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Sweet and Sensitive: Sensory Processing Sensitivity and Type 1 Diabetes

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ABSTRACT

Objective: 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. The aim of the study was to examine whether SPS is associated with an autoimmune disease such as type 1 diabetes (T1D).
Research design and methods: Participants were 128 adolescents (62 with T1D and 66 comparisons [without autoimmune disease]) and their parents who completed the Highly Sensitive Person Scale (HSPS) questionnaire, assessing SPS level.
Results: Higher levels of SPS were found in the T1D group than in the comparison group. Furthermore, the frequency of SPS trait was significantly higher in the T1D group than in the comparison group.
Conclusions: 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.

The fundamental way that individuals perceive and respond to their environment is through the processing of sensory information (Aron & Aron, 1997; Aron, Aron, & Jagiellowicz, 2012; Dunn, 2001; Jerome & Liss, 2005). People have different thresholds for perceiving, responding to, and becoming overwhelmed by sensations, which are reflected in individual lifestyles, moods, and temperaments (Dunn, 2001).

Sensory processing sensitivity (SPS), a recently proposed construct, refers to a tendency to more strongly and deeply process a variety of information including the arts, caffeine, other peoples' moods, hunger, and pain. Roughly 20% of the population is hypothesized to be highly sensitive. They tend to process and respond to lower thresholds of information and to better detect subtle differences in the environment. These processing differences are **genetically based, present at birth**, and located in the central nervous system (Aron et al., 2012; Aron & Aron, 1997), and polymorphisms both in the serotonin and in the dopamine systems have been implicated (Chen et al., 2011; Homberg, Schubert, Asan, & Aron, 2016). People high in SPS also tend to be more in tune with their own thoughts and emotions, to be more aware of the emotions of others, and to be prone to "pause to check" in new situations due to their predisposition to wariness (Aron et al., 2010; Aron et al., 2012; Aron & Aron, 1997).

SPS was comprehensively studied in a series of seven studies designed to create and validate the Highly Sensitive Person Scale (HSPS), a self-report measure of sensory processing style (Aron & Aron, 1997). Sensory processing is related, but not identical, to the constructs of behavioral inhibition (Carver & White, 1994; Gray, 1991), introversion (Eysenck, 1991), shyness (Kagan, 1997), and neuroticism (Aron & Aron, 1997). Aron and Aron (1997) argued that SPS has been confused with neuroticism and fearfulness because both highly sensitive and neurotic or fearful individuals may not proceed in the face of novel situations. Overall, highly sensitive people are more likely to experience anxiety disorders such as social phobia (Kinnealey & Fuiek, 1999; Liss, Mailloux, & Erchull, 2008; Neal, Edelmann, & Glachan, 2002), avoidant personality disorder (Meyer & Carver, 2000), and depression (Johnson, Turner, & Iwata, 2003). Thus, highly sensitive people are not necessarily prone to more negative emotional states, but they may be more sensitive to negative parental environments and are more prone to negative affectivity when exposed to negative environments (Aron, Aron, & Davies, 2005; Liss, Timmel, Baxley, & Killingsworth, 2005). Furthermore, SPS is positively correlated with levels of stress and symptoms of ill-health (Benham, 2006).

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Given that many SPS individuals experience sensory bombardment (Aron & Aron, 1997), they are more susceptible to environmental influences; for example, they are more affected than others by negative developmental experiences and environmental exposures (Hartman & Belsky, 2015) and may experience higher levels of stress (Aron et al., 2005; Liss et al., 2005), which can also lead to diseases and further life experiences that strongly affect the sensitivity genotype by increasing its phenotype expression (Pluess, 2015) and activating the sympathetic nervous system (Shoenfeld et al., 2008).

The present study examined whether SPS is associated with an autoimmune disease such as diabetes mellitus type 1 (also known as juvenile diabetes or type 1 diabetes [T1D]). Since Hawkins, Davies, and Holmes (1957) first proposed the link between stress and illness, stressful life events have been found to be positively associated with chronic diseases (Renzaho et al., 2014; Shoenfeld et al., 2008; Stojanovich & Marisavljevich, 2008).

The loss of body self-tolerance, as in the case of autoimmune diseases, is considered to be caused by genetic, hormonal, immunological, and environmental factors (Shoenfeld & Isenberg, 1989). >80% of patients reported emotional stress before disease onset (Stojanovich, 2010), and the disease can cause further stress that may in turn lead to other autoimmune diseases (Shepshelovich & Shoenfeld, 2006). The role of stress as a pathogenic factor in autoimmune diseases has been discussed in the literature (Carter, Herrman, Stokes, & Cox, 1987; Herrmann, Schölmerich, & Straub, 2000; Persson, Berglund, & Sahlberg, 1999; Wolfe, 1999), and it is presumed that neuroendocrine hormones triggered during stress may lead to dysregulation/altered or amplified cytokine production. This response can trigger the hypothalamic-pituitary-adrenal axis and sympathetic nervous system (Shoenfeld et al., 2008).

The current study focuses on T1D, as a case of autoimmune disease. T1D is one of the most common chronic diseases among children; 15,000 children are diagnosed each year in the United States alone (Juvenile Diabetes Research Foundation, 2016). The disease is caused by autoimmune destruction of insulin-producing beta cells of the pancreas, leading to deficient insulin production, which renders the body unable to control the amount of sugar in the blood. The total dependence on an outside source of insulin has short- and long-term implications (e.g., cardiovascular diseases and problems with the limbs, blindness, kidney failure, coma, and even death) (Compas, Jaser, Dunn, & Rodriguez, 2012).

Adolescents with T1D are more likely to be at risk of peer-group ridicule and violence than their healthy counterparts and to exhibit high internalizing and externalizing symptoms and high involvement in risky behaviors (Luyckx, Seiffge-Krenke, & Hampson, 2010; Silverstein et al., 2005; Storch et al., 2004). Furthermore, adolescents with T1D are at risk for diabetes complications, which can be exacerbated by their need for individualization and independence (Silverstein et al., 2005). Hence, the complex treatment and new lifetime regime affect the routines of the adolescent and his/her family, posing emotional challenges for and exerting stress on the whole family system (Cunningham, Vesco, Dolan, & Hood, 2011; Landolt, Vollrath, Laimbacher, Gnehm, & Sennhauser, 2005).

Taken together, the study objectives were to compare SPS levels between a group of adolescents with T1D and a comparison group (without autoimmune diseases) to investigate whether adolescents with T1D have higher SPS levels than those in the comparison group, and to compare the frequency of the SPS trait within each group. Thus, we hypothesized as follows:

Hypothesis: The T1D group will show significantly higher levels of SPS than the comparison group, and the frequency of SPS traits will be higher in the T1D group.

Research Design and Methods

Participants

Participants ($N = 128$) included 62 adolescents with T1D and 66 adolescents without an autoimmune disease for comparison, matched to T1D participants. All come from middle-class homes, and all speak Hebrew as a native language.

The T1D group ($n = 62$) includes 35 males (56.5%) and 27 females (43.5%); their average age was 16.06 years ($SD = 3.47$). Participants in the T1D group were diagnosed at a mean age of 10.97 years ($SD = 5.57$), and with an average Hb1Ac level of 7.17 ($SD = 1.54$). Participants diagnosed with diseases in addition to T1D were excluded from the study.

The comparison group ($n = 66$), participants without an autoimmune disease, includes 38 males (56.7%) and 29 females (43.3%); their average age was 15.09 years ($SD = 4.09$). Participants' demographics are shown in Table 1.

Instruments

a) **Demographic questionnaire.** A six-item questionnaire gathered information about gender, age, parents' marital status, socioeconomic status, and the T1D disease (i.e., onset of T1D, Hb1Ac level).
b) **The Highly Sensitive Person Scale (HSPS)—child report and parent report** (Aron & Aron, 1997). This 27-item questionnaire evaluates the SPS trait. Both versions, child and parent, have showed solid reliability and discriminant and convergent validity (Acevedo et al., 2014; Aron et al., 2005; Aron & Aron, 1997; Liss et al., 2005). Respondents are asked to respond to items using a Likert scale ranging from 1 (*not at all*) to 7 (*extremely*). Example items include "Do other people's moods affect you?" and "Are you easily overwhelmed by strong sensory input?" In the current study, Cronbach's alphas for the T1D group were $\alpha = 0.86$ (self-report), $\alpha = 0.89$ (parent report), and for comparisons $\alpha = 0.85$ (self-report) and $\alpha = 0.86$ (parent report). Self-report and parent report were highly correlated ($r = 0.73$, $p < 0.0001$).

Procedure

The Helsinki Committee for Experiments on Humans, at a major medical center, approved the study protocol. At the recruitment stage, families were invited to participate in the study by diabetes clinic staff. In the second stage, a comparison group with matched demographic characteristics was recruited, using social media and advertisements. After signing informed consent forms, participants (adolescents with T1D/healthy adolescents, and parents) were asked to complete questionnaires. Participants were told that their anonymity would be preserved throughout the study, that the data collected would be used for research purposes only, and that their names would remain confidential. They were also assured of their right to discontinue their participation in the study at any time and were offered the option to receive the final general research findings.

Results

Preliminary Analyses

To determine whether any demographic and personal characteristics might interfere with the analysis and thus should be controlled for in the analyses, we conducted some preliminary analyses. A *t*-test

Table 1
Participants' Demographic Data.

Characteristic	T1D group ($n = 62$)	Comparison ($n = 66$)
Gender		
Males, *n* (%)	35 (56.5%)	38 (56.7%)
Females, *n* (%)	27 (43.5%)	29 (43.3%)
Age, *M* (*SD*)	16.06 (3.47)	15.09 (4.09)
Diabetes onset age, *M* (*SD*)	10.97 (5.57)	–
Hb1Ac level, *M* (*SD*)	7.17 (1.54)	–

T1D = Type I diabetes.

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