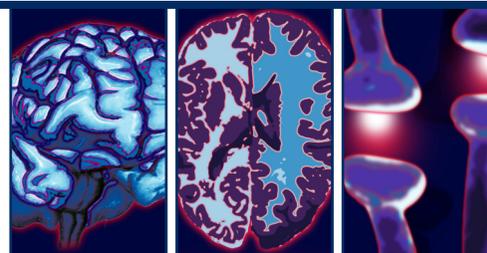


JOURNAL WATCH



Our expert highlights the most important research articles across the spectrum of topics relevant to the field of neuropsychiatry

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Rapee RM, Kennedy SJ, Ingram M, Edwards SL, Sweeney L: Altering the trajectory of anxiety in at-risk young children. *Am. J. Psychiatry* 167(12), 1518–1525 (2010).

There is increasing evidence suggesting the importance of risk factors for anxiety disorders, thus opening the possibility of prevention. A number of overlapping temperament styles (e.g., behavioral inhibition, social withdrawal, inhibition and shyness) may predict the development of anxiety disorders in general. Conversely, parental factors may also contribute to childhood anxiety. Rapee *et al.* performed a randomized controlled trial evaluating the 3-year effects of a parent-focused intervention for anxiety versus a monitoring-only condition in a sample of carefully assessed inhibited preschool-age children, most with a Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV) diagnosis of social phobia.

The intervention described in this study (six 90-min sessions) was conducted in groups of approximately six sets of parents and included a discussion on anxiety and its development, parent management techniques, the deleterious effect of overprotection in maintaining anxiety, application of exposure hierarchies and cognitive restructuring to the parents' own worries. The outcome measures included the number and severity of anxiety disorders, anxiety symptoms and degree of inhibition. Inhibited children whose parents received the intervention exhibited lower frequency and severity of anxiety disorders (particularly social phobia and generalized

anxiety disorder) and lower levels of anxiety symptoms according to maternal, paternal and child reports. These results comprise the first solid evidence showing that a brief low-cost intervention early in the child's life may produce sustained decreases in children's anxiety symptoms, and therefore have major public health implications.

Andersen SL, Greene-Colozzi EA, Sonntag KC: A novel, multiple symptom model of obsessive-compulsive-like behaviors in animals. *Biol. Psychiatry* 68(8), 741–747 (2010).

Obsessive-compulsive disorder (OCD) is currently conceptualized as a multi-dimensional syndrome that is associated with several abnormalities on symptom, neurocognitive and neurochemical levels. To date, animal models of OCD have failed to reproduce this heterogeneity and typically portrayed only a single symptom dimension (e.g., stereotypies, perseveration or anxiety). Based on the observation that exposure to a pharmacological agent during a neurodevelopmental-sensitive period produces opposite changes in the targeted neurotransmitter system, Andersen *et al.* exposed rats to an anti-OCD drug (clomipramine) or placebo during the neonatal period and assessed them in adulthood. Clomipramine exposure in immature rats produced a range of behavioral and biochemical changes that resembled OCD in humans, including increased anxiety, hoarding, perseverative behavior, working memory impairment and corticostriatal dysfunction. Remarkably, dopamine D2 receptors were elevated in the striatum,

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whereas serotonin 2C, but not serotonin 1A, receptors were elevated in the orbital frontal cortex. This study illustrates how it is possible to develop an animal model that reproduces OCD on a different level. This model may be of use for testing anti-OCD treatments that may be of potential benefit to humans. It also shows that drug exposure during a sensitive period can program disease-like systems permanently.

Nelson B, Yuen K, Yung AR: Ultra high risk (UHR) for psychosis criteria: are there different levels of risk for transition to psychosis? *Schizophr. Res.* 125(1), 62–68 (2011).

The ultra high risk (UHR) for psychosis criteria includes one or more of the following characteristics: attenuated psychotic symptoms (APS), in other words, subthreshold forms of positive psychotic symptoms during the past year; brief limited intermittent psychotic symptoms (BLIPS), in other words, frank psychotic symptoms that have lasted less than a week and have spontaneously subsided; and 'trait and state' risk factor (Trait), in other words, a first-degree relative with a psychotic disorder or a schizotypal personality disorder plus a significant decrease in functioning during the previous year. A number of studies have validated the UHR criteria; however, it was unclear whether any particular UHR syndrome (i.e., APS, BLIPS or Trait), or combination of criteria, was associated with a higher risk of transition to psychosis. This study analyzed data on UHR criteria and transition to psychosis status after 6 months of follow-up. A total of 817 patients (88%) had baseline information available for analysis. The percentage of subjects who presented with APS, Trait and BLIPS were 83, 27 and 4%, respectively. When the two intermediate groups (APS alone and APS + Trait) were combined, there was evidence that the risk

of transition increased in the order of Trait alone < APS < BLIPS ($p = 0.024$). This data indicates that particular attention may need to be paid to patients with BLIPS, especially early in the course of treatment.

Merikangas KR, He J-P, Burstein M et al.: Lifetime prevalence of mental disorders in U.S. adolescents: results from the National Comorbidity Survey Replication–Adolescent Supplement (NCS-A). *J. Am. Acad. Child Adolesc. Psychiatry* 49(10), 980–989 (2010).

Using data from the National Comorbidity Survey–Adolescent Supplement (NCS-A), this study aimed to examine the lifetime prevalence of DSM-IV mental disorders with and without severe impairment, their comorbidity across broad classes of disorder and their sociodemographic correlates. The NCS-A, a nationally representative face-to-face survey, includes data from 10,123 adolescents between the age of 13 and 18 years in the USA. DSM-IV mental disorders were diagnosed using a modified version of the fully structured WHO Composite International Diagnostic Interview. The most common condition was found to be anxiety disorders, followed by behavior disorders, mood disorders and substance use disorders. The same pattern was observed for age of onset, with anxiety disorders having the earliest age of onset and substance use disorders the latest. Comorbidity was common, with approximately 40% of patients diagnosed with one class of disorder also meeting criteria for another class. This study is of importance as it provides the first prevalence data for a range of mental disorders in a nationally representative US adolescent cohort. It highlights that common mental disorders in adults first emerge during childhood and, therefore, there is a need for research into prevention and early intervention in youths.

Lanzenberger R, Wadsak W, Spindelegger C et al.: Cortisol plasma levels in social anxiety disorder patients correlate with serotonin-1A receptor binding in limbic brain regions. *Int. J. Neuropsychopharmacol.* 13, 1129–1143 (2010).

Reduced glucocorticoid feedback, as a result of hypothalamic–pituitary–adrenocortical axis dysregulation, and alteration of the serotonergic system have been linked to mood disorders. This study aimed to investigate interactions between these systems to better understand the relationship between stress and depression. 5-hydroxytryptamine (HT)_{1A} receptor binding was measured in the anterior and posterior cingulate cortices, hippocampus, amygdala, medial orbitofrontal and retrosplenial cortices, and dorsal raphe nucleus of 12 male patients with social phobia and 18 matched controls. Patients with social phobia had significantly lower plasma cortisol levels than healthy controls and strong negative correlations between cortisol plasma levels and 5-HT_{1A} binding in the amygdala, hippocampus and retrosplenial cortex were observed in this group of patients. These associations in the amygdala and hippocampus were significantly higher in patients with social phobia than in healthy controls. A negative association between plasma cortisol levels and 5-HT_{1A} receptor distribution consistent with studies in rodents and nonhuman primates was confirmed by the study. The authors conclude that the vulnerability for mood disorders may be caused by dysregulation of the cortisol altering limbic 5-HT_{1A} receptors.

Financial & competing interests disclosure

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