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Social influences on neuroplasticity: stress and interventions to promote well-being

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Experiential factors shape the neural circuits underlying social and emotional behavior from the prenatal period to the end of life. These factors include both incidental influences, such as early adversity, and intentional influences that can be produced in humans through specific interventions designed to promote prosocial behavior and well-being. Here we review important extant evidence in animal models and humans. Although the precise mechanisms of plasticity are still not fully understood, moderate to severe stress appears to increase the growth of several sectors of the amygdala, whereas the effects in the hippocampus and prefrontal cortex tend to be opposite. Structural and functional changes in the brain have been observed with cognitive therapy and certain forms of meditation and lead to the suggestion that well-being and other prosocial characteristics might be enhanced through training.

Social influences are among the influences on brain structure and function that are most powerful in inducing plastic change. The vertebrate brain appears to be particularly sensitive to social influences and this sensitivity may be especially acute in primates[1].

The brain is constantly being shaped, wittingly and unwittingly, by environmental forces that impinge on organisms. The circuitry that has been implicated in social and emotional behavior appears to be importantly shaped by experience, and early experience in these domains is likely involved in governing differences among individuals in their vulnerability or resilience to future adversity. Studies in both animal models and humans provide a foundation for understanding how explicit interventions designed to promote prosocial behavior and well-being might induce plasticity-related changes in the brain. There is a growing corpus of evidence that suggests that interventions ranging from regular moderate physical exercise[2] to cognitive therapy[3,4] to interventions derived from ancient contemplative practices[5] induce plasticity-related alterations in the brain and support a range of positive behavioral outcomes.

There are many different mechanisms of plasticity and, at the human level, there are methodological constraints that limit the mechanisms that can be directly studied. Most human work has focused on alterations in different indices of brain structure that can be measured with modern magnetic resonance imaging (MRI). Enduring functional alterations can also be assessed using functional MRI (fMRI) and related techniques.

Experience-dependent influences on particular features of cognitive function, such as language learning, appear to have a robust sensitive period[6]. However, even a competence as clearly 'cognitive' as language acquisition is influenced by social context and social interaction (see ref. 6 for a review). The social deprivation of orphanages for abandoned children in Bucharest, Romania has been found to produce profound cognitive impairment that can be partially remediated by early placement in foster care[7]. The earlier the age of foster care placement and removal from the orphanage, the less severe was the observed cognitive deficit. The extent of such sensitive periods in the realms of social and emotional behavior is not yet known. However, there are some hints; for example, there is recent evidence in a rodent model that amygdala circuits are kept in an immature state in an infant by the presence of the mother, but can be stimulated to mature by corticosterone to promote maturation to allow aversive learning[8]. Once a developmental event has occurred, can it be reversed? Research on recovery of vision in adult amblyopic subjects suggests mechanisms that might be used to remove the brakes on adult plasticity, such as the use of behavioral interventions[9]. Whether similar mechanisms might be present to facilitate adult plasticity of social behavior has not been studied.

We do know that early stressful and nurturing environments have robust effects on the developing brain, some of which persist for the life of the organism. The effects of stress are the most well-characterized, and we review key findings at the animal level below.

Research at the human level that has focused on the experience-dependent effects of stressful life events has taken advantage of largely unintended environmental circumstances, such as child maltreatment or exposure to early stress. In addition to this corpus, there is now a growing literature on the effect of interventions explicitly designed to promote positive outcomes, such as physical exercise[2], cognitive therapy[3,4], social service programs for older individuals[10] and meditation[5,11]. There are also a growing number of interventions designed to promote prosocial behavior in children that include social-emotional learning[12] and executive function training[13]. The evidence for their efficacy is mostly behavioral at this point in time, and the mechanisms by which such interventions operate have not been systematically examined, although it is likely that some features of neuroplasticity will be important for at least some of the behavioral effects that have been described.

Here we review some key findings at the animal level that establish experience-induced structural plasticity in response to social influences. Although most of the findings have focused on stressful environmental influences, there are some data on specific environmental influences

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that appear to promote positive social and emotional behavior. We also review experience-induced plasticity in humans arising from both unintended influences, such as early life stress, and explicit intervention strategies that are designed to promote more effective coping with stress and salubrious social and emotional behavior. Some of these interventions are derived from ancient contemplative practices, whereas others have emerged from the modern research context. One important idea that we present is the notion that, just as we as a society are learning to take more responsibility for our physical health by engaging in regular physical exercise, we can also take more responsibility for our minds and brains by engaging in certain mental exercises that can induce plastic changes in the brain and that may have enduring beneficial consequences for social and emotional behavior. This also invites the perspective that qualities such as well-being ought to be viewed, at least in part, as a product of trainable skills and that interventions explicitly designed to promote well-being may have beneficial behavioral and biological effects. Although well-being and other similar constructs exhibit moderate stability in the absence of either unwitting or intentional influences, the evidence suggests that change can occur in the presence of such factors.

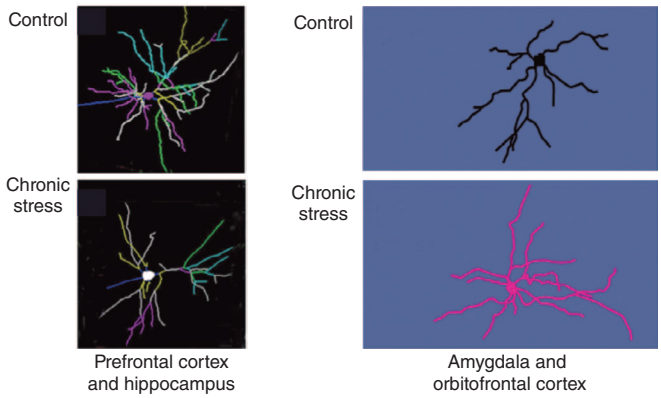

Figure 1 Chronic stress causes neurons to shrink or grow, but not necessarily to die. Representation of the chronic stress effects detected in animal models on growth or retraction of dendrites in the basolateral amygdala and orbitofrontal cortex (growth) and in the CA3 hippocampus, dentate gyrus and medial prefrontal cortex (shrinkage). These effects are largely reversible in young adult animals, although aging appears to compromise resilience and medial prefrontal cortex recovery[21].

Basic research at the animal level

Evidence that the healthy mature animal brain is capable of structural plasticity can be traced to the so-called enriched environment studies[14], which were in turn based on findings of enhanced problem solving behavior in rats living as pets in a complex environment[15]. Rats that lived for weeks in an environment filled with toys that were changed daily in a larger and more complex living space showed increased thickness of cerebral cortical areas. This was also true of aging rats[16]. Subsequent studies found that cortical neurons showed increased dendritic branching and complexity in such an environment compared with normal laboratory cages, as well as increased numbers of glial cells and increased blood supply[17].

More recent investigations have shown that both acute and chronic stress alter spine density and dendritic length and branching in brain regions, such as hippocampus, prefrontal cortex and amygdala[18]. Measured by conventional neuroanatomical methods, the time course of these changes were found to occur over days and are largely reversible, at least in young adult animals[18,19]. However, a recent study using transcranial two-photon microscopy to track the formation and elimination of dendritic spines *in vivo* after treatment with glucocorticoids in developing and adult mice revealed spine turnover in several hours that was higher in the developing barrel cortex, but was still very much present in the adult, and similar changes occurred in multiple cortical areas, suggesting a generalized effect that may occur in many brain regions[20]. Mechanisms for such dendritic and synaptic remodeling involve not only glucocorticoids, but also excitatory amino acids and other cellular mediators[18,21].

Sex hormones also promote structural plasticity in hippocampus, cerebral cortex, hypothalamus and other brain regions[22,23]. For example, ovarian hormones promote cyclic changes in spine density in the hippocampus, as well as in the primary sensory-motor cortex and prefrontal cortex of rodents and monkeys[24,25]. Mechanisms for these changes involve not only estradiol and progesterone, but also excitatory amino acids and other cellular mediators[22].

A major breakthrough in brain plasticity came with the rediscovery of neurogenesis in the adult dentate gyrus[26–29]. Dentate gyrus neurogenesis is stimulated by physical activity and environmental enrichment[30] and is inhibited by chronic physical and social stressors[18]. Regular physical activity also increases human hippocampal volume, possibly via stimulating neurogenesis[2].

Structural plasticity in the adult brain involving not only neurogenesis, but also dendritic and synaptic turnover, can be related to social interactions in the visible burrow system for rats[31] and in the tree shrew. In the tree shrew, a resident-intruder protocol shows the powerful effect on the intruder in terms of reduced neurogenesis and dendritic shrinkage in the hippocampus[32,33].

Although the hippocampus shows impaired neurogenesis and atrophy of dendritic trees after chronic stress, the same stressor causes dendritic growth in the basolateral amygdala, along with increased anxiety (**Fig. 1**) and aggression, whereas neurons in the medial prefrontal cortex shrink and those in the orbitofrontal cortex grow[18,34,35]. These are largely reversible changes, at least in young adult animals, although aging compromises the reversibility of neuronal atrophy in the medial prefrontal cortex[19].

Stress-induced changes in the circuitry of these brain regions alters the balance between different neural systems that are activated by experiences[18,36]. For example, low self esteem in humans has been associated with a smaller hippocampus, impulsiveness and poor executive function have been associated with a defective prefrontal cortex, and aggression and anxiety have been associated with an overactive amygdala[36].

Early life experiences are potent in this regard[37]. In both animal models and humans, experiences, good and bad, shape these circuits and their connectivity, and experiences can trigger adaptive or maladaptive responses depending on the health and balance of those interconnections. In animals, early life events related to maternal care, as well as parental care in humans, have a powerful role in later mental and physical health, as demonstrated by the adverse childhood experiences study[38]. Prenatal stress impairs hippocampal development in rats, as does stress in adolescence[39]. Abusive maternal care in rodents and the surprising attachment shown by infant rats to their abusive mothers appears to involve an immature amygdala[40], the activation of which by glucocorticoids causes aversive conditioning response to emerge. Maternal anxiety in the variable foraging demand model in rhesus monkeys leads to chronic anxiety in the offspring, as well as signs of metabolic syndrome[41,42].

There is also structural plasticity in the mesolimbic reward system that is affected by social defeat and leads animals to increased drug self-administration. As a result, medium spiny neurons in the nucleus accumbens show altered dendritic spine formation[43]. Social defeat, along with maternal separation in infancy, increases vulnerability to substance self-administration[44]. Drugs of abuse alter the morphology of many brain regions[45], which may or may not drive addictive behavior or reflect compensatory changes[46]. Notably, there

is cross-sensitization of appetitive stimuli in that induction of need-free salt appetite leads to altered dendritic morphology in the shell of the nucleus accumbens and sensitizes the animal to amphetamine self-administration[47].

In addition to findings that underscore the deleterious effect of early life stress on later development, some animal studies have found protective effects of nurturing environments, as well as resilience-enhancing effects of exposure to mild stress early in life. Animal models have contributed enormously to our understanding of how the brain and body are affected[48,49]. Epigenetic, transgenerational effects transmitted by maternal care are central to these findings. Beside the amount of maternal care, the consistency over time of that care and the exposure to novelty are also very important, not only in rodents[50,51], but also in monkeys[52]. A recent study found that the rat pups who received high levels of licking and grooming during the first week of postnatal life had higher levels of glucocorticoid mRNA expression in the hippocampus as young adults and enhanced induction of synaptic plasticity in the dentate gyrus *in vitro*[53].

Beneficial effects of early exposure to mild stress have been observed in squirrel monkeys. After exposure to mild stress from postnatal weeks 17–27, the mildly stressed animals displayed decreased anxiety as young adults, as measured by decreased maternal clinging, enhanced exploratory behavior and increased food consumption. Moreover, animals exposed to early mild stress had lower basal plasma adrenocorticotropic hormone and cortisol, and lower cortisol following stress exposure[54]. Animals exposed to early mild stress exhibited enhanced prefrontally dependent response inhibition as young adults, suggesting that the early exposure to mild stress enhances prefrontal regulatory mechanisms that facilitate stress inoculation[55]. In this same squirrel monkey model, mild stress exposure early in life results in increased ventromedial prefrontal cortex (vmPFC) volume during the peripubertal period[56]. The increased vmPFC volume reflects surface area expansion of this PFC zone rather than an increase in cortical thickness. Moreover, increased white matter myelination in this region was detected with diffusion tensor imaging[56].

One of the longest held notions of brain plasticity is that certain critical periods or windows exist in development, during which circuitry is laid down that lasts for the lifetime. However, a more recent set of findings suggests that developmentally induced plasticity, at least certain kinds, can be reversed by re-opening those windows. For example, ocular dominance imbalance resulting from early monocular deprivation can be reversed by patterned light exposure in adulthood that can be facilitated by fluoxetine[57] and food restriction[58], in which reducing inhibitory neuronal activity appears to be important[59]. Investigations of underlying mechanisms for the re-establishment of a new window of plasticity are focusing on the balance between excitatory and inhibitory transmission and removing molecules that put the brakes on such plasticity[9].

Depression is more prevalent in individuals who have had adverse early life experiences[38]. Neurotrophic factors such as BDNF may be an important feature of the depressive state, and elevation of such factors by diverse treatments ranging from antidepressant drugs, such as fluoxetine, to regular physical activity may be a key feature of treatment[60]. In addition, there are other potential applications, such as the recently reported ability of fluoxetine to enhance recovery from stroke[61]. An important aspect of this new view[62] is that the drug is opening a window of opportunity that may be capitalized by a positive behavioral intervention; for example, behavioral therapy in the case of depression or intensive physiotherapy to promote neuroplasticity to counteract the effects of a stroke.

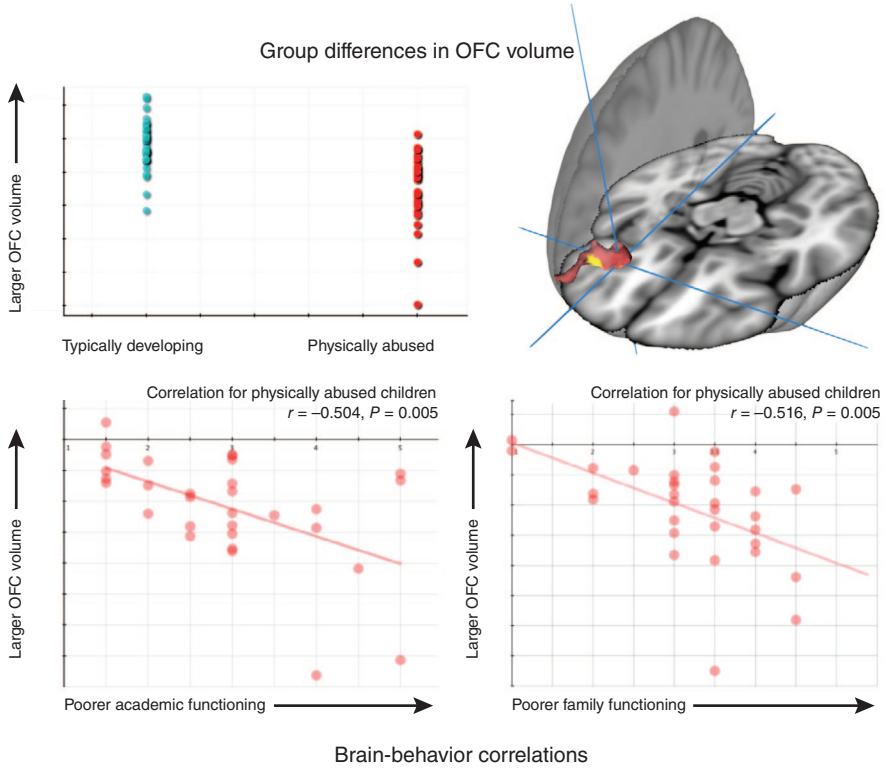

Figure 2 Physically abused children show alterations in orbitofrontal (OFC) volume compared with typically developing children, and volume shrinkage in this region is related to measures of family stress. Top, physically abused children show reductions in orbitofrontal cortex compared with typically developing controls. Bottom, among physically abused children, those showing poorer academic function and poorer family functioning (greater family stress) exhibit less volume in orbitofrontal cortex[68]. Note that because the voxel-wise analysis was a between-groups comparison and the correlational analysis was conducted with the abused children only, this does not suffer from the 'double-dipping' problem.

Plasticity in human social brain

Experience-induced effects of adversity and stress. The social and emotional circuitry of the brain is continuously being shaped by forces that impinge on the nervous system during prenatal development and throughout life. The fact that experience-induced plasticity has been documented in the social brain in a variety of animal models provides the foundation for examining similar effects in humans. There is now a substantial body of evidence on the effect of stressful environments on the developing human brain and associated behavior[62–67]. For example, in a sample of 31 physically abused and 41 typically developing teenage children who underwent structural MRI scanning using diffeomorphic image normalization and tensor-based morphometry, the abused children had smaller orbitofrontal volumes and, furthermore, the smaller the orbitofrontal volume in the abused sample, the more severe the social stress as reported by children and parents on a structured interview (**Fig. 2**)[68].

Early life stress modulates the hypothalamic-pituitary-adrenal axis, particularly cortisol as an output measure of this system, although the

effects on this system are complex and depend on the chronicity and timing of the stress[69]. Evidence that child abuse is associated with alterations in the epigenetic regulation of the glucocorticoid receptor was obtained in a study of postmortem tissue extracted from the hippocampus of suicide victims with a history of child abuse and those with no abuse history along with controls[70]. In hippocampus, decreased levels of glucocorticoid receptor mRNA were observed, as well as mRNA transcripts bearing the glucocorticoid receptor 1F splice variant and increased cytosine methylation of an *NR3C1* promoter[70].

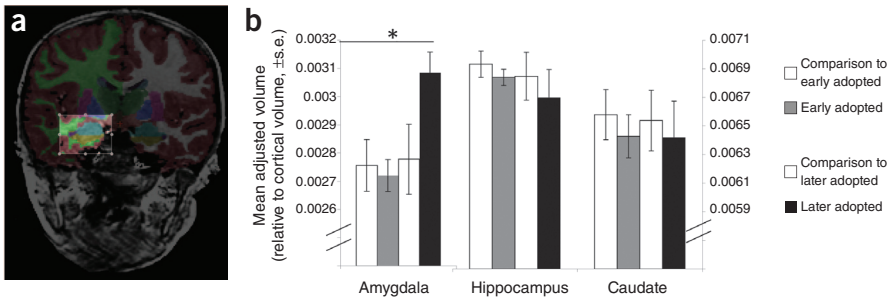

Figure 3 Anatomically segmented amygdala volumes are larger in later-adopted post-institutionalized children. (**a**) Anatomical segmentation of the amygdala. (**b**) Later-adopted post-institutionalized children show larger amygdala volume compared with both early adopted children and with typically developing controls. No differences among groups were found in hippocampus or caudate[71]. Asterisk indicates that the later adopted group exhibited significantly larger amygdala volume compared with each of the comparison groups.

One study capitalized on unfortunate circumstances and examined 38 post-institutionalized children who were raised in impoverished orphanages in either Eastern Europe or Asia and 40 non-institutionalized children[71]. At the time of testing, the children were 8.5–9.5 years and were institutionalized, on average, at 2.5 months of age. Using an automated segmentation algorithm, the authors specifically looked at volumetric measures of the amygdala, hippocampus and caudate. When the post-institutionalized sample was compared with controls, no overall differences between groups were found for any of the three structures examined. However, they also divided the post-institutionalized sample into those who were adopted at an early age versus those adopted later on (<15 months versus >15 months at age of adoption, respectively). When participants were divided in this way, the later adopted post-institutionalized children were found to have significantly larger amygdale than those adopted early on and their control counterparts. There were no significant differences among any of the groups in the volumes of the hippocampus or caudate (**Fig. 3**). When examined continuously, the authors found that the age at adoption was positively correlated with amygdala volume, such that those adopted at a later age had larger amygdala volumes. Higher parental ratings of internalizing behavior and anxiety were also correlated with larger amygdala volume. A similar pattern of results was obtained from a sample of 10-year-old children, some of whom were continuously exposed to maternal depressive symptoms from birth and others who had no exposure to maternal depressive symptoms[72]. At 10 years of age, children who had been continuously exposed to maternal depressive symptoms from birth had significantly larger left and right amygdale than children with no such exposure. There were no significant differences in hippocampal volume between these groups. The mean depression score of the mother computed over 7 years predicted amygdala volume of her child at age 10 such that mothers with higher levels of depressive symptoms had children with larger amygdala volume.

These findings are consistent with the idea that early life stress induces structural changes in the developing brain. The two most prominent structural findings from human research suggest that amygdala volume is increased and that sectors of the prefrontal cortex are decreased. Some caution regarding these findings in the amygdala is warranted because of methodological complications resulting from automated segmentation algorithms with subcortical structures such as the amygdala[73]. Moreover, the precise ages at which these effects occur needs to be carefully studied, as, particularly for the amygdala, early hypertrophy and enlargement may occur in response to adversity, and, perhaps in part because of excitotoxic processes, premature volume reduction may be produced[74]. Such a developmental pattern in the amygdala has been suggested to occur in the autistic brain[75,76]. The amygdala and prefrontal cortex and their interconnections have been strongly implicated in emotion regulation[77–79] and well-being[80], and dysfunctions and/or structural abnormalities in their interconnections have been implicated in psychopathology[81–83].

Prosocial intervention and training. An important question replete with both theoretical and practical relevance is whether explicit interventions or training designed to foster prosocial behavior and well-being, or more naturally occurring forms of positive social interaction and social support, can induce neuroplastic changes in the brain. A study examining the effect of holding the hand of one's spouse found a significant attenuation of the neural response to the threat of shock in several threat-sensitive brain regions, including the anterior insula and ventral anterior cingulate cortex, in women when they were holding their spouse's hand compared with controls that included holding a stranger's hand and an alone condition[84]. As this and other similar studies examined the effect of an acute manipulation, the effects are likely to be phasic and short-lived, but they raise the question of whether cumulative exposure to social support would induce beneficial plastic changes[85]. Other forms of social support, such as maternal care, appear to modulate the effect of prenatal risk on hippocampal volume, at least in women[86].

There is a growing literature documenting functional and structural changes in the brain with specific interventions and training regimes. The behavioral evidence in support of such interventions and training provides a reasonable foundation for the exploration of neural changes that support these behavioral outcomes. For example, interventions designed to promote prosocial behavior, such as effective emotion regulation, have been developed for incorporation in school curricula to support the development of more positive social and emotional trajectories in K-12 school children. A recent meta-analysis of 213 programs involving more than 270,000 school children found that, compared with controls, participants in social emotional learning programs demonstrated significant gains in social and emotional skills and performed, on average, 11% better on standardized measures of academic achievement[12]. Other evidence suggests that cognitive therapy for depression[4], as well as well-being therapy[87], specifically helps to improve positive affect.

A recent review presented evidence consistent with the view that cognitive therapy enhances prefrontal function and via this enhanced prefrontal activation, amygdala activation is inhibited[88]. A recent study examined the effect of cognitive therapy on patients with chronic fatigue syndrome in a short-term longitudinal study[89]. At baseline, these patients showed decreased gray matter volume compared with healthy controls. Patients then underwent 16 1-h sessions of cognitive therapy and were rescanned following treatment. Increases in lateral prefrontal volume were found in the patients following treatment that were correlated with improvements in digit symbol substitution and in

a choice reaction time task[89]. Unfortunately, changes in mood or social behavior were not reported.

The effect of secular training derived from meditation traditions that emphasize the cultivation of positive affect, such as compassion and kindness, has received increased empirical attention. A recent review concluded that such exercises, which are oriented toward enhancing the positive emotions compassion and kindness, do indeed increase positive affect and decrease negative affect[90]. It has been shown that 1 d of compassion meditation training increases prosocial behavior in a virtual game compared with a 1 d of memory training control condition[88]. Collectively, these findings raise the possibility that such interventions and training programs designed to explicitly decrease stress and enhance certain forms of positive emotion may produce specific plasticity-related alterations in brain function and structure.

A recent study of functional brain alterations with compassion meditation in expert practitioners who have been meditating for more than 10,000 h over the course of their lifetime, compared with novices who were just learning to meditate, found that, during a mental practice explicitly designed to enhance compassion, the practitioners showed enhanced gamma oscillations and gamma synchrony compared with controls[91]. Enhanced BOLD signal was detected with fMRI in response to emotional sounds in brain regions, including the insula and temporoparietal junction, that have been implicated in previous studies of empathy[92]. The increase in gamma oscillations and gamma synchrony might reflect its role in synaptic plasticity[93] and suggest a general enhancement of synaptic plasticity through this form of mental practice.

Other research suggests that mindfulness meditation may operate via a distinct neural mode of self-referencing that favors momentary nonjudgmental present-moment experience over narrative self-focused mentation. This form of mental training has been found to decrease anxiety and increase positive affect[94]. A study tested this idea by comparing novices and participants who attended an 8-week course in mindfulness meditation (mindfulness-based stress reduction, MBSR). fMRI was measured in response to a task that contrasted an 'experiential focus' to a narrative self-focused condition in response to trait adjectives. The MBSR participants exhibited reductions in medial prefrontal activation and increased activation of the insula and lateral prefrontal cortices during the experiential versus narrative conditions[95]. Consistent findings using a different methodological strategy were obtained in a recent study comparing experienced mindfulness meditation practitioners to novices. The experienced practitioners showed decreased medial prefrontal activity in the baseline default BOLD signal compared with the novices[96]. Other findings indicate that activation of the medial prefrontal cortex at baseline is associated with mind-wandering[97] and that mind wandering is associated with unhappiness[98]. A major limitation of all of the studies described above on is that they relied on between group comparisons of a meditation group compared with a control group. To more firmly establish that differences are the results of meditation training *per se* and not of self-selection and other factors that might confound between group comparisons, longitudinal investigations of changes over the course of meditation training are needed. Such a design was used to examine whether certain forms of meditation may operate via effects that are opposite to those produced by stress. As we noted above, early life stress increases amygdala volume. In a longitudinal study of 26 participants undergoing an 8-week training in MBSR, MRI scans were obtained before and after the 8 weeks of training. Reductions in perceived stress following MBSR were correlated with reductions in gray matter volume in the right basolateral amygdala that were obtained from MRI scans performed before and after the 8 weeks of training (**Fig. 4**)[11]. These findings suggest that the plasticity-related alterations in brain regions that have been implicated in stress can occur with as little as 8 weeks of mindfulness meditation training.

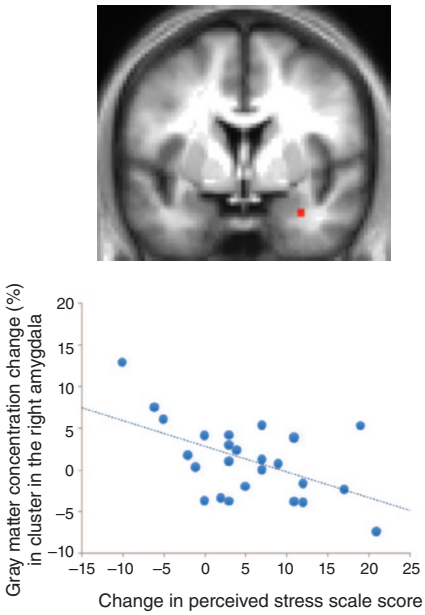

Figure 4 Change in gray matter volume in the right basolateral amygdala from pre to post 8 weeks of MBSR was associated with decreases in perceived stress over this same time period. Individuals undergoing MBSR who showed the largest decreases in perceived stress also showed the largest decreases in basolateral amygdala gray matter volume[11].

Summary, conclusions and implications

It has been known for more than a century that social and emotional behavior is modified by experience. Abundant evidence exists that stress and adversity, particularly early in life, can produce enduring alterations in behavior. It has also been claimed for thousands of years that specific forms of mental training can produce robust beneficial and enduring effects on behavior. The rigorous investigation of such effects and the neural mechanisms responsible for producing them has only recently become a serious focus of neuroscientific study. The findings that we discuss underscore the structural plasticity of emotional circuitry in response to both acute and chronic stress, particularly alterations of spine density and dendritic length and branching in hippocampus, amygdala and prefrontal cortex. Evidence at the animal level has identified several different mechanisms of plasticity, including dendritic and synaptic turnover and neurogenesis. The animal and human evidence is consistent in demonstrating that many forms of stress promote excessive growth in sectors of the amygdala, whereas effects in hippocampus tend to be opposite. Whether critical or sensitive periods exist for plasticity in response to social influences has not been thoroughly addressed and more systematic developmental studies are required. Moreover, the reversibility of structural changes following alterations in social and emotional conditions has not been systematically examined.

At the human level, research is beginning to document the effect of explicit interventions designed to decrease stress and promote prosocial behavior and well-being on brain structure and function. These studies are consistent with basic research in demonstrating increases in specific sectors of prefrontal activation and decreases in amygdala activation. These functional alterations are accompanied by structural changes that show increases in prefrontal volume and decreases in amygdala volume. The precise differences among the various interventions that have been developed for this general purpose have not been systematically studied,

nor has the relation between functional and structural changes been carefully documented. Moreover, it is apparent that both structural and functional connectivity between prefrontal regions and subcortical structures is extremely important for emotion regulation and that these connections represent important targets for plasticity-induced changes. This is likely to be an important focus of future studies.

Finally, the studies on interventions explicitly designed to promote positive emotional qualities, such as kindness and mindfulness, imply that such qualities might best be regarded as the product of skills that can be enhanced through training, just as practice will improve musical performance and produce correlated regionally specific anatomical changes. Whether these interventions simply modulate the adverse effects of stress or whether they result in a profile of neurobehavioral functioning that is better than normal will require considerably more evidence, although the available evidence points toward the latter possibility. This perspective can lead to the view that social and emotional characteristics can be educated in ways that are not dissimilar from certain forms of cognitive learning. Many forms of meditation and cognitive therapy can enhance self-control or self-regulation[99]. Such improvements in self-control are particularly apparent in social and interpersonal contexts. It is in these contexts that attentionally demanding stimuli typically occur and where self-regulation is especially important. A recent study of a cohort of 1,000 participants assessed from birth to 32 years of age found that childhood measures of self-control predicted physical health, substance dependence, personal finances and criminal offending outcomes at 32 years of age[100]. The authors defined self-control as a family of processes that include delay of gratification, impulse and attentional control, executive function, and willpower. They suggest that early interventions that enhance self-control might reduce a panoply of societal costs, save taxpayers money and promote prosperity. The mental training at the core of the techniques described above might constitute ideal interventions to promote early self-control and improve later adult prosocial outcomes. For example, mindfulness meditation has been found to strengthen selective and other aspects of attention and executive function[5]. Whether such interventions can produce changes that have lasting consequences is a possibility that requires extensive empirical investigation.

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COMPETING FINANCIAL INTERESTS

The authors declare no competing financial interests.

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