

Cortisol and the early years **by Robin Balbernie, Consultant Child Psychotherapist** **November 2006**

Cortisol is a steroid hormone designed to mobilise bodily resources in the face of immediate physical danger, and it also has a part to play in regulating the stress response once this has been triggered by the amygdala, the appraisal centre of the brain. This is the normal cascade of physiological events that have evolved to promote survival and subsequently restore equilibrium. The production of cortisol is initiated by the hypothalamus, a region of the diencephalon (part of the limbic system) which acts to regulate visceral functions. It manufactures corticotrophin-releasing hormone (CRH) in response to stress-related signals from the amygdala. The first response of the hypothalamus is to activate the sympathetic nervous system and trigger the release of adrenaline and noradrenaline from sympathetic nerves and the adrenal glands so they can mobilise the body for an instant and rapid response. CRH is secreted several minutes later, and this triggers the pituitary to release ACTH (adrenocorticotrophic hormone) into the bloodstream, causing cortisol to be produced by the cortex of the adrenal glands. This limbic-hypothalamic-pituitary-adrenocortical (L-HPA) system has been implicated in the aetiology of drug and alcohol abuse. (Gunnar and Donzella, 2002) In the brain cortisol binds to receptors in the hippocampus, and when sufficient numbers are occupied signals go to the hypothalamus telling it to stop producing CRH. There is a normal daily variation in cortisol levels from early on in life; this can be non-intrusively measured by saliva swabs. High levels of cortisol are generally observed in the morning and low levels are present in the evening for young children, and it is thought that this pattern may reflect the establishment of sleep / wake patterns.

Early experiences, pre- and post-natal, affect how many cortisol receptors are present in the infant's brain. It follows, therefore, that the ability of a child to cope with stress could be a reflection of the number, or availability, of cortisol receptors. It is normal for a baby to get stressed and produce cortisol, but what can vary is the ability to reduce the level before it causes damage. Over the first year of life children generally learn to dampen their cortisol response to experiences of stress, and this ability is linked to the quality of the caregiving they have received. Some toddlers exhibit raised cortisol levels in response to an unpleasant stimulus, such as the prick of a needle when they are given a routine inoculation. Significantly these children are those who have an insecure relationship with their caregiver. And: 'Behaviours reflecting distress, such as crying, proximity seeking, and withdrawal and parent reports of negative emotionality on temperament inventories tend to predict elevations in cortisol only when care quality is poor or the attachment relationship between parent and child is insecure.' (Gunnar and Donzella, 2002: 207) In other words 'infants in secure attachment relationships are less likely to elevate cortisol, even if they are distressed, whereas infants in insecure relationships do.' (Gunnar and Cheatham, 2003:204) The performance does not necessarily match physiological events. Even if left with an unfamiliar babysitter for the first time babies will cry but not elevate cortisol levels if the babysitter is jolly and sensitive; but it is a different story if the babysitter is distant. All infants experience stress and subsequently broadcast distress – the exceptions being such children as have been terrorised by direct abuse or domestic violence or numbed into silence by such practices as "crying it out", may respond by dissociation (i.e. 'switching off') at the end of the stress response.

Signalling distress by crying is the way evolution has programmed babies, and implies an "expectation" that they have the ability to organise the behaviour of their parents. 'In the healthy newborn, large increases in cortisol, heart-rate, and crying to stressors... herald the

development by 6 months of a well-organised, easy to-manage infant.’ (Gunnar and Barr, 1998:6) The parent has kept their side of the bargain, and acted as an external regulator for the child’s internal states. This is because ‘(u)nder conditions of sensitive and responsive caregiving, the high cortisol responsivity of the newborn diminishes and it becomes difficult to provoke increases in cortisol to many stressors by the end of the first year of life... Nevertheless, when young children are exposed to moderately less sensitive and responsive care, increases in cortisol are observed.’ (Gunnar and Donzella, 2002:215) This increase is most evident in children with negative emotional temperaments.

The L-HPA system is programmed by early relationship-based experiences, and it can adapt to repeated emotional stress so that cortisol levels cease to match observed behaviour. ‘By the end of the first year, events that elicit distress, wariness and inhibition of approach no longer produce elevations in cortisol on average. This dampening of the cortisol response results in dissociation of behavioural distress and negative emotionality from activation of the L-HPA axis, at least at the adrenal level.’ (Gunnar and Donzella, 2002:206) So manifest distress does not necessarily imply an increase in cortisol levels, and these usually get smaller over the pre-school years as social competence grows. However, in a study that looked at the correlation between cortisol levels and quality of day care, where the poorest settings in the sample actually scored about average on official childcare quality indices, it was found that ‘(f)or 3- to 5-year old children in full-day centre-based childcare, large increases from morning to afternoon are observed in centers of poorer quality.’ (Gunnar and Donzella, 2002:210) Recent research from Australia (Parry, Sims and Guilfoyle, 2006) confirms the deleterious effects of poor quality childcare, with results that show how ‘(m)edium quality care produced steady cortisol levels suggesting low level stress which could affect development.’ While ‘(u)nsatisfactory service delivery is associated with increases in cortisol levels, indicating chronic stress and the risk of negative long-term developmental outcomes.’ This will affect vulnerable children more, as ‘children with more negative emotional temperaments and poorer self-regulating competence were the ones who showed the most marked increase in cortisol over the day in less than optimal childcare homes’ (Gunnar and Chatham, 2003:205), although sometimes high-risk children often do not show the expected level of cortisol when under similar stress because of the inherent adaptive capacity of the L-HPA axis to maintain cortisol levels within the normal range in response to chronic (persistent) stress.

Prolonged and excessive cortisol levels over the normal variation in young children can have serious long term consequences. ‘Elevated glucocorticoids alter the physical structure of the hippocampus, shrinking dendrites and facilitating processes that lead to cell death.’ (Gunnar and Barr, 1998:4) Long periods of upset compromise the stress-control function of the hippocampus. The hippocampus is also the part of the brain involved with laying down episodic, or declarative, memory. ‘High levels of stress not only transiently block hippocampal functioning, but excessive and chronic exposure to stress hormones may lead to neuronal death in this region, possibly producing decreased hippocampal volume, as found in patients with chronic posttraumatic stress disorder.’ (Siegal, 1999:50) Research has demonstrated that ‘infants with higher cortisol levels produce smaller electrical charge in their brain when they are forming memories for new visual information.’ It has also been found that ‘(c)hildren who produce higher levels of cortisol during normal days at nursery school have a harder time sustaining attention than do children with lower cortisol levels...(and) these children are also poor at self-control or inhibition of unwanted behaviour.’ (Gunnar and Barr, 1998:5) This fits with the observation that a ‘flood of stress hormones can produce toxic effects on the development of brain systems responsible for self-regulation.’ (Siegal, 1999:295) Although it is unlikely to occur in any daycare facility, the extremes of cortisol levels triggered by maltreatment do sound a warning note. ‘Increased

corticosteroid levels during infancy selectively induce neuronal cell death in “affective centers” in the limbic system and produce permanent functional impairments of the directing of emotion into adaptive channels.’ (Schore, 2003:33) Cortisol is especially toxic to growing brain cells (it helps to break down protein stores, liberating energy for use by the body), and so can have a corrosive effect over the first two years of life, the period of greatest and most plastic brain growth. This will especially hit the developing limbic system which is crucial for attachment functions and emotional regulation. Schore (2003:289) concludes that the ‘(e)levated corticosteroid levels that accompany relational trauma would induce increased activity of the proapoptotic death inducer, Bax, known to accelerate programmed cell death in the developing limbic system, particularly in the early developing right brain.’ This interesting study gives us yet more tangible evidence of the importance of secure relationships in the development of the young child’s brain.

References.

Gunnar, M. R., & Barr, R. G. (1998) Stress, early brain development, and behaviour. *Inf. Young Children*. 11 (1), 1-14.

Gunnar, M. R. & Donzella, B. (2002) Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology*. 27, 199-220.

Gunnar, M. R. & Cheatham, C. L. (2003) Brain and behaviour interface: stress and the developing brain. *Infant Mental Health Journal*. 24 (3), 195-211.

Parry, T. S., Sims, M. & Guilfoyle, A. (2006) Salivary cortisol levels in children as a biomarker of quality child care. Poster Presentation. WAIMH Congress, Paris.

Siegel, D. J. (1999) *The Developing Mind: Towards a Neurobiology of Interpersonal Experience*. New York: The Guilford Press.

Schore, A. N. (2003) *Affect Dysregulation and Disorders of the Self*. New York: W. W. Norton & Company.