

What Happens to Disinhibited Social Engagement Disorder Over Time?

Helen Minnis, PhD

Dis inhibited social engagement disorder (DSED) is one of the most intriguing syndromes in child and adolescent psychiatry but also one of the least understood. We know little about its developmental course in different groups of children, so I was delighted to see the incisive longitudinal work from the Bucharest Early Intervention Study by Guyon-Harris and colleagues.¹ My own interest in DSED was sparked back in the early 1990s, when a travel bug had taken me to Guatemala and I had found a job in an orphanage. I was also the family doctor to the surrounding villages, in which children were considerably more materially deprived than their peers in the orphanage who had three meals a day, chlorinated running water, and a reliable school. What the orphanage children did not have was family care—instead they experienced rotating shifts of carers, many of whom were travelers like me, some only there for one or two weeks. Prior to my trip, a senior child psychiatrist had armed me with Bowlby's *Attachment and Loss*² and a photocopy of the ICD-10 classification of "disinhibited attachment disorder." My dual role in the orphanage soon made it obvious that the social behavior of many of the institutionalized children was grossly different from that of their family-reared peers. Strangers could not walk into the orphanage until they had peeled off the hands of the small children who had crowded them. In contrast, children in local family homes peeked out from behind their mother's skirts—just as John Bowlby had said they would. Indiscriminate behavior was adaptive in the orphanage setting, because otherwise emotionally neglected children received bursts of attunement from whichever carer was available. The same indiscriminate behavior was, however, highly maladaptive outside the orphanage setting, as children would wander off without checking back and put themselves in danger.

Research has already shown that symptoms of DSED can persist across childhood^{3,4} and even into adulthood,⁵ but the Guyon-Harris *et al.* study goes deeper: it adds new knowledge by examining different profiles in the course of DSED over time, and takes a closer look at factors that might be associated with reduction or persistence of DSED symptoms.¹ This is crucial information for clinicians, social care practitioners, and researchers: we already know that DSED symptoms can persist even into adulthood—what makes this more or less likely?

The study examined how the DSED cluster of symptoms develops with age under different care conditions (the variable-centered approach), and also how groups of young children with DSED develop from early to late childhood (the person-centered approach). Symptoms of DSED were examined in 193 children: 124 children raised initially in institutions and randomized into family foster care or care as usual (usually remaining in the institution, although sometimes placed in foster care later on), as well as 69 age- and sex-matched community comparison children who had never been institutionalized. Children were assessed with the same measure of DSED symptoms (the Disturbances of Attachment Interview)

four times in the preschool years then again at ages 8 and 12 years. The research group followed up well over 90% of the children originally involved to 12 years, so trajectories are unlikely to be much affected by sample attrition. As predicted, both groups institutionalized in early life began with high symptom scores for DSED compared their never-institutionalized peers, and these symptoms reduced significantly over time in the group randomized to foster care after about age 20 months. The person-centred approach took these findings further by identifying 4 profiles: "minimal," with consistently low DSED symptoms over time; "persistent modest," with some low-to-moderate DSED symptoms persisting over time; "early decreasing," with a marked reduction in DSED symptoms in early childhood; and "elevated," with high symptom scores throughout childhood, decreasing somewhat in adolescence. Not surprisingly, the minimal profile contained most of the children who had never been in institutional care. The early decreasing group contained mostly children who had been in institutional care as preschoolers but who were randomized at around 20 months to foster care—as well as a few of the never-institutionalized group. The elevated and persistent modest groups contained mostly children who had been raised in institutions and either remained there or were randomized to foster care. In other words, the DSED symptoms of some children who were randomized to foster care decreased quickly, whereas DSED symptoms were persistent in other children, despite their placement in foster care. More total time spent in institutional care, placement into foster families at an older age, and the number of placement disruptions were associated with elevated or modest symptoms of DSED persisting across development.

This is important work in a young field. The randomized controlled trial design means that we can have confidence that early placement in foster care from an institutional context leads to a marked reduction in DSED symptoms in many children. What is still less clear is the relationship between later foster care disruptions, beyond the trial, and persistence of DSED symptoms. On one hand, it could be that unstable, poor-quality foster care allows DSED to persist: this would chime with the work of Dozier and colleagues demonstrating the importance of foster carer commitment for the development of secure attachment in new foster placements.⁶ On the other hand, children with DSED might be more difficult to care for: in other words, DSED symptoms might contribute to foster care breakdown. We still know little about the impact of persistent DSED symptoms as children develop. Recent work from the European-Romanian Adoption Study suggests that DSED symptoms confer little impairment in adulthood in the absence of comorbid disorders,⁵ but the small corpus of available research suggests that DSED is usually accompanied by comorbid disorders.^{5,7} Both behavioral and molecular genetic work^{8,9} has suggested that genetics may have a role in the emergence of DSED in some maltreated children, and there is also now evidence that attention-deficit/hyperactivity disorder may be

particularly closely linked to DSED. Although it has been extremely helpful for the Guyon-Harris study to focus solely on DSED in order to examine developmental profiles, especially at this early stage of the field, future studies will need to consider the role of comorbidity and child temperament in the development of DSED, and, to achieve this, larger samples including family-reared children with DSED will be required. Because of the challenges inherent in recruiting such samples, pooling of data across studies may have an important future role here.

Children and adolescents with DSED symptoms who do not recognize social boundaries and are indiscriminate in their social behavior are clearly at higher risk than their peers from those few predatory adults looking to prey on vulnerable children. What data we have also suggests that children with DSED often have complex comorbid profiles that may make them challenging to care for.⁷ Treatment trials aiming to reduce DSED symptoms are ongoing, but so far we have no clear evidence base for treatment. This important study has shown that one key way of preventing DSED from emerging to begin with is to place young

children in need of substitute care in family foster care, rather than in institutions. This has wide-ranging implications for childcare practice around the world.

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Professor Minnis is with the Institute of Health and Wellbeing, University of Glasgow, Scotland.

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Correspondence to Helen Minnis, PhD, Institute of Health and Wellbeing, University of Glasgow, University Avenue, Glasgow G12 8QQ, UK; e-mail: helen.minnis@glasgow.ac.uk

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