AMERICAN PSYCHOLOGICAL ASSOCIATION

Journal of Psychopathology and Clinical Science

ISSN: 2769-7541

2022, Vol. 131, No. 6, 575–587
https://doi.org/10.1037/abn0000598

Experience-Driven Plasticity and the Emergence of Psychopathology: A Mechanistic Framework Integrating Development and the Environment Into the Research Domain Criteria (RDoC) Model

Katie A. McLaughlin[1] and Laurel Gabard-Durnam[2, 3]
[1] Department of Psychology, Harvard University
[2] Division of Developmental Medicine, Boston Children's Hospital
[3] Department of Psychology, Northeastern University

Despite the clear importance of a developmental perspective for understanding the emergence of psychopathology across the life-course, such a perspective has yet to be integrated into the Research Domain Criteria (RDoC) model. In this paper, we articulate a framework that incorporates developmentally specific learning mechanisms that reflect experience-driven plasticity as additional units of analysis in the existing RDoC matrix. These include both experience-expectant learning mechanisms that occur during sensitive periods of development and experience-dependent learning mechanisms that may exhibit substantial variation across development. Incorporating these learning mechanisms allows for clear integration not only of development but also environmental experience into the RDoC model. We demonstrate how individual differences in environmental experiences—such as early life adversity—can be leveraged to identify experience-driven plasticity patterns across development and apply this framework to consider how environmental experience shapes key biobehavioral processes that comprise the RDoC model. This framework provides a structure for understanding how affective, cognitive, social, and neurobiological processes are shaped by experience across development and ultimately contribute to the emergence of psychopathology. We demonstrate how incorporating an experience-driven plasticity framework is critical for understanding the development of many processes subsumed within the RDoC model, which will contribute to greater understanding of developmental variation in the etiology of psychopathology and can be leveraged to identify potential windows of heightened developmental plasticity when clinical interventions might be maximally efficacious.

General Scientific Summary

We present a framework that incorporates developmentally specific learning mechanisms that reflect experience-driven plasticity as additional units of analysis in the Research Domain Criteria (RDoC) matrix. Incorporating these learning mechanisms allows for both development and environmental experience to be integrated into the RDoC model. This experience-driven plasticity framework can stimulate progress in understanding the development of many processes subsumed within the RDoC model, contribute to greater understanding of developmental variation in the onset of psychopathology, and can be leveraged to identify developmental windows of heightened plasticity when clinical interventions might be maximally efficacious.

Keywords: development, environment, adversity, early life stress, sensitive periods

To develop normally, children require a wide variety of inputs from the environment. Some of these experiences must occur during specific periods of development when the human brain depends upon input from the environment to develop certain capacities. Perceptual development provides an illustrative example. Early in life, visual input to the eyes is required for the visual system to develop normally; this input must occur during a sensitive window that occurs during the first months of life. Numerous other processes also exhibit this type of experience-expectant development, in which particular types of environmental experiences occurring during specific windows of time are required to foster adaptive development. When these expected experiences are absent or when atypical or unexpected experiences occur—such as exposure to trauma—development can be fundamentally altered in ways that increase vulnerability to psychopathology.

Katie A. McLaughlin https://orcid.org/0000-0002-1362-2410
Laurel Gabard-Durnam https://orcid.org/0000-0002-4564-8068

This work was supported by the National Institute of Mental Health (R01-MH103291, R01-MH106482, R56-119194, R37119-194 to Katie A. McLaughlin) and support from the Tokyo University International Research Center (to Laurel Gabard-Durnam).

Correspondence concerning this article should be addressed to Katie A. McLaughlin, Department of Psychology, Harvard University, 33 Kirkland Street, Cambridge, MA 02138, United States. Email: kmclaughlin@fas.harvard.edu

periods enable tuning and narrowing of the brain's responsiveness to specific types of expected environmental inputs (e.g., language, responsive caregivers), after which additional tuning to new inputs is diminished and requires extensive exposure. Third, they occur for specific brain circuits only during specific windows of development, although their timing is itself malleable, as discussed below. Fourth, sensitive periods are consolidated by molecular and structural regulators that protect the experience-modified circuitry and produce enduring effects on brain function and behavior, although other learning mechanisms may modify function further via residual plasticity following a sensitive period.

Sensitive periods are carefully orchestrated processes that unfold across RDoC levels of analysis from genes to behavior (Figure 1A). Sensitive period initiation is regulated by molecular pacers and triggers. Pacers inhibit sensitive period initiation to prevent precocious plasticity and maintain healthy developmental momentum (Takesian & Hensch, 2013). Conversely, triggers promote sensitive period initiation and increase neuroplasticity (e.g., through increased brain-derived neurotrophic factor [BDNF], a growth factor involved in synaptic transmission and brain plasticity; Hanover et al., 1999). Critically, exposure to the expected environmental experience is also required to initiate sensitive periods. In fact, the timing and quality of the expected experience impacts when and how sensitive period learning occurs (Werker & Hensch, 2015). That is, sensitive period timing and plasticity are themselves malleable as a function of experience (Figure 1B). A delay of the expected experience results in delayed sensitive period initiation. However, the system cannot wait for the experience indefinitely, and prolonged deprivation can result in sparse or no learning. Even if the experience occurs at the optimal time, the quality of that experience matters. Enriched experience may initiate sensitive periods more quickly and involve neural changes that produce greater functional tuning than inconsistent or poor-quality experiences.

Once a sensitive period is successfully triggered, additional mechanisms facilitate rapid structural and functional reconfiguration and tuning to the expected experiential inputs (Takesian & Hensch, 2013). For example, dramatic synaptic pruning occurs during sensitive periods to eliminate inefficient and unnecessary connections as circuit function becomes tuned by environmental experience. Continued exposure to expected experiences within the sensitive period is necessary to sculpt healthy brain function via these mechanisms (Schwarzkopf et al., 2007). Sensitive periods are then closed to stabilize the experience-driven function. Sensitive period neuroplasticity is downregulated by a number of molecular and structural factors (e.g., peri-neuronal nets, myelination) that actively inhibit plasticity thereafter (Takesian & Hensch, 2013).

Experience-Dependent Learning

In contrast to experience-expectant learning, experience-dependent plasticity facilitates learning at all points in life (Greenough et al., 1987). Experience-dependent learning involves changes induced by experience without prior preparation (e.g., learning to meditate). Although these mechanisms have no ontogenetic constraints in availability, their degree of plasticity does change with age and as a function of the environmental trigger (Figure 1C). It is well-established that experience-driven plasticity diminishes as the brain matures (Fu & Zuo, 2011). Moreover, at a given age, the intensity and duration of an experience can impact the degree of experience-dependent plasticity and subsequent learning that occurs (Figure 1D). Meditation skill will be greater for someone who practices daily for years than someone who practices sporadically. In cognitive–behavioral therapy, skill acquisition and symptom reduction are directly related to the degree of engagement in homework (i.e., skill practice) outside of session (Neimeyer et al., 2008). Notably, experiences that trigger experience-expectant learning early in development (e.g., language input required for phoneme discrimination) can trigger subsequent experience-dependent learning later in development (e.g., learning new words; Werker & Hensch, 2015).

Experience-dependent learning operates through varied mechanisms. Some forms of experience-dependent learning require changes in BDNF in response to experience, whereas others are BDNF-independent (Aarse et al., 2016). Neural changes include modulating the strength of neural connections (Fu & Zuo, 2011) and creating or pruning neural connections, but less extensively than in experience-expectant learning (Trachtenberg et al., 2002). Indeed, the number of new neural connections formed during experience-dependent learning is strongly correlated with behavioral performance on that task (Fu & Zuo, 2011; Xu et al., 2009). Experience-dependent learning can also induce structural changes like new myelination, though to a lesser degree than experience-expectant learning (Mount & Monje, 2017; Takesian & Hensch, 2013). For neural circuitry that has undergone experience-expectant learning, subsequent experience-dependent learning is limited by the neural structure and function established during the sensitive period.

Thus, there is precise mapping of brain and behavioral changes in development depending on age and the experience-driven plasticity mechanisms invoked by particular environmental experiences. This specificity highlights why a nuanced approach to characterizing the environment is critical to uncover how the biobehavioral processes in the RDoC domains develop in ways that underpin psychopathology.

Leveraging Individual Differences in Early-Life Experience to Understand Mechanisms of Plasticity

Although animal models of experience-driven plasticity experimentally manipulate the type and timing of environmental inputs to identify experience-expectant or -dependent changes in neural and behavioral development, such experimental manipulation of environmental experiences is largely infeasible in human studies. Instead, naturally occurring individual differences in the type and timing of experiences can be leveraged to study experience-driven plasticity in humans. The predominant approach to studying these individual differences in processes underpinning the development of psychopathology has relied on children exposed to different forms of early life adversity. This approach can shed light on how the quality, timing, and nature of early experiences influence experience-expectant and experience-dependent learning mechanisms.

Early life adversity involves negative environmental experiences that are either chronic or severe, that reflect a deviation from the expectable environment, and that are likely to require adaptation by a child (McLaughlin, 2016; Nelson & Gabard-Durnam,

experiences, such as interpersonal violence), and deprivation—the absence of expected inputs from the environment, such as social and cognitive stimulation and emotional nurturance (McLaughlin et al., 2014; Sheridan & McLaughlin, 2014). This model posits that experiences of threat and deprivation influence emotion, cognitive, and neurobiological development in ways that are at least partially distinct. As described below, these experiences also have fundamentally different implications for the mechanisms underlying experience-driven plasticity (McLaughlin & Sheridan, 2016; McLaughlin et al., 2014).

Deprivation

Experiences of deprivation provide a unique opportunity to identify sensitive periods of experience-expectant learning and to determine the types of environmental experiences that are required for specific cognitive, emotional, and social capacities to emerge. Animal models of sensory development demonstrate that when the required environmental experience does not occur during a sensitive period, it leads to a dramatic reorganization of neural circuits and behavior that persist once the sensitive period has closed. This suggests that timing of exposure is particularly important when studying forms of adversity involving deprivation.

Although deprivation involving a complete absence of experiential substrates needed to drive plasticity within a sensitive period is relatively rare in humans, it exists in some sensory domains that are experience-expectant, such as vision. For example, sensitive periods in human visual development have been characterized by studying children born with dense cataracts that result in visual deprivation in either one or both eyes (Lewis & Maurer, 2005). Children deprived of exposure to language in early life, either due to being born deaf to hearing parents or in extreme cases of neglect, have similarly revealed sensitive periods in language development. This work along with research on developmental variation in learning a second language (Newport, 1990; Pierce et al., 2014) has convincingly demonstrated that multiple sensitive periods exist for language development during which specific types of environment input are required for normal development (Werker & Hensch, 2015).

Deprivation models can be extended to identify the specific environmental inputs and their timing that are required to scaffold development of processes that have relevance to psychopathology. Much of this work has focused on previously institutionalized children, as the timing of this exposure is well-defined and easily quantified. However, deprivation in social and cognitive inputs—including low levels of cognitive stimulation, exposure to complex language, parental scaffolding of child learning, and environmental enrichment—is commonly experienced among children who are neglected (McLaughlin et al., 2017) and also occurs at higher rates in children raised in poverty than children from families with higher socioeconomic status (Bradley et al., 2001; Romeo et al., 2018; Rosen et al., 2020). Children raised in institutions experience deprivation of many kinds, including exposure to language, supervision and interaction with adults, cognitive stimulation, and learning opportunities (Smyke et al., 2007). Perhaps most profound is the absence of a sensitive caregiver who responds contingently to the child—a type of caregiving necessary for the development of secure attachment (McElwain & Booth-LaForce, 2006). The Bucharest Early Intervention Project—a randomized controlled trial in which some children were removed from deprived orphanage settings and placed in families while others experienced prolonged institutional care—evaluated whether a sensitive period exists for attachment security. Indeed, children removed from institutional care and placed in a family by the age of 22 months were just as likely to develop secure attachment as children raised in families from birth; in contrast, a minority of children placed after 22 months of age developed a secure attachment and were no more likely to become securely attached than children who experienced prolonged institutional rearing (Smyke et al., 2010). This finding suggests the presence of a sensitive period in the first 2 years of life for the development of an attachment to a caregiver, such that a majority of children who experience responsive caregiving during the first two years of life develop secure attachment, whereas a minority of children who experience responsive caregiving for the first time after this period develop secure attachment.

Evaluating whether sensitive periods exist for other processes that contribute to the emergence of psychopathology, such as aversive learning, reward sensitivity, and cognitive control is more challenging than for domains of sensory development or attachment security. Unlike vision, where the required environmental input is relatively obvious, the psychosocial inputs required to scaffold cognitive control, for example, are likely psychosocial in nature, complex, and multifaceted, and as such are not yet fully understood. Moreover, children exposed to psychosocial deprivation in the form of neglect, separation from caregivers, or institutional rearing are typically not completely deprived of social and cognitive stimulation or emotional nurturance. Rather, these children experience infrequent, low-quality, or anomalous inputs (Smyke et al., 2007; see Figure 1B). Determining how deprivation in these types of early experiences shapes the emergence of emotional, cognitive, and social capacities, and associated neural circuit development, can extend models of experience-driven plasticity to the more complex and varied psychosocial experiences that drive development of the biobehavioral processes in the RDoC model.

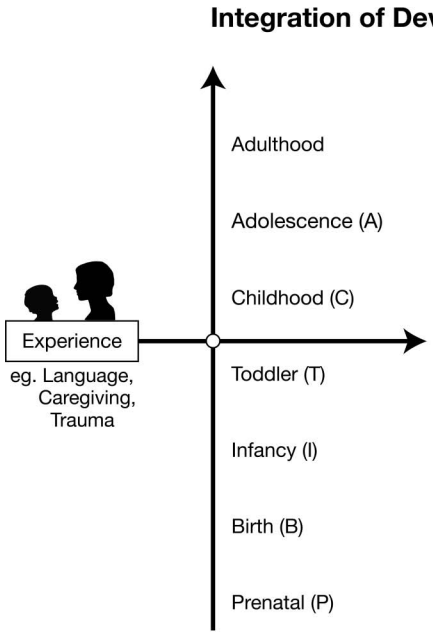
Threat

In contrast to deprivation, exposure to traumatic events that involve a high degree of threat are unlikely to reflect an experiential substrate for which a sensitive period exists. Given that the ability to identify threat cues in the environment is essential for survival, it is unlikely that the ability to learn about sources of threat and mobilize defensive responses to them would develop only when threatening experiences occur during a specific point in development. As such, there is unlikely to be a sensitive period for some negative valence processes (i.e., a specific point in development when the brain "expects" to experience environmental threats). Instead, these processes are most likely experience-dependent.

However, this does not mean that the plasticity mechanisms through which trauma influences development are age-invariant. Indeed, exposure to trauma during childhood is much more likely to produce lasting neural changes than when exposure happens in adulthood (Tottenham & Sheridan, 2010). Scant research has examined whether the timing of exposure within childhood and adolescence is important, although one domain where such timing

Figure 2

Integration of Development and Environmental Experience Into the Research Domain Criteria (RDoC) Framework



RDoC Domain	Unit of Analysis: Experience-Expectant Learning	Experience-Dependent Learning	Genes	Molecules	...
Negative Valence					
Threat Processing Constructs					
Threat Detection	X	All Ages			
Stress Reactivity	I - T	C - A			
Fear Extinction	I - T	C - A			
Emotion Regulation Constructs					
Implicit Regulation	C	B - A			
Explicit Regulation	X	All Ages			
Cognitive Systems					
Perception Constructs	P - I	T - A			
Language					
Discrimination (v. non-language)	P - I	T - A			
Phoneme Discrimination	I	T - A			
Phonological Categories	T	C - A			
Vocabulary	X	All Ages			
Cognitive Control Constructs	?	All Ages			
...					

Note. The current iteration of the RDoC framework as a matrix of domains and units of analysis lacks clear integration of developmental processes or the influence of environmental experience. We present a revised schematic of the RDoC framework that incorporates developmental and experiential contributions to the biobehavioral processes in the matrix through experience-driven plasticity mechanisms. This revised RDoC matrix allows for both the nature and timing of environmental experience to be specified along the developmental axis and also affords the benefit of allowing developmental variation in other units of analysis already included in the model to be directly incorporated into the matrix. Experience-driven plasticity across development is specified as two additional units of analysis that are measurable across all domains in the RDoC framework: experience-expectant learning and experience-dependent learning (negative valence and cognitive systems are illustrated here). Developmental stages in which experience-expectant or experience-dependent mechanisms influence processes within each domain are specified; if a form of plasticity does not occur at a particular stage of development, it is indicated by an "X". Just as cells within the standard RDoC matrix can contain further details (as elements contained within a cell), so too can these additional cells. The learning mechanisms specified as new units of analysis can include additional elements like developmental trajectory charts and biobehavioral processes involved in the learning process at each developmental stage.

ence of a supportive other dampens HPA axis responses to threat (Hostinar et al., 2014). Evidence from children exposed to deprivation involving institutional rearing suggests that the presence of a sensitive and responsive caregiver during a sensitive period in the first 2 years of life may be required for typical development of the HPA axis (McLaughlin, Sheridan, et al., 2015). Children removed from institutional care before the age of two exhibited patterns of cortisol reactivity to stress in late childhood that were no different than children raised in families from birth; in contrast, children placed into families after age 2 exhibited markedly blunted cortisol responses to stress that did not differ from children who remained in prolonged institutional care (McLaughlin, Sheridan, et al., 2015). Interestingly, recent evidence suggests that adolescence may be a second period of developmental plasticity for the HPA axis, where recalibration is possible for children in supportive families even after exposure to early life deprivation (Gunnar et al., 2019).

Negative valence processes also centrally involve a neural network comprised of the amygdala and medial prefrontal cortex (mPFC). Twin studies indicate that the development of this circuit is influenced largely by environmental rather than genetic factors (Achterberg et al., 2018). What remains unclear is which environmental influences on the amygdala-mPFC circuit and associated emotional processes are experience-expectant or experience-dependent (Tottenham & Gabard-Durnam, 2017). Fear extinction processes may reflect experience-expectant learning mechanisms. Early in life, extinction learning is capable of erasing fear memories; by adulthood, extinction produces a competing memory trace but does not eliminate the original fear memory (Kim & Richardson, 2010). The developmental shift from fear erasure to extinction coincides with the formation of perineuronal nets—a key molecular brake on sensitive period plasticity (Gogolla et al., 2009). The degree to which certain salient environmental experiences (e.g., music) are capable of recruiting mPFC and producing anxiolytic effects in animal models also exhibit sensitive period plasticity (Yang et al., 2012), a finding recently replicated in humans (Gabard-Durnam et al., 2018). Moreover, social isolation during this same period has been shown to alter mPFC function and myelination patterns persistently, even with subsequent return to social environments (Makinodan et al., 2012). These findings suggest that some aspects of amygdala-mPFC circuit regulation in humans may be experience-expectant, although the suite of experiential substrates that drive this plasticity remain to be characterized.

and linguistic stimulation in the home environment (Romeo et al., 2018; Rosen et al., 2018, 2020; Sheridan et al., 2012).

Sensitive periods have been clearly established in a range of perceptual domains. In addition to visual acuity and contrast (Lewis & Maurer, 2005), sensitive periods also occur for face perception. Specifically, infants can discriminate between individuals of other species (e.g., macaques) until only 9 months of age (Pascalis et al., 2002), unless they are exposed to faces of other species regularly during the sensitive period (Pascalis et al., 2005). Sensitive periods are also well-documented for language discrimination and perception (Werker & Hensch, 2015). For example, infants are able to discriminate the sounds of all languages before the age of 6 months; by 10 months, infants retain the ability to discriminate only the sounds of their native language (Kuhl et al., 1992). This perceptual tuning is driven by experience and can be altered by exposing infants to a non-native language during the sensitive period (Kuhl et al., 2003; Pierce et al., 2014). Sensitive periods in language development have also been identified for audio-visual matching and phonological categorization (Werker & Hensch, 2015). In contrast, vocabulary development is solely experience-dependent, can be acquired throughout development, and is unique to each individual based on the words they encounter throughout life.

Little is known about the experience-driven plasticity mechanisms that underlie the development of working memory and cognitive control. Although some of the molecular mechanisms regulating sensitive period plasticity have been identified in cortical regions that underlie these higher-order cognitive processes, including the PFC (Larsen & Luna, 2018), it is unclear to what degree these abilities are experience-expectant versus experience-dependent. A central unanswered question is whether there are specific experiential inputs required for the development of executive functions. Given the clear associations of early life deprivation with poor executive functioning, some have argued that social and cognitive stimulation that occurs in the context of early caregiver interactions creates learning opportunities that scaffold the development of these skills (Bernier et al., 2010; McLaughlin et al., 2017; Rosen et al., 2019). Although early life deprivation has lasting influences on executive functioning (Lengua et al., 2015), these abilities and the fronto-parietal networks that support them continue to develop throughout the second decade of life (Luna et al., 2010). These developmental patterns have led some to argue that adolescence is a sensitive period for the development of executive functions (Larsen & Luna, 2018). Consistent with this possibility, evidence from a longitudinal study of previously institutionalized children followed from infancy suggests that the association of the caregiving environment with executive functioning is stronger during adolescence than in childhood (Colich, Sheridan, et al., in press), suggesting heightened plasticity and the possibility for improvement in these abilities during adolescence among those in supportive environments. One intriguing possibility is that multiple sensitive periods exist throughout childhood and adolescence for executive functions. Whether this development is experience-expectant, what the required environmental experiences are, and precisely when in development such inputs are expected, however, remain to be elucidated.

Clinical Relevance of an Experience-Driven Plasticity Framework

Incorporating experience-driven learning mechanisms into the RDoC framework will not only foster progress in characterizing the developmental mechanisms that contribute to psychopathology and the specific types of environmental experiences that scaffold these biobehavioral processes, but also has direct implications for identifying intervention targets.

Substantial progress has been made in characterizing the emotional, cognitive, social, and neurobiological processes that contribute to the emergence of psychopathology. Progress has lagged considerably behind, however, in specifying the core underlying dimensions of environmental experience that influence the development of these biobehavioral mechanisms. Greater research into patterns of experience-driven plasticity will shed light on the specific types of environmental experiences that shape these processes, and the experience-expectant and experience-dependent mechanisms that drive their development. Such an approach also has relevance for intervention development to prevent the onset of psychopathology. The intervention approach inherent in the RDoC framework emphasizes targeting underlying mechanisms (e.g., blunted sensitivity to reward or attention biases toward threat) that contribute to the onset and maintenance of psychopathology. This mechanistic approach is essential for developing novel treatments and early interventions. However, it does not present easy options for screening and identifying people who may be at risk for psychopathology, as measuring these mechanisms often requires behavioral tasks, biological assays, or neuroimaging measures. Greater understanding of the environmental determinants of these biobehavioral processes may present far more realistic targets for screening and early identification (McLaughlin, DeCross, et al., 2019). For example, blunted sensitivity to reward occurs in depression and prospectively predicts depression onset (Gotlib et al., 2010; Nelson et al., 2016). Behavioral activation targets this reduced motivation to pursue pleasant activities directly (Dimidjian et al., 2006), and thus may be a promising approach for preventing depression. However, determining who might benefit from this type of behavioral intervention through screening with behavioral or neuroimaging tasks is challenging. As noted earlier, accumulating evidence demonstrates that early life social and cognitive deprivation is strongly linked to this pattern of blunted reward sensitivity (Sheridan et al., 2018). Of course, not all children exposed to deprivation (or with blunted reward sensitivity) will go on to develop depression, but screening for exposure to neglect, food insecurity, low cognitive stimulation, and other forms of deprivation (e.g., within pediatric primary care or early childcare settings) may be more feasible in terms of identifying those who could benefit from these early interventions than screening for blunted reward processing.

Perhaps most importantly, progress in understanding patterns of experience-driven plasticity will inform not only who is in need of early intervention but *when* such interventions might be maximally efficacious. We provide several illustrative examples. First, evidence for a sensitive period in the first 2 years of life for the development of an attachment to a caregiver (Smyke et al., 2010) suggests that parenting interventions aimed at improving sensitive and responsive caregiving may have the most pronounced effects when administered during this developmental window. In contrast,

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Received May 8, 2020
Revision received July 31, 2020
Accepted September 1, 2020 ■