



Obesity and Diabetes Risk In Children With Sensory Processing Disorders

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Abstract

Background: Sensory Processing Disorders represent a widespread abnormalities of behavior, social interaction, communication and learning development rising among people in the whole world. It is reconized that there is a significant number of children who were not diagnosed with Sensory Processing Disorders; also that early interventions have an essencial impact on the quality of life of this target population. In this context, food selectivity and sensory sensivity could be associated to eating problems. **Objective:** The aim of this review was to provide a concise summary of the research published in the years of 2014-2018, with an emphasis on the risk of Obesity and Diabetes in children with SPD. **Methods:** This Minireview considered the Pubmed, Lilacs and Scielo bases, with the keywords Autism, Attention Deficit Hyperactivity Disorder, Obesity, Diabetes and Child. **Results and Conclusions:** 10 papers were selected. There seems to be a consensus in the literature about the possibility of an association between these variables. However more strong scientific evidences are necessary.

Keywords: Autism; Attention deficit hyperactivity disorder; Obesity; Diabetes

Abbreviations: ADHD: Attention Deficit Hyperactivity Disorder; SPD: Sensory Processing Disorders; TD: Typical Development; ASD: Autism Spectrum Disorder; FP: Feeding Problems; ID: Intellectual Disability; BMI: Body Mass Index; SPS: Sensory Processing Sensitivity; T1D: Type 1 Diabetes

Introduction

Sensory Processing Disorders or SPD have been more diagnosed in children during the last decades, with significant growing. In this context, there are still lacks of needed information on this subject; particularly the impact on the quality of life of those affected and their families.

This Minireview raises the risk of obesity and diabetes in this target population, considering scientific articles published between the years of 2014-2018, with an emphasis on the risk of Obesity and Diabetes in children with SPD. We considered Pubmed, Lilacs and Scielo bases, with the descriptors "Autism", "Attention Deficit Hyperactivity Disorder", "Obesity", "Diabetes" and "Child". The exclusion criteria were articles repeated in databases, editorials and letters that did not appear in scientific paper format.

Little et al. [1] examined sensory processing in 239 children ages 3-14 years with Autism Spectrum Disorder (ASD), Attention Deficit Hyperactivity Disorder (ADHD), and Typical Development (TD). These authors related that children with ASD showed the highest rate of oral processing differences.

Children with autism demonstrated significant feeding inflexibility. They are often described as picky eaters with low intake of fiber-rich foods, including fruits and vegetables, but with a higher intake of carbohydrate [2,3].

The study of Riederer et al. [4] indicated that lower white matter connectivity within white matter tracts of insula-fronto-striatal taste-reward circuitry are associated with obesity as well as greater connectivity within white matter tracts connecting the amygdala and Anterior Cingulate Cortex. The specificity of regions suggests sensory integration and reward processing are key associations that are altered in and might contribute to obesity. Bell, Couthard & Wildbur [5] investigated the relationship between self-disgust and sensory processing within eating psychopathology.

According to their results, significant associations would be found between reported self-disgust and sensory processing and these associations were predicted to vary between different eating disorders. Higher anxiety and lower sensation seeking were found to be significantly

associated with higher self-disgust scores, which demonstrate a possible association between those who are both anxious and less likely to seek sensation within their environment with feelings of self-disgust across the eating psychopathology spectrum.

Crasta et al. [6] compared the prevalence and profile of Feeding Problems (FP) and their relationship with sensory processing in 97 children with autism and Intellectual Disability (ID), between ages 3- to 10-year-old. According to these authors the prevalence of FP were 61.0% and 46.4% among children with autism and ID respectively. Feeding Problems were severe among children with autism, especially in young children; disruptive meal-time behaviors and food over select. Feeding Problems and various dimensions of sensory processing were significantly associated.

Obesity is a public health concern for the population in general and for children with ASD specifically. Lawson & Foster [7] investigated the relationships between sensory patterns, obesity, and physical activity engagement of 77 children with ASD. Their results showed that almost half (42.2%) of the children were overweight or obese, and sensory avoiding behaviors were related to higher Body Mass Index (BMI). Shedlock et al. [8] evaluated an increased risk of obesity, type 2 diabetes mellitus, hypertension, hyperlipidemia, and nonalcoholic fatty liver disease/nonalcoholic steatohepatitis in children with ASD. The authors findings suggested that children with ASD have an increased risk of obesity and obesity-related metabolic disorders.

According to Goldberg et al. [9], Sensory processing sensitivity (SPS) is a recently proposed construct that refers to a genetically influenced tendency to more strongly and deeply process a variety of information. These authors examined whether SPS was associated with an autoimmune disease such as Type 1 Diabetes (T1D) in 128 adolescents, T1D is associated with higher levels of SPS. Hence, there is a need to develop interventions, treatments, and care focused on the needs of T1D patients with SPS temperament, aimed at better treatment adherence. Furthermore, longitudinal research is needed to evaluate whether SPS is a risk factor in the development of T1D.

Sukasem et al. [10] explored the association of genetic polymorphism of genes related to pharmacokinetics or pharmacodynamics with insulin resistance in 89 children and adolescents with ASD and treated with risperidone. Their results revealed that 5 (5.62%) patients presented with hyperglycaemia and insulin resistance was detected in 15 (16.85%) patients. This finding suggested that Brain-Derived Neurotrophic Factor

(BDNF 196G $\neq$ A) polymorphism may be a genetic marker for predicting insulin resistance before initiating treatment in patients treated with risperidone. This is one of the most attractive neurotrophic factors because of its role in neuroplasticity and brain repair. But it is necessary more robust and reliable evidences.

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