PERSPECTIVE
https://doi.org/10.1038/s41562-018-0384-6

nature human behaviour

Corrected: Author correction

Growing a social brain

Shir Atzil[1]*, Wei Gao[2], Isaac Fradkin[1] and Lisa Feldman Barrett[3]*

It has long been assumed that social animals, such as humans, are born with a brain system that has evolved to support social affiliation. However, the evidence does not necessarily support this assumption. Alternatively, social animals can be defined as those who cannot survive alone and rely on members from their group to regulate their ongoing physiology (or allostasis). The rather simple evolutionary constraint of social dependency for survival can be sufficient to make the social environment vitally salient, and to provide the ultimate driving force for socially crafted brain development and learning. In this Perspective, we propose a framework for sociality and specify a set of hypotheses on the mechanisms of social development and underlying neural systems. The theoretical shift proposed here implies that profound human characteristics, including but not limited to sociality, are acquired at an early age, while social interactions provide key wiring instructions that determine brain development.

Humans are a social species. We cooperate with each other[1]. We form long-term pair bonds with selected individuals[2]. We bear and care for children over an extended period of time[3]. Sociality is a survival strategy, which optimizes securing the resources that are necessary for growth, protection and reproduction[4]. Consequently, many human psychological features can be best understood in a social context. Here, we propose an evolutionary theory of social affiliation. We begin by defining a social species as one where animals regulate one another's fundamental physiological processes (or allostasis), and therefore their survival depends on social bonds. Allostasis is the ongoing adjustment of an individual's internal milieu that is necessary for survival, growth and reproduction[5], and social animals gradually learn to regulate their own and others' allostasis using social communication[6]. Therefore, we hypothesize that social affiliation is rooted in allostasis[6]. Social dependency for allostasis regulation is a coevolutionary plan that inherently relies not only on the individual's physiology for survival, but also on their social environment. This evolutionary plan is adaptive because as a result of a relatively simple evolutionary feature (for example, inability to control your own allostasis), it maximizes social motivation and developmental flexibility in learning culturally relevant knowledge and behaviours needed to survive in a specific community or social niche.

All mammals and most birds are social to some extent, as newborns cannot survive without at least one dedicated caregiver. In newborns, the presence of the caretaker is to actually keep newborns alive, which could have robust implications for social development. Mothers establish and control allostasis in their offspring during embryonic development[7], in eggs or in the womb. This physiological dependency continues once the offspring are born or hatched. Our definition of a social species using the idea of allostasis dependence is not meant to restrict or simplify the definition of a social species but to add an important dimension to social neurobiology. The idea that sociality is evolutionarily related to allostasis is supported by comparative evidence linking a central feature of allostasis, energy metabolism, to social strategies. The literature indicates that across different species, higher allostatic demands are associated with more complex sociality (Table 1). Given mammalian ontogeny, newborns depend on their mothers, and the initial social dyad is designed to physically regulate allostasis in the infant, including energy expenditure, temperature and immune function[8], and mothers implicitly provide bio-behavioural regulation to their offspring, a phenomenon previously referred to by Myron Hofer as maternal 'hidden regulators'[9]. For example, human mothers feed their infants to regulate their diet, and sing and touch their infants to regulate their temperature, heart rate[10], sleep and arousal[11] (that is, control many aspects of infants' autonomous nervous system). Importantly, while a mother regulates her infant's allostasis she provides nutrition, soothing and comfort. In effect, a caregiver's allostatic support is rewarding[12], which makes social interactions a strong reinforcement. With repeated care, the infant gradually builds an internal model of the caregiver[9]. As the experience with the caregiver is repeatedly associated with a vigorous reward (that is, allostasis regulation), we hypothesize that the internal model of the caregiver is acquired as rewarding, which promotes infant attachment and motivation towards social interactions. Altogether, we propose here that parental care is directed towards infant allostasis, and thus provides an optimal incentive for brain development and learning, as via allostasis the social dyad encourages the acquisition of new behaviours and concepts that are necessary for social affiliation.

In support of the theory suggested here, we review three lines of evidence. First, the social neuroimaging literature and how the brain processes social information. We propose that neural systems supporting social behaviours overlap with those supporting allostasis[13,14]. According to our framework, social affiliation is acquired and develops as a result of an inborn dependency in allostasis. Allostasis is thus considered a domain-general process, which is an important ingredient of sociality. Correspondingly, neural systems that support allostasis represent a crucial neural ingredient that wires social behaviours[15]. Then, a review of the brain development literature demonstrates that the neural circuitry needed for social affiliation is not evident in newborns, and develops throughout childhood[16]. We propose that this potentiates its susceptibility to environmental input. The third line of evidence is the developmental neurobiology literature, which provides compelling evidence that early social experience (particularly maternal care) determines the offspring social biology and behaviour in adulthood. Altogether, these lines of literature indicate that current theories of social bonding are not supported by empirical evidence, and there is a need to consider an alternative theory.

[1]Hebrew University of Jerusalem, Jerusalem, Israel. [2]Cedars-Sinai Medical Center, Los Angeles, CA, USA. [3]Northeastern University, Massachusetts General Hospital and Harvard Medical School, Boston, MA, USA. *e-mail: shir.atzil@mail.huji.ac.il; l.barrett@neu.edu

Table 1 | Comparative association between allostasis and social strategies across species

Level of observation	Evidence	References
Comparative relationship between social strategy and metabolism	There is a relationship between social status and standard metabolic rate in juvenile Atlantic salmon, such that socially dominant fish have higher metabolic rates.	195
	Large brains in primates are associated with both increased metabolism and increased social demands.	196,197
	Sloths have an extremely low standard metabolic rate compared with other mammals. Accordingly, social interactions among sloths are rare, and sloths are known for their solitary habits.	198
	High energy requirements imposed on reproductively active females are an important determinant of the phenomenon of female social dominance in the sifaka *Propithecus uerreauxi*, a Madagascar primate.	199
Similar genetic basis for social development and energy metabolism	Changes in gene expression in primates, which were suggested to accompany social evolutionary development include genes controlling energy metabolism (for example, abnormal spindle-like microcephaly associated (ASPM) gene, glutamate dehydrogenase 2 (GLUD2) gene and microcephalin 1 (MCPH1) gene)	200
Social deficits in bees and humans share a neural gene expression signature	Social deficits in non-social bees and autistic spectrum disorder humans are associated with the gamma-aminobutyric acid (GABA) receptor and voltage-gated ion channel genes, which are involved in allostasis[201].	202

Synthesizing new and longstanding ideas, we propose a developmental trajectory that is socially crafted and powered by allostasis. In particular, with early life experience, the brain assembles predictive models, which enables the development of a conceptual system as a prerequisite for social development. We argue that in a next step, development of concepts promotes human social development by acquiring social concepts (such as the 'mommy'), and social skills (such as synchrony). Integrating empirical findings about the developmental trajectories of neural networks and social competency, we introduce the hypothesis that brain development and social development are two manifestations of the same phenomenon: becoming social experts. Thinking of social affiliation as an acquired skill has scientific, clinical and societal implications.

Unexplained findings in social brain development studies

According to our framework, social animals are not born with a predetermined 'social brain', but rather biologically adapt to become social as a result of allostasis dependency. In this section, we describe neural circuits involved in social processing, and review evidence demonstrating that they are not innate and that early social experience effectively determines their functional outcome (as well as behaviour) in adulthood.

What is the 'social brain' and what is it for? The fully developed adult human brain is organized as anatomically connected and functionally coupled intrinsic networks[13]. These networks are held together by thick, long-range axonal tracks[17]. Of specific importance to both allostasis and social processing are the salience network[13] and the default mode network[18] (also called the mentalizing network[13]). The salience and default mode networks together make up an integrated network for implementing allostasis and represent its sensory consequences, called interoception[14]. These two networks are considered domain-general core networks in the sense that they are consistently involved in a variety of psychological phenomena, including social functioning[13]. These domain-general networks are connected to each other and to other parts of the brain, via cortical nodes called 'rich club hubs'[19], which integrate information from across each network, and between the different networks[19]. The rich club hubs are also heavily connected to each other and to the sensory and motor networks of the brain[19], and are thought to function as a high-capacity backbone for synchronizing neural activity to integrate information across the entire brain[20].

The core intrinsic networks and hubs, specifically in the default mode and salience networks, have been repeatedly demonstrated to participate in social brain processing, including maternal bonding[21–23], social cognition[13] and social network size[24], and to be impaired in patients with social deficits such as in autistic spectrum disorder (ASD)[25,26]. A recent meta-analysis of social neuroimaging studies searched across the literature for a distinct 'neural fingerprint' for social processing. The results from this meta-analysis showed that neural circuits associated with social processing across the neuroimaging literature are similar to domain-general circuits, namely the default mode and the salience networks, which are also involved in allostasis[14] and other mental capacities (S.A. et al., manuscript in preparation). Thus, evidence from human neuroimaging studies suggests an overlap between the neural system that supports social behaviours and the one that supports allostasis.

The brain is malleable in early life and sensitive to social input. It has been demonstrated in the past that the infant brain is not a miniature version of an adult brain[27]. Key aspects of adult brain functional architecture, including long-distance functional synchronization and rich club hubs within the core networks (that is, salience, default), are missing in newborns[28–30] (Fig. 1). Consistently, from a structural development perspective, the myelination of the long-distance axon tracts that allow for the fast, efficient information transfer throughout the networks develops for the most part after birth[27,28,30–32]. In general, sensory and motor-control networks become synchronized early in life, even during the prenatal period, and show adult-like spatial topology shortly after birth[32,33]. However, the core networks, mostly residing in association cortices, develop more slowly over time[34]. For example, it has been reported that major nodes of the default mode network do not become synchronized until six months of life[34] and that the network continues to develop well into childhood and young adulthood[35]. This is also consistent with the developmental course of dendritic arborization and synaptogenesis, which similarly show earlier peaking in primary sensory cortices but prolonged growth in prefrontal and other association cortices[36]. Overall, empirical data suggest that humans are born without the neural infrastructure that supports adult sociality.

Human brain development is a protracted process that starts in utero and lasts for up to 25 years postnatal[37]. During the first perinatal weeks, the brain is characterized by maximized plasticity. There is a massive acceleration of brain growth, myelination,

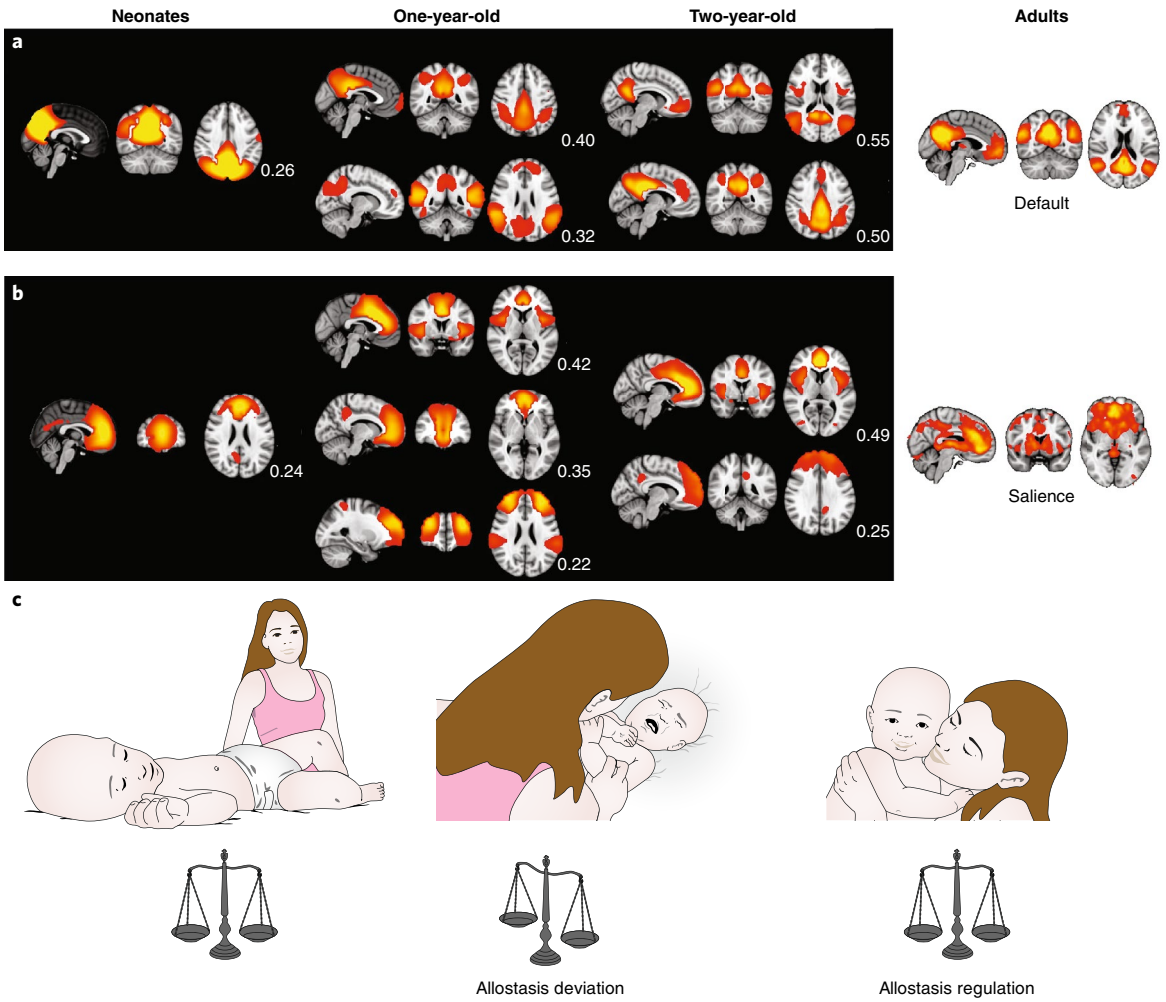


Fig. 1 | Growing a social brain. a,**b**, Development of the default mode network (**a**) and salience network (**b**) during the first two years of life derived from a data-driven independent component analysis technique. The spatial correlation value of each map with the corresponding adult network map is shown at the bottom right of each map. These panels demonstrate that key aspects of adult brain functional architecture that support social processing, including long-distance functional synchronization and rich club hubs, are missing in newborns. Despite the numerous studies showing that neural development depends on social experience (see Supplementary Table 1), the mechanistic role of social care on the developmental trajectory of the default and salience networks was never directly assessed. **c**, Given the pliability of neonates' brains, we hypothesize that the intensive social care during critical years of development have a dominant role in sculpting these networks. Panels **a** and **b** reproduced from ref. [203], Springer Nature Ltd.

enrichment of neural synapses, cortical volume expansion and cortical folding[27,38,39], all of which are critical to normal cognitive development[38,40,41]. The extended developmental course in humans, along with massive neural plasticity, makes brain development susceptible to environmental input[37,42,43], suggesting that the social environment could have a dominant role in determining the outcome — the fully formed adult brain.

Early-life social care determines social behaviour in adulthood. It is well-established that in social animals, social interactions are critical to development[44]. Compelling empirical evidence shows that provision of early life care shapes brain anatomy[45–48], function and development[49–53] (for a review see ref. [54] and for a list of evidence see Supplementary Table 1). In addition to brain development, early social care also determines the behavioural phenotype of the offspring. The development of social–behavioural repertoire in adulthood depends on early life social experience[55]. Even 'typical' social behaviours, such as maternal behaviours, sexual behaviour and peer interaction[56], would not develop without maternal care[57]. However, not only the complete deprivation of social care shifts the trajectory of social development. Individual differences in maternal care have the power to shape social development in mammal offspring[58]. For example, rats demonstrate individual differences in maternal behaviour, as some dams are spontaneously more maternal than others[58]. Female rats that provide high levels of maternal behaviours towards their pups received high levels of maternal care[59]. This intuitive trajectory of cross-generation transmission of maternal behaviours could rely on a genetic mechanism, that is, the dam could have genetically inherited a 'high social genetic profile' from her parents. Alternatively (or additionally), the mechanism could be acquired, as the dam could have been 'programmed' to interact with her pups during her own rearing. Cross-fostering studies confirm the role of postnatal experience in mediating this transmission. Females born to dams that provide low levels of maternal behaviour and are fostered by dams that provide high levels of maternal behaviour will perform high levels of maternal behaviour towards their own pups[60,61].

Human studies have also demonstrated that variation in maternal behaviour impacts children's social development[62]. Children of mothers with postpartum depression, a condition that impairs

maternal behaviour[63], have altered social development (including long-term susceptibility to social problems such as separation anxiety and social withdrawal[64]). On the contrary, children who receive optimal maternal care showed improved social development along with optimal physiological organization across childhood and adolescence[44,62]. A recent longitudinal study in humans showed that individuals who experienced sensitive, responsive and supportive caregiving exhibited improved bio-behavioural regulation with their adult romantic partners, 37 years later[65]. This study makes two important points. First, even small fluctuations in early care are powerful enough to cause variation in social behaviour in adulthood. Second, the parent–infant dyad is not only responsible for the cross-generation transmission of parental behaviours, but also plays a general role in moulding social behaviours at large.

To summarize the first section, allostasis regulation is a rewarding process[12], and as such can potentially motivate learning and development. In social animals, social regulation of allostasis is proposed here to motivate social learning and the maturation of associative neural networks, which are reportedly involved in social functioning in adulthood. We hypothesize that infants will show facilitated network development when their allostatic needs are sensitively regulated. This hypothesis is supported by literature demonstrating that child development is optimized and even accelerated where provision of parental care is sensitively attuned to the infant needs[66]. In the next section, we outline how social care in early life can potentially support social development, via the acquisition of concepts.

An alternative framework for social development

According to our framework, infants are not born with 'core social knowledge'[67,68], but rather need to learn about social agents and social behaviours. As a result, a prerequisite of social development is the acquisition of rudimentary concepts, which start as multimodal representations (such as a face) and become more abstract with development. We will describe our proposed course of development from birth onwards, starting with the development of a conceptual system, and then describe how such a conceptual system promotes social development.

Neural prediction as a potential mechanism for how experience sculpts the developing brain. An increasingly popular hypothesis in neuroscience is that the brain runs internal models that function as Bayesian filters for incoming sensory input, driving action and constructing perception and other psychological phenomena[15,69,70]. This hypothesis is often called predictive coding[70–75]. Prediction signals (also known as 'top down') are embodied, whole-brain representations that continuously anticipate (1) populations of upcoming sensory events from inside and outside the body and (2) populations of best action to deal with those events. Unanticipated information is a prediction error signal that tracks the difference between the prediction and the actual incoming input from the world and the body (also known as 'bottom up' signal). Predictions are generated in agranular association cortices and propagate to primary sensory cortices, always preparing for the next moment[74].

A key feature of the predictive coding model is the interaction between the forward and backward flow of information: the backward flow delivers predictions while the forward flow computes the residual errors between prediction and sensory inputs[74,76]. In early life as infants' sensory pathways become intact (infants' sensation starts in utero and continues after birth), without sufficient sensory experience to form valid predictive models, most sensory input is considered 'prediction error', simply because the brain cannot predict it. This idea was conceptualized by Alison Gopnik as 'lantern consciousness', or how babies take in everything around them[77]. With experience, infants start to detect and predict sensory patterns based on co-occurrence probability. This is called 'statistical learning'[78–80]. Detecting structure within the environment is a critical step in development[81] as from a meaningless stream of unpredicted sensory information, populations of instances are grouped together and mentally represented as concepts[82]. The infant's experience shifts from 'lantern consciousness' to 'spotlight consciousness', or intentional selection of perceptual input[77].

Of special importance for the development of sociality is neural prediction within the interoceptive system, which is the sensory consequence of allostasis. Importantly, the term allostasis was originally defined in terms of prediction[5]. Allostasis, as defined by Sterling and Laughlin[83], is "the core task of all brains to regulate the organism's internal milieu by anticipating needs and preparing to satisfy them before they arise"[83]. The predictive nature of allostasis is one way in which allostasis differs from the concept of homeostasis. Others have marked the importance of predictions in allostasis[84–87], and interoceptive predictions about allostasis have been suggested to underlie decision-making and motivational behaviour[85]. We propose that in newborns, interoceptive information about allostasis is regularly associated with exteroceptive information about caregivers. This conditioning prompts the infant's brain to regulate the internal milieu by attending to social information. Deviations from physiological balance (for example, low glucose levels) elicit distress behaviour (for example, cry), which (ideally) elicits social care and dramatically raises the chances of survival[88,89]. Thus, according to our theory, for infants raised in social dyads, interoceptive perception of allostasis is temporally associated with exteroceptive perception of the caregiver (see Box 1 and Fig. 2). With experience, infants learn to predict about allostasis given social information. A recent comparative study supports the importance of maternal care predictability by demonstrating that when infants (humans and rats) can predict maternal sensory input, they develop optimally[90]. This suggests that efficient Bayesian models about maternal sensory input (computed by an infant's brain) promote optimal bonding and development. Accordingly, developmental settings that obstruct the conditioning between the caretaker and allostasis, such as prematurity, postpartum or developmental psychopathology, or orphanhood, are predicted to interfere with social development. We hypothesize that with development, as top-down predictive models gradually govern infants' experience, infants' allostasis and allostatic independence will exponentially increase. Computationally, this process might involve a gradual decrease in the salience of interoceptive prediction errors. Empirical evaluation of the relationship between the predictive coding approach and allostasis during development is warranted.

Forming Bayesian models about the social (and non-social) environment depends on temporal and multi-modal contingencies in the infant brain. Multi-modal sensory inputs (exteroceptive and interoceptive) are integrated in agranular association cortices, which rely on those learned contingencies to generate Bayesian models and issue predictions[74]. The agranular association cortices, which integrate information from across the brain, become predictive 'hubs'[19] (Fig. 2). Multi-modal integration is not evident in newborns[91]. Moreover, resting-state functional magnetic resonance imaging reveals that core networks and predictive hubs are also not traceable in newborns (Fig. 1). This supports our hypothesis that the development of prediction infrastructure relies on postpartum experience that potentially determines the actual multimodal associations.

In the absence of multi-modal association or predictive models, we hypothesize that infants' experience mostly includes bottom-up information, or prediction errors. Sensory information is mostly processed in primary sensory cortices[91]. This idea is supported by empirical data on brain development. Thalamo-cortical connections, which deliver bottom-up sensory information to the primary sensory cortex (for example, prediction error), are the first to develop in the embryo[31], while the cortico-cortical connections that

Box 1 | A predictive coding approach to social development in which infants learn social concepts as generative models for allostatic regulation

According to models of predictive brain function[70–75], the brain constantly constructs multi-modal[207] hypotheses about the world (that is, predictions) and then samples evidence from the environment that are measured against these hypotheses (that is, prediction errors)[15,69,70].

A common mathematical formulation of these dynamics involves Bayesian inference, where the brain's hypotheses regarding the environment can be generally described by:

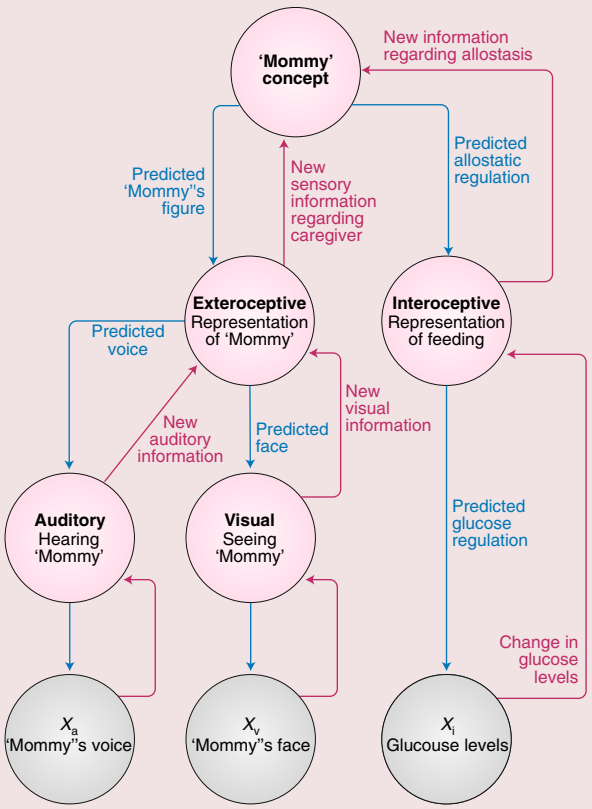
$$\mu_1 = \mu_0 + (x - \mu_0) \times \tau \quad (1)$$

where μ_0 denotes the brain's prior prediction, x denotes the sensory signal, such that the brackets represent the prediction error, and μ_1 denotes the brain's updated hypothesis. τ denotes the weight of the prediction errors, often called precision, which is a function of the ratio of the uncertainty of the prediction, and the uncertainty and saliency of the prediction error[74,207]. Thus, prediction errors are given a lesser weight when the predictions are relatively certain.

In the allostasis framework, the ultimate goal of the brain's predictive models is to optimize physiological demands and gain[74]. When a caregiver impacts the infant's allostasis, she/he effectively minimizes interoceptive prediction errors. With experience, the infant learns that exteroceptive cues regarding the caretaker (for example, smell, voice) predict allostatic regulation. Consistent care thus creates relatively certain predictions regarding allostatic regulation that reduce the precision of interoceptive prediction errors. Then, exteroceptive cues regarding the caretaker are given a higher importance than cues not impacting allostasis. At higher levels, this multimodal information is integrated in the infant's mind in the form of a concept (see Fig. 3), which can be formalized by:

$$P(\text{'Mommy'}|\text{Interoception, Exteroception}) \propto P(\text{'Mommy'}) \times P(\text{Interoception}|\text{'Mommy'}) \times P(\text{Exteroception}|\text{'Mommy'}) \quad (2)$$

Corresponding with Bayesian models of the brain, equation (2) shows that the infant's concept of 'mommy' is conditional on interoceptive and exteroceptive information. This concept will get more robust and reliable (that is, with lower uncertainty) as the caretaker's behaviour affecting the infant's interoceptive and exteroceptive sensations involves statistical regularity and consistency. Learning occurs as the concept of a 'mommy' given these inputs, [P('Mommy'|Interoception, Exteroception)], becomes the exteroceptive and interoceptive multi-modal prediction regarding the caregiver, [P('Mommy')]. When caretakers are not successful in social regulation of allostasis (for example, due to potential parent-related illness such as postpartum depression), statistical regularity between the exteroceptive input about the caregiver and the interoceptive input about allostasis is impaired. According to the framework suggested here, we predict that this will cause a developmental impairment in acquiring social concepts.



A schematic Bayesian model of organizing social information in infants. Infants associate exteroceptive and interoceptive information to form social concepts, such as 'mommy'. The social concept of 'mommy' represents a computational predictive model. Based on previous experience, an association between exteroceptive information about the caregiver and interoceptive information about allostasis is made, and the brain can issue predictions (downward blue arrows) regarding upcoming allostasis changes using social information and vice versa (to make social predictions based on interceptive information). For example, by the age of a few days old, infants have already gained repeated experience in feeding, and learned that exteroceptive information about 'mommy' is useful to predict about upcoming changes in their plasma glucose levels (for more detail, see Fig. 2). Upward red arrows represent prediction errors used to update the brain's model and predictions. For simplicity, we present here only two examples of exteroceptive modalities and one example of interoceptive input, whereas a full model incorporates a range of interoceptive and exteroceptive modalities. We propose that the brain acquires and uses such concepts (social and others) to optimize information processing, with the ongoing ultimate goal of allostasis regulation.

deliver top-down predictions form after birth[31,92], and in social animals are subjected to social impacts. Intriguingly, thalamo-cortical functional connections in one year olds, particularly those between the thalamus and salience network regions, show unique predictive values for cognitive development outcomes at two years of age[93,94]. Cortico-cortical connections are shaped by experience[95], and organize throughout childhood with significant dendritic growth and synaptogenesis around critical periods of cognitive development[92]. Multi-modal integration is not genetically predetermined, and its emergence and maturation critically depend on cross-modal experiences that shape the neural circuits in such a way that is optimized for the immediate environment in which the animal will function[91].

Notably, agranular association cortices, which issue most neural predictions[74], such as the anterior insula, anterior cingulate cortex and ventro-medial prefrontal cortex[74], are commonly considered major nodes of the 'social brain', and have been linked to social

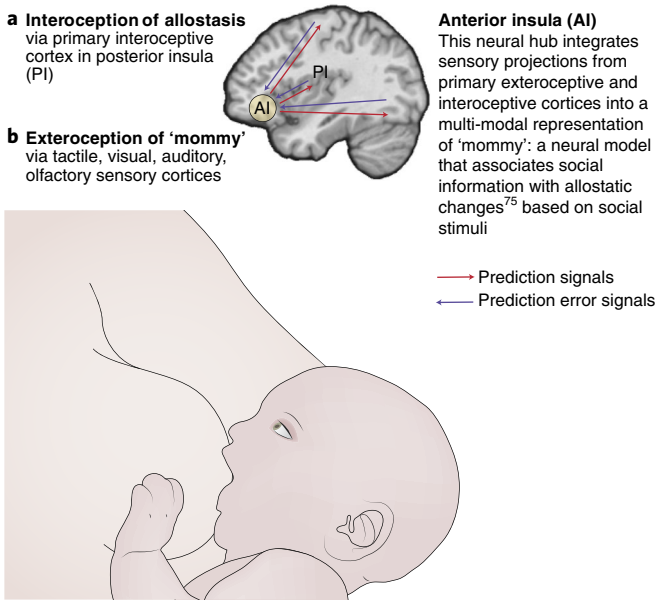


Fig. 2 | The anterior insula is a hub that integrates exteroceptive to interoceptive inputs. The anterior insula is a part of a multi-modal integration network, which overlaps with the salience network[109] (also including the dorsal anterior cingulate cortex and dorsal operculum[204]). **a**, Interoception: ascending sensory signals from the internal milieu of the body are carried to primary interoceptive cortex in the posterior insula[14,74]. The association cortex in the ventral anterior insula has bidirectional connections to primary interoceptive cortex in the posterior insula, which enable a bidirectional flow of information between the two regions: the ventral anterior insula sends 'top down' anticipated interoceptive prediction signals to the primary interoceptive cortex in the posterior insula, while 'bottom up' ascending sensory inputs from the body goes from the posterior insula to the ventral anterior insula[74]. **b**, Exteroception: the anterior insula is also involved in integration of sensory processing of the outside world[205], and in perception of all sensory modalities[206]. The involvement of this associative cortical region in both interoception and exteroception[85,205,206] can mark its role in integration of allostatic and social information. We hypothesize that with repeated care, infants learn to associate interoceptive input about allostasis and exteroceptive input about the caretaker into one multi-modal experience. One example of social regulation of allostasis is feeding. The feeding of a human newborn (like all mammals) inherently involves a caretaker. With every feeding, the caretaker regulates the infant's glucose levels. The newborn's experience of feeding conditionally comprises interoceptive digestive information (for example, glucose levels) and exteroceptive information about the caretaker (for example, caretaker's face, smell). There is a temporal conditioning between a caretaker and allostasis, which will be processed by the infant as one multi-modal experience. We hypothesize that based on the temporal conditioning between social and allostatic information, infants imbue social information into allostatic processes, and the association cortex that integrates the multi-modal input learns to predict about allostasis, based on social input. To prepare for the next feeding, the presence of the caretaker will already trigger the infant's anterior insula to issue predictions about upcoming changes in glucose levels. The predictions will propagate to the posterior insula, and other brain regions that assist to prepare for the upcoming allostatic changes, and achieve stability through physiological (for example, insulin release) or behavioural (for example, crying, sucking, salivating) changes.

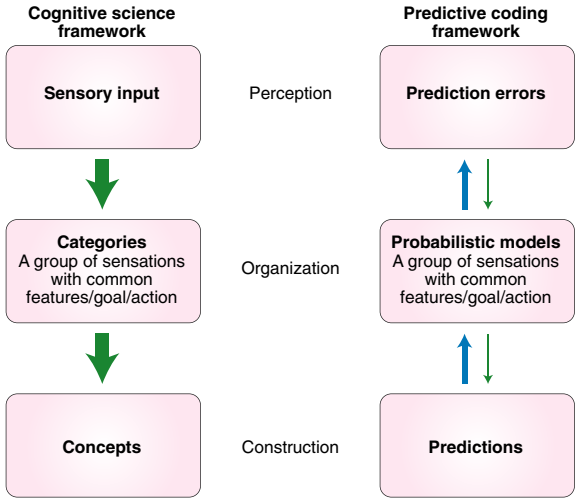


Fig. 3 | Concepts as predictions. Similar ideas about information processing are described using different terminology in the predictive coding and cognitive science frameworks. At the level of perception, actual incoming sensory input from the world and the body (also known as bottom-up signal in cognitive science) is considered in the predictive coding framework as prediction error. At the next level of processing, information is organized into 'categories' (cognitive science), or 'probabilistic models' (predictive coding). At the higher level of processing, the mental representations of categories are 'concepts'[105]. Just like concepts, predictions are also whole-brain representations that organize new incoming sensory input[75]. Importantly, in the predictive coding framework, the organization of sensory input is a predominately top-down process that starts with a probabilistic model. The model incorporates new sensory data only when it is not predicted (that is, prediction error), which in turn updates the model[74].

competencies such as mental inference, empathy and person perception[13,96–98]. The same brain regions, which are cortical rich club hubs of the salience and default mode networks, are also suggested to regulate the autonomic nervous system, the immune system and the neuroendocrine system, as part of a predictive allostasis regulation neural system[14,15,74,75,85,99]. Moreover, the amygdala, nucleus accumbens and hypothalamus, which are also considered key regions in social processing[100], have a key role in allostasis regulation[99] and are thought to compute prediction error and motivate behaviour[101]. We hypothesize that these regions' involvement in social processing reflects an underlying process that occurs simultaneously: preparing the organism for upcoming changes in allostasis.

In support of our hypothesis, the association hubs that are involved in social processing are not exclusive to social processing. While social information is very useful for allostasis prediction, other types of 'non-social' information (for example, food) can also be useful to predict allostasis. This would explain the consistent involvement of these association cortices and limbic regions in general affective experiences not necessarily related to social experience (for a meta-analysis see ref. [102]). According to our framework, consistent provision of social care can impact neural plasticity and promote neural associations between these regions into 'core' large-scale networks that implement an acquired system for the purposes of allostasis. Thus, the 'social brain' is really the predictive brain, which develops as a function of social experience aimed at allostasis regulation.

Neural prediction supports the development of a conceptual system. When a brain is 'processing information' to construct perceptions and plan action, it is asking 'What is this new sensory input most similar to?' relative to situated, past experiences (see refs [103,104]). The dis/similarity of the current sensory array is computed with reference to the past to estimate the potential energy costs and rewards

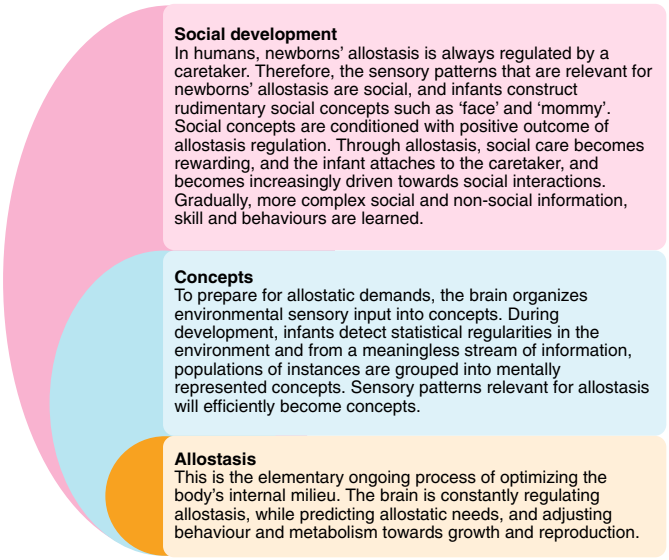


Fig. 4 | Relationship between allostasis, concepts and social development. Allostasis is an elementary process needed for survival and is thus the first order of the diagram. Concepts: to prepare for allostatic demands, the brain uses past experience as a model, and organizes incoming sensory input into mentally represented concepts. Organization of sensory input is governed by allostasis, and underlies social development, and is thus the second order of this diagram. Social development: in social species, allostasis is (by definition) regulated in a social environment. Consequently, social animals first and foremost learn social concepts. Allostasis-driven learning is rewarding, and that promotes social bonding. Social development relies on allostasis-driven concepts and is thus the higher order of this diagram.

for the body. That is, a prediction is considered a partially completed category that is used to classify incoming sensory signals to construct sensory perceptions. Similarly, in cognitive science, a category is a population of events or objects in the world that are treated as similar because they all serve a particular function or goal in some context. The mental representations of categories are concepts[105]. In effect, then, it has been proposed that when the brain assembles populations of predictions, it is constructing concepts[15], or what L. Barsalou refers to as 'ad hoc' concepts[106,107] (Fig. 3). It was previously established that prediction errors prompt learning because they modify the future predictions to incorporate the new information[76,108]. A concept, then, represents a group of sensory predictions (Fig. 3), and concept learning is the encoding of sensory prediction errors[109]. In this view, every event of new learning (for example, the processing of prediction error) is categorized into a concept.

Allostasis-driven learning of social concepts. According to our framework, the first step in social development is acquisition of rudimentary social concepts. At early infancy, the infant gains experience interacting with the caretaker, and most interactions will be implicitly or explicitly aimed towards allostasis regulation. With consistent ongoing care, infants detect the statistical regularities of their social environment[80]. For example, by repeated gazing at the mother, infants will gradually recognize the spatial organization of her facial features and form a rudimentary concept of a face. The brain categorizes sensory information to predict about allostasis, and thus we propose that sensory regularities (such as a face) will become concepts more rapidly if they impact allostasis. The association between allostasis and a human agent will result in learning an important social concept: 'mommy' (similarly, this concept can be 'daddy' or any other caretaker and will be referred to as the 'mommy' concept regardless of the primary caretaker gender/s or familial relation; Fig. 4 and Box 1).

Allostasis-driven learning of social competencies. Through social regulation of allostasis, a child experientially acquires not only social concepts (like a face or 'mommy'), but also social competencies. One of the basic social competencies infants gain is synchrony. Bio-behavioural synchrony is an important aspect of mother–infant attachment[44], and has been shown to be important for shaping optimal developmental outcomes of physiological regulation, executive functions and social aptitude[110].

Here, we consider synchrony as one efficient strategy for social regulation of allostasis. Starting from gestation, a mother controls her foetus's allostasis via mother–foetus physiological synchronization[7]. After birth, mammalian mothers continue to regulate the infants' allostasis[111,112] using the same strategy. Mothers regulate their infants' temperature by holding them close so that their temperatures synchronize[111]. Mothers regulate their infants' immune function by breastfeeding, synchronizing their gut microbiota and antigen-specific antibodies[113]. Mothers regulate infants' arousal with voice (by singing, or speaking loudly or softly)[114], synchronizing their heart rates[10]. Within a healthy dyad, the infant quickly gains a lot of experience in synchrony, and will progressively learn to willingly synchronize. Learning synchrony is one of the first social competencies that infants acquire, as within several months, infants not only learn to intentionally synchronize with others to regulate their own allostasis, but also start using synchrony to intentionally impact others' allostasis.

Among synchrony, another fundamental social competency infants acquire is joint attention[115]. Infants learn conceptual knowledge by synchronizing their attention with others. Attention is defined as a neural computation that biases certain features out of competing environmental information[116]. Attention is a learned cognitive skill that is plastic and shaped by experience[116]. Once attention develops, and the infant learns to synchronize, they learn the mental ability of sharing their attention with a caretaker at around six months[116]. Using joint attention, caretakers direct the infant's statistical learning towards relevant cultural and social information. Joint attention is a precursor for additional social competencies, like 'theory of mind'[117]. However, joint attention is a social competency that is fundamental not only for the development of social cognition, but also to many aspects of cognitive development[118], as within the medium of joint attention with caregivers and peers, infants are introduced to all knowledge and proficiency needed to survive in their environment. Mothers explicitly and implicitly teach infants new concepts as they influence the spontaneous statistical learning by providing regulatory social input, such as vocalizations[119], gaze[120] and touch[121].

Learning is an inherent aspect of allostasis, as the term 'allostasis' explicitly incorporates learning and predictive responding[5,74,122]. It was previously demonstrated that attaining physiological stability reinforces learning[12], and that physiological manipulation (for example, deviation from allostasis, for example, hunger) motivates learning[123,124] and prosocial behaviours in social animals[125,126]. Sensory input with allostatic implications (that is, likely to impact survival, offering reward or threat) will be learned, to support allostasis better in the future[14,127]. Accordingly, we hypothesize that stimuli with higher predictive value for allostasis will be learned quicker than stimuli with lower impact on allostasis. During development, infants learn social concepts and skills to prepare for allostatic needs, as caretakers introduce all the culturally relevant concepts, using language (Fig. 4).

Allostasis-driven development of abstraction. Allostasis-driven learning marks a special case for humans, compared with

non-human animals. All animals depend on allostasis and engage in allostasis-driven learning (including social learning). However, humans, on average, have a brain that is three times larger than a chimpanzee brain[128,129]. While humans and chimpanzees have comparable sensory and motor networks, in humans these networks are connected to an expanded core brain system of association cortices[130], which could imply an advanced capacity for multi-modal integration. These have been suggested to have evolved to sustain the relatively complex demands of the human social niche[131]. The advanced capacity for integration could underlie human ability for abstraction. Among non-human mammals, allostasis-driven learning is limited to sensory concepts with immediate physical impact on allostasis (such as food or pain). Humans can construct high-level abstract concepts (for example money, love, pride, god and other cultural concepts) and link them to allostasis (for example, have bodily representations of abstract ideas). Among 'socially entrained' humans, a word or an idea could be sufficient to regulate or disturb allostatic balance. The link between abstract entities such as words for physical regulation of allostasis could underlie language acquisition in humans[132]. It has been previously suggested that only as children develop social competencies, they can learn to understand the meaning of abstract concepts[133]. We hypothesize that social bonding promotes abstraction because social dyads provide the medium in which abstract concepts become meaningful by physically linking them to allostasis. Ad hoc investigations about allostasis regulation and social learning and abstraction in human infants are warranted.

According to our framework, during a critical developmental window, not only is spoken language acquired, but also culture. It has been demonstrated in comparative studies that longer developmental courses favour extended social learning[134–137]. As a result of human ontogeny, a human newborn's brain is highly immature, and thus fellow humans contribute to infant allostasis and brain development for an extended period during development[131]. This allows social input and culture to wire the brain in a more extensive way in humans than in any other animal. Individuals tend to conform to certain cultural schemes and synchronize according to a collective set of concepts, including norms, beliefs and manners[138]. The caretakers, and other members of the group, are responsible for cross-generation transmission of such relevant cultural knowledge. As part of the cultural scheme, and reinforced by allostasis, mentally rich human cognition is acquired. In addition to social learning, allostasis-driven learning can also shape other human features such as cognition, emotion and culture[15,139]. Empirical evidence suggests that mental and cognitive capacities (such as emotion, sociality and cognition) are not universal[140,141] and deep cultural differences exist between groups. According to our proposed framework, emotion and social concepts are environmentally constructed in each culture, and transferred between generations in social dyads during early life social 'training'. A smooth roll out of such a mechanism can ensure efficient cross-generation transmission of the deepest aspects of human emotion, cognition and sociality, while staying flexible and robust in face of change.

Social, cognitive and neural developmental trajectories

Despite evidence that social experience affects both neural and cognitive development, the structure–function developmental mechanism remains poorly understood. Characterizing the neural mechanisms of cognitive development is a pending empirical task. However, several neural developmental milestones temporally match the development of new cognitive skills. Of specific interest to social cognition is the temporal contingency between the developmental trajectories of the default mode network and of cognitive abilities such as conceptualization[13]. The default mode network is believed to construct mental representations of concepts[142], including complex representations about other people's minds (for example, theory of mind)[143]. The adult default mode network cannot be functionally traced in newborns[29,33]. The default mode network starts from an isolated posterior cingulate region that can be traced in neonates, and evolves to become a synchronized network later in life[29]. More precisely, most of the core nodes of the default mode network become synchronized by six months of age, making the default mode network among the first domain-general networks to achieve qualitatively adult-like spatial topology[144]. It has been demonstrated that the connectivity and volume of the major default mode network nodes (the posterior cingulate cortex and medial prefrontal cortex) are immature at birth[145]. The grey matter volumes as well as functional and structural connectivity in the default mode network continue to develop during childhood, reaching full maturity in late adolescence[33,146], right when social cognition abilities and mentalizing mature[146,147]. Intuitively, it has been postulated before that changes in brain connectivity might be linked to cognitive development[93,148,149], and that the absence of a default mode network in early infancy indicates the absence of cognitive skills such as conceptualization and theory of mind[33]. Accordingly, default-mode network maturation is potentially a crucial developmental step, which serves as the precursor for social and cognitive development. Importantly, network plasticity seen during development is not sufficient to conclude the lack of an inborn social system. A circuit or network can be innate and emerge only with experience. Alternatively, it is possible that a capacity, such as sociality, can be a byproduct of something else that is innate, such as allostasis. The degree of plasticity in brain network connectivity during the early years of life, combined with the importance of allostasis for reinforcement learning, together suggest that researchers should reconsider the common assumption that a network or system for sociality is inborn. Empirical investigation of mechanisms facilitating the impact of dyadic care on brain and social development is warranted.

The literature on social development in rodents provides robust support for social care having a mechanistic role in brain development, and specifies hard mechanistic evidence for how parental care physically controls brain development in the offspring (including receptor expression, plasticity and cortical folding, see Supplementary Table 1). By integrating cognitive and neural developmental literatures, we hypothesize that social care controls social and cognitive development via maturation of whole-brain neural networks. Specifically, we propose that parental care, which is consistently reinforced by allostasis, is necessary for the infant to build and refine a multisensory mental representation of concepts[82,133]. Starting from first simple multisensory concepts, such as a face, and then 'mommy', infants will gradually learn to represent more abstract concepts, including words and ideas, by linking them to allostasis via dyadic interaction. The developmental neural shift from primary motor-sensory circuitry seen in newborns to association networks in adults suggests a potential dramatic shift in the development of human experience: from undefined raw sensory experience in early infancy to constructed cognition in adulthood.

Growing a social brain is adaptive and promotes affiliation

In this Perspective, we present a theoretical framework for social affiliation. Specifically, social affiliation is learned and allostasis is the incentive. Social attachment could be the result of one evolutionary feature: helplessness in achieving and maintaining physiological stability. Such social codependency creates an ultimate driving force for attachment and learning. The mother–infant dyad is a social 'boot camp' for learning social affiliation, which is extremely efficient as infants' lives depend on their caregiver. While caretakers teach newborns the behavioural repertoire of sociality (including bonding, but also more complex social behaviours such as cooperation, competition, aggression and so on), they also programme their biology, helping them to 'grow a social brain'. Infants will learn to identify humans as important, and to synchronize with

them. They continue to learn through synchronizing conceptual knowledge and mental abilities with their caregivers. The caregiver explicitly and implicitly teaches the newborn a saliency road map of the world. Gradually, infants are trained not only to regulate their own allostasis, but also to become experts in decoding and attending to other people's allostasis, as they become socially functioning adults. However, not only social development relies on social regulation of allostasis, but also other cognitive and emotional developmental trajectories. During childhood and within social dyads and other members of their social group, such as family members and friends, infants are trained to acquire all skill and knowledge needed as adults. Social care is therefore not merely responsible for shaping a 'social brain'. We propose that social care is needed to grow a brain. Consequently, the entire brain, which is potentially sculpted by ongoing social transactions, can be thought of as a social brain. Human brains are transactive and cannot be considered outside the context of other human brains. Transactive brains form a collective and flexible system that sustains many of our human features, including knowledge[150], skill and biology.

Early life is a critical window for social learning due to the acute nature of the social dependency. As long as children's lives depend on social communication, their brains attend to learn the complex social information to survive[151,152]. As such, the social dyad catalyses social-related statistical learning. With maturation, the 'life or death' motivation for social learning gradually subsides, and the window of immense learning narrows. With that, social regulation of allostasis is not limited to early life and there is evidence from rodents and apes (including humans) for a social buffering effect (a regulatory social impact on an individual's physiology and behaviour) on different types of allostatic processes, including hypothalamic pituitary adrenal axis activity, oxytocin levels, affect and immune function (for a review see ref. [153]). In highly social species such as humans, adults regulate each other's allostasis as well[154,155]. As such, socially mediated learning is a life-long process[156,157].

The potentially crucial role of social experience during infancy in shaping brain and social development suggests that social animals do not necessarily rely on a predetermined specialized brain system to support affiliation[158,159]. Instead, according to our framework, domain-general neural systems implement a conceptual system to regulate allostasis, and that underlies social behaviour. Moreover, as cultural changes evolve much faster than natural selection does, evolution is more likely to select for flexible biological systems that are robust to unexpected environmental change[160]. A transactive human brain growing in the context of other human brains and genetically predisposed to wire itself to the environment[161] is just such a system. Thinking about bonding and social development as temporal conditioning between social information and allostasis is a hypothesis. This hypothesis is based on the integration of literature on allostasis and bonding, but also on learning. For example, a recent review about social learning in non-human animal models examined experimental work in animal models of learning, and concluded that domain-general associative mechanisms that track the predictive relationships and contingency between two salient events mediate learning about both social and asocial cues. Moreover, the authors concluded that while associative learning mechanisms are genetically inherited, at least in non-human animals there are no mechanisms dedicated to social versus asocial learning[162]. This supports our idea that any stimulus that conditionally impacts allostasis (for example, salient) will be learned, and that in social species, these stimuli are prominently social.

This Perspective introduces a theoretical framework about how the developing brain respects the physical and social surroundings as wiring instructions, along with a series of hypotheses about the early allostatic experiences with caregivers as one source of these wiring instructions. Our framework sets the ground for new research and ad hoc empirical evaluation targeted at allostasis, and its mechanistic role in the development of social affiliation. It also provides a different interpretation to existing findings and not everyone will agree. In particular, disentangling environmental from genetic effects is challenging, and further empirical investigations on the role of social care in child brain and behaviour development are warranted. While we predict that social experience is a major determinant in social development, genetic predisposition is not overlooked. We distinguish between genetic predisposition of a socially pre-wired brain (limited to domain-general processes, such as allostasis dependency), versus genetic determinism of a hard-wired brain (coding for detailed social behaviours or neural circuits). For example, the cross-fostering studies from the Meaney group[49,60,61,163,164] as well as the Lorenz imprinting studies[165,166] suggest that social behaviour does not depend on hardwired social 'programmes', and that particular phenotypes are flexible. While some studies that have suggested that social knowledge is hardwired in our genes and that newborns have innate social preference and behaviour[167,168], others have resisted this idea and provided evidence that there is no social preference in newborns[169–175]. It is possible that newborns' behaviours that are typically interpreted as 'social' are in fact 'allostatic' at first and only become social once infants learn to associate social information with allostasis. Accordingly, innate individual differences that are seen in infants relate to allostasis[176,177], and the developmental literature applies the term temperament[178] to describe traits such as 'irritability', 'positive affect' and 'activity level'[179]. This supports the idea that infant's genetic predisposition is limited to domain-general processes of allostasis, which interact with social experience to determine social development. Synthesizing human and non-human evidence about allostasis, bonding, learning and brain function establishes considerable doubt in the common view (attributing a dominant genetic account to social phenotype) and encourages research to test the alternative hypothesis suggested here (by which the social phenotype is shaped by social experience, driven by the genetic predisposition of allostasis dependency).

The framework suggested here could have implications for research in the field of neuroscience because it assumes that neural circuits that support sociality are not inborn. For example, across evolution, social affiliation depends on cortico-striatal (or anatomically homologous) pathways[151]. Accordingly, recent human functional magnetic resonance imaging studies have demonstrated that social affiliation depends on the neural association between domain-general circuits of the mesolimbic system and the default mode network[23]. The dopaminergic mesolimbic system has been shown to underlie allostatic processes[180–182], while the default mode network is important for conceptualization[183]. Thus, social affiliation relies on the connectivity within a multi-function neural system supporting allostasis and conceptualization.

Considering that the 'social brain' is not an isolated module, and that the entire brain is wired with respect to the social environment, could have implications for psychopathology. For example, current studies of ASD focus on boosting the 'social brain' with 'social drugs'[184], yet to date there are no successful pharmacological interventions aimed at the core social deficits of ASD[185]. Instead, it has been previously suggested that social impairments seen in ASD are attributed to domain-general processes, such as deficits in organization of sensory input into efficient predictive models[186]. Patients with ASD show deficits in categorizing information into concepts, manifested as atypical language acquisition or naming, which are accompanied by weaker connectivity of association networks[187]. Similarly, learning disorders, such as dyslexia, were suggested to be computationally understood as a deficit in integrating prior information (for example, forming efficient prediction models) with noisy observations (for example, prediction errors)[188]. In dyslexia patients, an atypical ratio between the a-prior model and the noisy input may account for patients' perceptual deficits[189]. A brain with

limited ability to use past knowledge for prediction is also limited in conceptualization, and thus social bonding. Empirical assessments for this potential mechanism are called for. Moreover, in cases of postpartum depression, where mothers are avoidant, or cases of postpartum anxiety, where mothers are intrusive[21], infants' allostasis is constantly disregulated. Distorted allostasis regulation can shift infants' developmental trajectories, including neural, social and cognitive development[190]. A clinical evaluation of infants' allostasis regulation within mother–infant dyads could support diagnosis and treatment for both mothers and infants, and help determine the developmental prognosis in maladaptive dyads.

Finally, acknowledging the environmental impact on social development has implications for societal issues such as race, family structure and religion. For example, treating 'mommy' as a learned concept, and not a biological imperative, makes a baby born to a family of two fathers not deprived of any natural necessity[191]. As a society, we construct many abstract concepts, which are powerful because they impact allostasis. Completely abstract ideas, such as god, race, money or love, materialize to become concrete (that is, have immediate allostatic implications), powerfully motivating human behaviour. This is because via social interactions humans learn to link those abstract concepts to their allostasis to survive and prosper in their culture. This can potentially explain how beyond the immediate dyadic bond with the caregiver, extended social effects, including social class or economic status, may carry powerful effects on child development[15,2,192], and even brain development[193]. Future research on the role of allostasis as part of a very complex control system of social behaviour, and on familial and extra-familial impacts on biology and behaviour of child development is warranted. Moreover, realizing the powerful potency of programming our children's brains and concepts can impact education, and raise the issue of social and cultural responsibility.

'Growing a social brain' is at the basis of every human's well-being. Early infancy is a critical time for establishing the biology of a healthy mind. However, the brain is plastic throughout life and so are many aspects of behaviour and cognition[194]. The human 'social brain' is not a pre-determined organ, but rather a plastic product of ongoing acculturation. As humans, taking responsibility on shaping our (social) brains can potentially impact science, societies and our children's education.

Received: 26 April 2017; Accepted: 19 June 2018;
Published online: 6 August 2018

References

1. Rand, D. G. & Nowak, M. A. Human cooperation. *Trends Cogn. Sci.* **17**, 413–425 (2013).
2. Johnson, Z. V. & Young, L. J. Neurobiological mechanisms of social attachment and pair bonding. *Curr. Opin. Behav. Sci.* **3**, 38–44 (2015).
3. Hawkes, K. Grandmothers and the evolution of human longevity. *Am. J. Hum. Biol.* **15**, 380–400 (2003).
4. Dunbar, R. I. & Shultz, S. Evolution in the social brain. *Science* **317**, 1344–1347 (2007).
5. Sterling, P. Allostasis: a model of predictive regulation. *Physiol. Behav.* **106**, 5–15 (2012).
6. Atzil, S. & Barrett, L. F. Social regulation of allostasis: Commentary on "Mentalizing homeostasis: the social origins of interoceptive inference" by Fotopoulou & Tsakiris. *Neuropsychoanalysis* **19**, 1–24 (2017).
7. Rao, P. N. S., Shashidhar, A. & Ashok, C. In utero fuel homeostasis: lessons for a clinician. *Indian J. Endocrinol. Metab.* **17**, 60–68 (2013).
8. Winberg, J. Mother and newborn baby: mutual regulation of physiology and behavior — a selective review. *Dev. Psychobiol.* **47**, 217–229 (2005).
9. Hofer, M. A. Hidden regulators in attachment, separation, and loss. *Monogr. Soc. Res. Child Dev.* **59**, 192–207 (1994).
10. Feldman, R., Magori-Cohen, R., Galili, G., Singer, M. & Louzoun, Y. Mother and infant coordinate heart rhythms through episodes of interaction synchrony. *Infant Behav. Dev.* **34**, 569–577 (2011).
11. Feldman, R., Eidelman, A. I., Sirota, L. & Weller, A. Comparison of skin-to-skin (kangaroo) and traditional care: parenting outcomes and preterm infant development. *Pediatrics* **110**, 16–26 (2002).
12. Keramati, M. & Gutkin, B. Homeostatic reinforcement learning for integrating reward collection and physiological stability. *eLife* **3**, e04811 (2014).
13. Barrett, L. F. & Satpute, A. B. Large-scale brain networks in affective and social neuroscience: towards an integrative functional architecture of the brain. *Curr. Opin. Neurobiol.* **23**, 361–372 (2013).
14. Kleckner, I. et al. Evidence for a large-scale brain system supporting allostasis and interoception in humans. *Nat. Hum. Behav.* **1**, 0069 (2017).
15. Barrett, L. F. *How Emotions are Made* (Houghton Mifflin Harcourt, Boston, MA, 2017).
16. Gao, W., Lin, W., Grewen, K. & Gilmore, J. H. Functional connectivity of the infant human brain plastic and modifiable. *Neuroscientist* **23**, 169–184 (2016).
17. Bullmore, E. & Sporns, O. The economy of brain network organization. *Nat. Rev. Neurosci.* **13**, 336–349 (2012).
18. Yeo, B. T. et al. The organization of the human cerebral cortex estimated by intrinsic functional connectivity. *J. Neurophysiol.* **106**, 1125–1165 (2011).
19. van den Heuvel, M. P. & Sporns, O. Rich-club organization of the human connectome. *J. Neurosci.* **31**, 15775–15786 (2011).
20. van den Heuvel, M. P. et al. Abnormal rich club organization and functional brain dynamics in schizophrenia. *JAMA Psychiatry* **70**, 783–792 (2013).
21. Atzil, S., Hendler, T. & Feldman, R. Specifying the neurobiological basis of human attachment: brain, hormones, and behavior in synchronous and intrusive mothers. *Neuropsychopharmacology* **36**, 2603–2615 (2011).
22. Atzil, S., Hendler, T. & Feldman, R. The brain basis of social synchrony. *Soc. Cogn. Affect. Neurosci.* **9**, 1193–1202 (2013).
23. Atzil, S. et al. Dopamine in the medial amygdala network mediates human bonding. *Proc. Natl Acad. Sci. USA* **114**, 2361–2366 (2017).
24. Bickart, K. C., Hollenbeck, M. C., Barrett, L. F. & Dickerson, B. C. Intrinsic amygdala-cortical functional connectivity predicts social network size in humans. *J. Neurosci.* **32**, 14729–14741 (2012).
25. Uddin, L. Q. et al. Salience network–based classification and prediction of symptom severity in children with autism. *JAMA Psychiatry* **70**, 869–879 (2013).
26. Di Martino, A. et al. The autism brain imaging data exchange: towards a large-scale evaluation of the intrinsic brain architecture in autism. *Mol. Psychiatry* **19**, 659–667 (2014).
27. Dubois, J. et al. The early development of brain white matter: a review of imaging studies in fetuses, newborns and infants. *Neuroscience* **276**, 48–71 (2014).
28. Gao, W. et al. Temporal and spatial evolution of brain network topology during the first two years of life. *PLoS ONE* **6**, e25278 (2011).
29. Gao, W. et al. The synchronization within and interaction between the default and dorsal attention networks in early infancy. *Cereb. Cortex* **23**, 594–603 (2013).
30. Gao, W. et al. Temporal and spatial development of axonal maturation and myelination of white matter in the developing brain. *Am. J. Neuroradiol.* **30**, 290–296 (2009).
31. Smyser, C. D., Snyder, A. Z. & Neil, J. J. Functional connectivity MRI in infants: exploration of the functional organization of the developing brain. *Neuroimage* **56**, 1437–1452 (2011).
32. Fransson, P., Aden, U., Blennow, M. & Lagercrantz, H. The functional architecture of the infant brain as revealed by resting-state fMRI. *Cereb. Cortex* **21**, 145–154 (2011).
33. Fransson, P. et al. Resting-state networks in the infant brain. *Proc. Natl Acad. Sci. USA* **104**, 15531–15536 (2007).
34. Elton, A., Alcauter, S. & Gao, W. Network connectivity abnormality profile supports a categorical-dimensional hybrid model of ADHD. *Hum. Brain Mapp.* **35**, 4531–4543 (2014).
35. Fair, D. A. et al. The maturing architecture of the brain's default network. *Proc. Natl Acad. Sci. USA* **105**, 4028–4032 (2008).
36. Tau, G. Z. & Peterson, B. S. Normal development of brain circuits. *Neuropsychopharmacology* **35**, 147–168 (2010).
37. Stiles, J. & Jernigan, T. L. The basics of brain development. *Neuropsychol. Rev.* **20**, 327–348 (2010).
38. Dubois, J. et al. Primary cortical folding in the human newborn: an early marker of later functional development. *Brain* **131**, 2028–2041 (2008).
39. Finlay, B. L. & Uchiyama, R. in *Evolution of Nervous Systems* 2nd edn (ed. Kaas, J. H.) 123–148 (Elsevier, Oxford, 2017).
40. Rogers, C. E. et al. Regional cerebral development at term relates to school-age social-emotional development in very preterm children. *J. Am. Acad. Child Adolesc. Psychiatry* **51**, 181–191 (2012).
41. Woodward, L. J., Clark, C. A., Bora, S. & Inder, T. E. Neonatal white matter abnormalities an important predictor of neurocognitive outcome for very preterm children. *PLoS ONE* **7**, e51879 (2012).
42. Curley, J. P. & Champagne, F. A. Influence of maternal care on the developing brain: mechanisms, temporal dynamics and sensitive periods. *Front. Neuroendocrinol.* **40**, 52–66 (2016).

43. Johnson, M. H. Functional brain development in humans. *Nat. Rev. Neurosci.* **2**, 475–483 (2001).
44. Feldman, R. Parent–infant synchrony and the construction of shared timing; physiological precursors, developmental outcomes, and risk conditions. *J. Child Psychol. Psychiatry* **48**, 329–354 (2007).
45. Tomoda, A. et al. Reduced prefrontal cortical gray matter volume in young adults exposed to harsh corporal punishment. *Neuroimage* **47**, T66–T71 (2009).
46. Whittle, S. et al. Positive parenting predicts the development of adolescent brain structure: a longitudinal study. *Dev. Cogn. Neurosci.* **8**, 7–17 (2014).
47. Teicher, M. H., Anderson, C. M. & Polcari, A. Childhood maltreatment is associated with reduced volume in the hippocampal subfields CA3, dentate gyrus, and subiculum. *Proc. Natl Acad. Sci. USA* **109**, E563–E572 (2012).
48. Luby, J. L. et al. Maternal support in early childhood predicts larger hippocampal volumes at school age. *Proc. Natl Acad. Sci. USA* **109**, 2854–2859 (2012).
49. Champagne, F. A. et al. Maternal care associated with methylation of the estrogen receptor-α1b promoter and estrogen receptor-α expression in the medial preoptic area of female offspring. *Endocrinology* **147**, 2909–2915 (2006).
50. McGowan, P. O. et al. Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. *Nat. Neurosci.* **12**, 342–348 (2009).
51. Pena, C. J., Neugut, Y. D., Calarco, C. A. & Champagne, F. A. Effects of maternal care on the development of midbrain dopamine pathways and reward-directed behavior in female offspring. *Eur. J. Neurosci.* **39**, 946–956 (2014).
52. Insel, T. R. Oxytocin — a neuropeptide for affiliation: evidence from behavioral, receptor autoradiographic, and comparative studies. *Psychoneuroendocrinology* **17**, 3–35 (1992).
53. Webb, A. R., Heller, H. T., Benson, C. B. & Lahav, A. Mother's voice and heartbeat sounds elicit auditory plasticity in the human brain before full gestation. *Proc. Natl Acad. Sci. USA* **112**, 3152–3157 (2015).
54. Teicher, M. H., Samson, J. A., Anderson, C. M. & Ohashi, K. The effects of childhood maltreatment on brain structure, function and connectivity. *Nat. Rev. Neurosci.* **17**, 652–666 (2016).
55. Suomi, S. J. Early determinants of behaviour: evidence from primate studies. *Br. Med. Bull.* **53**, 170–184 (1997).
56. Arling, G. L. & Harlow, H. F. Effects of social deprivation on maternal behavior of rhesus monkeys. *J. Comp. Physiol. Psychol.* **64**, 371–377 (1967).
57. Harlow, H. F. Total social isolation: effects on macaque monkey behavior. *Science* **148**, 666 (1965).
58. Champagne, F. A., Francis, D. D., Mar, A. & Meaney, M. J. Variations in maternal care in the rat as a mediating influence for the effects of environment on development. *Physiol. Behav.* **79**, 359–371 (2003).
59. Champagne, F. A. Epigenetic mechanisms and the transgenerational effects of maternal care. *Front. Neuroendocrinol.* **29**, 386–397 (2008).
60. Champagne, F. & Meaney, M. J. Like mother, like daughter: evidence for non-genomic transmission of parental behavior and stress responsivity. *Prog. Brain Res.* **133**, 287–302 (2001).
61. Pena, C. J., Neugut, Y. D. & Champagne, F. A. Developmental timing of the effects of maternal care on gene expression and epigenetic regulation of hormone receptor levels in female rats. *Endocrinology* **154**, 4340–4351 (2013).
62. Feldman, R. The adaptive human parental brain: implications for children's social development. *Trends Neurosci.* **38**, 387–399 (2015).
63. Granat, A., Gadassi, R., Gilboa-Schechtman, E. & Feldman, R. Maternal depression and anxiety, social synchrony, and infant regulation of negative and positive emotions. *Emotion* **17**, 11–27 (2016).
64. Herba, C. M. Maternal depression and child behavioural outcomes. *Lancet Psychiatry* **1**, 408–409 (2014).
65. Raby, K. L., Roisman, G. I., Simpson, J. A., Collins, W. A. & Steele, R. D. Greater maternal insensitivity in childhood predicts greater electrodermal reactivity during conflict discussions with romantic partners in adulthood. *Psychol. Sci.* **26**, 348–353 (2015).
66. Feldman, R. Parent–infant synchrony: biological foundations and developmental outcomes. *Curr. Dir. Psychol. Sci.* **16**, 340–345 (2007).
67. Carey, S. & Spelke, E. Science and core knowledge. *Philos. Sci.* **63**, 515–533 (1996).
68. Spelke, E. S. & Kinzler, K. D. Core knowledge. *Dev. Sci.* **10**, 89–96 (2007).
69. Clark, A. Whatever next? Predictive brains, situated agents, and the future of cognitive science. *Behav. Brain Sci.* **36**, 181–204 (2013).
70. Hohwy, J. *The Predictive Mind* (Oxford Univ. Press, Oxford, 2013).
71. Rao, R. P. & Ballard, D. H. Predictive coding in the visual cortex: a functional interpretation of some extra-classical receptive-field effects. *Nat. Neurosci.* **2**, 79–87 (1999).
72. Friston, K. The free-energy principle: a unified brain theory? *Nat. Rev. Neurosci.* **11**, 127–138 (2010).
73. Clark, A. Are we predictive engines? Perils, prospects, and the puzzle of the porous perceiver. *Behav. Brain Sci.* **36**, 233–253 (2013).
74. Barrett, L. F. & Simmons, W. K. Interoceptive predictions in the brain. *Nat. Rev. Neurosci.* **16**, 419–429 (2015).
75. Chanes, L. & Barrett, L. F. Redefining the role of limbic areas in cortical processing. *Trends Cogn. Sci.* **20**, 96–106 (2016).
76. Friston, K. A theory of cortical responses. *Philos. Trans. R. Soc. London Ser. B* **360**, 815–836 (2005).
77. Gopnik, A. *The Philosophical Baby* (Bodley Head, London, 2009).
78. Siegelman, N. & Frost, R. Statistical learning as an individual ability: theoretical perspectives and empirical evidence. *J. Mem. Lang.* **81**, 105–120 (2015).
79. Krogh, L., Vlach, H. A. & Johnson, S. P. Statistical learning across development: flexible yet constrained. *Front. Psychol.* **3**, 598 (2012).
80. Saffran, J. R., Aslin, R. N. & Newport, E. L. Statistical learning by 8-month-old infants. *Science* **274**, 1926–1928 (1996).
81. Kirkham, N. Z., Slemmer, J. A. & Johnson, S. P. Visual statistical learning in infancy: evidence for a domain general learning mechanism. *Cognition* **83**, B35–B42 (2002).
82. Tenenbaum, J. B., Kemp, C., Griffiths, T. L. & Goodman, N. D. How to grow a mind: statistics, structure, and abstraction. *Science* **331**, 1279–1285 (2011).
83. Sterling, P. & Laughlin, S. *Principles of Neural Design* (MIT Press, Cambridge, MA, 2015).
84. Carpenter, R. Homeostasis: a plea for a unified approach. *Adv. Physiol. Educ.* **28**, 180–187 (2004).
85. Gu, X. & FitzGerald, T. Interoceptive inference: homeostasis and decision-making. *Trends Cogn. Sci.* **18**, 269–270 (2014).
86. Seth, A. K. Interoceptive inference, emotion, and the embodied self. *Trends Cogn. Sci.* **17**, 565–573 (2013).
87. Seth, A. K., Suzuki, K. & Critchley, H. D. An interoceptive predictive coding model of conscious presence. *Front. Psychol.* **2**, 395 (2012).
88. Finlay, B. L. & Syal, S. The pain of altruism. *Trends Cogn. Sci.* **18**, 615–617 (2014).
89. Lummaa, V., Vuorisalo, T., Barr, R. G. & Lehtonen, L. Why cry? Adaptive significance of intensive crying in human infants. *Evol. Hum. Behav.* **19**, 193–202 (1998).
90. Davis, E. P. et al. Exposure to unpredictable maternal sensory signals influences cognitive development across species. *Proc. Natl Acad. Sci. USA* **114**, 10390–10395 (2017).
91. Stein, B. E., Stanford, T. R. & Rowland, B. A. Development of multisensory integration from the perspective of the individual neuron. *Nat. Rev. Neurosci.* **15**, 520–535 (2014).
92. Petanjek, Z., Judaš, M., Kostović, I. & Uylings, H. B. M. Lifespan alterations of basal dendritic trees of pyramidal neurons in the human prefrontal cortex: a layer-specific pattern. *Cereb. Cortex* **18**, 915–929 (2008).
93. Alcauter, S. et al. Development of thalamocortical connectivity during infancy and its cognitive correlations. *J. Neurosci.* **34**, 9067–9075 (2014).
94. Alcauter, S., Lin, W., Keith Smith, J., Gilmore, J. H. & Gao, W. Consistent anterior-posterior segregation of the insula during the first 2 years of life. *Cereb. Cortex* **25**, 1176–1187 (2015).
95. Trachtenberg, J. T. & Stryker, M. P. Rapid anatomical plasticity of horizontal connections in the developing visual cortex. *J. Neurosci.* **21**, 3476–3482 (2001).
96. Singer, T. & Lamm, C. The social neuroscience of empathy. *Ann. N. Y. Acad. Sci.* **1156**, 81–96 (2009).
97. Andrews-Hanna, J. R., Smallwood, J. & Spreng, R. N. The default network and self-generated thought: component processes, dynamic control, and clinical relevance. *Ann. N. Y. Acad. Sci.* **1316**, 29–52 (2014).
98. Shamay-Tsoory, S. G. The neural bases for empathy. *Neuroscientist* **17**, 18–24 (2011).
99. Ganzel, B. L., Morris, P. A. & Wethington, E. Allostasis and the human brain: integrating models of stress from the social and life sciences. *Psychol. Rev.* **117**, 134–174 (2010).
100. Bickart, K. C., Dickerson, B. C. & Barrett, L. F. The amygdala as a hub in brain networks that support social life. *Neuropsychologia* **63**, 235–248 (2014).
101. Schultz, W. & Dickinson, A. Neuronal coding of prediction errors. *Annu. Rev. Neurosci.* **23**, 473–500 (2000).
102. Lindquist, K. A., Satpute, A. B., Wager, T. D., Weber, J. & Barrett, L. F. The brain basis of positive and negative affect: evidence from a meta-analysis of the human neuroimaging literature. *Cereb. Cortex* **26**, 1910–1922 (2015).
103. Bar, M. The proactive brain: memory for predictions. *Philos. Trans. R. Soc. London Ser. B* **364**, 1235–1243 (2009).
104. Bar, M. Predictions: a universal principle in the operation of the human brain. Introduction. *Philos. Trans. R. Soc. London Ser. B* **364**, 1181–1182 (2009).
105. Murphy, G. *The Big Book of Concepts* (MIT Press, Cambridge, MA, 2004).
106. Barsalou, L. W. Ad hoc categories. *Mem. Cogn.* **11**, 211–227 (1983).

107. Barsalou, L. W., Kyle Simmons, W., Barbey, A. K. & Wilson, C. D. Grounding conceptual knowledge in modality-specific systems. *Trends Cogn. Sci.* **7**, 84–91 (2003).
108. Hollerman, J. R. & Schultz, W. Dopamine neurons report an error in the temporal prediction of reward during learning. *Nat. Neurosci.* **1**, 304–309 (1998).
109. Barrett, L. F. The theory of constructed emotion: an active inference account of interoception and categorization. *Soc. Cogn. Affect. Neurosci.* **12**, 1–23 (2017).
110. Feldman, R., Rosenthal, Z. & Eidelman, A. I. Maternal-preterm skin-to-skin contact enhances child physiologic organization and cognitive control across the first 10 years of life. *Biol. Psychiatry* **75**, 56–64 (2014).
111. Levin, B. E. Metabolic imprinting: critical impact of the perinatal environment on the regulation of energy homeostasis. *Philos. Trans. R. Soc. London Ser. B* **361**, 1107–1121 (2006).
112. Bauman, D. in *Ruminant Physiology: Digestion, Metabolism, Growth and Reproduction* (eds Dobson, A. & Dobson, M. J.) 238–256 (Comstock Publishing Associates, Ithaca, NY, 2000).
113. Arrieta, M. C., Stiemsma, L. T., Amenyogbe, N., Brown, E. M. & Finlay, B. The intestinal microbiome in early life: health and disease. *Front. Immunol.* **5**, 427 (2014).
114. Nakata, T. & Trehub, S. E. Infants' responsiveness to maternal speech and singing. *Infant Behav. Dev.* **27**, 455–464 (2004).
115. Tomasello, M. in *Joint Attention: Its Origins and Role in Development* (eds Moore, C. & Dunham, P.) 103–130 (Psychology Press, New York, NY, 1995).
116. Amso, D. & Scerif, G. The attentive brain: insights from developmental cognitive neuroscience. *Nat. Rev. Neurosci.* **16**, 606–619 (2015).
117. Baron-Cohen, S. The development of a theory of mind in autism: deviance and delay? *Psychiatry Clin. North Am.* **14**, 33–51 (1991).
118. Belmonte, M. K. et al. Autism as a disorder of neural information processing: directions for research and targets for therapy. *Mol. Psychiatry* **9**, 646–663 (2004).
119. Trehub, S. E. & Gudmundsdottir, H. R. in *The Oxford Handbook of Singing* (eds Welch, G. & Sergeant, D.) 1–20 (Oxford Univ. Press, Oxford, 2015).
120. MacLean, P. C. et al. Mother–infant mutual eye gaze supports emotion regulation in infancy during the still-face paradigm. *Infant Behav. Dev.* **37**, 512–522 (2014).
121. Mantis, I., Stack, D. M., Ng, L., Serbin, L. A. & Schwartzman, A. E. Mutual touch during mother–infant face-to-face still-face interactions: influences of interaction period and infant birth status. *Infant Behav. Dev.* **37**, 258–267 (2014).
122. Ramsay, D. S. & Woods, S. C. Clarifying the roles of homeostasis and allostasis in physiological regulation. *Psychol. Rev.* **121**, 225 (2014).
123. Muenzinger, K. F. & Fletcher, F. M. Motivation in learning. VI. Escape from electric shock compared with hunger-food tension in the visual discrimination habit. *J. Comp. Psychol.* **22**, 79 (1936).
124. Petrinovich, L. & Bolles, R. Deprivation states and behavioral attributes. *J. Comp. Physiol. Psychol.* **47**, 450 (1954).
125. Okanoya, K. in *Evolution of the Brain, Cognition, and Emotion in Vertebrates* (eds Watanabe, S., Hofman, M. A. & Shimizu, T.) 207–224 (Springer, Tokyo, 2017).
126. Scott, J. P. Critical periods in the development of social behavior in puppies. *Psychosom. Med.* **20**, 42–54 (1958).
127. Li, S. S. Y. & McNally, G. P. The conditions that promote fear learning: prediction error and Pavlovian fear conditioning. *Neurobiol. Learn. Mem.* **108**, 14–21 (2014).
128. Preuss, T. M. The human brain: rewired and running hot. *Ann. N. Y. Acad. Sci.* **1225**, 182–191 (2011).
129. Spocter, M. A. et al. Neuropil distribution in the cerebral cortex differs between humans and chimpanzees. *J. Comp. Neurol.* **520**, 2917–2929 (2012).
130. Barrett, L. F. The theory of constructed emotion: an active inference account of interoception and categorization. *Soc. Cogn. Affect. Neurosci.* **12**, 1–23 (2017).
131. Finlay, B. & Uchiyama, R. *Evolution of Nervous Systems* (Oxford Academic Press, Oxford, 2017).
132. Hauser, M. D., Chomsky, N. & Fitch, W. T. The faculty of language: what is it, who has it, and how did it evolve? *Science* **298**, 1569–1579 (2002).
133. Bloom, P. Precis of How children learn the meanings of words. *Behav. Brain Sci.* **24**, 1095–1103; discussion 1104–1034 (2001).
134. Lupfer, G., Frieman, J. & Coonfield, D. Social transmission of flavor preferences in two species of hamsters (*Mesocricetus auratus* and *Phodopus campbelli*). *J. Comp. Psychol.* **117**, 449–455 (2003).
135. Galef, B. G. & Laland, K. N. Social learning in animals: empirical studies and theoretical models. *AIBS Bull.* **55**, 489–499 (2005).
136. Uller, T. Developmental plasticity and the evolution of parental effects. *Trends Ecol. Evol.* **23**, 432–438 (2008).
137. Wolf, J. B. & Brodie, E. D. The coadaptation of parental and offspring characters. *Evolution* **52**, 299–308 (1998).
138. Stigler, J. W., Shweder, R. A. & Herdt, G. (eds) *Cultural Psychology* 1–44 (Cambridge Univ. Press, New York, NY, 1990).
139. Atzil, S. & Gendron, M. Bio-behavioral synchrony promotes the development of conceptualized emotions. *Curr. Opin. Psychol.* **17**, 162–169 (2017).
140. Gendron, M., Roberson, D. & Barrett, L. F. Cultural variation in emotion perception is real: a response to Sauter, Eisner, Ekman, and Scott (2015). *Psychol. Sci.* **26**, 357–359 (2015).
141. Russell, J. A. Culture and the categorization of emotions. *Psychol. Bull.* **110**, 426–450 (1991).
142. Andrews-Hanna, J. R. The brain's default network and its adaptive role in internal mentation. *Neuroscientist* **18**, 251–270 (2012).
143. Lombardo, M. V. et al. Shared neural circuits for mentalizing about the self and others. *J. Cogn. Neurosci.* **22**, 1623–1635 (2010).
144. Gao, W. et al. Functional network development during the first year: relative sequence and socioeconomic correlations. *Cereb. Cortex* **25**, 2919–2928 (2015).
145. Gao, W. et al. Evidence on the emergence of the brain's default network from 2-week-old to 2-year-old healthy pediatric subjects. *Proc. Natl Acad. Sci. USA* **106**, 6790–6795 (2009).
146. Supekar, K. et al. Development of functional and structural connectivity within the default mode network in young children. *Neuroimage* **52**, 290–301 (2010).
147. Blakemore, S. J., den Ouden, H., Choudhury, S. & Frith, C. Adolescent development of the neural circuitry for thinking about intentions. *Soc. Cogn. Affect. Neurosci.* **2**, 130–139 (2007).
148. Alcauter, S. et al. Frequency of spontaneous BOLD signal shifts during infancy and correlates with cognitive performance. *Dev. Cogn. Neurosci.* **12**, 40–50 (2015).
149. Uddin, L. Q., Supekar, K. S., Ryali, S. & Menon, V. Dynamic reconfiguration of structural and functional connectivity across core neurocognitive brain networks with development. *J. Neurosci.* **31**, 18578–18589 (2011).
150. Wegner, D. M. in *Theories of Group Behavior* (eds Mullen, B. & Goethals, G. R.) 185–208 (Springer, New York, NY, 1987).
151. Syal, S. & Finlay, B. L. Thinking outside the cortex: social motivation in the evolution and development of language. *Dev. Sci.* **14**, 417–430 (2011).
152. Dunbar, R. I. M. The social brain hypothesis. *Evol. Anthropol.* **6**, 178–190 (1998).
153. Gunnar, M. R. & Sullivan, R. M. The neurodevelopment of social buffering and fear learning: integration and crosstalk. *Soc. Neurosci.* **12**, 1–7 (2017).
154. Coan, J. A., Schaefer, H. S. & Davidson, R. J. Lending a hand: social regulation of the neural response to threat. *Psychol. Sci.* **17**, 1032–1039 (2006).
155. Master, S. L. et al. A picture's worth: partner photographs reduce experimentally induced pain. *Psychol. Sci.* **20**, 1316–1318 (2009).
156. Lantolf, J. P., Thorne, S. L. & Poehner, M. E. in *Theories in Second Language Acquisition: An Introduction* (eds VanPatten, B. & William, J.) 207–226 (Erlbaum, Mahwah, NJ, 2015).
157. Padilla, A. M. & Perez, W. Acculturation, social identity, and social cognition: a new perspective. *Hisp. J. Behav. Sci.* **25**, 35–55 (2003).
158. Adolphs, R. The social brain: neural basis of social knowledge. *Annu. Rev. Psychol.* **60**, 693–716 (2009).
159. Frith, C. D. The social brain? *Philos. Trans. R. Soc. London Ser. B* **362**, 671–678 (2007).
160. Whitacre, J. M., Rohlfshagen, P., Bender, A. & Yao, X. Evolutionary mechanics: new engineering principles for the emergence of flexibility in a dynamic and uncertain world. *Nat. Comput.* **11**, 431–448 (2012).
161. Boyd, R., Richerson, P. J. & Henrich, J. The cultural niche: why social learning is essential for human adaptation. *Proc. Natl Acad. Sci. USA* **108**, 10918–10925 (2011).
162. Heyes, C. & Pearce, J. M. Not-so-social learning strategies. *Proc. R. Soc. B* **282**, 1709–1715 (2015).
163. Champagne, F. A. & Meaney, M. J. Transgenerational effects of social environment on variations in maternal care and behavioral response to novelty. *Behav. Neurosci.* **121**, 1353–1363 (2007).
164. Francis, D., Diorio, J., Liu, D. & Meaney, M. J. Nongenomic transmission across generations of maternal behavior and stress responses in the rat. *Science* **286**, 1155–1158 (1999).
165. Lorenz, K. Der Kumpan in der Umwelt des Vogels. *J. Ornithol.* **83**, 289–413 (1935).
166. Lorenz, K. in *Leaders in the Study of Animal Behavior: Autobiographical Perspectives* (ed. Baerends, G. P.) 259–287 (Bucknell Univ. Press, Lewisburg, PA, 1985).
167. Morton, J. & Johnson, M. H. CONSPEC and CONLERN: a two-process theory of infant face recognition. *Psychol. Rev.* **98**, 164–181 (1991).
168. Braddick, O. Human development: faces in the womb. *Curr. Biol.* **27**, R704–R706 (2017).
169. Cook, R., Bird, G., Catmur, C., Press, C. & Heyes, C. Mirror neurons: from origin to function. *Behav. Brain Sci.* **37**, 177–192 (2014).

170. Turati, C., Di Giorgio, E., Bardi, L. & Simion, F. Holistic face processing in newborns, 3-month-old infants, and adults: evidence from the composite face effect. *Child Dev.* **81**, 1894–1905 (2010).
171. Gava, L., Valenza, E., Turati, C. & de Schonen, S. Effect of partial occlusion on newborns' face preference and recognition. *Dev. Sci.* **11**, 563–574 (2008).
172. Turati, C., Bulf, H. & Simion, F. Newborns' face recognition over changes in viewpoint. *Cognition* **106**, 1300–1321 (2008).
173. Cassia, V. M., Turati, C. & Simion, F. Can a nonspecific bias toward top-heavy patterns explain newborns' face preference? *Psychol. Sci.* **15**, 379–383 (2004).
174. Simion, F., Leo, I., Turati, C., Valenza, E. & Dalla Barba, B. How face specialization emerges in the first months of life. *Prog. Brain Res.* **164**, 169–185 (2007).
175. Turati, C. Why faces are not special to newborns: an alternative account of the face preference. *Curr. Dir. Psychol. Sci.* **13**, 5–8 (2004).
176. Gartstein, M. A. & Rothbart, M. K. Studying infant temperament via the revised infant behavior questionnaire. *Infant Behav. Dev.* **26**, 64–86 (2003).
177. Huffman, L. C. et al. Infant temperament and cardiac vagal tone: assessments at twelve weeks of age. *Child Dev.* **69**, 624–635 (1998).
178. Davidov, M., Knafo-Noam, A., Serbin, L. A. & Moss, E. The influential child: how children affect their environment and influence their own risk and resilience. *Dev. Psychopathol.* **27**, 947–951 (2015).
179. Rothbart, M. K. & Ahadi, S. A. Temperament and the development of personality. *J. Abnorm. Psychol.* **103**, 55 (1994).
180. George, O., Le Moal, M. & Koob, G. F. Allostasis and addiction: role of the dopamine and corticotropin-releasing factor systems. *Physiol. Behav.* **106**, 58–64 (2012).
181. Koob, G. F. & Le Moal, M. Drug addiction, dysregulation of reward, and allostasis. *Neuropsychopharmacology* **24**, 97–129 (2001).
182. Beauchaine, T. P., Neuhaus, E., Zalewski, M., Crowell, S. E. & Potapova, N. The effects of allostatic load on neural systems subserving motivation, mood regulation, and social affiliation. *Dev. Psychopathol.* **23**, 975–999 (2011).
183. Buckner, R. L., Andrews-Hanna, J. R. & Schacter, D. L. The brain's default network: anatomy, function, and relevance to disease. *Ann. N. Y. Acad. Sci.* **1124**, 1–38 (2008).
184. Young, L. J. & Barrett, C. E. Neuroscience. Can oxytocin treat autism? *Science* **347**, 825–826 (2015).
185. Farmer, C., Thurm, A. & Grant, P. Pharmacotherapy for the core symptoms in autistic disorder: current status of the research. *Drugs* **73**, 303–314 (2013).
186. Pellicano, E. & Burr, D. When the world becomes 'too real': a Bayesian explanation of autistic perception. *Trends Cogn. Sci.* **16**, 504–510 (2012).
187. Verly, M. et al. Altered functional connectivity of the language network in ASD: role of classical language areas and cerebellum. *Neuroimage Clin.* **4**, 374–382 (2014).
188. Jaffe-Dax, S., Frenkel, O. & Ahissar, M. Dyslexics' faster decay of implicit memory for sounds and words is manifested in their shorter neural adaptation. *Elife* **6**, e20557 (2017).
189. Jaffe-Dax, S., Raviv, O., Jacoby, N., Loewenstein, Y. & Ahissar, M. Towards a computational model of Dyslexia. *BMC Neurosci.* **16**, O12 (2015).
190. Leerkes, E. M., Su, J., Calkins, S. D., O'Brien, M. & Supple, A. J. Maternal physiological dysregulation while parenting poses risk for infant attachment disorganization and behavior problems. *Dev. Psychopathol.* **29**, 1–13 (2016).
191. Tasker, F. Lesbian mothers, gay fathers, and their children: a review. *J. Dev. Behav. Pediatr.* **26**, 224–240 (2005).
192. Bornstein, M. H. & Bradley, R. H. *Socioeconomic Status, Parenting, and Child Development* (Routledge, New York, NY, 2014).
193. Merz, E. C., Tottenham, N. & Noble, K. G. Socioeconomic status, amygdala volume, and internalizing symptoms in children and adolescents. *J. Clin. Child Adolesc. Psychol.* **47**, 312–323 (2018).
194. Kolb, B., Gibb, R. & Robinson, T. E. Brain plasticity and behavior. *Curr. Dir. Psychol. Sci.* **12**, 1–5 (2003).
195. Metcalfe, N. B., Taylor, A. C. & Thorpe, J. E. Metabolic rate, social status and life-history strategies in Atlantic salmon. *Anim. Behav.* **49**, 431–436 (1995).
196. Leonard, W. R. & Robertson, M. L. Evolutionary perspectives on human nutrition: the influence of brain and body size on diet and metabolism. *Am. J. Hum. Biol.* **6**, 77–88 (1994).
197. Dunbar, R. I. The social brain hypothesis and its implications for social evolution. *Ann. Hum. Biol.* **36**, 562–572 (2009).
198. Soares, C. A. & Carneiro, R. S. Social behavior between mothers' young of sloths *Bradypus variegatus* Schinz, 1825 (Xenarthra: Bradypodidae). *Braz. J. Biol.* **62**, 249–252 (2002).
199. Richard, A. F. & Nicoll, M. E. Female social dominance and basal metabolism in a Malagasy primate. *Propithecus verreauxi*. *Am. J. Primatol.* **12**, 309–314 (1987).
200. Curley, J. P. & Keverne, E. B. Genes, brains and mammalian social bonds. *Trends Ecol. Evol.* **20**, 561–567 (2005).
201. Schulkin, J. *Allostasis, Homeostasis, and the Costs of Physiological Adaptation* (Cambridge Univ. Press, Cambridge, 2004).
202. Shpigler, H. Y. et al. Deep evolutionary conservation of autism-related genes. *Proc. Natl Acad. Sci. USA* **36**, 9653–9658 (2017).
203. Gao, W., Alcauter, S., Smith, J. K., Gilmore, J. H. & Lin, W. Development of human brain cortical network architecture during infancy. *Brain Struct. Funct.* **220**, 1173–1186 (2015).
204. Sepulcre, J., Sabuncu, M. R., Yeo, T. B., Liu, H. & Johnson, K. A. Stepwise connectivity of the modal cortex reveals the multimodal organization of the human brain. *J. Neurosci.* **32**, 10649–10661 (2012).
205. Xu, P. et al. Different topological organization of human brain functional networks with eyes open versus eyes closed. *Neuroimage* **90**, 246–255 (2014).
206. Sterzer, P. & Kleinschmidt, A. Anterior insula activations in perceptual paradigms: often observed but barely understood. *Brain Struct. Funct.* **214**, 611–622 (2010).
207. Angelaki, D. E., Gu, Y. & DeAngelis, G. C. Multisensory integration: psychophysics, neurophysiology, and computation. *Curr. Opin. Neurobiol.* **19**, 452–458 (2009).

Acknowledgements

We thank K. Toledano for his contribution to the illustrations.

Author contributions

S.A., W.G. I.F. and L.F.B. contributed to writing the manuscript.

Competing interests

The authors declare no competing interests.

Additional information

Supplementary information is available for this paper at https://doi.org/10.1038/s41562-018-0384-6.

Reprints and permissions information is available at www.nature.com/reprints.

Correspondence should be addressed to S.A. or L.F.B.

Publisher's note: Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.