The impact of poverty and socioeconomic status on brain, behaviour, and development: a unified framework

Abstract: In this article, we, for the first time, provide a comprehensive overview and unified framework of the impact of poverty and low socioeconomic status (SES) on the brain and behaviour. While there are many studies on the impact of low SES on the brain (including cortex, hippocampus, amygdala, and even neurotransmitters) and behaviours (including educational attainment, language development, development of psychopathological disorders), prior studies did not integrate behavioural, educational, and neural findings in one framework. Here, we argue that the impact of poverty and low SES on the brain and behaviour are interrelated. Specifically, based on prior studies, due to a lack of resources, poverty and low SES are associated with poor nutrition, high levels of stress in caregivers and their children, and exposure to socio-environmental hazards. These psychological and physical injuries impact the normal development of several brain areas and neurotransmitters. Impaired functioning of the amygdala can lead to the development of psychopathological disorders, while impaired hippocampus and cortex functions are associated with a delay in learning and language development as well as poor academic performance. This in turn perpetuates poverty in children, leading to a vicious cycle of poverty and psychological/physical impairments. In addition to providing economic aid to economically disadvantaged families, interventions should aim to tackle neural abnormalities caused by poverty and low SES in early childhood. Importantly, acknowledging brain abnormalities due to poverty in early childhood can help increase economic equity. In the current study, we provide a comprehensive list of future studies to help understand the impact of poverty on the brain.

Keywords: amygdala; hippocampus; cortex; prolonged stress; low- and middle-income countries (LMICs); generational poverty

1 Introduction

Poverty is defined as a state of deprivation, characterised by a lack of basic needs and resources (Dufford et al. 2019, 2020). Synonymous with poverty, socioeconomic status (SES) refers to the social standing of a family or individual, which is a combination of income, educational level, and occupational attainment. Along these lines, SES is also measured as an individual’s or family’s economic and social position in relation to others, based on their income, educational level, and occupation (Gibbings et al. 2009). SES also includes factors that are influenced by the economic and social resources available to a person and can influence the neighbourhood (e.g., postcode) as well as the schools and universities they attend. According to Palacios-Barrios and Hanson (2019), poverty is multifaceted, identified by economic, social, and psychological challenges. As noted by Palacios-Barrios and Hanson (2019), poverty is typically operationalised in research through financial, educational, as well as occupational metrics. It is important to note that low SES and poverty impact both developing and developed countries. For example, approximately 20% of people in the US and UK live in poverty (Cox et al. 2018). The rate is much higher in developing countries (Alvaredo and Gasparini 2015).
SES of an individual can affect the quality and availability of various resources and services such as access to physical and mental health care facilities, and educational and occupational opportunities (Letourneau et al. 2013; Newacheck et al. 2003; Villatoro et al. 2018). While SES has been implicated in many psychosocial contexts (education, language development, access to healthcare facilities), the impact of SES on physiological and neural development is less understood (Noble et al. 2012b). However, neural evidence has identified that individuals (specifically children and adolescents) from lower SES backgrounds often display atypical differences in neural processing and cognitive performance (Hackman et al. 2010; Noble et al. 2015). This suggests that the effect of SES is not just confined to one’s social status, and access to quality health care and educational opportunities but also has a significant impact on brain functioning, cognitive aptitude, and educational outcomes (Rakesh and Whittle 2021).

The primary objective of this paper is to establish a comprehensive framework that elucidates the intricate connections between child poverty, delayed neural development, impaired language and educational achievement, and adverse mental health outcomes. This framework seeks to highlight the pivotal role of early childhood brain changes in comprehending the multifaceted consequences associated with poverty.

### 1.1 A unified framework of the impact of poverty on brain and behaviour

In this section, we provide a unified framework on how poverty and low SES may impact brain and behaviour. Based on an extensive review of the literature, we developed a framework that explains how poverty and low SES impact the brain and behaviour (see Figure 1, for details).

As can be seen in Figure 1, children live in poverty may experience severe stress, are exposed to poor diet, and/or socio-environmental hazards (McCurdy et al. 2010). These factors negatively impact brain development in early years of life. These semi-permanent brain changes, in turn, impact a number of important developmental outcomes, such as language development, educational attainment, and put people at risk of developing psychological and physical disorders. Neural damage in early stages of development can also lead to the development of several psychological disorders (including depression and substance use disorders) as well as old-age disorders (including mild cognitive impairment and Alzheimer’s disease).

![Figure 1: A framework of poverty-related factors and future consequences, such as delay language development, poor educational attainment, and neural abnormalities. An integrative framework of the links between brain and behavioural abnormalities due to poverty.](image-url)
It is important to note that the poverty-related outcomes section of Figure 1 feeds back into poverty, in a circular fashion. In other words, children living in poverty are at a disadvantage in terms of educational, occupational, and health outcomes when they are adults (Graham and Power 2004). In addition, poor inadequate educational, occupational, and health factors in adults lead to poverty of their children, thus creating generational poverty (Gal 2014).

Generational poverty is defined as parents growing up in poor environmental impacts educational attainments of their children, which in turn, make them also poor in the future (Beegle 2003; Gal 2014). For example, several studies have shown that poor diet (including breastfeeding) in children is related their parents’ educational level (Kollataj et al. 2011; Nelson 2000). This is possibly the case due to poor education is usually associated with low income, making it difficult to, for example, obtain healthy food products, in urban regions. Along these lines, there is a plethora of studies showing that psychopathological disorders and low educational attainment in parents is associated with poor diet and education of their children (Alderman and Headey 2017; Kunwar and Pillai 2002; van Ansem et al. 2014). For example, research has also shown that eating disorders in parents were found to impact normal healthy development of children (Watson et al. 2018). Further, depression in parents was found to impact normal healthy development of children (Fletcher et al. 2011; Fredriksen et al. 2019; Orvaschel et al. 1980). This in turn does perpetuate poverty in children.

Below, we unpack Figure 1 in order by discussing each segment of the figure (from left to right), respectively.

2 Poverty-related factors: stress, poor diet, and socio-environmental hazards

Based on literature review, the main elemental impacts of poverty are an increase in stress levels, poor diet, and an increase in exposure to socio-environmental hazards (see left side of Figure 1). Below, we discuss each in order.

2.1 Stress

Poverty is associated with an increase in stress levels (Schmidt et al. 2021). Several studies have shown that poverty impacts stress levels of parents thus impacting caring duties of their children (Blair and Raver 2012). Importantly, the impact of parental stress on children can start as early as in pregnancy. For example, stress during pregnancy was found to impact brain development of the foetus (Gross et al. 2016; Lefmann and Combs-Orme 2014). In addition, stress in childhood was found to negatively impact physical and psychological development in adulthood. For example, according to Johnson et al. (2016), younger adolescents exposed to high cumulative life stress during childhood have been shown to demonstrate poorer executive functioning, which has been found to result in smaller prefrontal volumes. Further, cumulative and prolonged stress negatively affects neurogenesis in the hippocampus (Lajud and Torner 2015), which in turn impacts learning abilities.

Studies conducted with low SES children in rural areas, who experienced material deprivation and stress (poor housing, family instability) showed higher basal cortisol, which is indicative of higher stress levels (Liberzon et al. 2015). However, positive parenting (as in high SES families) has been associated with lower cortisol levels in children (Liberzon et al. 2015). In the same study, it was found that cortisol levels mediate the relationship between low SES and poor executive functioning, suggesting that SES may mediate executive functioning and prefrontal cortex development. Several studies show that stress in children can impact several processes including self-regulation (Evans and English 2002). Poor self-regulation is related to the development of several psychopathological disorders, as we discuss below.

As related to stress, childhood trauma has also been reported in children from low socioeconomic status (Assari 2020a; Jahanshahi et al. 2022). Childhood trauma involves emotional, physical, and sexual abuse as well as neglect. While child neglect is strongly associated with socioeconomic status, there are several studies also reporting that emotional, physical, and sexual abuse are more common in families with low socioeconomic status (Lacey et al. 2022; Maguire-Jack et al. 2021). This perhaps can explain the prevalence of psychopathological disorders in families in low socioeconomic status, as childhood trauma in poor children could cause these disorders (see discussion below on this topic).

2.2 Poor diet

Poverty and low SES are associated with poorer diets and, in severe cases, malnutrition (Cuevas-Nasu et al. 2019; Van de Poel et al. 2007; Vilar-Compte et al. 2021; Wei et al. 2018). For example, due to their high cost, healthy foods are often less accessible to individuals from disadvantaged economic backgrounds (Ketttings et al. 2009). Such dietary limitations can negatively affect mental health, leading to depression,
anxiety, and cognitive impairments (Firth et al. 2020; Sparling et al. 2021). Research has recently proposed an agenda that identifies the complex association between food security, access to nutritious food, and mental health (Sparling et al. 2021). This relationship suggests that health and access to food are influenced by SES, and individuals living in poverty often experience the most significant disparities. Despite these findings, Sparling et al. (2021) emphasize that many initiatives aimed at enhancing food security, nutrition, and overall health frequently focus on physiological outcomes, often neglecting the potential mental health implications of poor nutrition.

Nonetheless, there is a growing body of research that acknowledges the relationship between mental health, food security, and nutrition (Pourmotabbed et al. 2020; Tevie and Shaya 2018). For instance, groups such as low-income individuals, the elderly, pregnant and post-natal women, children, and adolescents face challenges accessing affordable, healthy diets, putting them at greater risk of mental illnesses (Emerson et al. 2017; Herbison et al. 2012; Jacka et al. 2011; O’ Neil et al. 2014; Oellingrath et al. 2014). Considering these insights that link poverty and low SES to poor diets, malnutrition, and adverse mental health outcomes, we will examine how poor nutrition affects brain structures, educational achievements, and psychopathology (i.e., mental health).

Poor diet and lack to access to health nutrition can have significant impacts on brain development throughout the lifespan. For example, there is evidence that poor nutrition or malnutrition in mothers can significantly affect prenatal brain development (Fitzgerald et al. 2020; Ho et al. 2014; Oellingrath et al. 2014). Considering these insights, Sparling et al. (2021) found that link poverty and low SES to poor diets, malnutrition, and adverse mental health outcomes, we will examine how poor nutrition affects brain structures, educational achievements, and psychopathology (i.e., mental health).

Poverty is associated with an increase exposure to lead and nitrogen dioxide (Luby et al. 2022), which was found to impact brain development and lead to the development of several brain disorders (Sanders et al. 2009; Song et al. 2023). Several studies show that exposure to air pollution (which is not uncommon in poor regions and countries) is associated with damage to several cortical areas (de Prado Bert et al. 2021). The impact of air pollution was found to impact young children under 1 year old (Pérez-Crespo et al. 2022).

In a study conducted in Mexico City, known for a high index of air pollution, researchers discovered a concerning link between air pollution and prefrontal lesions in both children and dogs, highlighting the widespread impact of environmental factors on brain health. In addition, it has been found that exposure to air pollution can lead to the development of neurological disorders including Parkinson’s and Alzheimer’s disease (Jeremy 2017). In addition to air pollution, noise pollution was found to impact brain function (Jeremy 2017). Importantly, several studies found that noise pollution is more common in areas with low SES (Dale et al. 2015). Noise pollution was found to impact the frontoparietal network (Nußbaum et al. 2020) and cognition (Thompson et al. 2022). Noise pollution was also found to be associated with the development of Alzheimer’s disease (Crous-Bou et al. 2020). Besides the well-documented issues of air and noise pollution, empirical evidence has shown that limited access to healthcare services plays a significant role in influencing child development (Coye and Edmunds 1998; Moore et al. 2015), and this relationship is reciprocal. Specifically, children facing vulnerabilities, particularly those
from socioeconomically disadvantaged backgrounds, exhibit reduced utilization of healthcare resources (Bull et al. 2022). As will be elaborated upon in subsequent sections, it is worth noting that weak health has been identified as a hindrance to achieving optimal educational outcomes.

There are few studies on the impact of poverty (mostly socio-environmental hazards) and SES on neurotransmitters. According to Johnson et al. (2016), children raised in poor families are likely to live in areas in which they are likely to be exposed to environmental toxins and at a greater risk of iron deficiencies. Low iron increases the body’s ability to absorb harmful neurotoxins such as lead. This in turn alters the transmission of glutamate and dopamine, resulting in changes of neuroplasticity and synaptic communication in low socioeconomic status individuals. In animal studies, it has been found that impoverished environments can lead to a reduction in dopamine levels (Calakos et al. 2022; Green et al. 2010). Other studies have investigated reward processing in animals and humans, which is associated with dopamine function (Erber et al. 1980; Narisada 2017; Spence 1972). A recent study has also reported low socioeconomic status is associated with reduced reward sensitivity (Perera-WA et al. 2022). Similar findings were also reported by Decker et al. (2024). Along these lines, one study found that D2 receptors in the striatum are associated with socioeconomic status (Wiers et al. 2016). Another study has reported that socioeconomic status and social rank are related to ventral striatum activity, and possibly reward processing (Ly et al. 2011). As related to socioeconomic status, one study found that social rank is associated with dopamine availability, such that dopamine D2 availability is higher in dominant female than in male monkeys (Nader et al. 2012); the authors found the opposite results with dopamine transporter (DAT) availability, as it decreased in dominant female monkeys. Similar results were found by the same authors regarding kappa opioid receptors in humans (Johnson et al. 2023), which is also associated with reward processing. Furthermore, several studies have reported a relationship between dopamine D4 receptors and socioeconomic status (Guo and Tillman 2009; Nobile et al. 2007; Sweitzer et al. 2013), yet it is not clear on what the causes or implications of these relationship are. Unlike prior results, some studies have shown that individuals with a low income-to-poverty ratio pay more attention to reward information (White et al. 2022).

Some other socio-environmental hazards that are associated with poverty include population density and lack of green spaces in poor neighbourhoods. Several government reports and research studies have shown that there are higher population density in lower and middle income countries than in developed countries (Jedwab et al. 2021). The same also applies to developed countries, as regions with lower socioeconomic status have a larger population density than regions with higher socioeconomic status. Other studies have shown that population density is negatively correlated with mental health (Evans et al. 2001; Hong et al. 2014). As similar and related to population density, there are fewer green spaces in regions with lower, rather than high, socio-economic status (Dai 2011; Hoffmann et al. 2017; Schüle et al. 2017). Studies have also shown that the shortage of green spaces is related to health problems, including mental health (Collins et al. 2020; Nutsford et al. 2013; Vanaken and Danckaerts 2018).

2.3.1 The impact of poverty and low SES on the brain in general

In this section, we discuss the middle segment of Figure 1, which is focused on the impact of poverty, and its related effects (e.g., high stress, poor diet, and exposure to socio-environmental hazards) on the brain. There have been many studies on the impact of poverty on brain function (Lipina and Posner 2012; Pitts-Taylor 2019). SES can have a significant influence on brain functioning throughout an individual’s lifespan. The socioeconomic environment during early development can have a profound and long-lasting impact on brain development and subsequent education performance well into adulthood (McLean et al. 2012). The impact of poverty can be detected even at children under 9 months old (Tomalski et al. 2013). For example, using foetal MRI methods, it has been found that 1-month infants whose families have low socioeconomic have a smaller volume of grey matter (Betancourt et al. 2016; Hanson et al. 2013).

Children raised in impoverished conditions often display changes in brain functioning, especially in regions such as the hippocampus and amygdala, leading to a number of negative implications, such as cognitive and emotional disturbances, as well as emotional dysregulation (Almquist et al. 2010; Dike 2017; Gard et al. 2020; Kiernan and Mensah 2009; Kolb and Gibb 2016; Luby et al. 2013; Najman et al. 2009). The influence of poverty on the functionality of the hippocampus and amygdala may be attributed to the heightened levels of stress, thereby leading to an elevation in cortisol production, which has been previously identified as a factor with detrimental effects on these specific regions of the brain (Fowler et al. 2021; Huang et al. 2009; Lebedeva et al. 2018; Resmini et al. 2016). Notably, lower SES is associated with a diminished cognitive performance and structural changes in the brain, such as reduced cortical surface area and grey matter volume and a reduction in amygdala and hippocampal volume (Farah et al. 2006; Noble et al. 2012a). In addition, some studies found that poverty and low
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SES impact the connectivity between the amygdala and prefrontal cortex (Tian et al. 2021).

### 2.4 Obesity, poverty, and brain function

As mentioned above, obesity disproportionately impacts poor individuals. Given the link between SES and dietary habits, it is imperative to consider the potential impact of these diets on brain development and function. Building on the socioeconomic factors influencing obesity, the neural implications of this condition further underscore its complexity. For instance, when obese individuals encounter food cues, such as visual or olfactory stimuli, they often display abnormal activity in brain regions linked to memory, emotion, reward, motivation, and executive functioning (Li et al. 2021). Notably, areas such as the prefrontal cortex, anterior cingulate cortex, caudate, putamen, thalamus, amygdala, and hippocampus show abnormalities in obese individuals when presented with food cues (Dimitropoulos et al. 2012; Li et al. 2021; Moreno-Padilla et al. 2018; Stoeckel et al. 2008). Specifically, compared to weight control groups (i.e., individuals with healthy BMI ranges), obese individuals demonstrate an increase in the activation of the hippocampus and amygdala both when exposed to high-calorie food cues and when not exposed to food cues (Li et al. 2021). Further, a meta-analysis of 14 fMRI studies showed that when obese/overweight individuals are exposed to high-calorie foods, they typically exhibit stronger activation in their amygdala, hypothalamus, orbital frontal gyrus, and caudate — areas associated with reward and motivation. These findings suggest that obese individuals have a stronger propensity to crave high-calorie foods. This might be due to abnormal activity in the hippocampus and amygdala, which play pivotal roles in food consumption and weight regulation through established learning, memory, and stimulus-response pathways.

Studies have confirmed that high-calorie diets common in obese populations, correlate with increased activation in the hippocampus and amygdala (Yang et al. 2021). However, it is not clear if obesity preceded and caused the dysfunction in the hippocampus and amygdala, or if abnormal activity in these regions caused individuals to crave high-calorie diets leading to obesity, or if there is a reciprocal relationship between these brain regions, diet, and obesity. Li et al. (2021) provided some insight by revealing that the relationship between obesity (i.e., BMI) and activation of the amygdala and hippocampus was fully mediated by participants' baseline hippocampal and amygdala activity. This suggests that individuals with higher baseline activation in these regions are more prone to opt for high-calorie foods.

Additionally, their study indicated that obese participants exhibited higher baseline activity in the pre-frontal cortex, posterior cingulate cortex, anterior cingulate cortex, bilateral insula, inferior frontal gyrus, and supplementary motor area. However, these regions did not display any differences compared to healthy controls when exposed to food cues. These findings show that the hippocampus, and to a lesser extent, the amygdala, have a significant influence of in dietary choices. However, while there is evidence pointing to the sensitivity of the hippocampus to dietary changes, fewer studies have focussed on the potential mental health effects that could arise due increased hippocampal activation, and smaller hippocampal volume because of poor dietary choices and poor nutrition. There is strong evidence that obesity can contribute to abnormal brain activity, functionality, and volume, particularly in the hippocampus, amygdala, and prefrontal cortex. Given that obesity disproportionally affects individual from lower SES due to the cost of health eating and proximity to cheaper unhealthy foods there is an increased risk that individuals from lower SES could experience detrimental effects in their brain development, brain functionality, and brain volume due to their diets.

### 2.5 Differential impacts of poverty and socioeconomic status on brain-based outcomes

Extending our discussion of the impacts of poverty on brain-based outcomes more broadly, it is important to note that poverty does not impact brain-based functioning and behaviour in an identical manner across populations (Johnson et al. 2016). That is, the effects of poverty exposure can result in a multitude of differential impacts. The concept of differential impacts in this context illustrates the multifaceted manner in which groups or populations of people exposed to poverty may be impacted (Boyle 2016). Evidently, we observe this to be the case through the varying degree of how groups or population are exposed to poverty. For example, the time in life at which an individual encounters poverty, the severity of the impact, as well as the types of poverty encountered can vary drastically. Namely, in limited access to educational resources, opportunities for employment, nutritional deficiencies, and exposure to stressful family and environmental conditions.

These factors can also be encountered collectively or individually. For example, an individual, group, or population may experience all or some of these conditions. These factors can also be encountered in isolation. In other words, an individual, group, or population may reside in a low-socio
economic location, and as a result is limited in access to quality of education, though may not be exposed to stress at home (Yu et al. 2020). The varying degree of exposure to poverty can impose minimal to intense developmental impacts, relating to plasticity in the early stages of development (Lipina and Posner 2012). This means that the brain is highly sensitive to external stimuli, both positive and negative. Butterworth et al. (2011) note that chronic stress associated with living in poverty can alter the brain’s structure and function, affecting areas critical for memory, executive function, and emotional regulation. This impairment can lead to challenges in academic environments, where memory plays a key role in understanding and retaining new information (Dike 2017).

Impulsivity and inhibitory control are also markedly affected by the conditions of poverty. The prefrontal cortex, primarily responsible for executive functions such as planning, decision-making, and impulse control, alters functional connectivity in response to environmental stimuli (Dégéilh et al. 2020). In poverty-stricken environments, where immediate survival often takes precedence over long-term planning, the development of this brain region can be compromised. The consequence of this impact can foster a perpetuating experience of poverty (Mani et al. 2013). Substance use and its relationship with poverty further illustrate the complex interplay between environmental stressors and brain development. Individuals living in poverty are found to be at a higher risk of substance abuse, partly due to the neurobiological changes induced by chronic stress and the lack of access to mental health services (Manhica et al. 2021). Substance use, in turn, can exacerbate the negative impact on cognitive functions (Dagher 2015; Wahler 2012), and can result in at-risk behaviour and criminality (Dokkedal-Silva et al. 2021; Manhica et al. 2021) creating a vicious cycle that impedes the ability to break free from poverty. Conveying the intricate interactions and outcomes in an accessible manner is crucial for developing targeted interventions, as it highlights the need to consider the diverse needs and circumstances of those affected by poverty. Such as, in the development of policy, or developing programs aimed at mitigating the effects of poverty.

Several studies investigating the links between poverty and brain changes were conducted using the Adolescent Brain Cognitive Development (ABCD) Study (Kim et al. 2022). For example, using the ABCD datasets, it was found that the impact of SES varies across different racial groups in the USA, such that Caucasian Americans with high SES have low level of stress and better educational resources and health. However, most black Americans, regardless of their SES, have higher level of stress, worse educational resources, and work health conditions (for discussion, see Assari 2018). This phenomenon is known as the minorities’ diminished return (Assari et al. 2020a, 2021b), which applies to black Americans, immigrant groups, indigenous people, and refugees. The minorities’ diminished return is also associated with brain changes, including to the amygdala (Assari et al. 2020b).

### 2.5.1 The impact of poverty and low SES on specific brain regions

As we show below, most of the studies on the impact of poverty on the brain focus on the hippocampus, amygdala, and cortex. Furthermore, many studies also show that poverty and low SES impacts educational attainment and language development, as well as the development of psychopathological disorders. Figure 1 provides a framework on the relationship between brain and behavioural deficits caused by poverty.

#### 2.5.2 Hippocampus

The association between hippocampal volume and SES has received considerable attention in the literature (e.g., Hanson et al. 2011; Jednoróg et al. 2012; Noble et al. 2012a, 2015; Weissman et al. 2023; Yu et al. 2018). Typically, research has observed a positive correlation between SES and hippocampal volume, indicating that higher SES is often associated with increased hippocampal size. Conversely, lower SES is associated with smaller hippocampal volume. When circumstances interfere with the development of the hippocampus (e.g., poverty and early life stressors associated with SES), there is a potential for a reduction in hippocampal volume (Dégéilh et al. 2020; Noble et al. 2012a).

The hippocampus is important for academic performance, primarily due to its integral role in memory formation, rational learning, and emotional regulation (Assari 2020b; Moustafa et al. 2010, 2013b). A meta-analysis examined the association between hippocampal volume and memory in children and adolescents (Botdorf et al. 2022). Their results identified a small significant positive correlation between memory performance and hippocampal volume in children and adolescents. A reduction in hippocampal volume, in turn, results in impairments in memory retention and the ability to understand complex concepts (Assari et al. 2021a; Ward et al. 1999; Yu et al. 2018). Such a reduction may also lead to elevated stress levels, diminished motivation, and a compromised attention span. These factors collectively suggest that a reduced hippocampal volume could substantially hinder academic performance. Although considerable research has established links between hippocampal volume and SES, as well as
between hippocampal volume and educational outcomes, learning, and memory, there is limited literature that examines the interplay among these three factors (Yu et al. 2018).

One study examined the direct impact of SES on hippocampal volume in children (8–12 yrs) and young adults (18–25 yrs) (Yu et al. 2018). Their study found a significant age \( \times \) hippocampal volume interaction, where the reduction of hippocampal volume was dependent on participants’ age. Specifically, there was a reduction in hippocampal volume for the children but not young adults. This suggests that as individuals age some of the effects of SES on hippocampal volume are diminished, especially when children experience a high level of parental support. While these mitigating factors were speculative, they were not empirically tested in the study. However, the finding is supported by the broader literature which indicates that the negative effects of SES can be mitigated by strong positive parental involvement (Luby et al. 2013). This indicates that while hippocampal volume is positively associated with SES, the impact of SES on hippocampal volume is mediated by how involved parents are in their child’s development and education (Luby et al. 2013; Yu et al. 2018).

The relationship between SES and hippocampal volume varies across the lifespan.

Research has shown that hippocampal volume is relatively stable after the age of four (Daugherty et al. 2016; Gogtay et al. 2006). However, children from lower SES backgrounds have smaller hippocampal volumes compared to their higher SES peers (Daugherty et al. 2016; Gogtay et al. 2006; McLean et al. 2012). Interestingly, the impact of SES on hippocampal volume seems to be most pronounced during childhood (McLean et al. 2012). In contrast, studies have found that SES does not significantly affect hippocampal volume in adults (Yu et al. 2018). This suggests that early life circumstances related to SES may have a lasting impact on hippocampal volume. Moreover, the absence of protective factors, such as parental educational attainment, may cause these reduced volumes to persist into adulthood for those from lower SES backgrounds (Vannini et al. 2011). Therefore, these findings highlight the importance of early intervention in lower SES populations to potentially mitigate long-term neural disparities and possible implications associated with educational attainment and hippocampal volume and functionality. Evidence suggests that lower SES (e.g., exposure to poverty during early childhood) is associated with atypical hippocampal development, volume, function, thus impacting learning and memory. For example, hippocampal volume has been implicated with educational outcomes (Botdorf et al. 2022; O’Shea et al. 2018).

Further, smaller hippocampal volumes have been linked to poorer memory and learning performance (Antoniades et al. 2018). However, while larger hippocampal volumes are associated with better learning and memory, the effect of hippocampal volume on learning and memory performance is often mitigated by an individual’s level of education. For example, the performance of individuals with relatively smaller hippocampal volumes on learning and memory tasks are often similar to others when they have received more educational training (e.g., O’Shea et al. 2018). Raffington et al. (2019) conducted a longitudinal analysis to address some of the prominent limitations within the literature to investigate if both environmental (e.g., SES) and genetic factors are associated with cognitive performance in children. Specifically, they aimed to test if associative memory and hippocampal volume were related to educational achievement, after controlling for genetic predispositions toward educational achievement. Their study focussed on children aged 6–7 with a follow-up at ages 8–9, respectively. Their results showed that children from lower SES families experience poorer memory performance and smaller hippocampal volume and this effect did not change across time. However, this association was not dependent on a genetic predisposition for educational attainment and was likely due to their lower SES.

Noble et al. (2012b) conducted a study to explore the relationship between hippocampal volume and educational attainment across various life stages. Prior research had established a negative correlation between educational level and exposure to life stressors, as well as between life stressors and hippocampal volume. Extending these findings, Noble et al. (2012) investigated whether educational attainment directly influences hippocampal volume. The study revealed that the effect of age on hippocampal volume differs based on one’s level of education. Specifically, individuals with lower educational levels showed a significant decline in hippocampal volume with age, whereas those with higher educational levels experienced less pronounced age-related reductions. These findings suggest that higher education may serve as a protective factor against age-related brain changes. However, this study did not establish causality. For instance, it is also possible that smaller hippocampal volumes across the lifespan could lead to lower educational attainment due to early developmental deficits. Such structural changes are likely influenced by an individual’s SES during early childhood and adolescence and their subsequent level of educational attainment.

According to Johnson et al. (2016), studies that have explored the relationship between childhood SES and cortical thickness in the hippocampus have identified that children raised in higher SES status in childhood is correlated with greater volume in the hippocampus where as low SES status in childhood is correlated with reduced...
hippocampus volume. Accumulative evidence suggests that parental hostility and support may mediate the relationship between lower SES and childhood hippocampal volume, suggesting that, in the presence of environmental and financial stress, parenting style may have a direct impact of psychophysiological evolution of this region.

### 2.6 Poor diet and the hippocampus

As discussed above, poor diet is quite common in impoverished communities. The hippocampus seems to be affected by poor diet to a greater extent than other brain regions (Gainey et al. 2014; Sloan et al. 2021) and recent research has aimed to understand the complex relationship between dietary habits and hippocampal function. For example, in a mice study, Gainey et al. (2014) compared the effects of high and low-fat diets on hippocampal and amygdala function through cognitive performance tasks (i.e., memory for novel object location and novel object recognition). Their results showed that there were significant impairments for the amygdala and hippocampus for the high-fat diet group compared to the low-fat diet group. However, they also found that after a 6-week period of dieting the mice were able to mitigate the negative effect of the high-fat diet on amygdala functionality. However, the hippocampus remained adversely affected by the high-fat diet and continued to exhibit cognitive impairments. These findings, while found in mice, suggest that high fat diets have a particularly detrimental impact on hippocampal function to a greater extent than other brain regions.

Sloan et al. (2021) conducted a randomised control study to assess the impact of a high flavanol diet, rich in antioxidants, on cognitive ageing. They recruited 255 healthy adults aged 50–75 years for a 20-week period, comprising 12 weeks of dieting followed by 8 weeks off the diet. Their findings suggest that dietary flavanols are associated with hippocampal functionality, memory performance, and normal cognitive decline. Notably, participants with a poor baseline diet quality exhibited significant improvements in hippocampal learning performance after the introduction of the flavanol diet. This indicates the significant impact of diet on the hippocampus, particularly the dentate gyrus, but also shows that dietary changes later in life can lead to marked changes in brain functionality. However, the effects of the flavanol diet varied across different brain regions. While the flavanol diet enhanced hippocampal performance, there was no significant improvement observed in the function of the pre-frontal cortex. This study shows that a healthy diet, rich in flavanols, can bolster hippocampal health, whereas other research displayed that a diet high in fatty acids and carbohydrates might harm hippocampal function. Furthermore, their findings support the notion that the hippocampus is more susceptible to dietary influences than other brain regions but suggest memory function can be improved through specific dietary nutrients that improve hippocampal function.

#### 2.6.1 Amygdala

Research indicates a complex relationship between SES and amygdala structure and function, particularly in young adults and children (Dégeilh et al. 2020). Studies have consistently shown that higher SES is positively correlated with larger amygdala volume, especially in adolescents aged 13–21 (Hao et al. 2022). This suggests that SES may play a role in emotional development during this critical period. However, the impact of SES is more pronounced in individuals from lower SES backgrounds, who often exhibit smaller amygdala volumes, experience more negative emotions, and attenuated amygdala growth (Rakesh and Whittle 2021). This is supported by findings that children aged 6–12 from lower SES backgrounds have reduced amygdala volume compared to their high-SES counterparts, even when controlling for age, cortical volume, and biological sex (Noble et al. 2012, 2013). While SES is linked to amygdala volume, it does not appear to influence the functional aspects of the amygdala, such as emotion regulation (Hao et al. 2022). This is further complicated by the fact that lower SES is associated with increased amygdala activation when exposed to emotionally salient stimuli, indicating a potential deficit in emotional regulation (Javanbakht et al. 2015).

The amygdala plays an important role in emotional processing and regulation, social interactions, and learning (Yu et al. 2018). For example, the amygdala demonstrates activation during aversive learning, emotional learning, and associative learning (Calandreau et al. 2005; Straube et al. 2007; Wen et al. 2022). In particular, associative learning (i.e., operant and classical conditioning) is integral to an individual’s ability to acquire new knowledge and is related to education outcomes (Chau and Galvez 2012). However, there is also evidence of SES having a direct impact on amygdala volume which could lead to deficits in learning ability (Hao et al. 2022; McLachlan et al. 2020; Rakesh and Whittle 2021). Higher SES is generally associated with larger amygdala volume, which is particularly significant for adolescents and those from lower SES backgrounds. However, SES does not seem to influence the functional aspects of the amygdala, although it may affect emotional regulation as evidenced by increased amygdala activation in lower SES individuals.

Fewer studies have investigated the long-term trajectories of SES on amygdala volume and the impacts of these
throughout the lifespan. Butterworth et al. (2011) examined 431 adults (aged 44–48 years). Their study showed that adults who were from lower-SES backgrounds (i.e., financial hardship) presented with significantly smaller amygdala volumes compared to those from higher-SES backgrounds, even after controlling for well-known aged-related factors that contribute to amygdala atrophy. These findings highlight the nuanced ways in which SES can impact both the structural and functional aspects of the amygdala, with implications for emotional and psychological well-being, and educational attainment (Laakso et al. 1995; Noble et al. 2012b, 2013). In terms of education, few studies have investigated how SES and the Amygdala (volume, function, and activation) interact to predict educational outcomes of children, adolescents, or adults. However, based on the known functions of the amygdala, we can speculate that a reduction in amygdala volume, resulting in reduced functional connectivity may directly impact an individual's ability to learn.

The role of the amygdala in associative learning is directly implicated with oral and written language development, memory consolidation, skills acquisition (mathematical, scientific, and linguistics), and understanding of complex concepts – especially when it requires making connections between ideas (Ortiz-Mantilla et al. 2010). However, the role of the amygdala can also have indirect impacts on learning ability (Chau and Galvez 2012; Farley et al. 2016; Ortiz-Mantilla et al. 2010). For example, because the amygdala is heavily involved in emotional and behavioural regulation, an individual's learning environment can influence how they engage with the learning process. For instance, when a child's learning environment positively reinforces study habits, study effort, and academic achievement, young people are more likely to develop a positive association with learning and indirectly improve their educational performance through increased intrinsic academic motivation (Park et al. 2017). Conversely, when an individual's learning environment does not create a positive association, this can fundamentally change how they appraise learning and could affect their ability to develop intrinsic motivation. However, while these indirect impacts can be observed behaviourally, these effects may be more pronounced in individuals from lower SES because of their contribution to structural changes within the amygdala. Therefore, it is important for future research to test the possible moderating or mediating role of the effect of SES on educational outcomes through amygdala volume.

According to Olson et al. (2021), severe economic hardship may be a risk factor for altered amygdala functioning and connectivity is observed as early as the first few months of life. Observed through fMRI conducted on naturally sleeping infants $n = 32$ (10 weeks old), extremely poor families ($n = 16$), affluent families ($n = 16$). Findings identified that infants from extremely poor families displayed less negative functional connectivity between amygdala and precuneus. According to Johnson et al. (2016), enlarged amygdala volume is highly correlated with behavioural disorders. Functional studies are identified to be more consistent, finding that lower childhood SES and risky family environments are associated with greater or less-regulated amygdala activation during emotion processing tasks. Chronic stress is identified as a risk factor in the relationship between childhood poverty and amygdala activity. Lower amygdala to hippocampus volume ratios are correlated with emotional dysregulation. Maternal depression and insecure infant attachment are associated with larger amygdala in childhood and young adulthood. Several studies found that there is a link between childhood SES and amygdala function, such as people with lower SES have smaller amygdala (Luby et al. 2013), but for conflicting findings, see Dufford et al. (2019).

2.6.2 Cortex

There are a plethora of studies linking volume and activation levels of the cortex to SES. For example, using near-infrared spectroscopy, Olson et al. (2021) found that SES (measured by parental level of education and household income) is related to differences in functional brain development of the prefrontal cortex of pre-schoolers during cognitive set-shifting tasks. Further, other studies have reported that poverty is related to low working memory performance and executive functioning, which is indicative of dysfunctional prefrontal cortex (Evans and Schamberg 2009; Evans et al. 2021). When SES was dichotomised into a categorical poverty versus no poverty measure, prefrontal activation (measured by significant changes in oxyhaemoglobin in the task-switch vs. the baseline/rest conditions) was observed in the no-poverty group only, whereas the children experiencing poverty showed no significant activation during the task conditions.

Furthermore, studies have reported reduced grey matter volume in individuals living under poverty line in the US (Hair et al. 2015), especially in parietal and frontal cortical areas (Hanson et al. 2013). It has been also reported that household income is related to grey and white matter volumes (Luby et al. 2013). According to Johnson et al. (2016), accumulating evidence regarding the structural changes in prefrontal cortex in relation to SES help explain the relationship between poverty, chronic stress and cognitive/behavioural outcomes. They also argued that material deprivation, as well exposure to stress and negative parenting behaviours are associated with reductions in prefrontal volume.
2.7 The impact of poverty and low-SES on language, educational attainment, and psychopathology

In this section, we discuss several studies on the impact of poverty on educational attainment, language development, as shown in the right panel of Figure 1.

2.7.1 Language delay and educational performance

A plethora of studies show that SES is associated with academic achievement and educational performance (Enger et al. 2006; Islam and Khan 2017; Nicolas et al. 2009; Rodríguez-Hernández et al. 2020). Students from high SES backgrounds tend to perform significantly better than their counterparts from lower SES backgrounds (Fernández Sanjurjo et al. 2018; Hadden et al. 2020). This trend is not only evident in standardised public examinations but also in the likelihood of these students advancing to subsequent educational levels (e.g., progressing to higher education and advanced degrees, Cardak and Ryan 2006). Importantly, Hair et al. (2015) found that brain abnormalities are mediating factors between poverty and poor academic performance (also see Ehler 2023). This finding is demonstrated in the model presented in Figure 1.

While SES is a strong indicator of educational performance, the exact mechanisms that contribute to the better performance of high SES students are multi-faceted and extend beyond economic advantage. For example, high SES students often exhibit heightened intellectual abilities, a greater sense of educational mastery, higher self-efficacy, and more efficient psychosocial coping styles (Bosma et al. 2007; Falci 2011). Such attributes might be because of their access to better learning environments, greater exposure to educational resources, or even genetic predispositions. While the impact of genetics is evident, the environment in which a student develops plays an equally pivotal role. Living conditions, especially those marked by poverty, can be detrimental. Poverty-stricken environments may expose children to health risks, hinder their brain development, and lead to behavioural challenges in educational settings (Dike 2017; Kolb and Gibb 2016).

Educational performance and language attainment are interrelated. It has been reported that language development can predict subsequent academic performance (Kastner et al. 2001; Pace et al. 2017). From as early as infancy, children from low SES demonstrate differences in brain activation related to language development. According to Olson et al. (2021), neural circuits subserving executive and language functioning are recognised as particularly sensitive to the socioeconomic environment. Thus, young children entering kindergarten from low socioeconomic backgrounds may already be at a disadvantage in terms of language and literacy skills. According to Johnson et al. (2016), literacy skills are among the strongest skills associated with SES.

2.7.2 Psychopathology

The negative consequences of exposure to poverty on psychological functioning are well documented and several decades of research have contributed to a broad understanding of the link between poverty and psychopathology. As discussed above, exposure to poverty is associated with reduced development and functioning in the amygdala, hippocampus and pre-frontal cortex ( Brito and Noble 2014; Duval et al. 2017; Vyas et al. 2002). A crucial outcome of a reduction in the development and functioning of the aforementioned are a decrease in functional connectivity between regions of the brain, which can be concisely defined as synchronicity and communication between these brain regions (Jolles et al. 2011), which is also associated with impaired psychosocial functioning (i.e., adaptive coping and stress management) (Evans et al. 2013; Gilman et al. 2002). This has been found to be especially important in the developing brain as the first 2 years of infancy demonstrate the highest level of plasticity. As such, disruptions of this process are linked to disruptions of brain based development (Gao et al. 2017). The long-term impacts of these factors have been associated to reinforced experiences of poverty, and as a result, can lead to in inter-generational experiences and transmission of poverty (Najman et al. 2018). Recent findings from Palacios-Barrios and Hanson (2019), demonstrated a relationship between impairments in neural development, as well as deficits in psychosocial functioning as a consequence of exposure to poverty. According to Palacios-Barrios and Hanson (2019), a link between poverty and psychopathology occur due to impairments in self-regulation as a consequence of exposure to poverty. Specifically, greater self-regulatory abilities result in greater attention to relevant information in one’s environment, which in-turn results in positive behavioural adjustments. Further, deficits in self-regulation may lead to an unawareness of the impacts of one’s behaviours and the consequences associated to these behaviours, as well as an inability to anticipate or detect negative outcomes.

The experiences of poverty can lead to a number of negative outcomes depending on the time in which the individual is exposed to poverty (Bradley et al. 2001). Importantly, Kim et al. (2016) noted that childhood experiences of poverty results in a number of consequences, namely, a dysregulated...
approach to stress, which has been linked to psychological illness in adult life. The time in which an individual is exposed to poverty was also linked to the development of maladaptive coping strategies. Exposure to poverty between early childhood to early teenage years was found to contribute to the development of negative coping strategies, such as disengagement coping (Kim et al. 2016). Disengagement strategies within the context of poverty are acknowledged as a risk factor which contributes to cyclical experiences of poverty. Santiago et al. (2012) argue that disengagement strategies enhance the negative experiences of the effects of poverty, which are typically anxiety, depression, and stress (Kim et al. 2016; Wadsworth et al. 2008).

Several disengagement strategies typically associated with poverty are strategies such as avoidance, dissociation, and high risk behaviour such as substance abuse (Votta and Manion 2004). This in combination with the negative effects prefaced, foster an environment which undermines the development of adaptive coping strategies in order to improve one’s own circumstance, such as willingness to access support, budgeting, planning, and seeking employment (Mayo et al. 2022). An important aspect of adaptive coping is the ability to regulate one’s own behaviour. In a study of low-income youth, Buckner et al. (2009) found adaptive coping to be strongly associated with positive outcomes, such as academic engagement, behaviour, and mental health. From a self-regulatory perspective, exposure to poverty has been linked to deficits in this important psychosocial skill. Palacios-Barrios and Hanson (2019) argue that greater self-regulatory abilities have been found to result in greater attention to relevant information in one’s environment, which in-turn results in positive behavioural adjustments. Whereas deficits in self-regulation may lead to an unawareness of the impacts of one’s behaviours and the consequences associated to these behaviours, as well as an inability to anticipate or detect negative outcomes (Palacios-Barrios and Hanson 2019). Skills such as those are important when making decisions or navigating stressors associated with limited financial resources (Yu et al. 2020).

Further, deficits in non-vocational skills such as attention create barriers to employment, often prolonging exposure to the economic pressures of poverty and financial hardship. This often leads to the onset of major depressive disorder, characterised by persistent experiences of sadness and loss of interest in activities (LeMoult et al. 2020). Furthermore, as a consequence of childhood exposure to poverty, factors like stress, limited socialisation, as well as reduced participation in extracurricular activities, can contribute to disengagement strategies, often leading to substance abuse and the development of substance use disorders (Manhica et al. 2021). Several studies have shown that substance abuse is related to low SES (Daniel et al. 2009; Gauffin et al. 2013), but for conflicting findings, see (Martin 2019).

Furthermore, the relationship between substance use disorders (i.e., drug addiction) and poverty is bidirectional (Moustafa 2019). Studies have shown that drug abuse is more common in neighbourhoods with low socioeconomic status (Chauhan and Widom 2012), including synthetic drugs that have damaging impact on the brain, such as methamphetamine (i.e., ice). Unlike cocaine for example, studies have shown that there are more people from low, rather than high, socioeconomic status that abuse methamphetamine (Rommel et al. 2015). Studies have also shown that methamphetamine has a more detrimental impact on several brain areas than cocaine, including dopamine and the basal ganglia (Fowler et al. 2008). Several studies have also shown that poverty in childhood increases drug abuse in adulthood (Arteaga et al. 2010; Barton et al. 2018; Manhica et al. 2021). Further, drug abuse in parents is associated with neglect of their children, childhood trauma, and an increase in susceptibility of drug abuse in the children (Font and Maguire-Jack 2020; Lee et al. 2021).

Several studies have also shown that there is a relationship between SES and the development of cognitive decline, dementia, and Alzheimer’s disease (Mortimer and Graves 1993), but for conflicting findings, see (Wilson et al. 2005). Educational level in particular was related to the development of Alzheimer’s disease (Letenneur et al. 1999), but for conflicting results see (Pavlik et al. 2006). However, one study in Denmark found that earlier diagnosis of Alzheimer’s disease was reported more in people with higher income (Petersen et al. 2021). However, this could be the case due to more awareness and being able to afford medical checkups.

3 Discussion

The goal of this study is to amalgamate a large body of research to explain the intricate interactions between poverty, socioeconomic status and brain functioning. By bringing the brain into the picture of poverty-related outcomes, we can perhaps in the near future, overcome poverty-related disparities. Our study shows that poverty impacts both the brain and behaviour, and that these changes are interrelated (also see Rakesh and Whittle 2021; Troller-Renfree et al. 2022). We also found that maladaptive neural changes occur early in childhood due to poverty and low socioeconomic environment. These neural changes have a large negative impact on language development, educational attainment, as well as the development of psychopathological disorders.

Our modelling framework (Figure 1) provides an explanation of generational poverty. As explained above,
Generational poverty is defined as parents growing up in poor environmental impacts educational attainments of their children, which in turn, make them also poor in the future (Beegle 2003; Gal 2014). Our model suggests that maladaptive brain changes during early childhood development has large impact on language development, educational attainment, and wellbeing, thus reinforcing poverty in adults and their children, leading to a vicious cycle of continuous generational poverty.

We here argue one way to minimise neural damage caused by poverty is by mitigating risk factors such as cumulative and prolonged stress, poor diet, and socio-environmental hazards. It has been found recently that anti-poverty policies can improve neural development (Weissman et al. 2023). Furthermore, prior studies have shown that changing dieting plans can change brain activity (DelParigi et al. 2007). Managing stress was also found to ameliorate brain function, including the hippocampus (Veena et al. 2009) and amygdala (Hölzel et al. 2010).

3.1 Future directions: an extensive list of studies to better understand poverty-brain interrelationships

Given that the interrelationship between poverty and brain processes is a budding research area, there are a multitude of future directions. Below, we provide a comprehensive list of future studies that can help further understand the poverty-brain interactions.

Future studies should investigate the complexity of Figure 1 by conducting a large-scale study as well as path analysis to understand the interactions between behaviour and brain changes due to poverty. Importantly, path analysis will also determine the strength among each all components in Figure 1. For example, future research should investigate exact contribution of stress and poor diet to brain function. Along these lines, several studies have shown that brain abnormalities mediate the relationship between poverty and academic performance (Ehler 2023; Hair et al. 2015). As discussed above, this in agreement with our modelling framework (Figure 1). However, future work should explore whether brain abnormalities mediate the relationship between poverty and other outcomes, such as the development of psychopathological disorders.

There are also other mediating factors in the relationship between poverty and educational, health, and occupational outcomes. For example, while SES is undoubtedly a pivotal factor in determining educational outcomes, its effects can be mitigated when parents invest in creating an environment conducive to learning. It is observed that parents from higher SES backgrounds, especially those with advanced educational qualifications, are in a position to invest more time and resources, such as tutoring and private education, for their children (Guryan et al. 2008; Park 2008). This socioeconomic advantage enables them to create a nurturing educational environment that instills strong beliefs and values about education in their children (Kim and Lee 2010; Zhang and Xie 2016). However, current research provides limited insights into the interaction between a student’s SES and the quality of parental involvement in their education, which should be investigated in future work. In addition, future work should also extend the current modelling framework in Figure 1 and all mediating factors between poverty, brain, and behavioural outcomes.

It is important to note that while some prior studies have investigated the impact of SES as an integrated factor, some other studies have considered each of its components – education, occupation, and income – separately (Wells and Lynch 2012). Future research should investigate whether each of SES components have a different impact on brain and behaviour. Along these lines, there is a notable distinction between the social and economic components of SES, as both of which have unique influences on educational outcomes. For instance, while household income does contribute to a student’s performance, parental educational attainment appears to be a more dominant factor (O’Connell 2019). This distinction necessitates a re-evaluation of how SES is defined and utilized in research. The same applies to the development of psychopathological disorders in low SES individuals. Future research should investigate which factors of low SES (high stress levels, poor diet, or exposure to socio-environmental hazards) are related to the development of psychopathological disorders in poor individuals. It is likely that stress in childhood plays a key role in the development of drug addiction, while poor diet and stress are related to the development of major depressive disorder in poor individuals.

Studies have also shown that reducing poverty can ameliorate brain function (Troller-Renfree et al. 2022). It is also important to test whether reducing poverty also impacts psychopathology, education, and language development. Based on Figure 1, it is expected that reducing poverty may improve wellbeing and lead to a reduction in psychological disorders. Future work should investigate whether mitigating factors related to poverty (e.g., excessive stress, poor diet, and exposure to environmental hazards) may normalise brain activity. While this was done in some studies in relation to stress and diet, it was not done with socio-environmental hazards (DelParigi et al. 2007; Hölzel et al. 2010; Veena et al. 2009). As mentioned above, stress management can ameliorate brain function (Hölzel et al. 2010; Veena et al. 2009). However, most of these studies were conducted on adults. While there are stress
reduction and management programs for children (Goh et al. 2015), these studies did not investigate changes in brain function due to these interventions. Future work should investigate whether stress reduction can ameliorate brain function in children and teenagers.

As discussed above, the impact of SES on hippocampal volume is mediated by the amount of parental involvement and their educational level (Luby et al. 2013; Yu et al. 2018). This is an important finding, because it means there are factors that can mitigate neural abnormalities caused by poverty. Along these lines, in rural settings in the U.S., parental investment in education was found to significantly mediate the association between SES and educational outcomes (Henry et al. 2011). Parents’ involvement, complemented by their beliefs and values about education, has a profound impact on the academic trajectory of students, regardless of their socioeconomic background (Hermida et al. 2019). Accordingly, future research should investigate whether the same applies to other brain areas. Furthermore, in addition to parents and caregivers, future research should also investigate whether teachers’ involvement and government support and aid can also mitigate brain abnormalities caused by poverty.

In terms of neural studies, future work should investigate which subregions of the amygdala and hippocampus are impacted by poverty. The amygdala has few areas, including basolateral amygdala, central nucleus of the amygdala, and intercalated cells (Corbit and Balleine 2005; Moustafa et al. 2013a; Sierra-Mercado et al. 2010; Strobel et al. 2015). These brain regions are impacted differently by stress and anxiety (Etkin et al. 2004). Future work should investigate how poverty and low SES impacts different regions of the amygdala and hippocampus.

More research is needed on the impact of socio-environmental hazards on brain function. Most of the research done on this area is related to the development of ageing disorders, as discussed above. However, exposure to air and noise pollution impacts the brain and body, making individuals more vulnerable to the development of disorders such as Alzheimer’s and Parkinson’s disease.

4 Conclusions

From the expanding neural research, we can identify that low SES negatively impacts brain development and functionality. In particular, SES is associated with maladaptive amygdala hippocampal, and cortical processing. When these brain regions are underdeveloped or have a reduced functional capacity due to an individual’s SES, this manifests into poor cognitive and socio-emotional functioning as well poor educational, occupational, and health outcomes. We have here provided here the first modelling framework to explain the complex relationship between poverty, brain processes, and occupational/educational/health outcomes. Our model explains generational poverty using neural studies. Importantly, we here provide a comprehensive set of future studies to further help explain the impact of poverty and how to mitigate its effects.

Research ethics: For this systematic review, ethical approval was not necessary as it involves secondary analysis of published data, which were collected under the ethical standards of the original studies.

Informed consent: Given the nature of this work as a systematic review, the study compiles and synthesizes findings from pre-existing research publications. Therefore, individual informed consent for this study was not required as the data were derived from published sources where informed consent had already been obtained by the original investigators.

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