The neurodynamics of emotion: delineating typical and atypical emotional processes during adolescence

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Abstract

The study of development is, in and of itself, the study of change over time, but emotions, particularly emotional reactivity and emotional regulation, also unfold over time, albeit over briefer time-scales. Adolescence is a period of development characterized by marked changes in emotional processes and rewiring of the underlying neural circuitry, making this time of life formative. Yet this period is also a time of increased risk for anxiety and mood disorders. Changes in the temporal dynamics of emotional processes (e.g. magnitude, time-to-peak and duration) occur during this developmental period and have been associated with risk for mood and anxiety disorders. In this article, we describe how the temporal dynamics of emotions change during adolescence and how they may increase risk for these psychopathologies. We highlight studies that illustrate how formalizing temporal neurodynamics of emotion may enhance links among levels of analyses from neurobiological to real-world, moment-to-moment experiences.

Research highlights

- Adolescence is a period of dynamic changes in emotional reactivity and regulation.
- The varying time-scales of these emotional processes map onto changes in cortical-subcortical circuitries.
- Formalizing the temporal dynamics of emotional processes may enhance links among levels of analysis, connecting neural circuits to behavior and real-word assessment of emotion.

Introduction

The emotional lives of adolescents can be erratic and unpredictable. Some teens show outwardly labile emotions, swinging from one mood to another in a matter of seconds or minutes. Others experience long bouts of sulking, ruminating or savoring over negative or positive thoughts for days and some retreat inwardly, inhibiting outward expressions of emotions over time despite strong internal feelings. The dynamic changes of these emotions in adolescents over time, whether expressed outwardly or not, vary by individual. We highlight studies that illustrate how temporal dynamics of emotional processes may inform our understanding of typical and atypical emotional processes. We focus on the period of adolescence – the developmental period when mood and anxiety disorders that involve maladaptive emotional processes peak in their prevalence (Casey, Oliveri & Insel, 2014).

Intense and frequent emotions are common during the early adolescent years (Larson, Moneta, Richards & Wilson, 2002; Steinberg, 2005). Learning to regulate these emotions without the buffer of the parent is thought to be a key milestone of this period as the adolescent prepares for adulthood (Casey, 2015). Emotion regulation refers to the capacity to redirect attention toward or away from emotional cues (Monk, 2008) or to reappraise emotional information and feelings (Silvers, McRae, Gabrieli, Gross, Remy et al., 2012). It has been described as a goal-orientated process (Frijda, 1988; Izard, 2009) requiring cognitive resources to be effective (Sheppes & Gross, 2011). An important feature of emotion regulation is that emotions are not fixed but rather unfold over time (Davidson, 1998; Solomon & Corbit, 1974). As Thompson (1994) notes, 'emotion regulation consists of the extrinsic and intrinsic processes

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responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and *temporal* features, to accomplish one's goals' (Thompson, 1994, pp. 27–28, emphasis added). Understanding the temporal dynamics of emotional processes may inform the cognitive capacity required to regulate an emotional episode (Schmeichel, 2007) and why one person is able to flexibly and adaptively regulate emotion while another cannot (Waugh, Shing & Avery, 2015).

In daily life, emotional regulation occurs in anticipation of emotional episodes as much as in response to them (i.e. anticipatory vs. reactive emotional regulation), as individuals sometimes ready themselves for expected affective events (Gross, 2011). Prolonged experience of an emotion is referred to as a mood state (Russell, 2003) and can become pathological depending on its duration and frequency (American Psychiatric Association, 2013). In this paper we describe how changes in basic temporal dynamics of emotions and their neural substrates during adolescence may give rise to pathological emotions.

Defining temporal neurodynamics of emotion

The neurodynamics of emotions unfold over seconds to hours. In their simplest form, emotions can be reduced to temporal parameters of the magnitude, rise-time, duration and habituation across repeated episodes (Davidson, 1998). Reducing the complex constructs of emotion into these simpler parameters affords mapping of core processes to their underlying neural substrates (Thompson, 1994). This approach builds on the framework of cognitive neuroscience (Gazzaniga, 2004). For example, the cognitive neuroscience of memory has been often divided into temporal parameters, separately examining the encoding, maintenance and retrieval of specific memories (Schneider & Pressley, 2013). This type of approach has demonstrated that the magnitude of prefrontal cortical activity during encoding predicts the later recall of information (Kirchhoff, Wagner, Maril & Stern, 2000; Wagner, Schacter, Rotte, Koutstaal, Maril et al., 1998), and that sustained dorsolateral prefrontal activity during delay periods predicts working memory performance (Braver, Cohen, Nystrom, Jonides, Smith et al., 1997; Cohen, Perlstein, Braver, Nystrom, Noll et al., 1997). Similarly, sustained experiences of emotion in the absence of an emotional stimulus engages sustained medial prefrontal cortex in adults (Waugh, Lemus & Gotlib, 2014). Advances in assessing affective neurodynamics are being facilitated by recent developments in statistical and methodological approaches to neuroimaging (e.g. Lindquist, Meng Loh, Atlas & Wager, 2009).

The human capacity to modulate emotions relies upon neural circuits that amplify and attenuate affective states. Frontolimbic circuitry implicated in emotion involves detection, processing and suppression of both positive and negative information. Traditionally, detection and processing of emotional information has been assigned to specific regions of the brain by valence, with the ventral striatum and amygdala being assigned to reward and threat detection, respectively. Yet converging animal (Paton, Belova, Morrison & Salzman, 2006), human imaging (Delgado, Nystrom, Fissell, Noll & Fiez, 2000; Levita, Hare, Voss, Glover, Ballon et al., 2009), and computational (Li, Schiller, Schoenbaum, Phelps & Daw, 2011) evidence suggests that these regions may not be valence specific, but rather specific to learning adaptive responses to positive and negative events. Regions of the prefrontal cortex (PFC) are thought to play a central role in regulating and suppression of emotional responses to these events (Ochsner & Gross, 2005; Ochsner, Silvers & Buhle, 2012).

Evidence from event related potential (ERP) components have indicated the rapid time-to-peak for the processing of emotion by revealing that there are early ERP components (~115 ms) signaling when emotional information is detected (Meeren, van Heijnsbergen & Gelder, 2005). Part of this rapid ERP component in response to emotional information is likely to resolve ambiguity (i.e. the valence of the stimulus; Olofsson, Nordin, Sequeira & Polich, 2008). Affective cues rapidly engage subcortical circuits including the amygdala and ventral striatum in which appraisal of the stimulus and coordinating adaptive behavioral output is paramount (Davis & Whalen, 2001; Haber & Knutson, 2010; Phelps & LeDoux, 2005).

Characterization of changes in the magnitude and habituation of neural responses over time using functional magnetic resonance imaging (fMRI) has begun to contribute to our understanding of emotional reactivity and regulation. For example, using positive and negative cues, Levita (Levita et al., 2009) found activity in both the ventral striatum and amygdala to cues of both valences. However, habituation to these cues, specifically to negative ones, was associated with symptoms of anxiety, not mean magnitude or time-to-peak. Time-to-peak in activity differed between these subcortical regions with activity in the ventral striatum peaking later than in the amygdala. This pattern is consistent with animal work confirming an unidirectional projection from the basolateral nucleus of the amygdala to the ventral striatum (Haber & Knutson, 2010) that has been associated with approach-related behavior irrespective of the valence of a cue (Stuber, Sparta, Stamatakis, van Leeuwen, Hardjoprajitno et al., 2011), while projections

from the basolateral nucleus to the central nucleus of the amygdala have been associated with withdrawal or freezing behavior (Davis & Whalen, 2001; LeDoux, 2000; Nieh, Kim, Namburi & Tye, 2013).

The behavioral response of approaching or withdrawing from positive and negative stimuli is thought to play a significant role in the development of adaptive and maladaptive emotional behavior (Casey, 2015). Specifically, adolescence has been linked to a time of reactivity and approach to emotional triggers, regardless of valence (Dreyfuss, Caudle, Drysdale, Johnston, Cohen *et al.*, 2014; Somerville, Hare & Casey, 2011), especially in males (Grose-Fifer, Rodrigues, Hoover & Zottoli, 2013). In contrast, habituation of this response to negative cues with repeated presentations has been associated with anxiety in adolescents who show less habituation of amygdala activity (Hare, Tottenham, Galvan, Voss, Glover *et al.*, 2008). Thus, examining the temporal neurodynamics of emotion provides insights for understanding the emergence of typical as well as pathological emotional processes during adolescence.

Development of temporal neurodynamics of emotion

The unfolding of emotions in response to stimuli – positive or negative, brief or prolonged – is embedded within lengthier developmental dynamics. Developmental changes across time frames of weeks, months and years are supported by neural and psychological processes that unfold over briefer time-scales of milliseconds to minutes. Figure 1 illustrates how distinct emotional neurodynamics may emerge during adolescence. Early in adolescence, subcortical circuitry may dominate emotional responses, as evidenced by heightened magnitude of subcortical regions in response to emotional cues. These subcortical dynamics are more resistant to regulation as they are associated with cortical afferents of briefer duration earlier in development (McRae, Gross, Weber, Robertson, Sokol-Hessner *et al.*, 2012; Silvers, Shu, Hubbard, Weber & Ochsner, 2015). As individuals enter early adulthood, the subcortical dynamics of early adolescence are modulated by emerging and more persistent cortical regulatory engagement (Casey, 2015; Ernst & Fudge, 2009). These more persistent cortical afferents are thought to facilitate improved emotional and behavioral regulation characteristic of adulthood.

The majority of functional imaging studies on the neural dynamics of emotion during adolescence have focused on the magnitude of responses to relatively brief emotional stimuli and tracked the neural habituation of this response over repeated presentations. Early work focused largely on negative affect. For example, Baird *et al.* (Baird, Gruber, Fein, Maas, Steingard *et al.*, 1999) showed activity in the amygdala to repeated passive presentation of fearful faces in adolescents. Later studies showed heightened amygdala responses to these cues of potential threat during adolescence relative to adults (Guyer, Monk, McClure-Tone, Nelson, Roberson-Nay *et al.*, 2008; Hare *et al.*, 2008; Monk, McClure, Nelson,
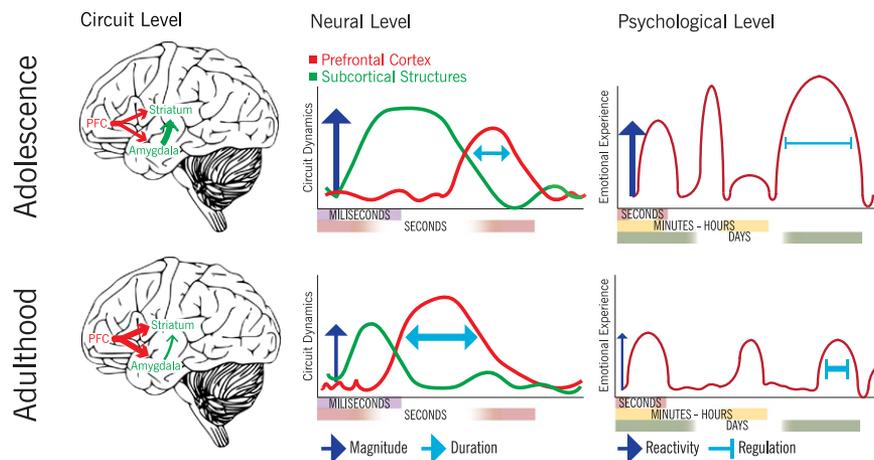

Figure 1 *A model of adolescent development of emotional temporal neurodynamics. At the circuit level, maturation of cortico-subcortical circuitry from adolescence to adulthood involves increased prefrontal input to subcortical circuitry. At the neural level, developmental changes in frontoamygdala connectivity are proposed to lead to more effective regulation of subcortical limbic regions via more sustained PFC engagement. At the psychological level, rapid fluctuation in emotions and moods during adolescence is attributed to less capacity to regulate heightened emotional reactivity.*

Zarahn, Bilder *et al.*, 2003). Across development, adolescents have exhibited a general pattern of heightened amygdala activity and slower behavioral responses to fearful faces as compared to children and adults (Hare *et al.*, 2008). This finding is consistent with studies of emotional reappraisal, suggesting increasing emotion regulation capacity from adolescence to adulthood (McRae *et al.*, 2012; Silvers *et al.*, 2015).

Examining the temporal dynamics of these responses over time (e.g. habituation of the amygdala response with repeated presentations of potential threat or extinction of a fear memory) implicates the ventromedial prefrontal cortex (vmPFC) as a key regulator of sustained amygdala activation (Phelps, Delgado, Nearing & LeDoux, 2004). Decreased activity in the amygdala with repeated presentation of empty threat has been associated with greater negative coupling within fronto-amygdala circuitry such that individuals with greater habituation of amygdala activity show more negative coupling between the vmPFC and the amygdala (Hare *et al.*, 2008). Tract tracing studies in rodents suggest that this inverse association between vmPFC and the amygdala represents greater suppression of amygdala activity via descending projections from the vmPFC (Amaral, Price, Pitkanen & Carmichael, 1992; Bouwmeester, Smits & Van Ree, 2002a; Bouwmeester, Wolterink & van Ree, 2002b; Dincheva, Pattwell, Tessarollo, Bath & Lee, 2014; Ghashghaei, Hilgetag & Barbas, 2007). These results and other recent developmental work (Gee, Gabard-Durnam, Flannery, Goff, Humphreys *et al.*, 2013a; Gee, Humphreys, Flannery, Goff, Telzer *et al.*, 2013b) highlight the importance of developmental changes in connectivity within fronto-amygdala circuitry in the modulation of heightened emotional responses during the period of adolescence.

Similar developmental findings regarding the magnitude of subcortical responses to threat have been reported in the context of rewards during adolescence (Barkeley-Levenson & Galván, 2014; Bjork, Knutson, Fong, Caggiano, Bennett *et al.*, 2004; Bjork, Smith, Chen & Hommer, 2010; Cohen, Asarnow, Sabb, Bilder, Bookheimer *et al.*, 2010; Ernst, Pine & Hardin, 2006; Galvan, Hare, Davidson, Spicer, Glover *et al.*, 2005; Galvan, Hare, Parra, Penn, Voss *et al.*, 2006; Galván & McGlennen, 2013; Geier, Terwilliger, Teslovich, Velanova & Luna, 2009; van Leijenhorst, Zanolie, Meel, Westenberg, Rombouts *et al.*, 2010). One of the first studies to test how reward-related neural processing occurs in adolescents relative to both children and adults (Galvan *et al.*, 2005, 2006) used a variant of a reward paradigm previously used in nonhuman primates to measure reward signals in dopamine rich neural circuitry. The amount of reward varied from small to large. There was an effect of reward in the ventral striatum (VS) and

orbitofrontal cortex (OFC) whereby these regions showed the most activity to the largest monetary reward (Galvan *et al.*, 2005, 2006). The effect was exaggerated in the ventral striatum for adolescents relative to both children and adults (Galvan *et al.*, 2006). This finding has been replicated across numerous labs (Cohen *et al.*, 2010; Ernst *et al.*, 2006; Geier *et al.*, 2009; van Leijenhorst *et al.*, 2010) and parallels the developmental evidence of an exaggerated amygdala response in adolescents to aversive stimuli (Hare *et al.*, 2008; Monk *et al.*, 2003). Finally, there was a monotonic decrease in the magnitude of PFC activity to reward with age and a decrease in response times specifically to cues that predicted the largest reward (Galvan *et al.*, 2005, 2006). Together these findings suggest an enhanced capacity to integrate valenced-information into appropriate action with development of frontolimbic circuitry.

Individual differences in the neurodynamics of emotion

Although as a group, adolescents tend to show intense and frequent emotions, emotional responses and their temporal dynamics vary by individual (Kosslyn, Cacioppo, Davidson, Hugdahl, Lovallo *et al.*, 2002; Underwood, 1975). Variation in how an adolescent reacts to emotional information can be adaptive (promoting well-being) or maladaptive (resulting in psychopathology). For example, persistent avoidance of cues that no longer signal a threat is a characteristic feature of anxiety and may rely on engagement of the amygdala and bed nucleus of the stria terminalis (Davis, Walker, Miles & Grillon, 2010). This pattern may generalize to emotional cues of both valences. Specifically, avoidance of potentially negative information or outcomes can lead to a pattern of behavior which in turn leads to missing opportunities for experiencing positive outcomes. This can become a habitual behavioral pattern and a pathway by which anxiety may precede anhedonic symptoms of depression (Fava, Rankin, Wright, Alpert, Nierenberg *et al.*, 2000). This work highlights the value of focusing on the behavioral output of the organism in addition to the incoming valence of the incoming information.

Adolescents' idiosyncratic life experiences, coupled with genetic predispositions, influence how brain circuits (e.g. subcortico-subcortical, cortico-subcortical and cortico-cortical networks) interact, and how they may give rise to individualized psychological and neurobiological trajectories (Masten & Cicchetti, 2010). Emotional experiences can modulate the strength of specific networks involved in emotion and emotional regulation. The repeated engagement of these networks as a result of

specific experiences can lead to approach and avoidance tendencies to emotional situations. With development, these networks become more fine-tuned (Dosenbach, Nardos, Cohen, Fair, Power et al., 2010; Fair, Cohen, Power, Dosenbach, Church et al., 2009) and the responses become more automatic, regardless of whether these responses are adaptive in the long-term.

Figure 2 provides a model for how changes in temporal neurodynamics of emotion may increase the risk for, and susceptibility to, psychopathologies that often emerge and/or peak during adolescence. Abnormalities in the persistence and the magnitude of activity in specific subcortical and cortical circuits are hypothesized to underlie susceptibility to anxiety and anhedonia. While there are several temporal parameters of emotion, much of the work to date focuses on the magnitude of brief emotional responses to transient cues at a moment in time or with repeated presentations over time. Yet other temporal parameters such as the duration of an emotional response (e.g. 'ruminating' for sustained negative affect and an inability to 'savor' positive experiences) often characterize psychiatric symptoms. The Diagnostic Statistical Manual (DSM), for example, explicitly incorporates the nature of duration in its definition of depression, stating that 'the essential feature of a Major Depressive Episode is a period of at least 2 weeks during which there is

either depressed mood or the loss of interest or pleasure in nearly all activities' (American Psychiatric Association, 2013). The diminished capacity to regulate limbic subcortical networks via cortical afferents is thought to underlie such aberrant emotions (Ochsner et al., 2012) and be at the very core of many affective disorders (Davidson, 1998) that emerge during adolescence (Casey, 2015; Silk, Steinberg & Morris, 2003).

Developmental timing also interacts with individual differences in emotional neurodynamics. The time at which pubertal maturation occurs influences emotional reactivity (Quevedo, Benning, Gunnar & Dahl, 2009; Silk, Siegle, Whalen, Ostapenko, Ladouceur et al., 2009; Susman, Inoff-Germain, Nottelmann, Loriaux, Cutler et al., 1987), stress reactivity (Stroud, Foster, Papandonatos, Handwerger, Granger et al., 2009), and the likelihood of risk-taking behaviors (Downing & Bellis, 2009). Early puberty is associated with an increased risk for depression (Ge & Natsuaki, 2009; Natsuaki, Biehl & Ge, 2009), an effect that is more pronounced among females than males (Hankin, Abramson, Moffitt, Silva, McGee et al., 1998). Late pubertal timing appears to heighten the risk for depression in males (Natsuaki et al., 2009). This growing literature underscores how developmental processes specific to adolescence interact with emotional dynamics which together give rise to the
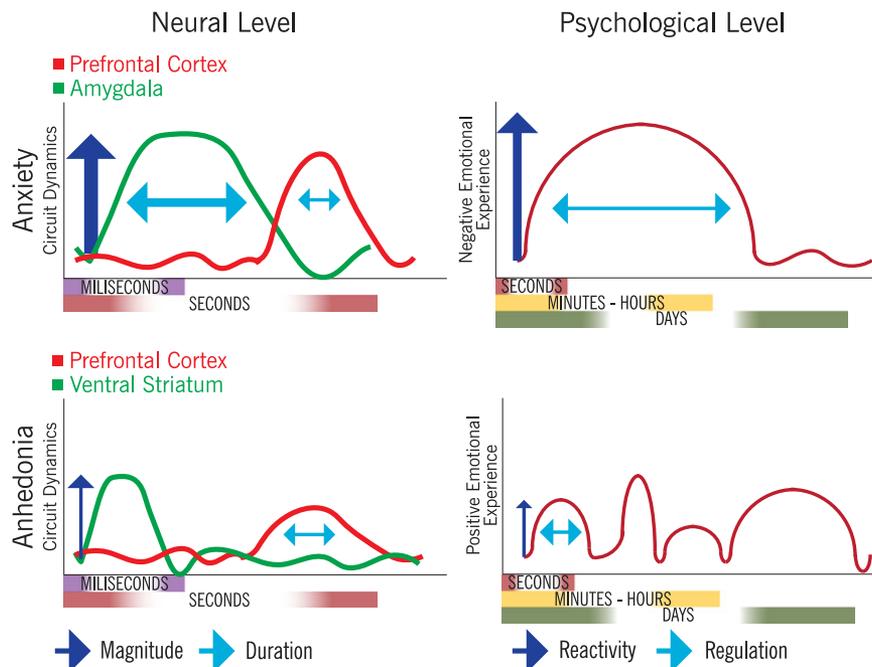

Figure 2 *A model of individual differences in the temporal neurodynamics of emotion. In response to aversive stimuli, those at-risk for anxiety disorders may show heightened amygdala activity that is sustained with delayed or reduced prefrontal activity. In response to appetitive stimuli, those at-risk for anhedonia may show lower magnitude and less persistent ventral striatal activity coupled with delayed or reduced prefrontal engagement.*

individual differences during this period (Arnett, 1999; Casey *et al.*, 2014).

Most studies of individual differences in emotion regulation examining temporal dynamics have focused on habituation of emotional responses to repeated presentations of brief emotional cues over time. For example, in the developmental study by Hare *et al.* (2008) (Figure 3 [panels: a–c]), the temporal dynamics of the neural responses to cues of threat were associated with self-reported anxiety. Specifically, decreases in amygdala activity to fearful face stimuli with repeated presentations over time were negatively associated with self-reports of trait anxiety, such that adolescents with less habituation (more sustained amygdala activity) reported higher trait anxiety. As mentioned earlier, habituation of amygdala activity is associated with greater negative coupling within fronto-amygdala circuitry. This finding is consistent with several studies showing associations between altered fronto-amygdala connectivity and anxiety (Bishop, Duncan & Lawrence, 2004; Davidson, 2002; Davis, 2006; Kim, Loucks, Palmer, Brown, Solomon *et al.*, 2011; Somerville, Kim, Johnstone, Alexander & Whalen, 2004).

Further evidence for the role of temporal neurodynamics in the risk for anxiety disorders comes from a study by Blackford and colleagues (2009). They showed that behaviorally inhibited children (a risk factor for social and generalized anxiety disorders) had a more rapid onset (i.e. time-to-peak) and prolonged duration of amygdala activity to neutral faces than those who were not behaviorally inhibited as children (Blackford, Allen, Cowan & Avery, 2013; Blackford, Avery, Shelton & Zald, 2009). Lau (Lau, Guyer, Tone, Jenness, Parrish *et al.*, 2012) has found similar effects in amygdala responses of anxious adolescents. These anxious adolescents have prolonged amygdala reactivity compared with healthy
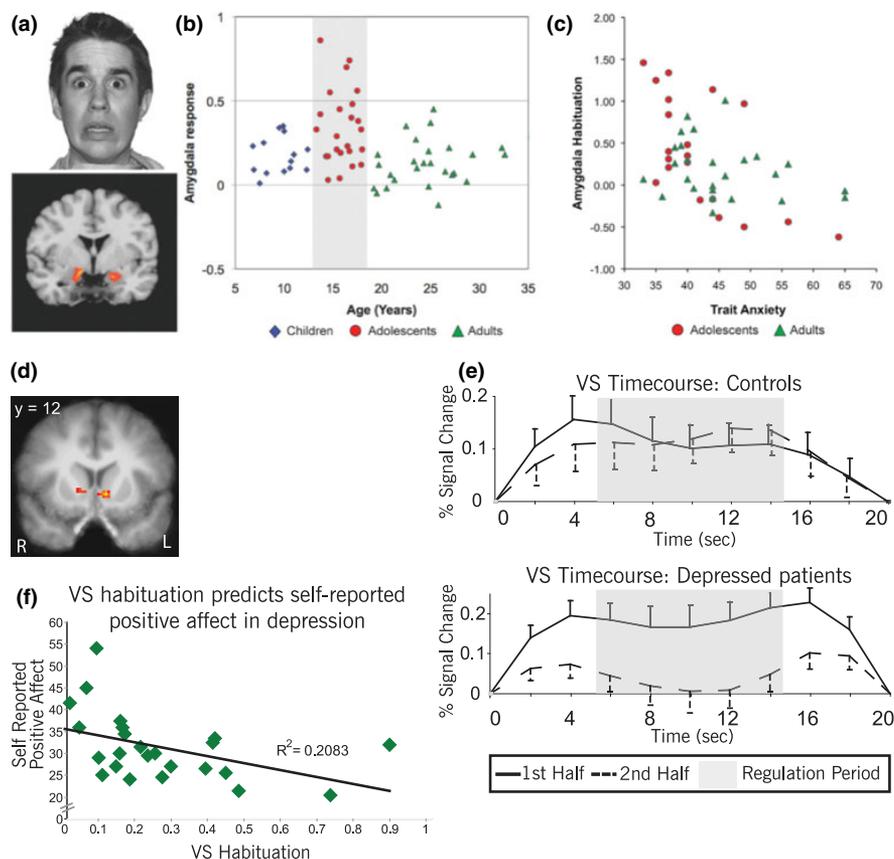

Figure 3   *Individual differences in the temporal neurodynamics of emotion. (a) Amygdala activity when detecting fearful faces is (b) elevated among healthy adolescents compared to children and adults and the lack of (c) habituation of this activity is associated with trait anxiety in adolescents (adapted from Hare et al., 2008). (d) Habituation of ventral striatal (VS) activity in response to appetitive images is greater for depressed adult patients (Heller et al., 2009) as shown in the (e) time course of VS activity for healthy controls and depressed patients (error bars indicate SEM). (f) The greater the habituation of VS activity the less self-reported daily positive emotion in depressed patients.*

controls following evaluations by peers. Overall, these data suggest that anxiety and risk for anxiety are associated with sustained amygdala responses.

Atypical patterns of fronto-amygdala activity have been implicated in depression as well. Amygdala reactivity in response to negative words appears to be more temporally sustained in depressed adult patients compared with healthy controls (Siegle, Thompson, Carter, Steinhauer & Thase, 2007). Depressed patients show sustained processing of negative information (Siegle, Granholm, Ingram & Matt, 2001) and delayed amygdala recovery after being exposed to idiosyncratic negative stimuli (Siegle, Steinhauer, Thase, Stenger & Carter, 2002). Sustained amygdala activity is also associated with trait rumination (Mandell, Siegle, Shutt, Feldmiller & Thase, 2014) – a risk factor in the development of depression (Nolen-Hoeksema, Wisco & Lyubomirsky, 2008). Together the evidence suggests that sustained amygdala activity to aversive cues predicts development of both depression and anxiety. This observation is consistent with the strong comorbidity between anxiety and depressive disorders (Brady & Kendall, 1992) and the tendency for anxiety to precede the onset of a depressive episode (Fava et al., 2000). However, how these symptomatically distinct disorders differ in affective neurodynamics could identify novel treatment targets.

Unlike the literature on anxiety disorders, the literature on mood disorders indicates that there are alterations in responses to positive-valenced cues and rewards, in addition to those involving negative-valenced cues. Most of these studies focus on anhedonia and the magnitude of regional brain activity to rewards rather than on temporal dynamics per se. For example, Telzer and colleagues examined the association between changes in depressive symptoms in adolescents over a one-year period and the magnitude of ventral striatum (VS) responses (Telzer, Fuligni, Lieberman & Galván, 2014). Those adolescents with the greatest VS activity when making pro-social (i.e. other-centered) compared with self-centered decisions were those who showed the largest decrease in depressive symptoms over a one-year follow-up. In addition to suggesting that the magnitude of VS activity may predict subsequent development of depressive symptoms in adolescents, these findings suggest that reward type is an important consideration when examining individual differences in neural dynamics of emotions.

A separate set of studies examining temporal dynamics of depression highlights the important role for habituation of reward-related circuits in psychopathology and presents a potential avenue for examining developmental processes. In a recent reappraisal study of adults with Major Depressive Disorder (Heller, Johnstone, Shackman, Light, Peterson et al., 2009), Heller showed that depressed patients relative to healthy volunteers had more rapid habituation of VS activity when upregulating (i.e. increasing) positive emotion. Depressed patients also displayed more rapid decoupling of VS-DLPFC connectivity compared with healthy controls when increasing positive emotion in response to viewing positively-valenced images (Figure 3[panels d-f]; Heller et al., 2009). The degree of habituation in VS activity correlated with self-reported positive affect in daily life with depressed patients who reported higher levels of positive affect showing less VS habituation. A second study followed these depressed patients over two months of antidepressant treatment (Heller, Johnstone, Light, Peterson, Kolden et al., 2013). Following pharmacological treatment, those patients making the greatest improvements in positive affect over the two months showed the largest reductions in habituation of VS activity and VS-PFC coupling (Heller et al., 2013). Critically, the effects reported above were not present when examining the mean activity (magnitude) across the scan session – the typical metric used in brain imaging analyses. Although this study was performed in adults, these findings highlight the utility of examining temporal dynamics of regulation of reward responses in psychopathology and how they map onto individual differences.

While VS activity to positively-valenced cues and outcomes has been associated with internalizing disorders, this neural signal has been associated with externalizing behaviors (e.g. risk taking) during adolescence as well (Chein, Albert, O'Brien, Uckert & Steinberg, 2011; Galvan, Hare, Voss, Glover & Casey, 2007). Specifically, heightened VS activity to potentially rewarding cues and situations has been associated with the higher likelihood of engaging in risky behavior during adolescence (Galvan et al., 2007, Chein et al., 2011). Although risk-taking behavior such as experimentation with substances can be normative, this behavior becomes pathological for some with subsequent craving and dependence following use (see, e.g. Crews, He & Hodge, 2007; Jacobus & Tapert, 2013; Luciana, 2013; Padmanabhan & Luna, 2014; Spear, 2000; Steinberg, 2004; US Department of Health and Human Services, 1999). Bechara (2005) has proposed an imbalance model of addiction that is reminiscent of the imbalance model of adolescence (Casey, Getz & Galvan, 2008). Specifically, Bechara (2005) suggests that neural circuitry signaling potential pain or pleasure is hyperactive in individuals with addiction. Accordingly, heightened activity in these brain regions can trigger involuntary signals that modulate, bias or even hijack the goal-driven cognitive resources supported by the prefrontal cortex. This imbalance between limbic subcortical or prefrontal cortical regions is thought to impair the capacity for self-control in adolescents, but in addiction it impairs the

capacity to resist drugs. Other theories of addiction similarly suggest the importance of sustaining emotional regulation in the face of craving substances (Baker, Piper, McCarthy, Majeskie & Fiore, 2004).

Together, these studies imply a significant role of sustained neural signals to salient emotional cues, regardless of valence, during adolescence that can give rise to different psychopathologies. A recent series of naturalistic lab-based studies have examined sustained positive and negative emotion referred to as 'emotional inertia' (Kuppens, Sheeber, Yap, Whittle, Simmons et al., 2012; Kuppens, Allen & Sheeber, 2010), and is defined by how long an emotion is sustained (i.e. the autocorrelation of emotional experience over time). Young adolescents were videotaped as they engaged in a 20-minute lab-based interaction with a parent. Behavioral coding of nonverbal affect and verbal content provided an estimate of both positive and negative emotional inertia. More persistent positive and negative emotional inertia predicted lower self-esteem ratings and risk of having depressive symptoms over two years later (Kuppens et al., 2012). This nonspecific valence effect of persistent positive and negative emotions predicting maladaptive outcomes suggests that difficulty regulating emotions over time, irrespective of valence, can be detrimental. This is consistent with models of emotion duration in psychopathology (Gruber, Eidelman, Johnson, Smith & Harvey, 2011) and our model of individual differences in the neurodynamics of emotion in development.

Linking levels of analysis: from biological to real-world measures of emotional neurodynamics

Formalizing emotions using temporal neurodynamics may help to link emotion processes of different time-scales across levels of analysis. A method that is particularly well suited for understanding how emotional dynamics change over prolonged time frames during adolescence is Ecological Momentary Assessment (EMA), often referred to as experience sampling. EMA is a method that can acquire self-report, arousal, location, ambient volume as well as other measures as individuals traverse their everyday life (Kaplan & Stone, 2013). Recent diary studies confirm that real-world emotions can last from a few seconds up to several hours (Verduyn, Delaveau, Rotgé, Fossati & Van Mechelen, 2015; Verduyn, Van Mechelen & Tuerlinckx, 2011; Verduyn & Lavrijsen, 2015).

EMA developmental research has found that adolescence, as compared to childhood and adulthood, is a period of increased variability in daily positive and

negative emotion (Larson, Csikszentmihalyi & Graef, 1980; Larson & Lampman-Petraitis, 1989). Moods become progressively more negative in early adolescence but this trend ceases around age 15 (Larson et al., 2002). Emotional variability declines in late adolescence and for most individuals this variability stabilizes in early adulthood (Larson et al., 2002). Stress has been shown to moderate these early to late-adolescent effects such that stressful life events are associated with greater real-world emotional instability across adolescence (Larson et al., 1980, 2002). This emotional lability has been suggested to amplify adolescents' vulnerability to the many emotional challenges they face (Gunnar, Wewerka, Frenn, Long & Griggs, 2009).

Within the period of adolescence there is significant variability in responses to emotional cues or events. EMA measures have been used to capture this variability in predicting risk for anxiety and mood disorders. For example, Silk and colleagues (Silk et al., 2003) found that adolescents who had greater difficulty regulating their negative affect (i.e. more prolonged self-reported negative emotion) in response to naturalistic stressors reported greater depressive symptom severity. In response to challenging events, children and adolescents with anxiety disorders report higher peak intensity of real-world negative emotion as compared with control youths (Tan, Forbes, Dahl, Ryan, Siegle et al., 2012). Similarly, children and adolescents with anxiety and depressive disorders who report higher real-world positive emotion in their daily lives show better treatment responses to Cognitive Behavioral Therapy (CBT; Forbes, Stepp, Dahl, Ryan, Whalen et al., 2012).

How do these measures of real-world emotional responses over minutes to hours relate to temporal neurodynamics of milliseconds to seconds? Forbes and colleagues examined associations between real-world emotion and brain activity in adolescents in the context of positive emotion (Forbes, Hariri, Martin, Silk, Moyles et al., 2009; Forbes, Ryan, Phillips, Manuck, Worthman et al., 2010). They found that the magnitude of striatal activity to winning money was predicted by real-world baseline positive emotion and was negatively correlated with depressive symptoms. They further found that medial PFC activity to winning money was positively correlated with depressive symptoms. This finding (Forbes et al., 2010) was moderated by pubertal status such that a more advanced pubertal status was associated with less striatal and more medial PFC reactivity.

In our own work we have combined EMA with imaging to examine associations between individual differences in real-world positive emotion persistence with the duration of reward-related activity (Figure 4; Heller, Fox, Wing, McQuisition, Vack et al., 2015). We

extended current EMA methodology to develop a real-world task whereby adult participants played a game each day in which they won (or did not win) money. This permitted a level of experimental control that had not been incorporated into EMA designs. To reconstruct each participant's individual positive emotion time-course, EMA sampling occurred frequently for 90 minutes after the game (every 10–12 minutes). Adults who sustained positive emotion the longest when winning $15 in their day-to-day life (over the course of minutes and hours) were those with the longest duration of ventral striatal activity when winning money (on the order of seconds). This study highlights one way of integrating emotional neurodynamics across multiple time-scales and levels of analysis that combines data from experimentally controlled studies in both real-world and laboratory-based settings.

In the future, EMA approaches will be an important assessment tool of an individual's functioning as they go about their daily life, especially as adolescents are facile with mobile device technology. These devices may be used to measure fluctuations in arousal, location, and self-reported emotion, all with minimal intrusion to individuals in the real world, in real time, as they go about their lives. EMA approaches can supplement current assessments of mental health, which require individuals to reflect over many weeks – a process that often does not reflect the person's true level of functioning (see, e.g. Redelmeier & Kahneman, 1996). The long-term potential of these technologies could be in supplementing psychotherapy, assessing medication compliance and providing remote anticipation of critical mental health events such as suicidality.

Converging methods approaches to understanding the neurodynamics of emotion

There are a variety of experimental methods (fMRI, ERP, pupil dilation, etc.) for examining temporal neurodynamics of emotional processes on the milliseconds-to-minute time-scale across development. While specific physiological components rarely exceed a few minutes, emotional experiences can persist for far longer (Nolen-hoeksema & Morrow, 1993; Verduyn et al., 2011). Thus, approaches that bridge multiple methods across these time-scales may elucidate the development of emotional dynamics.

Figure 5 illustrates a converging approach to the study of emotional neurodynamics. This figure separates imaging and behavioral methods for examining a variety of emotional and cognitive psychological processes. The temporal resolution of the imaging and behavioral methods are distinguished by their location on the y-axis of the figure. As such, certain imaging and behavioral methods may be more amenable to studying specific psychological processes but not others.

For example, a growing literature suggests that the timing and spatial distribution of specific temporal components of emotional processes change as individuals transition through adolescence into adulthood. These developmental changes in emotional reactivity and regulation are associated with differences in behavior as measured by reaction time, ERPs and pupil dilation that can predict risk for the development of mood and anxiety disorders (Hajcak, MacNamara & Olvet, 2010; Siegle, Steinhauer, Carter, Ramel & Thase, 2003). How these measures co-vary with one another and whether they each
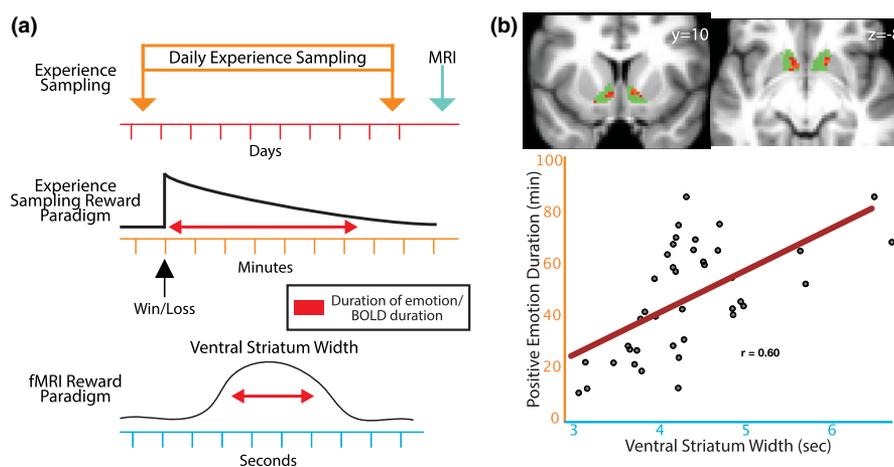

Figure 4 *Using temporal neurodynamics of emotions to formalize links between biological and real-world moment-to-moment experiences. (a) Sampling moment-to-moment real-world emotion using mobile technology during a reward paradigm and brain activity using fMRI during the same reward paradigm shows that (b) width of ventral striatal activity when winning money predicts the duration of real-world positive emotion reported when winning money (Heller et al., 2015).*

uniquely account for risk for psychopathology or resilience is an exciting area for continued investigation.

The high temporal precision of ERPs is a helpful tool for examining emotional neurodynamics. The late positive potential (LPP) is an ERP component that tracks attentional deployment to emotional stimuli and is attenuated when individuals regulate their emotions using strategies such as reappraisal (Hajcak *et al.*, 2010). The LPP has been separated into early (300-600 msec) and later components (> 600 msec) in which the early components are likely reflexive emotional processing while the later components are modulated by effortful emotional regulation (Hajcak *et al.*, 2010). This latter component is centered on the frontal cortex in adults, consistent with the previously reviewed fMRI findings of the importance of prefrontal circuitry in the development of emotion regulation (Kujawa, Klein & Hajcak, 2012). These data further illustrate the importance of timing, and highlight an important role for ERPs in examining the temporal dynamics of emotion together with other modalities.

As the pupil is innervated by neural structures implicated in emotional processing (e.g. the amygdala; Siegle *et al.*, 2003) and is associated with arousal (Kahneman & Beatty, 1966), measures of pupil dilation have examined the development of temporal dynamics in emotions. Sustained pupil dilation to negative stimuli is associated with anxiety (Price, Siegle, Silk, Ladouceur, McFarland *et al.*, 2013) and depression severity (Siegle, Steinhauer, Friedman, Thompson & Thase, 2011) in youth. Similar to the emerging ERP data, pupil dilation appears to be corroborating temporal dynamic effects

seen using imaging and EMA – showing associations between the persistence of certain emotional responses and risk for psychopathology. Using a combination of these tools (EMA, ERP, pupillometry, fMRI, etc.) may better position us to address questions about the neural dynamics of emotions during adolescence and which dynamics give rise to psychopathology and which are indicators of resilience.

Conclusions

Although several mental illnesses peak during the teenage years, the majority of adolescents weather the emotional turbulence of this period. The tension between subcortical limbic regions and the prefrontal cortex during the period of adolescence may have evolved to help the individual adapt to the many new social, physical, intellectual and sexual challenges of this period. The enhanced effects of rewards and threats on behavior and the brain may aid the adolescent in meeting these challenges. Changes in engagement of neural systems that activate the adolescent to meet the challenges of new social roles may be less adaptive today with earlier puberty and the relatively prolonged phase of adolescence in Western society. Our ability to engage in self-control, resist temptation and suppress fear requires opportunities to engage in these forms of regulation without the buffer of the parent in preparation for relative autonomy and survival as an adult. These capacities vary with development and experience, and vary by individual. Individuals who come into adolescence with poor self-control may be at greater risk for suboptimal decisions and actions that ultimately lead to poor outcomes. Conversely, those that are overly inhibited may not sufficiently test the social waters, leading to an anxiety disorder. A priority of future research will be to understand behavioral and brain changes during adolescence, employing a cognitive neuroscience approach that examines temporal dynamics of neural processes that could uncover patterns of potential clinical relevance. These data, together with EMA, could inform public health policies for modifying the environment, and guide treatments and interventions that would have lasting beneficial effects for our young people today and ensure a better future for them tomorrow.

One potential avenue for translation of findings pertaining to the temporal dynamics of emotion regulation is in personalization of psychotherapeutic intervention. Individual differences in personality, temperament and age may contribute to which emotion regulation strategy may be most effective for particular individuals. For example, such differences may predispose an individual to
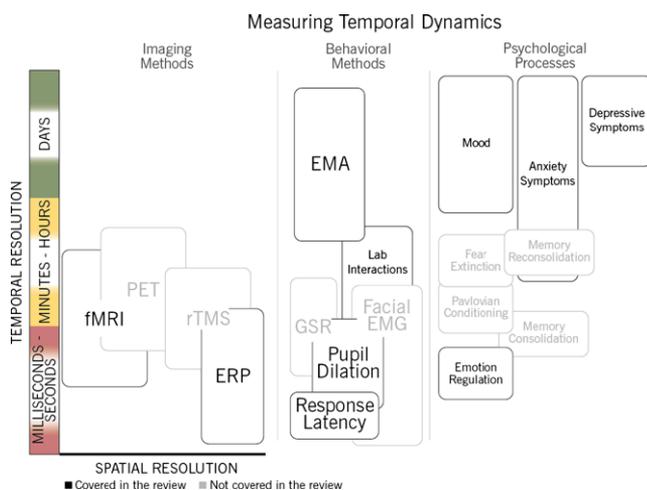

Figure 5 *Converging approaches to measuring the temporal neurodynamics of emotion. Psychological processes unfold over different time-scales and can be used to examine temporal dynamics of emotions.*

more easily maintain one regulation strategy over time due to it requiring fewer cognitive resources than another. As a result, it would logically follow that some individuals – at different points in development – may be more amenable to using some regulatory strategies or interventions compared with others. This may partially explain why some individuals benefit more from certain forms of therapy (e.g. CBT vs. interpersonal therapy vs. mindfulness vs. antidepressants) than others (Cuijpers, Reynolds, Donker, Li, Andersson et al., 2012). Some strategies may be easier to implement across age than others and some therapies may require more practice than others too. A central tenet of both CBT and mindfulness techniques is that with practice, these procedures for regulating emotions become easier, more rapidly engaged and automatic (Beck, 1979). With both adolescent and adult CBT, for example, patients begin treatment utilizing 'dysfunctional thought records' in a completely conscious, effortful manner. Initially, this is often cumbersome and takes time for patients to make their way through the worksheets. However, over time and with practice patients become more fluent and effective at recognizing the affective symptoms that necessitate the use of such CBT skills. As a result, patients are able to sustain negative or positive emotion regulation for longer periods. This account is consistent with our model of neurodynamics of emotion and may be exploited to promote faster or more effective and personalized treatment.

There is tremendous opportunity to better understand how emotional regulation develops during the period of adolescence. Parsing of emotional processes into simple elements of magnitude, duration and habituation is shedding new light on our understanding of how emotions change across development and are altered in individuals at-risk for psychopathology. Research examining temporal dynamics may benefit further from incorporating age and psychopathology status when examining age-specific changes in adolescence (e.g. Gee et al., 2013a; Jarcho, Romer, Shechner, Galvan, Guyer et al., 2015). The development of non-invasive imaging tools for assessing the developing and behaving human brain in real-time also has enhanced our understanding of circuit-driven changes in affective behavior during adolescence. Rapidly advancing mobile technology permits assessment of real-world functioning that could lead to novel diagnostics and treatments. Parsing affective phenomena into their core components and relating short-term biological events with longer-term psychological experiences may help to map the basic science of adolescent affective development and improve identification of those at-risk for psychopathology. Such improved understanding of both real-world dynamics of emotion and the neural circuits governing such experiences will inform our strategies for treating psychopathology and enhancing well-being.

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