Attention-deficit/hyperactivity disorder (ADHD) is the most common neurodevelopmental disorder in children and adolescents. It is characterized by age-inappropriate inattention/impulsiveness and/or hyperactivity symptoms. ADHD shows a high comorbidity with oppositional defiant disorder (ODD), a disorder that features symptoms of emotional lability. Due to this comorbidity, emotional lability was long considered a secondary consequence of ADHD, which could arise under the influence of environmental factors such as inefficient parenting practices, as part of an ODD diagnosis. In this model of heterotypic continuity, emotional lability was considered not to play any causal role regarding ADHD symptomatology.

LITERATURE FINDINGS:
As opposed to this view, it is now well established that a large number of children with ADHD and without any comorbid disorder exhibit symptoms of emotional lability. Furthermore, recent studies have found that negative emotionality accounts for significant unique variance in ADHD symptom severity, along with motor-perceptual and executive function deficits. Barkley proposed that ADHD is characterized by deficits of executive functions, and that a deficiency in the executive control of emotions is a necessary component of ADHD. According to this theory, the extent to which an individual with ADHD displays a deficiency in behavioral inhibition is the extent to which he or she will automatically display an equivalent degree of deficiency in emotional inhibition. However, not all children with ADHD exhibit symptoms of emotional lability, and studies have found that the association between emotional lability and ADHD was not mediated by executive function or motivational deficits. Task-based and resting state neuroimaging studies have disclosed an altered effective connectivity between regions dedicated to emotional regulation in children with ADHD when compared to typically developing children, notably between the amygdala, the prefrontal cortex, the hippocampus and the ventral striatum. Morphological alterations of the amygdala have also been reported in previous structural studies in children with ADHD.

DISCUSSION:
Emotional lability can result from different neurobiological mechanisms. In particular, bottom-up and top-down processes can be opposed. Bottom-up related emotional dysregulation involves an increased emotional reactivity, and is thought to be linked to the automatic evaluative activity of the amygdala. Top-down mechanisms are associated with the regulation of such activity, and rely on a prefrontal network including the lateral prefrontal cortex, the anterior cingulate cortex and the orbitofrontal cortex. Since various neuropsychological impairments and alterations in multiple brain networks have been implicated in the etiology of ADHD, contemporary models emphasize its neuropsychological heterogeneity. It is therefore likely that some but not all children with ADHD will exhibit neurobiological alterations in circuits dedicated to emotional regulation, possibly at different levels. Future research will have to identify the different causal pathways and to decide whether emotional lability represents a criterion to subtype ADHD diagnoses.

CONCLUSION:
Emotional dysregulation is now known to play a causal role regarding ADHD symptomatology. Along with executive functioning, reaction time variability and potentially delay aversion, emotional dysregulation should therefore be included in future theoretical models of ADHD, as well as in clinical practice when identifying the major impairments in this diagnostic group and when deciding therapeutic strategies.