

# *Risikofaktoren für Depression Bei Adoleszenten*



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# *Risk factors of depression occurrence in Adolescence*



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## □ Introduction and Epidemiology of Depression

- ❖ Depression in Adolescents: Causes, Treatment, and Prevention ( Abela & Hankin)
- ❖ Epidemiology of Depression in Adolescents



# □ Etiology of Depression



Adolescence is characterized by positive gains in cognitive maturity, better interpersonal skills, new experiences, increased autonomy, and hormonal changes (Feldman & Elliot, 1990).

Although these normative transitions can provide opportunities for further growth in cognitive, physical, psychological, and social domains, exposure to adverse experiences (e.g., peer pressure, difficulties in school performance, loss of a romantic relationship) *can place the adolescent at increased risk for the onset of a wide range of emotional and behavioral problems*, including the development of depressive symptoms (Steinberg, 2006).



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Depression in Adolescence - Understanding the origins of depressive symptoms in adolescents requires knowledge of how the transition from childhood to adolescence and from adolescence to adulthood serves as a backdrop for rising levels of depressive symptoms.





## ❖ Cognitive Vulnerability to Depression in Adolescents



Negative patterns of thinking and maladaptive information processing, termed cognitive vulnerabilities, have been shown to contribute to the development of depressive symptoms in adolescents who activate these vulnerabilities in response to negative life events (Abela, 2001; Hankin & Abramson, 2002; Lewinsohn, Rhode, & Seeley, 1994, 1998).





Cognitive Vulnerability Theories (Beck's Cognitive Theory, Hopelessness Theory, Cognitive Vulnerability-Transactional Stress Model) propose that individuals who possess cognitive vulnerability to depressive symptoms (belief systems focused on loss, failure, and worthlessness) are more likely to develop depressive symptoms following the occurrence of a negative life event than individuals who do not possess cognitive vulnerabilities.



Hankin and Abramson's (2001) Cognitive Vulnerability-Transactional Stress Model (CV-TSM) further developed the proposition that cognitive vulnerability was the key to the development of depressive symptoms.

The CV-TSM includes the concepts from Beck's Cognitive Theory (1987) (i.e., dysfunctional attitudes) and Abramson et al.'s (1987) negative inferential style, and hypothesizes a third cognitive vulnerability, namely, ruminative response style (Nolen-Hoeksema, 1997) .

## ❖ A Developmental Perspective on Interpersonal Theories of Youth Depression



- Social-Behavioural Deficits of Depressed Youth
- Relationship Disturbance in Depressed Youth
- Transactions between Interpersonal Dysfunction and Depression
  - Interpersonal Dysfunction as a Risk Factor for Depression



## ❖ Biological Vulnerability to Depression



- Normative development and functioning
- Structural Imaging findings
- Functional Imaging findings
- Neurochemical contributions
- An Integrative Framework: Bridging Biology and the analytic couch

## ❖ New Behavioural Genetic Approaches to Depression in Adolescence

- Quantitative Genetic methodology
  - Family Studies
  - Twin Studies
  - Adoption Studies

## ❖ Molecular Genetic Methodology



□ Review of research with focus on risk factors of depression occurrence in adolescence

❖ Review of research in world

❖ Review of research in Serbia and Macedonia





## □ Subclinical depression

Is this one and the same or two different clinical phenomena, and whether the differences are quantitative or qualitative?



Based on the previous discussion, we will further try to integrate the analysis and the explanation of results into a broader discussion about the *nature of subclinical depression*, or the old familiar question about *continuity or discontinuity of depression*.



Our findings on the existence of subclinical depression are consistent with the empirical findings that confirm the existence of depression in adolescents in a continuum (Hankin, Fraley, Lahey, & Waldman, 2005; Cuijpers, De Graaf, Van Dorsselaer, 2004a; Cuijpers & Smit, 2004b; Cuijpers, van Straten, & Smit, 2007 a; Cuijpers, Sander, Koole, Annemiek van Dijke, Roca, Li & Reynolds III, 2014).





According to Cuijpers and associates (Cuijpers, De Graaf, Van Dorsselaer, 2004a; Cuijpers & Smit, 2004b; Cuijpers, van Straten, & Smit, 2007 a; Cuijpers, Sander, Koole, Annemiek van Dijke, Roca, Li & Reynolds III., 2014), subclinical depression can be defined from at least three different perspectives.



In the context of the discussion, we are interested in the perspective for which it is assumed that depressive symptomatology exists on a continuum where on one side there are no symptoms, and on the other end of the continuum there is a major depression, and in-between there is subclinical depression (Gotlib, Lewinsohn, Seeley, 1995; Kessler, Zhao, Blazer, Swartz, 1997; Goldberg, 2000; Solomon, Haaga, Arnow, 2001).



There is a group of empirical studies showing that depression can be conceptualized as a continuum (Goldberg, 2000; Angst, Sellaro, Merikangas, 2000; Geiselmann, Bauer, 2000; Cuijpers, De Graaf, Van Dorsselaer, 2004a; Cuijpers & Smit, 2004b; Cuijpers, van Straten, & Smit, 2007 a), although it does not exclude the possibility of the existence of latent qualitative difference between clinical depression and subclinical depression (Geiselmann & Bauer, 2000; Cuijpers, De Graaf, & Van Dorsselaer, 2004a; Cuijpers, van Straten, & Smit, 2007 a).





In this context, according to Avenoli and associates (Avenoli, Knight, Kessler, & Merikangas, 2008) many studies of adults and adolescents have not been able to find strong support of categorical support of depression (Hankin, Fraley, Lahey, & Waldman, 2005; Ruscio & Ruscio, 2000 Slade & Andrews, 2005).



At the very end of discussion, we would like to point out that developmental psychopathology enables analysis and evaluation of biological, psychological, social and cultural processes and determines how interaction between these multiple levels of analysis can influence individual differences, continuity or discontinuity of adaptive or maladaptive behavior patterns and patterns through which normal and pathologically developmental outcomes can be achieved (Cicchetti & Sroufe, 2000; Cicchetti & Dawson, 2002).



According to Cicchetti and Koen (Cicchetti & Cohen, 2006), the central thing is the emphasis of the process of discovery of development, with the ultimate goal of understanding occurrence, progressive connecting and transformation of patterns of adaptation and maladaptation over time.





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THANK YOU!



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