Diagnostic shortfalls in early childhood chronic stress: a review of the issues

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Abstract
Clinical effects of early childhood chronic stress should be regarded as causing a developmental brain injury. However, current diagnostic constructs fail to capture the associated disabilities in emotional-behavioural regulation of stress and attachment functions adequately. We first focus on neglect as a prototypical early childhood chronic stressor; next we explore clinical associations of neglect; and finally we cite research pertaining to possible underlying pathophysiology of the effects of early childhood chronic stress. In addition, we discuss diagnostic labels that children with histories of early childhood neglect commonly acquire, and implications for treatment.

Introduction
Children with developmental and behavioural problems and histories of chronic early stress (e.g. neglect) face an uncertain diagnostic landscape. There is emerging evidence that the ‘toxic stress’ (Shonkoff & Garner 2012) of early adversity (e.g. poverty, discrimination, maltreatment) interacts with genetic vulnerabilities to cause permanent structural and functional changes to the developing brain. ‘From an evolutionary perspective, there may be nothing more threatening for a young child than the lack or loss of a trusted primary caregiver’ (Maheu et al. 2010). Chronic neglect in the early years is known to be associated with a wide range of clinical problems persisting into adulthood (Gunnar & Fisher 2006), albeit often with latent affects emerging at school age and beyond. Although the clinical picture resulting from early chronic psychological and emotional neglect is relatively clear, children continue to be poorly identified and, subsequently, treated. Certain diagnostic labels that children with developmental problems may acquire [e.g. autism spectrum disorder (ASD), learning disability, fetal alcohol spectrum disorder (FASD), intellectual disability] are often key to accessing supportive services and understanding, particularly in the school system, whereas others [e.g. attention-deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD)] often do not lead to increased formal support and carry damaging stigma that may outweigh benefit of the label. The diagnostic uncertainties perhaps stem from the confusing mix of risk factors (e.g. antenatal substances, family history of mental illness or cognitive difficulties, poverty) and symptoms. No one can ever randomize children experimentally to neglectful parents, so the clinical effects of chronic neglect and other stressors will be understood primarily through observational studies. Nonetheless, there is an important need to recognize this spectrum of pervasive developmental problems. Doing so will make it possible for parents, educators and health professionals to name, communicate about and better understand these disorders in order to facilitate appropriate care.
Scenario

Have you met this child? He is school aged, and likely has been diagnosed with ADHD and ODD. There are ongoing difficulties with a confusing mix of hostile and avoidant behaviours, family interactions (often in an already vulnerable household or foster home), peer relations and coping with demands and feedback of teachers. However, he may be described as often being ‘a really nice kid’, especially one-on-one. Tucked in the back of the thick medical chart are notes from a child protection services (CPS) investigation in infancy or toddlerhood citing concerns of inadequate parenting, maternal depression, neglect and/or exposure to violence. Despite intermittent CPS involvement, no maltreatment has ever been significant enough to warrant action, and the case is eventually closed, or perhaps the child has been apprehended and spent time in one or more foster homes.

School productivity is poor, yet psychoeducational testing confirms ‘average’ cognitive functioning and the absence of a formal learning disability. In clinic, the child is co-operative, compliant and seems behaviourally normal at first glance, or perhaps there is a hint of excessive behavioural lability or inhibition, or both. Little resource support is given at school because of a lack of ‘identification’. Given social skills deficits, tantrums and rigid behaviour, the question of an ASD arises, but developmental consultation does not support this. There is mention of possible FASD, but there are no characteristic facial features, growth parameters are normal, and the mother denies alcohol ingestion during the pregnancy. ADHD and ODD are considered, but treatment with stimulants, atypical antipsychotic medications and perhaps an antidepressant has produced modest results without seeming to modify a developmental trajectory tumbling worrisomely towards possible school failure, mental illness and criminality.

Clinical features of neglected children

Neglect is problematic to define (Dubowitz et al. 2002), is graded in terms of severity and duration, occurs frequently with other forms of maltreatment and is difficult to confirm in individual cases (Fallon et al. 2011). Ongoing suspicions of neglect often persist despite involvement by CPS. Without a psychologically available parent to model and help to regulate emotions and behaviours in the early years, children may acquire a permanent disability in reading and reacting to emotions and intentions in others and in regulating their own emotional states in response to stress (Stirling & Amaya-Jackson 2008). Dubowitz and colleagues (2002) found that at age 3, after controlling for mothers’ depression and sociodemo-

A biological basis for changes associated with early chronic stress

In humans, it is highly probable that many of the mental health symptoms and learning problems associated with child neglect arise in conjunction with alterations of the biological stress.
systems, as the stress response, emotional regulation and neurodevelopment are all dependent on these systems (De Bellis 2005). Children with a history of maltreatment tend to have a relative loss of the typical diurnal pattern of cortisol secretion (Bernard et al. 2010). Atypical production of cortisol has been associated with a multiple mental health disorders, including conduct disorder, anti-social personality disorder, substance abuse disorder and depression (Dozier 2006; Fisher et al. 2007).

The prefrontal cortex modulates reactions to stressful situations by facilitating calculated, executive function-driven responses in balance with the limbic system, which is involved in rapid, emotionally charged (fight or flight) responses. Neuropsychological studies in humans suggest that neglected children have deficits in prefrontal cortex function and difficulty with executive functions (De Bellis 2005). Eluvathingal and colleagues (2006), using diffusion tensor imaging, reported reduced integrity of the white matter tracts connecting the inferior frontal lobe to the anterior temporal lobe (including the amygdala) in seven caregiver-deprived youths who were adopted from Eastern European orphanages, compared with seven comparison youths. Maheu and colleagues in a study involving functional magnetic resonance imaging and exposure to emotionally evocative cues, found that youths with a history of caregiver deprivation and emotional neglect had significantly greater medial temporal lobe activation (amygdala and hippocampus), noting the accumulation of similar work strongly suggesting that caregiver deprivation as well as neglect and abuse influence the functioning of these specific brain regions (Maheu et al. 2010). In another study by Edmiston and colleagues (2011) 42 psychosocially at-risk adolescents without psychiatric diagnoses completed self-report child maltreatment questionnaires and underwent magnetic resonance imaging to measure the volume of brain structures. The researchers found that reductions in corticostrial-limbic grey matter were significantly associated with all subtypes of child maltreatment, suggesting that early maltreatment may cause changes to brain structures responsible for emotion and behaviour regulation with possible symptoms thereof in the absence of formal diagnoses of psychiatric illness.

There is accumulating neurobiological evidence of the details of the emotional and behavioural regulatory problems arising as a result of early chronic emotional stress. With these emerging data in mind, the effects of early chronic stress begin to look more like other conditions such as FASDs, in which an early insult is known to cause a spectrum of neurodevelopmental problems. The neuroendocrine milieu influenced by early excessive chronic stress (in our case, neglect) may be regarded as an insult causing brain injury, analogous to in utero exposure to alcohol. Continuing the brain injury analogy, exposure to chronic neglect may cause a spectrum of neurodevelopmental sequelae ranging from seemingly unaffected to markedly impaired development.

A complex overlap of diagnoses

As indicated in the scenario, children exposed to chronic stress associated with neglect may acquire multiple psychiatric labels. Even in combination, these diagnostic labels generally only capture certain facets of the clinical picture that may result from early chronic stress. It is, however, useful to provide an overview of the diagnostic entities that are frequently applied to these children to better understand the problem.

ODD/conduct disorder/disruptive behaviour disorders

Risk factors for high physical aggression include low income, low maternal education, family dysfunction and presence of younger siblings (Petitclerc & Tremblay 2009). These risk factors not surprisingly focus heavily on parenting vulnerabilities, hinting at neglect and emotional abuse. Disruptive behaviour disorders may be an accurate phenomenological description, but they fail to recognize aetiology or mechanism of functional-behavioural impairment (and subsequent marginalization from enriching social and academic participation). Most importantly, they fail to acknowledge what that information would tell us about management of specific cases. In other words, labelling a child with ODD is at best descriptive, but does not tell much about why they have difficulty and provides little information about interventions (e.g. does this child need more limit setting or more collaboration and flexibility; what modifiable environmental features are triggering the disruptive behaviour?). This formulation is key to addressing clinical problems and enhancing participation in age-appropriate activities (e.g. peers, academics, recreation, family activities), the continual marginalization from which is a potential risk factor for propagating the at-risk trajectory towards adverse outcomes.

Attention-deficit hyperactivity disorder (ADHD)

Maltreatment and ADHD likely share a complex bidirectional relationship: symptoms of ADHD may predispose to maltreatment, or be caused by maltreatment, and of course ADHD and maltreatment may share common aetiological factors (Ouyang et al. 2008) because ADHD has a strong genetic component (Sharp et al. 2009). However, ADHD is also associated with
maltreatment. For example, Ouyang found that ADHD symptoms were associated with neglect and abuse when controlled for psychosocial variables (Ouyang et al. 2008). The Multimodal Treatment Study of Children with ADHD (MTA) was a randomized trial with longitudinal follow-up involving treatment of children with ADHD. Statistical analysis found that a subgroup (i.e. of the ‘latent classes’) of children with the least favourable trajectory was predicted not by treatment randomization, but rather by other factors including family difficulties, drug exposure as well as high initial symptom scores, baseline aggression, lower IQs and lower social skills (Swanson et al. 2007). These ‘latent classes’ of the MTA trial hint at a heterogeneity within the population of children diagnosed with ADHD who may have a different mechanism for their presentations. Again, it is not clear how much of the ADHD in maltreated children is caused by ‘primary’ ADHD in the child versus the adverse environmental effects on the developing brain.

Nonetheless, we know that ADHD can be associated with external factors. For example, Gerring and colleagues (1998) found that children can develop ADHD following traumatic brain injury (distinct from pre-morbid ADHD). Kreppner and colleagues (2001) studied inattention and overactivity in adoptees as a possible ‘institutional deprivation syndrome’. They found that after controlling for low birthweight, malnutrition and cognitive impairment, inattention and overactivity were significantly associated with duration of early psychological deprivation, and that attachment disturbances correlated with inattention and overactivity symptoms.

Autism spectrum disorder (ASD)

Although much ignored, effects of early chronic stress such as neglect figure prominently into the differential diagnosis of ASD. In this clinician’s experience, referrals for possible ASD or questionable diagnoses of ASD are common in the context of deficits in social skills, emotion regulation and language and early chronic adversity. Reflecting on the features of ASD and consequences of early chronic stress, this confusion is not surprising. However, these entities are distinct (although not mutually exclusive), with the genetic aetiology of ASDs becoming clearer (El-Fishawy & State 2010) (as opposed to the now-discredited ‘refrigerator mother’ theory that assumed ASDs were caused by impaired mothering). From clinical experience, children with maltreatment histories without ASD tend to have clinically adequate social connectedness and non-verbal social skills (eye contact, facial expressions, gesturing, to-and-fro conversations etc.) given overall developmental functioning in low-stress situations, unlike children with ASD for whom these deficits are more consistent across environments.

Attachment disorders

Attachment remains a nebulous concept and no attachment-related diagnosis are regularly used by paediatricians, for example, in school-aged children – the age at which these children frequently present to medical attention for developmental and behavioural reasons. Reactive attachment disorder (RAD) begins to describe some of these children, but is limited to ‘severe’ cases, and does not apply to children over the age of 5.

In a review, Buckner and colleagues (2008) found that approximately 35–45% of maltreated children in foster care appear to have ‘clinically meaningful reactive attachment disorder symptoms’. Infants with disorganized attachment (as assessed in the ‘strange situation’ paradigm) are at increased risk for later externalizing behaviour problems and post-traumatic stress disorder (PTSD) symptoms (Zeanah et al. 2011). It is probable that children with RAD or even subthreshold attachment issues continue to experience ongoing difficulties that caused RAD or features thereof. How should we identify, characterize and describe them? Children with RAD (or subthreshold RAD) are less able to benefit from a given environment with their social-emotional difficulties, presumably leading to further deleterious effects on neurodevelopment. Dysfunction secondary to the damaged emotional stress regulatory system may emerge in subsequent social development, such as peer relationships (e.g. ‘I don’t want to play that game’ leading to aggression or isolation) or student–teacher relationships (e.g. instruction or criticism from teacher leading to acting-out or avoidance).

Post-traumatic stress disorder (PTSD)

Cumulative stress from chronic childhood maltreatment or multiple traumas puts children at high risk of PTSD symptoms, for which there is a significant symptom overlap with ADHD and ODD (Ford et al. 2000). Ford and colleagues (2000) found that maltreatment was more prevalent among children with ODD and ADHD treated in a psychiatric clinic, and that trauma exposure was associated with PTSD symptoms in children with ADHD and ODD. Does a child not listen to the teacher because of (i) a deficit of attention regulation (i.e. ADHD); (ii) a deficit of emotion and behaviour regulation (i.e. ODD); (iii) automatic overactivity and avoidance (i.e. PTSD); (iv) worries about performance (i.e. anxiety); or (v) because they have difficulty with relationship security (i.e. attachment)? Clearly, in
complex children with a mix of maltreatment histories, possible antenatal exposures, suboptimal pre- and post-natal nutrition, and genetic loading for developmental and mental health problems, sorting this out is generally impossible, and not practical in terms of treatment. Given the prevalence of this problem, when one also adds into the mix language, academic or cognitive difficulties, as is common in maltreatment, one has a real challenge that overwhelms services especially in systems that rely so heavily on distinct labels and single diagnoses to allocate support services.

From a traumatic stress perspective, van der Kolk (2005) published the proposed ‘developmental trauma disorder’, positing this diagnosis as an effect on the developing brain caused by ‘complex trauma’, described as multiple, chronic, prolonged early-life exposures include physical, emotional and educational neglect and child maltreatment. In this proposed diagnosis the complex traumatic exposure is followed by persistent cue-triggered social, emotional, behavioural and somatic dysregulation, persistent negative self-concept and outlook, and functional impairment across environments. Currently, ‘developmental trauma disorder’ does not seem to be a commonly known or accepted diagnosis, despite its consistency with the literature regarding sequellae of early chronic stress on emotional, behavioural and social functions (Stirling & Amaya-Jackson 2008).

**Implications for intervention**

Clearly, the most effective management strategy is prevention or avoidance of adverse environments. However, children come to attention at different points in their trajectories, and this is likely to continue occurring because of limitations in social programmes and the intrinsic difficulty in identifying and substantiating child neglect and psychological abuse. Fortunately, compared with an entity with pre-natal aetiology such as the FASDs, early chronic stress might be ‘turned off’, and secondarily preventative interventions may be added before the ‘critical periods’ of development have waned (Dozier 2006). Just as one might intervene later than the optimal moment in congenital hypothyroidism, iron deficiency or chronic lead toxicity, there is likely some permanent damage done but yet some window remaining to allow for reversing the damage and optimizing long-term outcomes.

A review of interventions is beyond the scope of the present article. However, interventions for the at-risk, early or ‘damage in progress’ cases may include early childhood education (Fantuzzo et al. 1996; Zoritch et al. 2000), therapeutic home visiting (Fantuzzo et al. 1996; van Doesum et al. 2008) and parent sensitivity training (Chaffin et al. 2006; Dozier 2006; van Doesum et al. 2008).

Emotional and behavioural difficulties bring to our attention many children with histories of neglect and other early chronic stressors. Clinically, we look back at the myriad of risk factors to development (i.e. antenatal, genetic, and early and current environments) and formulate the likely combination of such factors that may have led to their present conditions. A close look often reveals a recurrent pattern of emotional and behavioural difficulties that fits with early chronic stress. Although individual cause-and-effect pathways cannot be scientifically proven at present, this should not stop us, as in many areas of medicine we label and treat with less certainty than in the situations being discussed here. Despite the often-debilitating nature of their disabilities, these children often do not fall into any major diagnostic ‘boxes’ (i.e. autism, intellectual disability). Some labels, such as ODD, may actually discourage compassion and become a self-fulfilling prophecy by undermining educators’ and physicians’ attempts to help children and thus failing to address the real underlying problem.

The reality, unfortunately, is that complex formulations of neurobehavioural problems, although deserving, are not feasible for individual caregivers or systems other than those specialized in maltreatment or developmental pathology (of whom most doctors, schools and parents are not). Current diagnostic constructs, particularly categorical or phenomenological diagnoses, fail to capture what is truly a disability in everyday functions of attachment and emotional stress regulation due to the developmental brain injury of early chronic stress. Research is necessary to overcome the diagnostic shortfalls in early childhood chronic stress: we need to accurately define and diagnose this population in order to optimize timely management and to better their outcomes in life.

**Key messages**

- Early childhood chronic stress (e.g. neglect) is associated with long-lasting cognitive and behavioural difficulties with an increasingly well-recognized neurodevelopmental basis.
- An array of diagnostic labels are associated with early chronic stress.
- There is not currently a widely used or accepted diagnostic label that addresses the clinical features or mechanism of impairment in emotional stress regulation and attachment functions.
References


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