Psychoneuroendocrinology (2008) **33**, 530–539

Available at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/psyneuen

Cognitive development and cortisol patterns in mid-life: Findings from a British birth cohort

Chris Power[a,*], Leah Li[a], Clyde Hertzman[b]

[a]*UCL Institute of Child Health, Centre for Paediatric Epidemiology and Biostatistics, 30 Guilford Street, London WC1N 1EH, UK*
[b]*Human Early Learning Partnership, Faculty of Graduate Studies, University of British Columbia, Vancouver, BC, Canada V6T 1Z3*

Received 18 June 2007; received in revised form 18 January 2008; accepted 21 January 2008

KEYWORDS
Cortisol;
Cognition;
Education;
Childhood;
Adulthood;
Birth cohort

Summary
Life-course associations among cortisol, cognitive development and educational attainment in the general population are not well understood. Using the 1958 British birth cohort, our aim was to establish whether cortisol patterns at age 45 y are associated with childhood cognition and qualification level by adulthood. We measured salivary cortisol in 6527 individuals, 45 min after waking (t_1) and 3 h later (t_2). To identify lack of morning cortisol peak and diurnal rhythm, we defined groups with: (a) t_1 cortisol in the bottom 5% of the distribution, or (b) 'flat' t_1–t_2 cortisol. Data on cognitive tests at ages 7, 11 and 16 y and educational level were used. All childhood cognitive tests (maths, reading, verbal and non-verbal ability) were inversely associated, although not always significantly, with low t_1 and flat t_1–t_2 cortisol. For example, at age 11 for males, a standard deviation (SD) increase in maths score was associated with a 28% decreased odds for lowest t_1 cortisol, and with a 13% decreased odds of flat t_1–t_2 cortisol. Associations for lowest t_1 and flat t_1–t_2 cortisol were attenuated after adjustment for qualification level at 33 y among males, although adjustment for childhood socio-economic position had little effect. Weaker associations for lowest t_1 cortisol among females were either unchanged or strengthened after adjustment for qualification level. Our results for males, but less so for females, are compatible with a causal relationship in either direction, namely from cortisol to cognitive ability or vice versa.

1. Introduction

There are several reasons to expect that cortisol and cognition are related. The brain is a major target organ for corticosteroids (Belanoff et al., 2001). Glucocorticoids modulate neuro-transmitter systems, regulate the plasticity

Abbreviation: t_1, time 1 cortisol measure; t_2, time 2 cortisol measure; SEP, socio-economic position; SD, standard deviation; CI, confidence interval.

*Corresponding author. Tel.: +44 207 905 2106.
E-mail address: c.power@ich.ucl.ac.uk (C. Power).

0306-4530/$ - see front matter © 2008 Elsevier Ltd. All rights reserved.
doi:10.1016/j.psyneuen.2008.01.017

and circuitry of many brain regions, and influence the attention and concentration functions of the pre-frontal cortex (Davis et al., 2002; Lupien et al., 2005). In the short-term cortisol influences memory as well as the acquisition and consolidation of information. Children exhibiting increases in cortisol in response to a challenge have been shown to have higher performance on measures of executive function (a peg tapping inhibitory control task and an item selection task) than those who do not (Blair et al., 2005). Despite these insights, there is little population-based evidence linking the acquisition of cognitive skills and cortisol. However, from the limited evidence available to date, based on studies of children aged 6–14 years, the relationship appears to be bi-directional; that is, with effects of gluccorticoids on cognitive function and, conversely, effects of cognitive processing on gluccorticoid secretion (Lupien et al., 2005). Disruption of the normal diurnal rhythm of cortisol may also be important for cognition. The normal diurnal rhythm is characterised by a post-waking peak and subsequent decline over the day, but with approximately 10% of individuals lacking the post-waking peak (Stone et al., 2001). It has been argued that a lack of an early morning cortisol peak under non-experimental conditions could reduce a child's capacity to remain alert during the day (Gunnar and Vazquez, 2001). Haley et al. (2006) provide evidence that the ability to mount a cortisol response to a learning task is related to memory consolidation, at least in newborns. Brandtstadter et al. (1991) demonstrated that high salivary cortisol at 8am was associated with higher educational attainment. Several investigators have suggested that there is an inverted U-shape relationship, such that extremely low and high cortisol levels impair memory consolidation (Herbert et al., 2006). Lower levels are thought to lead to under-stimulation of hippocampal mediated learning and memory whilst higher cortisol levels lead to suppression as opposed to potentiation (Belanoff et al., 2001; Davis et al., 2002; Haley et al., 2006; Lupien et al., 2005).

Our interest here is in the relationship between cortisol and cognition across the life-course. Research on stimulation in early life (licking and suckling of baby rats during a critical period of development) shows alterations in the expression of genes that influence the development of response patterns in the HPA axis, and also of higher-order executive functions in the brain (Weaver et al., 2005). In humans, levels of environmental stimulation are known to vary by social group, such that lower social classes receive less stimulation (Hart and Risley, 1995). Low socio-economic position (SEP) is also related to higher levels of basal cortisol in children, emerging between ages 6 and 10 years (Lupien et al., 2000). However, this relationship may be unstable thereafter: between age 10 and 14 years, the direct association between basal cortisol and SEP is superseded by an indirect effect whereby SEP is associated with differences in cognitive processing style that, in turn, are associated with basal cortisol (Lupien et al., 2005). In respect of this latter proposed sequence, cortisol secretion could be an outcome rather than a precursor of cognitive function. In sum, the studies of SEP and cortisol at different stages in childhood suggest a dynamic bi-directional relationship between cortisol and cognition evolving over time. A SEP gradient in cortisol metabolism has been reported in adults, from the most to the least favourable SEP, with those in lower SEP having a slower rate of decline in cortisol over the day, resulting in higher evening levels (Cohen et al., 2006). This raises the prospect that reduced cognitive ability in childhood may lead to lower SEP in adulthood and, through that, to altered cortisol patterns. Finally, with respect to cortisol and cognition at later stages of the lifecourse, chronic exposure to high levels of cortisol is associated with memory impairments in the elderly (Seeman et al., 1997; Karlamangla et al., 2005; Lupien et al., 2005; Li et al., 2006).

Despite these insights, the life-course associations among cortisol, cognitive development and educational attainment in the general population are not well understood. This is not surprising because understanding the direction of association requires information on cortisol patterns in early life, followed by data on cognitive trajectories and educational outcome some decades later. To our knowledge, such true prospective data do not exist. Yet, cognitive trajectories have been delineated in several population-based studies (Jefferis et al., 2002; Richards and Sacker, 2003), providing an opportunity to examine the cognition, education and cortisol relationship retrospectively.

We anticipate that the social environment in early life affects cortisol patterns, which in turn, influence cognitive development in childhood, setting in train dynamic processes linking school success, and thence qualification level achieved by adulthood. We are not able to evaluate this process prospectively, rather we assess it indirectly, by investigating whether cortisol patterns in mid-adulthood are associated with cognition at different ages in childhood and with later educational achievement in the 1958 British birth cohort (Power and Elliott, 2006). Although we are unable to determine the temporal sequence, that is, whether cognitive function leads to cortisol patterning or vice versa, we can establish whether any associations between childhood cognition and adult cortisol are dependent on early social origins and adult educational achievement. Our previous work showed that less advantaged SEP in childhood is associated with poorer cognitive development and educational outcome (Jefferis et al., 2002), and also that adult cortisol patterns vary by SEP (Li et al., 2007), thus highlighting the need to take account of early social origins in studies of cognition and adult cortisol.

2. Methods

2.1. Study population

The 1958 cohort includes all children born in England, Scotland and Wales, in one week in March 1958. A population of about 17,000 live births were followed-up at ages 7, 11, 16, 23, 33, 42 y (Power and Elliott, 2006). More recently, a target sample of 11,971 participants identified as still in contact with the study, and at age 42 had not required a proxy interview (e.g. due to learning disability) were invited to a clinical examination undertaken in their home by a trained nurse; 9377 (78%) participants were seen September 2002–March 2004 the majority aged 45 y (68%), some aged 44 y (31%) and only a minority aged 46 y (<1%). Ethical