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Stress and depression: old questions, new approaches

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After decades in which diatheses dominated research on the diathesis–stress models of depression, increasing attention to stress and stress–depression mechanisms is in the forefront of efforts to understand depression and treat it effectively. Supplementing research on known risk factors and moderators (such as demographic, cognitive, relational, family, and personality characteristics) of the stress–depression association, much work now focuses on experiences of early life stress, acute stressors, and chronic stress and their developmental features and neurobiological mechanisms relevant to depression. The review briefly highlights the current status of risk factors, HPA axis, neural, and genetic approaches, noting conceptual and methodological challenges.

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Introduction

Stressful life events have long been recognized and studied as robust predictors of depression. However, most people who have major stressful events do not get depressed, leading researchers to refine the issue of why Person A develops depression following stressful experiences but Person B does not. Questions about the moderators of the stress–depression link have predominated until more recent years in which there has been increasing interest in mechanisms, especially biological, by which stress triggers depression. This brief review attempts to highlight recent developments and current issues for contemporary researchers in which stress is at the forefront of models of depression.

Developments in conceptualization and measurement of depression and stress

Changing constructs and methodologies applied to 'stress' and 'depression' clearly shape current research strategies. Some of the major findings of latter part of the 20th century were enabled by development of a reliable diagnostic classification system that sparked new areas of research, rapidly generating new theories of depression and major treatment advances. Nevertheless, there is widespread recognition that diagnosis-based research in depression has obscured enormous heterogeneity, which along with comorbidity, impedes precision and reproducibility of research findings [1,2]. Future research efforts are increasingly being directed toward alternative conceptualizations of the phenotype, including refining intermediate phenotypes [3,4,5•] and relating processes to transdiagnostic conceptualizations and dimensional measures [6].

Similarly, 'stress' conceptualizations evolved from focus on acute negative life events and easily administered but conceptually and methodologically limited assessments including checklists and questionnaires of perceived stress [7] to a much wider array of constructs and tools. Improvements include interview-based, objective measures of negative life event occurrence and severity [7]. Notably, there has also been increasing recognition of two additional but vitally important realms of stress exposure that had long passed largely below the radar in depression research: childhood adversities such as emotional abuse, and chronic, ongoing stressful conditions. There is increasing recognition that chronic stress, such as challenging marital, financial and work conditions, is a robust predictor of depression [8], as well as other psychopathologies, maladaptive behaviors, and poorer health outcomes [9,10]. Similarly, childhood adversity exposure predicts adolescent and adult psychopathology [11,12]. The impact of cumulative stress, especially measured as allostatic load, has also been the focus of efforts to understand depression and other forms of dysfunctional behavioral and medical health [13]. Thus, stress research warrants examination of early, chronic, continuing, cumulative, and proximal acute stress exposure. Moreover, there is growing evidence of the uniquely powerful role of interpersonal types of stress such as marital, family, and social relationships, losses, conflicts, and rejection in the prediction of depression especially in women [14,15].

Important shifts in definitions of stress and depression have also been accompanied by significant changes in models of associations between stress and depression. Much of research in recent decades was dominated by diathesis–stress paradigms, in which stress *predicts* depression, moderated by key vulnerability factors. Many research programs were largely focused on the diatheses, the vulnerability factors, with typically indirect or implicit

recognition of stress. As investigators tested the diathesis–stress models directly, it became clear that the stress–depression association, however, is not unidirectional, not static, and definitely not simple. Adults and children with histories of depression generate stressors in their lives, a bidirectional pattern portending continuing experiences of depression — and continuity of stress [16,17]. Depressive symptoms themselves contribute to the occurrence of stressful events in people's lives, but stable dysfunctional characteristics of the depressed person also predict disruptions and stressors, particularly in interpersonal relationships [18,19]. Thus, the link between stress and depression is bidirectional.

Furthermore, studies revealed that the stress–depression association is not static over time, with stressors playing progressively less of a role in triggering episodes over the clinical course of depression [20–22]. Also, some individuals appear to be 'sensitized' by experiences such as exposure to adversity in childhood, so that they are more likely to develop depression in response to later-occurring stressors compared to those not exposed or to develop depression at lower levels of exposure [23–25]. There is a growing body of research and theory on the ways in which stressful experiences including social relationships sculpt the brain and neuroendocrine systems at different developmental periods from prenatal through adolescence [26–28].

Psychosocial moderators of associations between stress and depression

Over the past few decades several variables repeatedly emerged as robust predictors of risk for depression, both as main effects and as moderators, and sometimes mediators, of the effects of stress on depression. Demographic factors, such as female gender, younger (rather than old) age, and lower socioeconomic status predict higher rates of depressive disorders and symptoms [29,30] in part because they are associated with acute and chronic stressors.

Clinical history factors, such as having a parent, especially a mother, with depression is a major risk factor for developing depression in youth [31,32], due to both genetic and parenting mechanisms, and family stress [33]. Personal history of prior depressive episodes is also a strong predictor of the risk of future depressions [34], with successive episodes reducing the association between stress and depression in a 'kindling' pattern [21].

For some years there has been a particular emphasis on depression-related cognition, biased and maladaptive perceptions emphasizing low self-worth, pessimism, futility, and exaggeration of the negative impact and meaning of events. Recent research on dysfunctional information-processing among depressed individuals reveals a variety of distinctive patterns including increased accessibility of, and greater difficulties disengaging from, negative material, and deficits in cognitive control leading to failure to inhibit irrelevant negative content, increasing the difficulty in 'recovering' from negative thoughts [35,36••].

Relatedly, ruminative response style is a dysfunctional trait-like pattern of response to negative experiences that typically intensifies and prolongs depressive symptoms [37]. Joining the list of prime predictors of depression or moderators of the stress–depression link, neuroticism is a core human trait reflecting the tendency to interpret the world as dangerous and threatening, coupled with negative emotional reactions in response to stress [38] and is highly predictive of mood and anxiety disorders [39,40]. Certain personality traits or interaction styles potentially predictive of dysfunctional (and stressful) interpersonal relationships have also been linked with depression outcomes, including excessive reassurance-seeking, dependency, rejection sensitivity, insecure attachment style, and related dispositions ([33]; see also [10]). Beyond the scope of the present review, a considerable amount of emerging research has focused on the genetic and neurobiological correlates of such traits and behaviors (e.g. [36••,41••]).

Research on biological risk factors and mechanisms of stress effects on depression

The various risk factors for depression and links between stress and depression are now under intense scrutiny of their biological mechanisms, foremost among them the HPA axis stress response processes, but also including imaging studies of brain structure and function, and hormonal and neurotransmitter systems, and genetic candidates that are known to be associated with stress reactivity and emotional responding. Vast volumes of human and animal research on both normal and abnormal processes are too extensive to cover, so a few brief comments focus on recent human research.

Hypothalamic-pituitary-adrenal (HPA) axis

It has long been observed that depressed adult patients show several indicators of dysregulation of the HPA axis — some state-dependent but others trait-like — including altered basal levels of cortisol, disrupted homeostatic mechanisms of the HPA axis evidenced in abnormal responses to an exogenous steroids and/or abnormal patterns of reactivity to stressors [42,43]. Glucocorticoids are widely dispersed throughout the brain, and in complex and developmentally programmed ways affect brain structures and functioning. Recent research also increasingly links HPA stress responses to immunological processes that may promote depression [14,42]. Numerous studies have explored associations between cortisol and stress, including both natural and experimentally induced social-evaluative stress, and depression. However, there have been many inconsistencies in findings likely due to variations in methods, samples, age groups, types and age of stress, and cortisol methods — as well as mediation by

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