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Commentary

Eating Disorders in Early Childhood

Yoba C*, Kamogh Zan Ane

Department of Pediatrics, CHU Yalgado Ouédraogo, Ouagadougou, Burkina Faso

DESCRIPTION

Eating disorders are complex heritable conditions that can be influenced by both genetic and environmental factors but are poorly studied in early development. Eating disorders in early childhood affect approximately 25% of children with typical development and 80% of children with developmental disorders. They may resolve spontaneously during development or produce more structured and severe problems for which late interventions would harm their outcome. These disorders are studied by focusing on both the child who, independently from food availability, feeds inadequately (Undereating (UE)) or excessively (Overeating (OE)), and possible early-arising maladaptive aspects of the parent-child relationship with associated low-quality interactions during feeding and play. During feeding, a negative maternal affective state and interactions characterized by conflictual, non-collaborative, and non-empathetic communication, may contribute to children's food refusal and to a negative emotional state. This makes feeding an unpleasant moment for mother and child, in which the maladaptive interaction and children's disordered eating reciprocally reinforce, ultimately leading to an ED. Maternal intrusiveness or withdrawal in interactive exchanges with the child may depend on a lack of pleasure in reciprocal interactions, probably produced by an impairments in the neurobiological circuits of reward. Conversely, parents' attention and sensitivity to the needs of the child, including the recognition of cues of hunger and satiety from breastfeeding and weaning, appear as key factors in the prevention of early EDs. Observational procedures for the assessment of the quality of mother-child interactions during feeding such as the Scale for the Assessment of Feeding Interaction have been used to evaluate at-risk patterns of caregiver-children's interactions associated with children's low ability to regulate affects and behavior, frequently correlated with EDs.

A genetic influence on EDs has been proposed as well. Maladaptive relational indicators may have a genetic basis, may emerge early during the first year of life and constitute a very significant risk factor for the appearance of children's disordered eating. Genetic studies, supported by the identification of neurotransmitters, hormones and peptides possibly regulating eating behavior and involved in the pathophysiology of EDs, have investigated, among others, genes encoding factors responsible for dopamine dynamics, including receptors, membrane transporters and metabolizing enzymes, due to DA activity in subcortical and hypothalamic circuits controlling brain reward systems, appetite and satiety pathways. Altered functioning of DA circuitry is involved in psychopathology, including abnormal feeding, and may be associated with the dysregulation of adaptive emotions and to internalized states of hypervigilance, withdrawal and inhibition, depression, anxiety and attachment insecurity.

Despite the large amount of information gathered on the genetics of major EDs in adults, data on young children are scarce or completely lacking. Indeed, studies on genetic variables associated with children's EDs would be particularly interesting in Developmental Psychology, allowing the proposal of paradigms intertwining psychological and biological variables.

Genetic analysis was focused on Variable Number of Tandem Repeats (VNTR) polymorphisms of genes encoding for the dopamine D4 receptor and Dopamine Transporter (DAT1).

Since DA availability in subcortical synapses depending on DAT1 activity may influence DRD4 responses, similarly to other studies carried out on inhibition/impulsivity or in hyperactivity, we observed the possible interaction between the two gene polymorphisms for UE and OE behaviors.

Genetic aspects were studied in association with behavioral features, the CBCL analyzes the presence of externalizing, internalizing and dysregulation symptoms in affective, behavioral and cognitive areas.

Correspondence to: Yoba C, Department of Pediatrics, CHU Yalgado Ouédraogo, Ouagadougou, Burkina Faso, E-mail: caoyoba@yahoo.fr

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