Emerging evidence for emotion context insensitivity in depression
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Depression is defined as a mood condition. One key problem in depression’s clinical description is determining how persistent mood changes influence emotional reactivity to ongoing environmental stimuli. Repeatedly, depressed individuals have been observed to exhibit diminished reactivity to change in the emotional context. These observations have spawned a theory called emotion context insensitivity (ECI). In this article we: (1) discuss the genesis of ECI theory, (2) consider recent convergent and divergent evidence that bears on ECI and related constructs, (3) sketch a synthesis out of these observations, and (4) outline several future directions that will consolidate our understanding of emotional functioning in depression.

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Introduction
Depression is a widespread mood disorder that affects nearly 20 percent of the population. Depression’s most characteristic changes involve alterations in mood, including persistent elevation in negative mood (e.g., sadness) and persistent lowering of positive mood (e.g., anhedonia) [1]. By all accounts, more accurate descriptions of what is disturbed in depression are needed to improve treatments for this condition. One important unresolved area in the clinical description of depression is to ascertain how persistent mood changes influence emotional reactivity to ongoing environmental stimuli.

Theoretical orientations
Emotions occur in a variety of eliciting contexts (e.g., a marriage, the workplace) and involve different response systems (i.e., changes in experience, behavior, and physiology). Over the last fifteen years, work drawing from affective science has sought to specify both the contexts and the response systems in which depressed people exhibit aberrant emotional reactivity. One initial handicap in tackling this question was that few theories were explicitly directed at emotional reactivity in depression. When we began this work, predictions based on emotion theory [2] and cognitive theories of depression [3] anticipated that clinically depressed moods will potentiate reactivity to negative emotional stimuli. Cognitive theories of depression offered the clearest signposts, postulating that negative cognitive structures guide preferential processing to negative stimuli thereby enhancing reactivity to negative stimuli in the environment [3].

Much has changed in fifteen years: a rich vein of work now contradicts the mood-potentiation hypothesis. Repeatedly, depressed individuals have exhibited diminished reactivity to change in the emotional context. These observations have spawned a theory called emotion context insensitivity (ECI). In this article we discuss the genesis of ECI theory and consider recent evidence (both converging and diverging) that bears on ECI and related constructs.

Foundations for ECI
Initial experimental work leading to the ECI hypothesis found that depressed participants reported smaller increases in sadness than healthy controls when responses from the neutral and sad films were compared [4]. A second study focused on the observable behavior of crying in response to a sad film. Given the common view among clinical commentators that depressed persons cry readily and intensely, we expected to see evidence of mood potentiation. Instead, there were no differences in the rate of observable crying behavior between depressed and nondepressed persons. Moreover, when reactivity was computed from a neutral reference, depressed criers showed smaller changes in their emotional experience and physiology than healthy criers [5]. Finally, in an important extension, we found reduced reactivity to sad stimuli in MDD even when the sad stimuli were constructed to be high in personal relevance [6*].

By 2008, enough laboratory studies had accumulated to allow a meta-analysis of emotional reactivity in depression and to allow estimation of effect sizes [7*]. Results from this meta-analysis confirmed the ECI pattern among experimental studies of depressed persons. Led by these data, we argued that MDD involves a generalized loss of emotional reactivity (lack of context-appropriate reactivity to both positive and negative elicitors), grounding our explanation of emotion context-insensitivity (ECI) in an evolutionary account of mood [9]. By this account, the evolved function of strong negative mood states, such as those seen in clinical depression, is to diminish action in risky or dangerous situations, or situations where behavior...
is not paying off. As a result, there are broad reductions in motivated action, including emotional reactivity to ongoing environmental stimuli [8].

**Corollaries of ECI**

Since our meta-analysis, research has supported ECI in several different ways. A first corollary of ECI concerns a prediction about the relationship between emotional reactivity and the severity and course of depression. If ECI reflects a fundamental problem in depression, it should be related to other depression-related impairments. Presumably, then, those depressed persons who exhibit the strongest ECI pattern should be the most impaired and have the worst future course. Indeed, data from several studies bear out these predictions [4,10**,11,12, see 13 for review].

A second corollary of ECI concerns the co-occurrence of depression with other mental disorders, an exceedingly common pattern [14]. Here, the prediction is that when other disorders co-present with depression, the emotional reactivity in the other disorders would resemble the ECI seen in depression alone. Notably, several studies find ECI to be observed in anxiety disorders when they co-occur with depression [15,16**,17,18].

A third corollary concerns application of the ECI hypothesis to the central nervous system. This corollary is important since initial evidence for the ECI hypothesis came predominantly from other response systems (i.e., the self-report of emotion experience, facial behavior, and autonomic physiology), in part because of challenges in operationalizing the magnitude of emotional reactivity in the brain. Nevertheless, there is recent evidence of ECI in measures of hypothalamic–pituitary–adrenal axis function (HPA; i.e., blunted cortisol [19]), and several studies of depressed peoples’ brain response to emotion-generative stimuli are likewise consistent with ECI [20**,21,22,23**, but see 24].

**Converging evidence**

Also heartening for the ECI hypothesis is that investigators pursuing independent lines of research have discovered other emotion-related phenomena that are consistent with ECI. For example, work by Peter Kuppens and colleagues [10**,13,25,26**,27,28*] has focused on the construct of emotional inertia, which pertains to the predictability of affect. Inertia is operationalized as the prediction of current emotional state from previous emotional state via autocorrelation modeling. Consistent with ECI, several studies find emotional inertia to be elevated in people who have symptoms of depression [25,26**,28*]. Moreover, persons who exhibit greater inertia at one point in time are more likely to have depression in the future [10**,26**,27, but see 30*].

A second ECI-related construct is emotion differentiation, which refers to the intercorrelation of emotional responses. Depressed persons have been shown to exhibit poorer emotion differentiation than controls, reflected in a greater intercorrelation among separate negative emotions at any time point than healthy controls [31**,32]. This finding of a lack of emotional differentiation is consistent with ECI’s flattened emotional landscape in which depressed persons report and display the same negative emotions across different contexts (i.e., a sunset, a sad movie). Future work should attempt to refine the interpretation of low differentiation in depression: does it truly reflect poor affective differentiation, or an inability to appreciate differences between related affective states, or does it reflect that, for depressed people, there is a greater tendency for negative affective states to be co-activated (i.e., triggered together)?

Finally, a third ECI-related construct is emotion network density. Here the focus is on the extent to which a person’s emotional responses are predictable from other previous emotional responses. For example, does a person’s current sadness predict his or her future anger or future fear? Depressed persons have been found to have a greater emotion network density than controls, particularly for negative emotional states [33**]. Although a distinct construct, this higher degree of predictability of negative emotions across time and context is consistent with ECI.

**Divergent evidence**

At the same time that convergent evidence has emerged, there has also been divergent evidence, which, potentially, presents challenges for the ECI theory.

First, not all laboratory studies find ECI. For example, although the blunting of the emotion-modulated startle has been among the most reliable of ECI findings [34,35], a recent startle study found enhanced startle to the anticipation of shock threat, suggesting there may be exceptional conditions where ECI does not hold ([36], i.e., specific high intensity threats).

A second area of divergent evidence comes from the study of emotion in everyday life, where several investigations appear to challenge a simple ECI view of depression. This is observed for research on affective instability, typically a measure of the magnitude of point-to-point variations in affect [37]. Depressed people have been found to report greater instability of negative affect in everyday life in typical assessments where the assessment points are about 90 min apart [29,38–41]. Furthermore, there is evidence that instability predicts a worse course [40] and is reduced with successful treatment [38]. Finding greater emotional instability in depression would appear to directly contradict the notion of greater inertia in depression. At the same time, it is important to clarify that inertia and instability are distinct operationalizations of emotion; each uses distinct statistical methods, and the two measures are only moderately
intercorrelated. While inertia considers how emotional states at one time predict emotional states at later times, instability considers the magnitude of moment-to-moment change in emotional states, using statistics such as the mean squared successive difference [26**,42].

Finally, and in some ways the most peculiar, observation arises from findings of depressed persons reporting greater mood brightening to everyday pleasant life events. Specifically, several investigations find that depressed persons exhibit larger decreases in negative affect after a positively appraised real life event than nondepressed people [43,44]. It should be noted that such pleasant events are relatively infrequent and do not completely relieve depressed persons’ dysphoria [44]. Nevertheless, this mood brightening finding is not predicted by any major theory of emotional functioning in depression; yet it has now been replicated for both participants with major and minor forms of depression, and across different methods for assessing affective experience [44].

Synthesis
Integrating the convergent and divergent evidence regarding the ECI hypothesis may help us forge a helpful synthesis about emotional functioning in depression, one that also clarifies the boundaries for ECI. Surveying what we know, ECI appears most likely and robust when normative, generic emotional stimuli are presented in the short term. Depression systematically reduces reactivity to stimuli which we know to affect the average non-depressed person, over a relatively brief time scale of seconds or minutes (a time scale we ordinarily associate with emotions [45**]).

The divergent observations, in turn suggest that depressed persons might have greater instability and be capable of greater reactivity in specific contexts. For example, greater instability or fluctuation in depression may be observed at the time scale of hours or days; this could reflect differences in slower, endogenous rhythms (i.e., circadian disturbance [41]) or be driven by factors that are hidden from external observers (i.e., idiosyncratic negative thoughts). Further, the mood brightening findings suggest another possible boundary for ECI: depressed persons may not be generally reactive to positive events or stimuli; however, when that rare event is appraised as positive, depressed persons can be reactive to it, a finding that has clear clinical implications.

Conclusions
Several future research directions would help to consolidate our understanding of ECI in depression. First, we need more intensive multi-method studies that assess emotional functioning in both laboratory and real life contexts in the same individuals [26**]. Such designs can both help us reconcile puzzling observations and check the viability of our proposed synthesis. Just as work in cardiovascular functioning has conducted lab studies and ambulatory studies to understand the exact conditions under which exaggerated reactivity might harm the heart [46], so, too, can such designs clarify the precise conditions under which ECI applies in depression.

Second, the theory of ECI arose originally from a series of observations, some of them unexpected. In this respect, the theory has had to play catch up to the data. An important future goal is to develop ECI into a rich, mature theory like cognitive theories of depression, which elaborates the ultimate origins, responsible proximal mechanisms, and exact conditions under which the theory applies. For example, while ECI was launched as theory of depression, we cannot say with certainty that the ECI pattern is specific to depression, in part because of a relative lack of comparative work with other mental conditions [18, for a broader discussion see 47].

Last, and relatedly, while there is good evidence for the fact of ECI, we are relatively early in our understanding of the responsible proximal mechanisms. Some early candidates include rumination (a cognitive mechanism [48]), diminished cardiac vagal control (a biological mechanism [49,50*]), and sleep disturbance [51]. In the future, to further broaden our understanding of mechanisms, it will be particularly important to consider brain-based substrates for ECI. One interesting preliminary idea is that depression may involve decreased connectivity between the amygdala and other areas of the brain such as the prefrontal cortex [24] and brain stem via reduced white matter in the solitary tract [23**]. This could explain why depressed people display diminished behavioral and autonomic reactivity to negative emotional stimuli despite robust amygdala reactivity across contexts [24].

Progress on these critical next steps will both help us deepen our understanding of ECI in depression and provide a broader picture of how depressed mood influences emotional reactivity to ongoing stimuli. Hopefully, what we learn from basic science (enhanced description and understanding of the responsible mechanisms) can be fed forward into clinical payoffs with the design of more effective interventions for this common mental condition.

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References and recommended reading
Papers of particular interest, published within the period of review, have been highlighted as:

- of special interest
- of outstanding interest


This study was the first to examine the ECI hypothesis with respect to both the negative potentiation and positive attenuation hypothesis of emotional experience in depression. The data supported ECI with some support for the positive attenuation hypothesis.


This study demonstrated consistent support for the ECI hypothesis in laboratory studies through meta-analysis. Depressed persons exhibited a greater deficit in reactivity to positive stimuli (d = 0.53) than to negative stimuli (d = -0.25) stimuli. The overall reduction in emotional reactivity (d = -0.37) was observed in self-report, behavior, and autonomic physiology.


This study found that never-depressed young adolescents who displayed inertia for happy, dysphoric, and angry emotions during a conflict resolution task with their parent were twice as likely to have developed depression at the 2.5 year follow up. This indicates that little change in emotional reactivity during a negative conversation prospectively predicted the onset of depression during adolescence.


This study examined cortisol stress responses to a speech stressor in individuals with social anxiety disorder with and without comorbid depression. Results demonstrated attenuated cortisol responses in the comorbid depression cases only. This indicates that MDD can attenuate reactivity in the context of anxiety.


This study used Diffusion Tensor Imaging to examine three tracts connected to the brainstem in MDD and MDD-S. Results demonstrated significantly decreased connectivity between the brainstem and the amygdala in MDDs compared to healthy controls, a pattern that may explain paradoxical features of emotional reactivity in MDD.


This study was path breaking in that it examined emotional reactivity in both laboratory and everyday life contexts in the same individuals. The results from both contexts were consistent with ECI, although results need to be replicated in a sample with diagnosed depression.


This study found that emotional inertia predicted depressive symptoms over and above both state and trait perseverative thinking. Together, this suggests that depressive symptoms are predicted by less variability in emotional experience on a daily basis and this prediction is not explained by common forms of cognitive inflexibility.


This study examined the relationship between inertia in positive affect and depressive symptoms. Over a multi-year follow up, greater inertia in positive affect was associated with recovery from depression in two separate clinical samples.


This study found that depressive with predicted difficulty differentiating negative emotions after controlling for decreased intensity and variability in

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emotional experience. Further, this study provides a heuristic model for considering different metrics of emotional functioning, i.e., variability versus differentiation.


This study found that relative to healthy persons, individuals with MDD had greater density in their emotion networks, such that reports of negative emotion at one time point predicted reports of negative emotion at the next time point. This indicates a greater continuity among different negative emotions across contexts in depression.


Provides a model for examining emotional reactivity and flexibility at three time scales: micro-level consisting of moment to moment fluctuations (seconds to minutes), meso-level consisting of context to context fluctuations (hours to days), and macro-level consisting of changes during developmental and life transitions (months to years). Current ECI research has straddled the micro and meso-levels, perhaps contributing to mixed findings.


This study examined respiratory sinus arrhythmia reactivity in currently and remitted depressed individuals and found that currently depressed individuals displayed less reactivity to a speech stressor task than both remitted and healthy control comparison groups. Results suggest that blunted RSA reactivity may mark the depressed state.