

BIOLOGICAL AND SOCIOCULTURAL DETERMINANTS OF NEUROCOGNITIVE DEVELOPMENT: CENTRAL ASPECTS OF THE CURRENT SCIENTIFIC AGENDA

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1. Introduction

The study of the influences of material and social deprivation on the central nervous system (CNS) has been an issue of interest in the agenda of neuroscience since the first half of the twentieth century. Early neuroscientific studies have begun to analyse how the exposure to complex, standard or deprived environments modifies the brain in the context of experimental animal models. At present, the same approach still applies to the analysis of how different rearing environments modulate the brain structure and function at molecular, genetic, cellular, network, individual and social behaviour levels (Mohammed *et al.*, 2002; Pang & Hannan, 2013; Sale *et al.*, 2009; Simpson & Kelly, 2011). Moreover, the study of stress regulation, which also has an extensive history in the neuroscience agenda, has addressed the impact of different threatening experiences on the hypothalamic-pituitary-adrenal axis (HPA axis) functioning (Feder *et al.*, 2009; Joëls & Baram, 2009; Karatoreos & McEwen, 2013; Lupien *et al.*, 2009). More recently, stress regulation analysis began to be applied to the study of poverty and cognitive development through different perspectives, such as vulnerability and environmental susceptibility (Ellis & Boyce, 2011; Sheridan *et al.*, 2013; Theall *et al.*, 2013), executive functions performance (Blair *et al.*, 2011; Evans & Fuller-Rowell, 2013), and child development policy (Shonkoff & Bales, 2011). During the twentieth century research programs emerged to analyse the influences of malnutrition (Antonov-Schlorke *et al.*, 2011; Georgieff, 2007) and the exposure to different types of pollutants (Hubbs-Tait *et al.*, 2005; Jacobson & Jacobson, 2004) and drugs (Thompson, 2009) at pre- and postnatal brain development stages, with significant implications for the neuroscientific study of social inequities. The neuroscientific study of human poverty, particularly child poverty, is an issue that has recently emerged (Gi-

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anaros & Hackman, 2013; Hackman *et al.*, 2009, 2010; Lipina & Colombo, 2009). Since the mid-nineties different researchers have begun to apply neurocognitive behavioural paradigms to compare the performance of children with disparate socioeconomic status (SES) (e.g., Farah *et al.*, 2006; Lipina *et al.*, 2004, 2005, 2013; Lawson *et al.*, 2013; Mata *et al.*, 2013; Mezzacappa, 2004; Noble *et al.*, 2005). Technological advances in neuroimaging allowed the incorporation of neural network analysis (e.g., D'Angiulli *et al.*, 2008, 2012b; Jednoróg *et al.*, 2012; Krishnadas *et al.*, 2013; Monzalvo *et al.*, 2012; Noble *et al.*, 2006, 2012, 2013; Raizada *et al.*, 2008; Rao *et al.*, 2010; Sheridan *et al.*, 2012, 2013; Stevens *et al.*, 2009).

Taking into consideration all these research efforts, the study of how different environmental conditions (e.g., disparate SES or poverty) influence brain organization and reorganization during development includes different approaches, such as neural plasticity, epigenetics, influence of environmental toxins, nutrition, stress regulation, poverty modulation of cognitive and emotional processing, and poverty perception of adults with a history of poverty at childhood (Hackman *et al.*, 2009, 2010; Lipina & Colombo, 2009). Some of the main questions currently included in this neuroscientific agenda focus on some topics already analysed in the fields of developmental psychology, cognitive psychology and health sciences, especially regarding the effects and mechanisms of mediation at the behavioural level of analysis (Bradley & Corwyn, 2002; Brooks-Gunn & Duncan, 1997; Evans *et al.*, 2013; Lipina & Colombo, 2009; McLoyd, 1998; Moffitt *et al.*, 2011; Reiss, 2013; Schreier & Chen, 2013; Yoshikawa *et al.*, 2012). Nonetheless, the intrinsically innovative aspect of the neuroscientific agenda is that neuroscience allowed the beginning of these explorations in terms of basic mental operations considering different levels of analysis (i.e., genetics, networks, behaviour). In this sense, during the last decade different researchers began to produce evidences regarding how disparate socioeconomic status or deprivations modulate different aspects of neural processing and neurocognitive performance in tasks with language and cognitive control demands – the two neurocognitive systems in which poverty modulation appears to be most frequent (Gianaros & Hackman, 2013; Hackman *et al.*, 2009, 2010; Lipina & Colombo, 2009; Lipina & Posner, 2012; Raizada & Kishiyama, 2010).

Addressing the mechanisms of mediation of the impact of child poverty on cognitive development is also a recent issue of interest and study in the context of neuroscience (Hackman *et al.*, 2010). In other related disciplines, such as the study of pollutant or toxic agent neurotoxicity and malnutrition, these issues began to be explored earlier. However, even in the latter case (i.e., nutrition) only recently have researchers begun to include current neu-

roimaging technologies, which would eventually allow improving its comprehension (e.g., Jackson & Kennedy, 2013; Sheinkopf *et al.*, 2009).

To generate changes in neurocognitive development, interventions have been recently implemented for the study of attentional disorders (e.g., Thorell *et al.*, 2009), dyslexia (McCandliss *et al.*, 2003; Shaywitz *et al.*, 2004; Temple *et al.*, 2003), dyscalculia (Wilson *et al.*, 2006), attentional development in healthy children (Rueda *et al.*, 2005), executive functions (Colombo & Lipina, 2005; Lipina *et al.*, 2012; Neville *et al.*, 2013b), and arithmetic performance (Wilson *et al.*, 2009) in samples of children from different SES backgrounds. In all these studies, the behavioural levels of analysis have been emphasized, and neuroimaging techniques (Rueda *et al.*, 2005; Shaywitz *et al.*, 2004; Temple *et al.*, 2003) and behavioural genetics (Espinete *et al.*, 2012; Neville *et al.*, 2013b; Rueda *et al.*, 2005, 2012) have been included in some cases.

Further research is required in terms of (1) the theoretical and methodological integration of developmental psychology and cognitive psychology hypotheses to the neuroimaging studies devoted to explore neurocognitive development, and intervention studies focused on better cognitive and emotional performances, both in disparate SES samples of infants, children and adolescents (Crone and Ridderinkhof, 2011; Gianaros & Hackman, 2013); and (2) the inclusion in this agenda of the exploration of how improve the comprehension of effects and mediation mechanisms beyond the most univariate approach proposed by neuroimaging techniques (e.g., Lipina & Posner, 2012; Noble *et al.*, 2013).

This chapter proposes a brief review of findings, conceptual and methodological contributions and challenges about the present neuroscientific approach of childhood poverty. The aim of this effort is to visualize target areas, which could potentially help to build a research agenda for the coming years. In this context of discussion, it would be wise to determine which is or would be the specific contribution of neuroscience that differs from that made by other disciplines.

2. Effects and mediators of material and social deprivation

Neural plasticity

The brain adapts to its environment based on experience (Hebb, 1949). In experimental settings, rodents and non-human primates exposed to motor, sensory and social stimulation in complex environments show several structural and functional changes in different neuronal and non-neuronal components, compared with those subjects exposed to deprived environments (Mohammed *et al.*, 2002; Pang & Hannan, 2013; Sale *et al.*, 2009; Simpson & Kelly, 2011). Specifically, exposure of different species to com-

plex, standard or deprived environments has been associated with several structural changes such as synaptic number and morphology, dendritic arborisation, cell morphology; number of astrocytes and glial-synaptic contacts, myelination, glial cell morphology; brain vasculature; brain cortex weight and thickness; rate of hippocampal neurogenesis; availability and metabolism of both neurotrophic factors and neurotransmitters in different brain areas; and neurotrophic and neurotransmitter gene expression as well. In turn, these multiple changes in neural structure have been repeatedly correlated with functional changes in motor, cognitive, and emotional outcomes at the behavioural level of analysis (i.e., learning, motor, self-regulation and attachment paradigms) (Mohammed *et al.*, 2002). Thus, development and learning would continue to exist, with each endpoint receiving inputs from experience-expectant (which would share common developmental time points across individuals of the same species) and experience-dependent mechanisms (which are more fluid in timing, as experiences and learning opportunities differ in developmental times among individuals) (Galván, 2010).

Neural plasticity in humans may also lead to use-dependent structural adaptation in cortical grey matter, in response to environmental demands (Bavelier & Neville, 2002). At the level of imaging studies, evidence exists that the brain may adapt dynamically to reflect environmental cognitive demands. For instance, neuroimaging studies evidence structural changes in specific areas after training in difficult motor tasks, such as the increased activation of motor, auditory and visual-spatial brain areas and white matter tracts as well, in professional musicians (Gaser & Schlaug, 2003; Imfeld *et al.*, 2009); or selective increases in grey matter volume in posterior hippocampus and concomitant spatial memory performance in licensed taxi drivers from London (Woollett and Maguire, 2011). During the last decade, the studies of developmental cognitive neuroscience aimed at analyzing the influence of poverty or SES on neural organization have been integrated into the research agenda of plasticity. These studies have made a specific contribution through the integrated analysis of different levels of analysis. Examples of such an integration are the study by Rao and colleagues (2010), which analyses the prediction of parent nurturing on memory demanding tasks and hippocampus volume; and the study by Sheridan and colleagues, which analyses the links among hippocampal function, HPA axis function and maternal SES (2013) or prefrontal function, HPA and home complexity language (2012).

Regarding white matter plasticity, and apart from the above studies of musicians, different mental and developmental disorders began to be de-

scribed in terms of their impact on cortical connectivity, using the diffusion-tensor imaging (DTI) technique and functional connectivity analysis. In this sense, the experience of a stressful event cannot be localized to single brain regions (Hermans *et al.*, 2011), but to a distributed system involving cortical and subcortical areas, and the neuroendocrine system as well. Thus, the stressing experience would depend upon sociocultural history and how it shapes the resting networks (Allen & Williams, 2011). In the specific context of poverty studies, Noble and colleagues (2013) have recently assessed to what degree white matter microstructure mediates the relationship between education attainment and performance in a cognitive control task.

Conceptually, current theoretical approaches propose that neural development often depends on neural activity, which in turn is mediated by experience. It is therefore assumed that cognitive and emotional processing and learning shape the neural networks responsible for this processing. In turn, this activity would change the nature of neural representations and their processing, which leads to new experiences and further changes in the neural systems. Therefore, in terms of neuroscience, this neuroconstructivist approach proposes that the basis of cognitive, emotional and learning development may be characterized by mutually induced changes between neural, cognitive, emotional and learning levels, in a complex ecological context involving social interactions with cultural specificities (Westermann *et al.*, 2007). Therefore, this complexity must be considered when trying to study each one of these dimensions in isolation or at a unique level of analysis.

Sensitive periods and epigenetics

One of the most promising areas to be faced by the field of poverty and neuroscience over the next years is that of the sensitive periods, which characterizes the structural and functional organization of those brain networks most affected by socioeconomic deprivation. Sensitive period refers not only to a time when human brain is especially sensitive to particular classes of external stimuli, but to a time window temporarily opened, during which the brain is particularly receptive to experience that contributes to its organization.

Neural networks are shaped by experience also during critical periods of early postnatal stages of development in different species. Timing, duration and closure of these plastic processes have been experimentally addressed by the analysis of the visual system. It has been hypothesized that these processes are run by the following principles: diversity of molecular mechanisms in different brain areas, role of structural consolidation, inhibitory and excitatory balance, functional competition between inputs,

regulation by experience and age, influence of motivation and cognitive control, and potential for reactivation of organizational processes in adulthood (Bavelier *et al.*, 2010; Hensch, 2004).

Recent behavioural studies have disclosed that critical periods are not necessarily fixed in terms of timing and object specificity. For example, the period of organization for the imprinting of any domestic bird – usually conceptualized as a critical period rather than sensitive – could be extended in time, should the appropriate stimulus be missing. Alternatively, imprinting can be reversed under certain learning conditions. This suggests that closure of this period is likely to constitute the natural consequence of a given learning process (Michel & Tayler, 2005).

At a neurocognitive level of analysis, several studies performed in humans show the expression of multiple sensitive periods in sensory systems, several aspects of speech development and face recognition (Peretz & Zatorre, 2005). A very important feature in the development of these sensory systems is that sensitive periods are not synchronized among sensory modalities. In spite of such differences in developmental timing, basic plastic mechanisms appear to be similar. The end of a sensitive period is often associated with the age at which a set of neural circuits subserving a given neural processing becomes specialized. For instance, between the 6th and 12th months of age, electrophysiological patterns associated with face recognition processes become specific for a given stimulus. In addition, and approximately at the same age, the number of cortical areas activated by the viewing of faces seems to decrease. This suggests that the end of the sensitive period of a neural substrate for a given modality processing coincides with specialization attainment (Johnson, 2005).

In the case of neural circuits involved in complex behaviours, the closure of sensitive periods seems to depend on their association or not with circuits performing either fundamental or high level computations. For instance, the sensitive period for circuits combining visual inputs from both eyes ends a long time before circuits responsible for recognizing biologically significant objects do so (Pascalis *et al.*, 2005). According to Knudsen and colleagues (2006), experience-dependent plasticity of high-level circuits – i.e. those related to language, cognitive and emotional processes – would depend on the type of information provided by those circuits, while experience-dependent plasticity is unable to attain completion until such circuits become stable.

Thus, time-scale and integration of different forms of plasticity would be targets for a neuroscientific agenda in the field of poverty and brain development aimed at exploring windows of intervention opportunities. This

analysis is time-consuming and requires methodological innovations for the exploration of molecular, system and behavioural events and phenomena at the same time, and throughout different stages of development. For example, in experiments with infants different tools are usually introduced to facilitate motor skills before the age at which these behaviours are typically observed. These studies provide behavioural information about how experience-expectant processes can be manipulated to happen before it is expected. Therefore, associated measures of neural activity could allow a better understanding of the emergence of the mechanisms responsible for these behaviours (e.g., Needham *et al.*, 2002; Rao *et al.*, 2010).

Current studies in the developmental neuroscience field continue to advance in the understanding of the mechanisms through which experience and environmental influences interact with genes, especially with DNA biochemical markers and histone proteins that regulate gene activity, which could be modified by early experience. Post-translational modifications of histones and DNA methylation are the most frequently analysed mechanisms, which are involved in interactions between gene activity changes and environmental factors, such as neurotoxin, nutrition and regulation of stress (Roth & Sweat, 2011; Zhang & Meany, 2010).

Preliminary studies of maternal care, caregiver maltreatment, mother-infant separation and prenatal stress in experimental animal models hypothesize that early environmental influences could produce lasting epigenetic modifications, stable changes in the CNS gene activity and behavior. For instance, in experimental models with rodents phenotypes of adult offspring raised by mothers providing high level of pup licking and grooming were attributable to molecular changes, such as hippocampal glucocorticoid receptor, transcription of the NGFI-A factor, corticotrophin releasing factor expressions, and glucocorticoid feedback sensitivity (Roth & Sweat, 2011; Zhang & Meany, 2010).

Different studies reported significant associations between childhood maltreatment and developmental disorders later in adolescence and adulthood. Neurocognitive approaches to adults with histories of childhood maltreatment suggest the modulation of this experience on different nodes of the HPA axis (Lupien *et al.*, 2009). In epigenetic animal models, Roth and colleagues (2009) found a significant methylation of the brain-derived neurotrophic factor (BDNF) in the prefrontal cortex, and a DNA hypermethylation paralleled a lasting deficit in expression of the gene as well. Moreover, pharmacological treatment with a DNA methylation inhibitor in adults that had experienced maltreatment resulted in the rescue of the aberrant DNA methylation and gene expression patterns incited by adver-

sity. These preliminary results suggest that experiences with an abusive caregiver during the very first stages of postnatal development can modify DNA methylation and gene expression.

Regarding mother–infant separation, recent evidence also supports the hypothesis that this experience can also modify DNA methylation and gene expression. In this sense, Murgatroyd and colleagues (2009) found that periodic mother–infant separations during a sensitive period of development modulate the methylation of the arginine vasopressin gene – a hypothalamic inductor of synthesis and release of adrenocorticotropin from the pituitary.

In other series of experiments, different studies have shown that prenatal experiences should be recognized for their profound effects on brain development, hypothesizing that glucocorticoids could be the mediator of such modulation. For example, Mueller and colleagues (2008) found that prenatal stress on adult HPA axis responsiveness and behaviour might be mediated by changes in hippocampal glucocorticoid receptor and hypothalamic corticotropin releasing factor genes expressions.

Learning and memory processes also evoke alteration of epigenetic markers in the adult CNS, as shown by animal models. For instance, Miller and Sweatt (2007) have used the contextual–fear conditioning paradigm to analyse epigenetic modulation of hippocampal genes. They found that following fear conditioning and during a period of fear memory formation, adult rats have a demethylation and transcriptional activation of the memory–enhancing gene *reelin*, and an increase in methylation and transcriptional silencing of the memory suppressor gene *Protein Phosphatase 1*.

The epigenetic analysis of the early experiences on brain development in humans is at its first stages, as many of the issues in the study of childhood poverty and brain development. McGowan and colleagues (2009) recently examined the gene expression and DNA methylation of the human glucocorticoid receptor (*Nr3c1*) gene in hippocampal samples from suicide victims with a history of childhood maltreatment. They found decreasing levels of mRNA hippocampal glucocorticoid receptor gene, correlated with increases in cytosine methylation of the *Nr3c1* promoter, which suggests that human caregiver experiences may program genes through epigenetic modifications. In another study, Oberlander and colleagues (2008) found that infants of mothers with high levels of depression and anxiety during the third trimester of pregnancy had increased methylation of the *Nr3c1* gene promoter in cord blood cells. Evidences of the modulation of epigenetic mechanisms during early development in different rearing conditions (e.g., disparate SES, stress exposure) have been recently incorporated into this research agenda. For instance, Essex and colleagues (2013) examined

differences in adolescent DNA methylation in relation to parent reports of adversity during childhood. They found that maternal stressors in infancy and parental stressors in preschool periods predicted differential methylation and gender differences. In addition, recent cumulative evidences have suggested differential susceptibility to rearing environment depending on dopamine-related genes (Bakermans-Kranenburg & Van Ijzendoorn, 2011). More recently, these frameworks have begun to be applied to analyse the association between dopaminergic polymorphisms and educational achievement (Beaver *et al.*, 2012).

Although many conceptual and methodological issues should be explored, the epigenetic approach supports the notion that epigenetic changes underlie at least partially the long-term impact of early experiences, and that epigenetic alterations are potentially reversible or modifiable through pharmacological and behavioural ways. This means that the understanding of the role of the epigenome in behavioural modifications driven by early experiences could contribute to the field of childhood poverty and brain development. However, the genetic polymorphisms in humans should be cautiously analysed because similar experiences could produce different outcomes in different people, which adds another level of complexity to the study of how behaviour is modulated by early experiences.

Poverty influences on self-regulation and language development

Regarding cognitive development, the most commonly described impacts of poverty were first associated with Developmental Psychology and Education, which are lower Developmental Quotients, children's verbal and achievement Intelligence Quotients (IQ) – verbal and executive, completed school years; and higher incidence of learning disorders and rates of school absence (Bradley & Corwyn, 2002; Brooks-Gunn & Duncan, 1997; Evans *et al.*, 2012; Yoshikawa *et al.*, 2012). With respect to language, in the same disciplines, current language development studies also show different patterns of socioeconomic modulation on several outcomes, such as vocabulary, spontaneous speech, grammatical development, and communication styles and skills (Hoff, 2006).

Viewing cognition as consisting of component codes, computed in different ways and programmed to perform complex tasks, leads to new ways of thinking about how the brain might organize thought and emotional processes (Posner & Raichle, 1994). Specifically, basic processes involved in early cognitive control and language development, such as the different sub-systems of attention, working memory, and flexibility, are the cornerstone of all forms of cognitive activity and social behaviour throughout the lifes-

pan in most cultural systems worldwide (Sperber & Hirschfeld, 2004). The main assumption from this neurocognitive point of view is that given the multiplicity of factors that influence and modulate brain development, it is most likely that the impact of poverty on cognition would have a neurocognitive basis, and that those more basic cognitive functions would be modulated by socioeconomic backgrounds.

Neuroscientific studies that began to assess associations between different forms of poverty and its impact on basic cognitive processing have been recently reported. Several studies verified the modulation of socioeconomic characteristics on different attentional, inhibitory control, working memory, flexibility, planning, phonological awareness, self-regulatory, decision making, and theory of mind processes related to different neurocognitive systems in infants, preschoolers, and school- and middle school-age children (D'Angiulli *et al.*, 2008, 2012b; Farah *et al.*, 2006; Herrmann & Guadagno, 1997; Jednoróg *et al.*, 2012; Kishiyama *et al.*, 2009; Lawson *et al.*, 2013; Levine *et al.*, 2005; Lipina *et al.*, 2004, 2005, 2013; Mata *et al.*, 2013; Mezzacappa, 2004; Monzalvo *et al.*, 2012; Noble *et al.*, 2005, 2007, 2006, 2012, 2013; Raizada *et al.*, 2008; Rao *et al.*, 2010; Sheridan *et al.*, 2012, 2013; Stevens *et al.*, 2009).

Among the most widely explored neurocognitive systems is the prefrontal/executive system, which includes subsystems such as the lateral prefrontal cortex and the anterior cingulate cortex related to working memory and cognitive control processing, respectively. In behavioural studies of preschoolers, first graders and middle school children, Farah, Noble and colleagues repeatedly found that low SES children had reduced performance on these tasks compared to middle SES children (e.g., Farah *et al.*, 2006; Noble *et al.*, 2005). These findings support the hypothesis that the prefrontal/executive system is one of the primary neurocognitive systems associated with social inequalities in early experience. Similar results have been observed in studies using specific paradigms designed to measure aspects of executive function. For example, Lipina and colleagues (2005) examined the performance of low and middle SES infants using a task of a delayed-response paradigm (i.e., AnotB), which incorporates the evaluation of processes such as working memory and inhibitory control. Findings showed that low SES infants had more errors associated with lower levels of performance in inhibitory control and spatial working memory, and errors associated with attention and search strategies. The effects of socioeconomic disparities on attention have been examined in several studies. For instance, Mezzacappa (2004) used an Attention Network Test (ANT) to investigate the effects of socioeconomic disparity on attentional processes in 6-year-old children. Re-

sults showed that low SES children had reduced of both speed and accuracy on measures of alerting and executive attention, indicating that SES modulated response conflict and inhibited distracting information.

The medial temporal/memory system was assessed by Farah and colleagues (2006) using an incidental learning paradigm in which subjects are not aware that memory will be tested during the learning phase of the task and both verbal and non-verbal stimuli can be employed (e.g., pictures and faces). Results showed that low SES first grade and middle school children had reduced performances, which was not initially found in kindergarten children. However, after adding a delay interval, this finding was observed in the older groups.

In some of these studies, researchers have reported that the modulation of SES on performance is neither similar in all the administered measures, nor uniform at all ages (e.g., Farah *et al.*, 2006; Lipina *et al.*, 2004, 2013; Noble *et al.*, 2005). Both aspects are worth considering for different reasons. Conceptually, this implies that poverty does not necessarily generate homogeneous and continuous changes in neurocognitive processing. This is consistent with temporal and regional differences in cortical organization throughout childhood and adolescence (Brain Development Cooperative Group, 2012; Gogtay *et al.*, 2004; Menon, 2013; Zhou *et al.*, 2013). At the same time, these findings are not consistent with the notion of low-SES performance as a deficit (D'Angiulli *et al.*, 2012a). In summary, the findings from the above behavioural studies indicate that SES disparities can adversely affect cognitive processes, such as language, executive function, attention and memory. This further suggests that specific brain regions may be associated with these processes, and that the paradigms used would be more specific than those used to measure general cognitive ability (e.g., scales of intelligence).

However, these evidences are still behavioural in nature and therefore present certain limitations. Thus, researchers can make only indirect inferences about brain function. In addition, many of these tests are multi-factorial and performance could be modulated for reasons other than those resulting from a specific alteration. Moreover, low correlations have been obtained among these tests, which mean that two tasks can engage the same system in different ways. Therefore, a deep examination of the impact of SES on the relationship between cognitive processes and brain function is needed. In this sense, magnetic resonance imaging (MRI), functional magnetic resonance imaging (fMRI) and electroencephalography (EEG) neuroimaging techniques applied to analyse the neural level of analysis would contribute to a better understanding of these relationships.

For instance, Noble and colleagues (2006) hypothesized that SES systematically influences the relationship between phonological awareness skills and brain activity in areas involved in reading. Specifically, researchers have predicted that the strength of the association between phonological awareness and brain activity will be increased in an environment with low exposure to literacy resources, and reduced in the opposite case. To test this hypothesis, researchers have examined fMRI responses during a pseudoword reading task in first- to third-graders from diverse SES backgrounds, who showed an equivalent level of phonological awareness scores from impaired to normal levels. Results suggested a significant phonological awareness-SES interaction in the left fusiform area, indicating that at similar low phonological awareness levels, children from higher SES were more likely to evidence increased responses in the left fusiform cortical gyrus, while children from lower SES were not. In another recent study of normal 5-year-old children performing an auditory rhyme-judgment task, Raizada and colleagues (2008) found that the higher the socioeconomic status, the greater the degree of hemispheric specialization in Broca's area, as measured by the left-minus-right fMRI activation during rhyming tasks. This suggests that the maturation of Broca's area in children may be ruled by the complexity of the linguistic environments in which they grow up. In turn, Monzalvo and colleagues (2012) compared fMRI activity to visual stimuli (i.e., houses, faces, written strings) and to sentences spoken in the native or a foreign language in 10-year-old dyslexic and normal children from disparate SES backgrounds. They found similarities in fMRI activation in both SES groups, which was interpreted by authors as the existence of a core set of activation anomalies in dyslexia, regardless of culture, language and SES. Additionally, Sheridan and colleagues (2012) found that complexity of family language – and salivary cortisol, a biological marker of stress regulation – was associated with both parental SES and prefrontal cortex activation during a stimulus-response mapping task. Finally, the same researchers (Sheridan *et al.*, 2013) verified associations between subjective social status and hippocampal activation in children during a declarative memory task. The latter findings add evidences to those by Gianaros and colleagues in college students and adults regarding the modulation of subjective social status on amygdaline response to angry faces (Gianaros *et al.*, 2008), and perigenual anterior cingulated cortex (Gianaros & Manuck, 2010), respectively.

More recently, different researchers have begun to explore the SES modulation of different structural brain measures using MRI techniques. For instance, Rao and colleagues (2010) examined the effects on later brain

morphology of parental nurturance and home stimulation in the context of a longitudinal data set of early experience during childhood (4 and 8 years) and MRI during adolescence. They found that parental nurturance at age 4 predicted the volume of the left hippocampus in adolescence (i.e., better nurturance was associated with smaller hippocampal volume). In contrast, home stimulation did not correlate with hippocampal volume. Importantly, the association between hippocampal volume and parental nurturance disappeared at 8, suggesting the existence of a sensitive period for brain maturation. In addition, Lawson and colleagues (2013) found evidences that parental education is associated with cortical thickness in some subareas of the prefrontal cortex. In turn, Noble and colleagues (2013) showed that educational attainment predicted the performance in an inhibitory control-like task, and that this association accounted for connectivity (i.e., DTI) previously linked to cognitive control. Finally, Jednoróg and colleagues (2012) explored the association between SES and brain anatomy applying MRI in 10-year-old children. Their results showed that low-SES condition was associated with smaller volumes and surfaces of grey matter in hippocampus, middle temporal gyri, left fusiform gyri and right inferior occipito-temporal gyri. In addition, they found local gyrification effects on anterior frontal regions, and no associations between SES and white matter architecture.

Complementary, recent studies of socioeconomic disparities have used electrophysiological techniques to obtain direct measures of brain activity. For example, D'Angiulli and colleagues (2008) examined the influence of socioeconomic disparities on attention using an auditory selective attention task and ERP techniques. In these studies, low and high SES children had to respond to target tones in an attended channel while withholding responses to all other tones in the attended and unattended channels. A negative difference waveform, reflecting selective attention, was observed for high SES but not for low SES children. These results suggest that high SES children selectively attended to relevant information, whereas low SES children attended equally to relevant and irrelevant information. In a similar approach, the effects of SES on attention were investigated by Stevens and colleagues (2009) using an adapted version of the selective auditory attention paradigm. In this case, children were instructed to attend to one of two narratives played simultaneously in speakers located to their left or right. They also observed that low SES children showed reduced ERP measures of selective attention, suggesting that this group has a reduced ability to filter or suppress irrelevant information. In a more recent study, D'Angiulli and colleagues (2012b) showed that frontal brain areas of high- and low-

SES children were differently activated during selective attention tasks. Tomarken and colleagues (2004) also found evidences of SES-related differences in the neural processing of emotion in low-SES adolescents. Specifically, researchers observed lower left-sided brain activity at rest, as measured by resting alpha-asymmetry at frontal sites. Finally, Tomalski and colleagues (2013) found SES disparities in frontal gamma power in infants as young as 6 months old.

In a recent study using a visual novelty oddball paradigm, Kishiyama and colleagues (2009) examined the impact of socioeconomic disparity on prefrontal-dependent ERP components. Interestingly, no behavioural differences were observed in measures of reaction time and accuracy between low and high SES children, and no differences were observed in target ERP responses. The behavioural results indicate that both groups could perform the task with a high degree of accuracy. In addition, group differences were predicted in prefrontal-dependent ERP components. Specifically, researchers found that low SES children had reduced amplitudes for early, attention-sensitive, visual ERP components and novelty ERP responses. These differences in results at distinct level of analysis reveal that environmental influences on neurocognitive performance and development could be differentially modulated. This implies that a complex approach with different methodologies is required in order to examine several levels of analysis (D'Angiulli *et al.*, 2012a; Gianaros & Hackman, 2013; Lipina & Posner, 2012). Although such an approach is methodologically and logistically difficult to implement, some recent examples can be verified (e.g., Neville *et al.*, 2013b; Noble *et al.*, 2012, 2013; Posner *et al.*, 2011; Rueda *et al.*, 2005; Rao *et al.*, 2010; Sheridan *et al.*, 2012, 2013; Voelker *et al.*, 2009).

Finally, there is still an important sort of disconnection between the large amount of behavioural and neural information, which represents not only a quantitative but also a qualitative issue. This means that the mere measurement of the neural correlates of SES disparities would be insufficient to advance in the field (Crone & Ridderinkhof, 2011; Gianaros & Hackman, 2013; Raizada & Kishiyama, 2010). As Raizada and Kishiyama propose (2010), information from the neural level of analysis may be useful for predicting performance changes – which anyway requires an integration of conceptual and methodological issues of development and plasticity. However, in cross-sectional comparative analysis and in behavioural interventions, the univariate approach of fMRI and ERP must be overcome using integrating alternatives, such as the analysis of its development in conjunction with neural connectivity analysis approaches.

Exposure to environmental toxic agents and drugs

The impact of different environmental toxic agents on children's cognitive development has been recently analysed. Several epidemiological and animal studies have reported the negative impact of different metals, plastics, legal and illegal drugs, and the lack of micro- and macronutrients on pre- and post-natal development of the CNS (Hubbs-Tait *et al.*, 2005; Walker *et al.*, 2011). At present, there is consensus about the negative effects on brain development of different neurotoxic agents such as lead (Magzamen *et al.*, 2013), mercury, manganese and cadmium (Hubbs-Tait *et al.*, 2005) – all of which cross the placenta. Although the documented impacts have been identified by high and low exposure to these neurotoxic agents at the behavioural and cognitive level, cognitive performance levels associated with toxic exposure are highly variable. Thus, further research is required concerning why some children are more susceptible than others are to certain neurotoxic agents. This would help to clarify the effectiveness of treatments and both regulatory and public policy interventions (Hubbs-Tait *et al.*, 2005).

Emphasis has been laid on the impact of alcohol consumption during pregnancy and its serious consequences on neurocognitive development throughout the lifespan, as evidenced by the many studies on foetal alcohol syndrome (Riley *et al.*, 2011). Specifically, prenatal alcohol exposure has been shown to have cognitive, social and emotional long-lasting impacts compared with other substances, which vary with the amount of consumption and the specific time during pregnancy at which exposure occurs (Irner, 2011).

Prenatal exposure to cocaine has been associated with various cognitive disorders. Schroder and colleagues (2004) studied children aged 8 and 9 years that were exposed to cocaine prenatally and found changes in the speed of response and procedural learning skills. In another study, Bennett and colleagues (2008) examined the effects of prenatal cocaine exposure on performance in a test of general intelligence in 231 children aged 4, 6 and 9 years. They found that prenatal cocaine exposure interacted with gender. Thus, boys had the lowest scores – especially in abstract reasoning, visual short-term memory and verbal reasoning tasks. Moreover, higher levels of home stimulation and mother's verbal IQs predicted higher scores. Finally, Sheinkopf and colleagues (2009) analysed prenatal cocaine exposure on performance on a task demanding inhibitory control in combination with fMRI techniques in children aged 8 and 9 years. Results showed no differences at the behavioural level between exposed and unexposed children. However, differences were observed at the level of neural activation during inhibition demands performance. Since these are preliminary findings, it is

necessary to be cautious regarding their implications. Nevertheless, results suggest that cocaine exposure during uterine life affects the development of those neurocognitive systems associated with the regulation of attention and inhibitory responses.

Other drugs with impacts on cognitive development are tobacco and marihuana. Fried and colleagues (2003) analysed the cognitive performance of 145 adolescents between 13 and 16 years exposed during prenatal life to both substances. The results showed a significant correlation between exposure to tobacco and general intelligence levels and auditory attention, and exposure to marihuana and performance on memory tasks, and analysis and synthesis processing. Recently, Barros and colleagues (2011) found that exposure to tobacco during pregnancy was associated with higher levels of excitability and emotional regulation difficulties at the neonatal stage. These studies – which also require the consideration of mediation analysis to explore their mechanisms of influence, suggest that prenatal exposure to tobacco and marihuana is associated with neurocognitive impairment during the first two decades of life. Furthermore, prenatal exposure to tobacco has also been associated with an increased risk of obesity, hypertension and gestational diabetes since adolescence (Cupul-Uicab *et al.*, 2011). These impacts could be sometimes reduced through the mediation of adequate parenting and the provision of environmental stimulation, even in the case of children grown up in contexts of poverty (Evans & Kutcher, 2011). Finally, Roussotte and colleagues (2012) studied the associations between prenatal methamphetamine and polydrug exposure and corticostriatal networks. Applying a functional connectivity MRI technique during a working memory task in children aged 7 to 15 years, they found differences in the patterns of activation – the putamen showed increased connectivity with frontal areas, while the caudate showed decreased connectivity with some of the same areas – in both groups of exposed children compared to controls.

According to the current agenda, a better understanding of these phenomena depends on the development and evaluation of models for the analysis of the simultaneous combined or cumulative effect of different neural components on different aspects of motor, cognitive and emotional processing (Evans *et al.*, 2013). Evidence shows that prenatal exposure to different legal and illegal drugs is associated with the development of various changes during the first two decades of life both at the behavioural and neural level of analysis (Mezzacappa *et al.*, 2011; Roussotte *et al.*, 2012). Since social vulnerability is associated with an increase in the use and abuse of these substances (Bradley & Corwyn, 2002; Evans *et al.*, 2013), this area of research significantly contributes to the study of the impact of poverty

on brain development. Although most of the studies are based on the application of behavioural methods, neuroimaging techniques have been recently applied for a better understanding of such impacts (e.g., Roussotte *et al.*, 2012).

Nutrition

Epidemiological research has shown that the correlation between nutrition and low SES modulates: (a) physical growth; (b) the potential occurrence of prenatal neural tube defects due to poor folic acid intake before pregnancy; (c) the prevalence of iron deficiency due to poor intake of food rich in this mineral; (d) cognitive process associated with memory demands after long episodes of poor nutrition; and (e) greater likelihood of developing insecure attachments and other emotional disorders due to chronic malnutrition (Bradley & Corwyn, 2002; Georgieff, 2007; Nyaradi *et al.*, 2013).

In neurobiological terms, nutrients and growth factors regulate brain development since the prenatal stage. The rapid brain development during the early stages of growth leads to greater vulnerability to poor nutrition. For example, experimental studies with nonhuman primates have recently demonstrated that moderate nutritional restriction of mothers during pregnancy is associated with various structural and functional disorders of the CNS (Antonov-Schlorke *et al.*, 2011). However, the detection of specific deficiencies depends on how each brain area or neural network is preferentially affected, and on devices to measure such potential impacts at the molecular, systemic or neural, and behavioural networks levels of organization. For example, iron deficiency has been associated with alterations in the synthesis of different neurotransmitters, cognitive speed processing, and performance on tasks with motor, emotional and cognitive demands. In addition, the impact caused by nutrient deficiency depends on the identification of that nutrient. To illustrate, zinc deficiency is associated with alterations in hippocampal development, but also in the cerebellum and autonomic regulation, while deficiencies of certain long-chain fatty acids affect myelin production and further contacts between neurons (Benton, 2008; Georgieff, 2007).

As in the case of other scientific approaches dealing with child poverty, it is difficult to determine the implications of the different nutritional deficit in the typical and atypical development, since children who lack proper nutrition also lack other resources. Specifically, a difficult issue to be determined is whether a condition associated with nutritional deficit occurs as a direct result of this deficit, or of inadequate prenatal care, difficulties to receive adequate medical treatment, or increased exposure to infectious agents (Adler & New-

man, 2002). For example, prematurity and low birth weight are also associated with the absence or reduction of prenatal care (Bradley & Corwyn, 2002). Many families living in poverty cannot afford health care services; therefore, they turn to emergency services at advanced stages of disease, thus increasing the risk of morbidity and mortality (Friel *et al.*, 2011).

Recent studies have explored the potential effects of diet on mental health transmitted across generations, and whether diet can influence epigenetic mechanisms. In this respect, a longitudinal study conducted in Sweden with records from 300 families living in relative isolation in the same region over a 100-year period; show that the risk for diabetes and early death was increased if paternal grandparents grew up in times of food abundance (Kaati *et al.*, 2007). Complementary to this finding, the experimental research on animal models also discusses issues such as the influence of diets rich in saturated fats on gene expression of different regulatory mechanisms of the hippocampus (Gomez-Pinilla, 2008).

Current studies of the effects of nutrition on brain function focus on the effects of breakfast on cognitive function and academic performance (Adolphus *et al.*, 2013), the effects of breakfast different components (e.g., rice versus bread) on neural activation (Taki, 2010), the role of insular cortex in food related processes (Frank *et al.*, 2013), and the impacts of long-term nutritional interventions. In this sense, the inclusion of multiple evaluation methodologies has been postulated, such as biological markers in combination with nutritional supplements and performance on cognitive tasks at different stages of development. This would allow the identification of potential sensitive periods during the lifetime (Prentice *et al.*, 2013) and pathways of impacts and interventions (Yousafzai *et al.*, 2013).

Regulation of stress response

Since the mid-twentieth century, many studies have analysed the regulation of stress response in both children and adults, as one of the most important mediating mechanisms of the effect of poverty on emotional, cognitive and social functioning (Fernald & Gunnar, 2009; Lupien *et al.*, 2009). Threats, negative life events, exposure to environmental hazards, family and community violence, change processes and family break-up and moves, job loss or instability and economic deprivation are more likely to occur in poverty conditions (Bradley & Corwyn, 2002; Evans *et al.*, 2013; Maholmes & King, 2012; Yoshikawa *et al.*, 2012).

Neural systems implementing this complex regulation include the hippocampus, amygdala and different areas of the prefrontal cortex (i.e., HPA axis). Together, these systems regulate the physiological and behavioural re-

response to stress, adapting to short- or long-term impacts caused by difficulties in adaptation processes, as in chronic situations of abuse or extreme poverty (Karatoreos & McEwen, 2013; Shonkoff *et al.*, 2012). These regulatory processes arise from communication between the brain and the immune and cardiovascular systems. On the one hand, bidirectional mechanisms regulating the stress response are protective through adjustments in the short term. On the other hand, these mechanisms may be associated with physiological mismatches under conditions of chronic stress, affecting recovery processes and overall health (McEwen & Gianaros, 2010). The stress and uncertainty caused by economic deprivation conditions increase the likelihood of negative emotional states, anxiety, depression and anger. In turn, this may induce more frequent negative parental control strategies, less sensitive emotional neglect and more difficulties in promoting appropriate socio-emotional adjustment in children (Shonkoff *et al.*, 2012). However, some studies show that even in poverty, maintaining proper child rearing can be a protective factor of development (e.g., Brody *et al.*, 2002), highlighting the environmental plasticity levels of these regulatory systems.

The analysis of the mechanisms mediating the stress has generated a number of guiding principles that can contribute to the understanding of child poverty. Ganzel and colleagues (2010) have suggested that the stressor properties (i.e., magnitude, duration and chronicity), and nature (e.g., social exclusion versus physical threat) modulate the type of differential impact on neural networks involved in acute and chronic responses to stress. In this regard, it is necessary to investigate the timing and specificity of the neural developmental sensitive to stress processes (Lupien *et al.*, 2009).

The current neuroscientific agenda in this area has begun to gradually incorporate the concepts and methodologies derived from the advances in epigenetics and the analysis of neural activation in both animal and human models. Three sets of problems have started to feed the agenda: prenatal programming of brain plasticity, reactivity of amygdala areas in threatening situations, and brain embodiment of adverse life experiences (Gianaros & Manuck, 2010).

In the context of child poverty analysis of long-term stressful experiences, Blair and colleagues (2011) found that cortisol levels combined with the quality of parenting contexts functioned as mediators of the effect of family income, maternal education and ethnicity on different cognitive control tasks. In addition, in a recent study including children in rural poverty contexts, Fernald and Gunnar (2009) found that cortisol levels decreased only in those cases where mothers had higher levels of depressive symptoms. This suggests that child poverty and maternal mental health modulate stress regulation: both

cortisol and mental health levels are two mechanisms that may improve the understanding of the associations between child poverty and stress.

The impact of moderate to chronic stress has been associated with the release of a diverse set of brain chemical modulators, which have specific temporal and spatial niches that generate complex phenomena not yet clearly identified (Joëls & Baram, 2009). Wismer-Fries and colleagues (2005) have recently found that the absence of opportunities for adequate attachment during the early stages of development is associated with hormonal changes involving neuropeptides vasopressin and oxytocin. These hormones are critical to the development of social bonds and the regulation of emotional behaviour. Specifically, experiences of physical and sexual abuse during early developmental stages have been associated with this complex pattern of stress responses, which are assumed mediators of increased susceptibility to psychiatric disorders in adulthood (Feder *et al.*, 2009). However, vulnerability and susceptibility to chronic moderate situations of stress vary individually according to epigenetic phenomena and the eventual presence of certain potentially protective factors, such as relationships with caring adults, self-regulation and social competences and the pleasantness of children (Evans & Fuller-Rowell, 2013; Luthar *et al.*, 2006).

During the last decade, the first neuroimaging studies were performed to explore how socioeconomic deprivation during childhood influences the stress response at different stages of life. Tottenham and colleagues (2011) evaluated the long-term neural correlates of adverse rearing conditions and performance on an emotional inhibitory control task that demanded the discrimination of threatening faces. The results showed that children raised in orphanages showed increases in amygdala activity, which was associated with emotional processing and decreased eye contact during dyadic interactions between children and adults. Previously, Taylor and colleagues (2006) had found that adults with histories of stressful childhood in families at risk of stress for physical and mental health presented specific patterns of reactivity in amygdala and orbitofrontal areas during observation of threatening faces. More recently, Butterworth and colleagues (2011) applying structural MRI found that adults exposed to poverty at childhood had modified the volumes in their hippocampus and amygdala nuclei; and Hanson and colleagues (2011) found the same pattern of results even in populations of school-age children.

3. Concluding remarks

The trajectories of typical and atypical neurocognitive development in disparate socioeconomic contexts generate different degrees of brain plas-

tivity (Gianaros & Manuck, 2010). The consideration of the sensitive periods for many of these plastic processes requires a revision of the agendas of those disciplines addressing child poverty based on the notion that the impacts are permanent and irreversible. Considering the latter as a starting point in the design of new studies and a scientific and public policy, does allow evaluating the opportunities that could enhance human development.

The notion that poverty impacts are irreversible, in contraposition to that postulating that the CNS plasticity promotes opportunities for emotional, cognitive and social optimization contributes to visualize the ethical and moral responsibilities of communities in the generation of those conditions. In this context of discussion, the neuroscientific approach helps to understand more specifically the extent of the impacts of social and material deprivation produced by society (Stephens, Markus & Phillipis, 2014), and the possibilities for preventive interventions to protect human development. Thus, neuroscience should actively participate in the ethical discussion on poverty, and may even enhance discussions about some basic moral rights and facilitate their exercise. Thus, access to adequate nutrition from prenatal stage encourages emotional and intellectual development in different contexts, such as home and school. It further promotes full social and educational inclusion, and comprises different aspects such as basic human rights, which have been considered by different disciplines.

Regarding the research agenda, there is still a trend focused on the effects of poverty on different processes, instead of the analysis of mediation mechanisms, and the use of reductionist conceptions of childhood poverty and development (D'Angiulli *et al.*, 2012a; Neville *et al.*, 2013a; Lipina *et al.*, 2011). These are some issues that require a deeper approach:

(1) Theoretical integration of developmental and cognitive psychology in experiments applying neuroimaging techniques, in order to promote and generate innovative hypotheses and research programs (Crone & Ridderinkhof, 2011; Gianaros & Hackman, 2013).

(2) Analysis of the development of neural connectivity in different socioeconomic rearing contexts (i.e., parenting), given its implications in the study of the impact of poverty, its mediators and intervention efforts (e.g. Jolles *et al.*, 2013; Lipina & Posner, 2012).

(3) Development of innovative research to analyse plasticity of complex affective and cognitive processes and their windows of opportunity (i.e., sensitive periods) in the context of intervention studies (D'Angiulli *et al.*, 2012; Lipina & Colombo, 2009; Lipina & Posner, 2012).

(4) Generation of alternative methodologies aimed at overcoming those obstacles associated with small sample sizes, application of longitu-

dinal designs, and integration of different levels of analysis (Gianaros & Hackman, 2013).

In all these areas – i.e., effects, mediators and interventions – research questions, and therefore the interpretation of data obtained by the applied molecular, behavioural, and neuroimaging techniques, seem to focus on the comparison of performance and degree of activation. In addition, most of the evidence is still based on cross-sectional or short-longitudinal designs, which makes it difficult to integrate and adjust the discussion on neurocognitive developmental phenomena. This sometimes leads to the mistaken notion that poverty is associated with neurocognitive deficits, which must necessarily be studied in a research context comprising neuroscientific approaches in the field of plasticity, development and transfer of interventions.

In particular, a need still arises to consider a proper and specific (neurocognitive) criticism on how to conceptualize the child poverty experience, to include and adjust the implications of poverty impacts and mechanisms according to different aspects of neurocognitive development (Lipina *et al.*, 2011). Although this issue has not yet been developed, the neuroscientific evidence on the impact and its progression during development contributes to an insight into poverty as a phenomenon much more complex and dynamic than the definitions proposed by other social and human scientific disciplines.

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