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EDITORIAL

Child psychiatry and the developmental perspective

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In 1963, J. Cotter Hirschberg, in a conference named "The basic functions of a child psychiatrist in *any* setting", highlighted that one of the several unique contributions which child psychiatrists may make in our work was the understanding of a symptom in the context of development [1]. Three years later, Lee Robins [2] published the landmark longitudinal study ''Deviant children grown up'', demonstrating the continuity between conduct problems in childhood and antisocial personality disorder in adulthood, and by that, clearly demonstrating to the mental health field the value of tracking people across the lifespan, since childhood, to understand psychopathology. In the following decades, Sroufe and Rutter [3], Garmezy [4], and Cicchetti [5] published influential papers that established the discipline of developmental psychopathology. In parallel, an upsurge of new technologies has unveiled the genome and the brain, paving the way to identify atypical neurodevelopmental processes underlying psychopathology. Today, a developmental perspective is still pivotal in our clinical work as child and adolescent psychiatrists, but not unique to us: a developmental perspective now shapes the current conceptualization and investigation of mental disorders, from those that are apparent in early childhood to senescence [6], and also the avenues to treat them [7].

Our field has benefited tremendously from investigations that took advantage of a developmental framework, and fortunately their number has been increasing dramatically over time. Several important examples have been published in the European Child & Adolescent Psychiatry in the past years, with emphasis on early predictors of later psychopathology [8, 9], continuity and discontinuity of psychopathology across development [10–13], risk and protective factors investigation [14], early intervention [15], and others approaches [16]. In this issue, we see two studies with this same perspective investigating the biological underpinnings of psychopathology.

The first study, by Walhovd and colleagues [17] from the University of Oslo, Norway, tested whether the dimensional nature of behavioral symptoms in childhood correlated with brain maturation. Built upon on the finds from Judith Rapoport, Philip Shaw and their team, who beautifully demonstrated that children with attention-deficit/hyperactivity disorder attained a maturational milestone (the peak of cortical thickness) later in comparison to control children [18], Walhovd and colleagues now tested cross-sectionally the association between cortical thickness and hyperactive, inattentive, and conduct symptoms in 107 healthy children from 8 to 19 years. The authors found no evidence of association between hyperactive and inattentive symptoms in healthy children and cortical thickness. Nevertheless, conduct symptoms were associated with cortical thickness controlling for age in left prefrontal and supramarginal areas. This association was being largely driven by younger children, where the interactions between conduct symptoms and age were reflected in regionally thinner frontal and temporal cortices bilaterally.

The second study, by Beitchman and colleagues [19] from the University of Toronto, Canada, tested the association between oxytocin genes and behavioral characteristics of children with antisocial behavior. Built upon on an extensive body of work that has demonstrated that oxytocin has important cognitive and behavioral effects, such as to facilitate social memory and afiliative behavior, which are

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impaired in disorders such as autism [20], Beitchman and colleagues now tested the association between six polymorphims on the *oxytocin* and *oxytocin receptor* genes and child aggression and callous-unemotional traits in 162 children with antisocial behavior compared to healthy adult controls. The authors found no polymorphism to be associated with aggressive behavior in the matched case–control analysis. Evaluating children with callous-unemotional trait, which reflect more severe emotional dysfunctions such as lack of empathy, lack of guilt, and shallow emotions, authors detected interesting results. The polymorphism rs237885 on the *oxytocin receptor* gene, and the haplotype consisting of this same polymorphism and also by rs2268493, were associated with significantly higher callous-unemotional trait in children with antisocial behavior. The haplotype analysis result was not significant when the sample was restricted to caucasians, but the single marker result was still significant, as well as when correction for multiple testing was applied.

The two studies published in this issue took advantage of relatively recent technologies—magnetic resonance imaging and genomic sequencing—to explore potential biological processes involved in the development of behavioral symptoms. The first study shows us that the presence of conduct symptoms even in children without a diagnosable mental disorder are reflected by impairment in the brain maturation. The second study shows us that *oxytocin receptor* gene is likely to be involved in the development of callous-unemotional trait. Their clinically oriented strategies of studying symptoms dimensionally and exploring a refined phenotype demonstrate how fruitful the dialogue between researchers and clinicians can be. New technologies that can deeper understand the brain, and consequently atypical neurodevelopmental processes, are now a reality. Genome-wide DNA and RNA analysis of human brain tissue and induced pluripotent stem cells are the two among several others with enormous potential. Their potential to impact their field, however, will also depend on a persistent dialogue among clinicians, clinical and basic investigators. As child and adolescent psychiatrists, our contribution is valuable and we shall work collaboratively and think innovatively to "*open new avenues and challenge the wisdom of the day*" [21].

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