AD/HD IN THE GENESIS OF CONDUCT DISORDER - DOES BIOPSYCHOSOCIAL APPROACH MAKE SENSE?

Dragan Mitrovic, Sladjana Martinovic-Mitrovic & Aleksandra Dickov
Institute of Psychiatry, Clinical Centre „Vojvodina”, Novi Sad, Serbia

SUMMARY
There are numerous theories approaching the source of mental disorders (including conduct disorder) from different perspectives – biological, psychological, social and multifactorial.

The question that arises is which theory is to be used to explain the issue. In the interpretation of phenomena in psychiatry, Kecmanovic discusses possibilities of different approaches (biological, psychological, social and biopsychosocial models) and concludes that none of them provide a complete solution as to how to approach different disorders. The question, therefore, is how to proceed? Although according to Kecmanovic, the biopsychosocial model, as Engel has formulated it, “provides only ingredients not a prescription”, it is our opinion that it indeed does not need to provide prescriptions- it is sufficient if it indicates the necessary ingredients. The prescription itself is to be found in novel scientific disciplines, in particular neuropsychology and epigenetics. Gilbert, on the other hand, points out that the bio-psychosocial approach is holistic, and more than that. „The bio-psychosocial approach addresses the complexity of interactions between different domains of functioning and argues that it is the interaction of domains that illuminates important processes” e.g. a hierarchical dimension of the model as one and development as another dimension provide the basis for a comprehensive perspective on psychiatric disorders, in this case of AD/HD as a risk factor for conduct disorder.

Key words: AD/HD - conduct disorder - bio psychosocial - child and adolescence

INTRODUCTION
Attention deficit hyperactivity disorder and conduct disorders

Conduct disorder is a psychiatric syndrome occurring in childhood and adolescence, characterized by a longstanding pattern of violations of rules and antisocial behaviour. As listed in the Diagnostic and Statistical Manual of Mental Disorders, 4th ed. (DSM-IV, 1994), symptoms typically include aggression, frequent lying, running away from home overnight and destruction of property.

Epidemiological data show the prevalence of attention-deficit/hyperactivity disorder (AD/HD) among conduct disorders, as diagnosed according to DSM IV classification, to be around 20% among children, in almost every other adolescent with conduct disorder, whereas in adults with conduct disorder the prevalence of AD/HD is again 20% (Froehlich 2007, Landover 2004, Mitrovic 1984). Klein et al. argue that 75% of ADD children with hyperactivity develop behavioural problems including 50% conduct disorder and 21% antisocial behaviour (Klein & Mannuzza 1991)

Our question is weather this relationship could be explained through a medical, psychological or social model or weather it cannot be understood without a holistic frame such as the bio-psychosocial model.

Medical model

Despite the increasingly articulated opposing views, Kecmanovic (2008) considers the medical model to be a dominant model in psychiatry over the last thirty years. Kecmanovic (2008) notes that biological theories form the conceptual basis of the medical model. This model posits that only what happens on the level of the structures and functions of individual organs and organ systems, primarily the brain, is relevant. Mental manifestations are epiphenomena of «deeper» biological activities taking place in the synapses and of cerebral neurochemical and neuropsychological processes (Kecmanovic 2008).

AD/HD is a highly heritable disorder (Neuman et al. 1999) Genetic factors explained 76% and 92% of the covariance between hyperactivity and inattention (Rietveld et al. 2004). Specific regional alterations in the process of membrane phospholipids synthesis in AD/HD which show progression over time and suggest an ascending dysfunction, with the basal ganglia impairing the fine-tuning of cortical control processes or executive functions in the slower-developing prefrontal cortex of children with AD/HD have been recorded (Stanley et al. 2008).

There is a lot of evidence that behavioural problems constitute a part of AD/HD:

- Early hyperactivity predicts later aggression and together with early aggressive behaviour disorder
significantly predisposes a child to persistent antisocial behaviour (Simonoff et al. 2004).

- A degree of impulsiveness (a component of AD/HD) correlates with aggression and self-harming (Barrat et al. 1999).
- Impulsiveness and aggression present stable signs and predict conduct disorder and delinquency (Moffit 1993)
- Behaviour disorders accompanied with AD/HD are usually more persistent and accompanied with more pronounced aggression, compared to ADD without hyperactivity (Lynam 1998, Taylor et al. 1996).

Herpertz et al. (2001) found that boys with ADHD plus CD showed a psycho physiological -response pattern that is very similar to that reported from studies with psychopathic antisocial personalities. Their findings dispel the commonly held belief that attention-deficit/hyperactivity disorder itself leads to delinquency. This statement is in concordance with Moffit’s (1993) findings that only a minority of children with conduct disorder, showing aggressive behaviour at school start up, have also shown early neuropsychological deficits which are represented through hyperactivity, impulsivity and decreased self control.

Contrary to this, Aney et al. (2008) examined three measures of conduct problems in 1,043,963 autosomal markers in order to identify candidate genes that may be important in AD/HD and AD/HD-related traits, such as conduct problems, and did not find genome-wide statistical significance (P<5x10^-7) for any of the tested markers and the three conduct problem traits.

The above evidence is close to the theories of Lombroso’s inborn criminals (Milovanovic 2005), while at the same time they speak little about the possible interaction of biological and psychosocial factors.

So far, only two recently published studies have dealt with this issue.

In the U.S. Department of Education Survey, Scheffler et al. (2009) followed 594 children with AD/HD and found that children with AD/HD who were on medication treatment scored on average 2.9 points better on a maths test and 5.4 points better on a reading test, in comparison with children who did not have any treatment. This research speaks in favour of the biological approach; however, there are studies indicating that this issue is not as simple, especially regarding behavioural problems.

Molina et al. (2009) expanded an initial 14-month study including 579 children with AD/HD into an eight-year follow-up, and concluded that there were no differences in the symptoms and functioning between the children classified into different groups based on the treatment, and that one-year treatment for AD/HD did not predict future function regardless of the type and intensity of the disorder. Children with AD/HD had considerably more academic and social problems, as well as more frequent behaviour problems, including more contacts with the police, more depressive conditions and psychiatric hospitalizations, compared to their peers without AD/HD, suggesting that the biological approach cannot solve all the problems associated with AD/HD.

Additionally, review of early family/parent training, which is partly based on the notion that quality of parent-child relations will facilitate learning to control impulsive, oppositional, and aggressive behaviour, indicates that early family/parent training is an effective intervention for reducing behavioural problems among young children and the effect size is in the 0.23 to 0.45 range, depending on the weighting procedure employed, corresponding to approximately 50% recidivism for the control group compared with 39% and 28% recidivism in the experimental group, respectively (Piquero 2008).

Psychological model

The psychological model represents a personalized model where symptoms are treated as manifestations of «deeper» processes, whereby in this case deeper processes do not involve cerebral structures and functions, but rather early individual experience, in particular early childhood experience (Kecmanovic 2008).

Aggressive behaviour among preschool children is common (Blader et al. 2007). According to Trembley et al. (2004), only one third of three-year-olds do not show aggressive behaviour, whereas one third of all boys and one fifth of all girls at this age show pronounced physical aggression. At older preschool age, and with starting school, aggression reduces (Trembley et al. 2004), which correlates with the process of socialization and highlights its significance for the peer behaviour in the period from the age of three to seven. This is not inconsistent with the fact that aggressive adolescent behaviour often begins in childhood (Lahey et al. 2005).

Concerning hyperactivity disorder, studies (The MTA Cooperative Group 1999) dealing with multimodal treatment of AD/HD children aged 7-9.9 years, lasting 14 months, found that pharmacotherapy for basic AD/HD symptoms was superior to behavioural treatment and routine social interventions.

Furthermore, combined treatment did not show significantly greater benefit for children compared to the lone treatment of AD/HD basic symptoms (The MTA Cooperative Group). However, although psychosocial treatment had only a moderate influence on basic symptoms of AD/HD, it helped alleviating and solving problems that were equally disabling for children as AD/HD itself (in particular, opposition and relations with peers and parents). Insisting, therefore, that, like the medical model, AD/HD is only a psychological problem, and hence the statement that AD/HD is not a deficiency, defect, or a neurological disorder, but more truly a disorder of problem-solving skills (Weathers & Weathers), can, on the other hand, pose ethical as well as profit-related questions in the same way and to the same extent as medical model did.
Rutter and Rutter (1993) state that major psychological theories have significant shortcomings. None of them provides a complete explanation of the developmental process and most of them ignore the social life of children, despite the fact that social life represents a crucial part of our heritage as social beings. In addition, the problem of „major theories“ is also that they all have been proven wrong or at least as having serious deficiencies as regards their very conceptual issues (Rutter & Rutter 1993).

**Social model**

Not all events have the potential to produce a mental disorder; it is primarily socio-cultural events that include a sudden change of the socio-cultural environment, a loss, socio-cultural disorganization, ambivalence or ambiguity of social norms, and those that prevent people from satisfying their primary and secondary needs, over either a short or prolonged period, that have this potential. According to the social model, the core issues are relations between an individual and relevant social groups, as well as individual reactions to different unfavourable social circumstances, be it a so-called life-event or a great man-made or natural disaster (e.g. earthquake, war) (Kecmanovic 2008).

A key finding of this line of research deals mainly with certain risk/protective factors, distinguished between trajectory groups. Thus, Nagin and Tremblay (2001) found that there is a high probability that boys born to poorly educated mothers who began childbearing as teenagers will follow a chronic offender trajectory.

While the risk factors outlined have been shown to be implicated in the development of conduct disorders, it is important to note that not all children exposed to these factors develop one. Rather, the evidence suggests that in children who do develop conduct disorders, aetiology consists of a combination of biological, psychological and social factors (Webster-Stratton & Dahl 1995).

**Biopsychosocial model – does it make sense?**

Elements of the bio-psychosocial model can be conceptualised as a systems’ hierarchy entailing levels of organisation. General systems theory is axiomatic about such hierarchies of knowledge. Lower levels of organisation are necessary for the existence of the higher ones, but not sufficient to describe, or explain, their nature (Pilgrim 2002).

According to Suls and Rothman (2004), the purpose of the bio-psychosocial model is to provide a framework for multisystem and multilevel study of human functioning, denoting that its role is not to give a recipe, and so the question arises as to who should provide solutions and prescriptions. In their opinion, the biopsychosocial model would also present a model of the “work in progress”. For Dobzhansky (1999), the dichotomy between environmental and genetic factors is false; their relationship should be determined in each individual case, and in this sense behaviour genetics has a crucial role. Rutter et al. (1999) cites Lyons’s study from 1995, where antisocial behaviour of American soldiers was examined analyzing the life cycle of an individual. The structural analysis model showed that genetics accounted for 7% of variance in childhood disorders, but also for 43% of variance in antisocial personality disorders in adulthood. As regards hyperactivity disorders, behavioural genetics (Rothenberger et al. 2004) provides data on the biological share in the AD/HD syndrome.

So far it is clear that behavioural disorders are biopsychosocially founded, however, the question remains whether the bio-psychosocial model can explain how different factors interrelate e.g. is there a recipe?

**Where is the recipe?**

**Neuropsychology or biopsychology?**

Neuropsychology studies brain-behaviour relations. In such a way, it covers the bio-psychological dimension of the biopsychosocial model. As early as the 1990s, Gottfredson and Hirschi (1990) ascribed problematic behaviours throughout one’s lifetime to diminished capacity of self-control. Later neuropsychological studies showed association between the problem of self-control and executive functions, which are related with the prefrontal area. Stahl (2008) associated hyperactivity with the activity of the prefrontal motor cortex, impulsivity with the orbital part of the prefrontal motor cortex, and attention with anterior cingulate (selectivity) and dorsolateral prefrontal cortex (attention maintenance).

**From the environment to the genes – epigenetics or biosocial model**

Caspi et al. (2002) studied the genes that affect activity of monoamino oxidase (MAO) in relation to manifestations of antisocial behaviour under the influence of negative life events, and found that negative life events had significantly higher impact in the case of low MAO activity than in the case of high MAO activity. Despite the recent controversial findings about the environmental effects on genes, Rutter (2006) thoroughly analyzed all aspects of the relationship between inherited and acquired, highlighting epigenetic phenomena and giving evidence of gene- environmental connections.

**Neurobiology and psychotherapy: inevitable dialogue**

According to Fuchs (2004), Bowlby’s attachment theory offers grounds for the psychobiological model of social development of the brain, where psychotherapy
can be viewed as a new form of bonding that could bring about affective homeostasis and restructuring of the implicit memory associated with attachment. Indeed, neuroimaging studies have demonstrated that psychotherapy can significantly change the structure and functioning of the brain, in ways that differ from the effect of pharmacotherapy.

**Where is the recipe – which factors should also be taken into account?**

Engel’s (1970) model represents, in a way, a vertical dimension, a hierarchical arrangement from the smallest (molecular) to the biggest (exosystem) parts. On the other hand, it needs another, or horizontal, dimension which would complete the framework. Time, or developmental process, should be an integrative part of the bio-psychosocial model.

A key observation in longitudinal studies of antisocial behaviour, delinquency, and crime indicates that chronic disruptive behaviour emerging early in the life course leads to frequent and oftentimes serious delinquency and crime in childhood, adolescence, and adulthood (McCord et al. 2001, Piquero et al. 2003).

Meyer (in Pilgrim 2002) argued that the elucidation of the patient’s problems must be in relation to their personal history, not merely their current mental state. For example, Tasman (2009) considers timeframe to be an important factor in assessing the effects of stress on personality development, while Niederhofer and Reiter (2003) point out a connection between attachment and temperament at the age of 6 months.

In the frame of bio-psychosocial factors (as vertical dimension) and development (as horizontal dimension), there is a range of additional factors. Rutter and Rutter (2001) say that protective and risk factors can appear as mediators, moderating, independent, overlapping, or proxy, which indicates the necessity for a great number of factors to be methodologically defined. Finally, we are active beings, capable of a certain degree of selecting and processing of our own experience.

**CONCLUSION**

Kecmanović (2006) summarized current criticism of the bio-psychosocial model as “it gives ingredients without a recipe”, thus arguing that this model is insufficient for theoretical and practical use. We accept the conclusion of Suls and Rothman (2004) that the purpose of the bio-psychosocial model is to provide a framework for multisystem and multilevel study of human functioning. The bio-psychosocial model is basically a hierarchical one. It is sufficient that the knowledge from different science can be incorporated in it. In psychiatry, this includes the knowledge of developmental psychology, neuropsychology, epigenesis, methodology…

Should we accept that all the factors are interrelated and interdependent (Logo 2008), we are directed towards careful analysis of their relationships, in spite of not having a final solution, and if we look at them developmentally we can find how they work together.

**REFERENCES**


41. The MTA Cooperative Group: A 14-Month Randomized Clinical Trial of Treatment Strategies for Attention-Deficit/Hyperactivity Disorder. Arch Gen Psychiatry 1999; 56:1073–1086.


Correspondence: