are often limited to older, less effective medications, and adherence is suboptimal due to financial and social constraints. These factors, coupled with higher exposure to stress, environmental toxins, and poor living conditions, contribute to worse outcomes, including lower quality of life and higher rates of premature death.

MANAGEMENT

Diagnosis

Lower SES is closely linked to delayed diagnosis, referral, and attendance at specialist clinics for childhood epilepsy (Figure 2). Limited education, health illiteracy, and lack of awareness are the initial barriers to seeking appropriate care. Parents may misinterpret seizures as behavioral issues or dismiss symptoms, such as nocturnal seizures, altogether. Socio-cultural belief systems influence the interpretation of abnormal neurological events: local traditional health practitioners may be consulted first.[66,67] Practical challenges families face (e.g., difficulty taking time off work, limited childcare, and inadequate transportation) exacerbate the delay in accessing care.

The next barrier is the lack of and unequal access to pediatric neurologists and neurodiagnostic services. The median number of pediatric neurologists globally is just 0.07 per 100 000 population, distributed unequally.[68] In Africa, the median is as low as 0.01 per 100 000 population. Within countries, neurologists are concentrated in urban centers. General practitioners, pediatricians, psychiatrists, and adult neurologists manage most cases despite limited or no specific training. Referrals to specialists are suboptimal: a study found that none of the referral letters for people with previous seizures included any comments on seizure history, reflecting significant gaps in primary seizure care.[69] Suboptimal clinical characterization and higher rates of misdiagnoses of epilepsy mimics are exacerbated in low-resource settings.[70]

Neurodiagnostic tests are often inaccessible and unaffordable. A drop in World Bank income classification correlates with a 29% decrease in the proportion of the population able to afford neurodiagnostic tests.[71] EEGs (Electroencephalograms)

and MRIs are markedly less available in low-resource settings. Computed tomography scanners are often more accessible and frequently used for initial seizure evaluation, but their cost is prohibitive for many[71] Even when available, lower SES correlates with increased missed neurodiagnostic appointments. Genetic and advanced metabolic testing are confined mainly to academic centers in high-income countries, and expertise in accurately interpreting test results is in short supply.

Treatment

Ideally, the selection of ASM (Antiseizure medication) is guided by efficacy, safety, tolerability, and the specific type or syndrome of epilepsy. In practice, choices are often limited by availability,[72,73] accessibility, and affordability, although a lack of awareness about the condition also plays an important role (Figure 2). Many families cannot afford newer or more effective treatments, and a significant proportion—up to a quarter—receive no ASM at all. While at least older ASMs are available in most countries, access varies significantly by income level.[73] In many LMICs, phenobarbital and carbamazepine remain the only widely accessible treatment options. Newer ASMs, which may offer better tolerability profiles and fewer drug interactions, are largely unavailable in these settings. Older ASMs are often not enzyme-neutral and can significantly interact with other essential medications, including antimicrobial drugs. Levetiracetam, a usually well-tolerated newer ASM, has recently been included in the World Health Organization (WHO) essential medicine list, but its uptake in individual countries remains to be seen.

Medication adherence is often suboptimal and consistently associated with SES and education level.[74-76] SES level is not correlated with knowledge of disease but with adherence.[77] Surveys have shown that parents are ill-aware of the nature, treatment objectives, duration of treatment, side effects, and the importance of adherence to drugs. Lack of transport,[78] language barriers.[75,79] Other social contexts (e.g., family conflict level) further contribute to this.

More specialized treatments are generally unavailable and often less effective in low-resource settings. Individuals with the lowest SES improve the least with a ketogenic diet.[80] Access to pediatric epilepsy surgery is delayed, lowering the odds of improvement.[81,82] Inadequate access, rural location, language barriers, lack of transportation, and the inability of parents to take time off work have been cited as reasons.[83] There is also unequal use of brain stimulation treatment based on insurance status.[84]

Outcomes

Long-term management requires good clinical monitoring to achieve the best outcome (Figure 2). Adherence to specialist clinic appointments strongly depends on parental income and education level.[85,86] Higher rates of missed appointments are associated with higher mergency department visits in children.

Many SDHs may influence the seizure threshold. Children living in poverty experience higher stress, contributed by food insecurity, higher parental and child anxiety, household chaos, and social adversity.[87-91] Inflammation levels are higher in families with lower SES, linked to factors like poor housing conditions, fewer green spaces, and stressful childhood events.[92-94] Sleep health disparities also correlate with low SES, driven by poor neighborhood air quality, lack of green spaces, and food insecurity.[95-98] Additionally, low-income households face greater exposure to environmental toxins, including endocrine disruptors from pesticide use.

Seizure control alone does not guarantee a good outcome. Many studies suggest that lower SES is associated with a lower quality of life.[99] Diagnostic delay reduces IQ in young children.[100] A poorer developmental outcome is seen in infants with infantile spasms, with more behavioural problems.[101] Poorer school performance in children with epilepsy is more related to family variables rather than seizure control. Benign childhood epilepsy is not so benign in some cases, as atypical long-term neuropsychological outcomes are more common in low SES.[102]

People with epilepsy in the lowest SES group die prematurely by an average of 17 years. Sudden Unexpected Death in Epilepsy rates are almost 3 times higher in the lowest SES quartile than in the highest SES quartile.[103] Status epilepticus risk increases with each point increase in the Index of Multiple Deprivation in a UK population-based study of childhood status epilepticus.[104]

ADDRESSING SDH IN EPILEPSY

Addressing SDH is critical to reducing health inequality and improving outcomes across the social gradient in epilepsy. Regional and national-level policy solutions, backed by sustained political will and funding, are crucial, challenging, and beyond the scope of this article. Local health professionals and the neuroscience community can offer differentiated, more targeted, and equally impactful solutions.

As a community, we need a far greater understanding of the role of SDH in epilepsy. We need a far greater breadth of investigations beyond the few academic centers or seemingly random geographical areas. We need far greater depth and specificity. The types and effects of SDH are highly heterogeneous, often individual, time and culture-specific. Poverty is usually treated as a single undifferentiated construct. Equally, epilepsy is a symptom complex comprising various disorders, precipitants, and outcomes—studies of SDH in epilepsy rarely differentiate them. Advancing the field requires studies to move beyond just showing that poverty or low education is associated with various facets of epilepsy. This is already clear. Proximate, more quantitative, and mechanistic level understanding is lacking. For example, it would be helpful to know which specific air pollutants, at which specific concentration threshold, can increase the risk of cortical malformations. We need international research standards and frameworks to allow data to be pooled, analyzed and interpreted meaningfully. In the broader research environment where funding and bandwidth are limited, we must urgently prioritize this area of research.

Local-level solutions are possible. Multiple seemingly small local changes are cumulatively powerful. Practical logistics issues permeate all aspects of epilepsy care. Families vulnerable to logistical failure should be identified, e.g., a lack of transport and work flexibility to come to an appointment at a specific time or place. They should be linked with transport services and offered more flexible appointment options (out-of-hours clinics, drop-in clinics, community satellite clinics, and tele-clinics). Transport funding for the most vulnerable

may provide a high return on investment (less wasted clinician time, better hospital flow, fewer ED visits, and improved health outcomes). Wider use of tele-neurology technology should be proactively offered. Given the widespread adoption and affordability of mobile phones and internet coverage, tele-neurology should be mobile first. More investment is needed to improve technical, user interface, and user experience issues. Better language translation and more personalized communication targeted at the individual's literacy and familiarity levels may soon be within reach. Improvements in distribution, accuracy, and affordability of large language models (LLMs)-based artificial intelligence solutions may address current limitations in access to language interpreters and the quality of clinical communication materials, e.g., questionnaires and leaflets.[105]

Lastly, every interaction of the local health system with an individual should be seen as a rare opportunity to start exploring and addressing the individual's and family's broader social context. Even simple interventions, e.g., a directory of local social needs and specific services, are valuable. Clinical bandwidth is often concentrated on the disease and the 'presenting complaint'. These are usually symptoms of much more profound underlying determinants of health. Spending extra minutes understanding the wider context can transform lives and improve health outcomes.

Availability of Data and Materials: This is a review article, and no new data were generated or analyzed during this study. All data supporting the findings are included in the manuscript, which is based on publicly available literature and sources cited within the review.

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APPENDIX

***SdHIELD** (**S**ocial **d**eterminants of **H**ealth **I**n **E**pi**L**eptic **D**isorders) Research Group**

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