Biological Psychology xxx (xxxx) xxx–xxx

Contents lists available at ScienceDirect

Biological Psychology

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

Children's cortisol and externalizing stress symptoms are predictors of adiponectin evolution over two years

Nathalie Michels*, Isabelle Sioen, Kirsten Schraven, Annelies De Decker, Carola van Aart, Stefaan De Henauw

Department of Public Health, Faculty of Medicine and Health Sciences, Ghent University, Ghent, Belgium

ARTICLE INFO

Keywords:
Adiponectin
Cortisol
Emotions
Conduct problems
Children
Longitudinal

ABSTRACT

Background: Adiponectin is an anti-inflammatory, insulin-sensitizing and energy-regulating adipocytokine. Consequently, the link between psychosocial stress and inflammatory diseases like the metabolic syndrome might be partially explained by lower adiponectin levels in stress. Nevertheless, the stress-adiponectin association has seldom been tested and no clarity exists about the directionality.

Methods: In the Belgian ChiBS study, serum adiponectin and stress levels were measured in 348 children (5–10y) at baseline and in 168 of them after 2-year follow-up. Psychosocial stress was reported with the Strengths and Difficulties Questionnaire (parental report on emotional, peer, and conduct problems), negative emotions (anger, sadness, anxiety) and negative events. In addition, salivary cortisol diurnal patterns were available from 2 days with each 4 samples. Longitudinal linear regression analyses were performed including step-wise adjustment for age, sex, socio-economic status, body fat%, physical activity and snack frequency.

Results: Despite some positive cross-sectional associations, high daily cortisol output (beta = −0.285), anger (beta = −0.233) and conduct problems (beta = −0.182) were associated with less adiponectin increase over time, in most cases independent of the tested confounders. The other directionality was not significant: no longitudinal prediction of stress by adiponectin.

Conclusion: In healthy children, daily cortisol output and externalizing stress symptoms were negative predictors of adiponectin evolution. These findings highlight the health-compromising effects of psychosocial stress.

1. Introduction

Apart from genetic and lifestyle predictors (Hong, 2010), there is more and more evidence that psychosocial stress factors might lead to metabolic dysregulations (Rosmond, 2005), even in children (Pervanidou & Chrousos, 2011). First of all, chronic stress has been associated with comfort food preference (Adam & Epel, 2007) and emotional eating, resulting in unhealthier food intake which can dysregulate energy intake, lipid and glucose homeostasis (Adam & Epel, 2007). This stress-appetite nexus is cortisol driven: cortisol can directly influence the reward pathways (e.g. in the mesolimbic system) through increased levels of opioids (role in hedonic evaluation of food) and dopamine (role in motivational aspects of eating), while cortisol also acts indirectly through its influence on other hormones (e.g. insulin, leptin and NPY) that regulate appetite and reward (Adam & Epel, 2007; Epel, Tomiyama, & Dallman, 2012). Second, the stress-hormone cortisol stimulates the lipoprotein lipase enzyme, induces adipogenesis (and thus adiposity) and decreases insulin production (Anagnostis, Athyros, Tziomalos, Karagiannis, & Mikhailidis, 2009). Third, the activation of the sympathetic nervous system during stress increases blood pressure and insulin resistance (Lambert & Lambert, 2011). Finally, sustained stress exposure also increase inflammation (Tian, Hou, & Yuan, 2014). In contrast to the fully matured adult brain in which stress may result in sensitization, chronically stressed children with developing brains may have permanent alterations of biological stress systems (Teicher, Andersen, Polcari, Anderson, & Navalta, 2002). Therefore, insight in underlying factors of stress and its link to health is pivotal.

Adiponectin is an anti-inflammatory, insulin-sensitizing and energy-regulating adipocytokine (Nigro et al., 2014) and thus low adiponectin might be a risk factor for the metabolic syndrome (Brooks et al., 2007). Next to these biological effects, it also potentially plays a role in the stress-appetite nexus. After all, one study showed higher adiponectin values to be associated with less disinhibited eating (Chearskul et al., 2012) and less craving (Hillemacher et al., 2009). The physiological mechanisms for adiponectin's relation with craving and addiction are largely unknown. First and most importantly, an interaction with the

* Corresponding author at: Ghent University, De Pintelaan 185 – 4K3, 9000 Gent, Belgium.
E-mail address: Nathalie.michels@ugent.be (N. Michels).

http://dx.doi.org/10.1016/j.biopsycho.2017.05.016
Received 14 June 2016; Received in revised form 6 April 2017; Accepted 28 May 2017
0301-0511/ © 2017 Elsevier B.V. All rights reserved.

dopaminergic mesolimbic system is suggested, a shared system for addiction and food craving since it is responsible for reward regulation (Brownell & Gold, 2012; Hillemacher et al., 2009). Second, adiponectin might contribute to better satiety signaling due to its insulin sensitivity-enhancing effects since insulin has an anorexigenic effect and since enhanced insulin sensitivity blocks the cortisol-induced food intake (Adam & Epel, 2007). Third, adiponectin's anti-inflammatory effect might intervene in the inflammation-induced stimulation of appetite in the hypothalamic centers (Cai & Liu, 2011). Taken together, lower adiponectin levels in stress might explain the link between stress and several disease outcomes like addictive eating behaviors and the metabolic syndrome.

Nevertheless, the stress-adiponectin association has seldom been tested in healthy humans and no clarity exists about the time-direction (cause-effect) and sign-direction (positive versus negative association). So far, only two longitudinal studies were found i.e. with negative mood in postmenopausal women (Adam, Schamarek, Springer, Havel, & Epel, 2010) and with motor-accident induced stress in children (Pervanidou, Margeli, Lazaropoulou, Papassotiriou, & Chrousos, 2008) being predictors of lower adiponectin. Most animal and culture studies show cortisol as an inhibitor of adiponectin (Degawa-Yamauchi et al., 2005; de Oliveira et al., 2011; Shi et al., 2010). Apart from a general negative association, non-significant associations with depression/anxiety (Domingues, Duarte, Rocha, & Teixeira, 2015), urinary cortisol (Adam et al., 2010), serum cortisol (Weber-Hamann et al., 2007), positive mood and perceived stress (Adam et al., 2010) have been described and even positive associations with depression (Jeong et al., 2012), anxiety (Wilhelm, Choi, Huckans, Manthe, & Loftis, 2013), serum cortisol (Fernandez-Real, Lopez-Bermejo, Casamitjana, & Ricart, 2003), in cell-culture cortisol (Lee & Fried, 2014) and in a chronic stress rat model (Sato et al., 2011). In mice, some evidence in the opposite time-directionality exists: adiponectin administration reduced basal cortisol levels (Li et al., 2009) and had anti-depressant effects (Liu et al., 2012). Discrepancies might be due to different conceptualization of stress (stressors versus emotions; positive versus negative valence; clinical versus non-clinical.), low quality of biomarker collection (e.g. just a single serum or single salivary cortisol sample without taking into account the diurnal rhythm) and the lack of adjustment for confounders like adiposity and diet. Overall, the literature lacks research in healthy children, with a longitudinal perspective, using valid stress biomarkers, and testing the relevance of stress-induced dietary changes herein.

Consequently, the current paper tests the longitudinal association between stress (using both stress reports and cortisol patterns) and adiponectin over two years in a population-sample of children. The rationale for the focus on children is that children can be more vulnerable for biological stress effects, that there is still less complexity with chronic illness/medication in childhood and in the perspective of early prevention. A first aim was to check the time-direction in the hypothesis that stress predicts lower adiponectin rather than adiponectin predicting stress. A second aim was to check whether this association was independent from socio-demographic data, body composition, physical activity and diet. Finally, an explorative analysis with disinhibited eating was performed to check the role of adiponectin in the stress-appetite nexus (as a possible link to health).

2. Methods

2.1. Design

Participants of this study were Belgian children recruited for the longitudinal ChiBS study (year 2010 and 2012). Children were between 4.5 and 10.8 years old at baseline and between 6.7 and 12.2 years old at follow-up. More details on the ChiBS study and its measurements have been described elsewhere (Michels, Vanaelst et al., 2012). Corticosteroid users and participants with diabetes were excluded. In 2010, 348 children (48% boys) had available data on adiponectin and stress. Of them, 168 (46% boys) had information on those variables in 2012. Those with full data in 2010 were not significantly different in socio-economic status, stress and body fat%, but were older than those without adiponectin values since younger children more often refused blood-withdrawal. Children with follow-up data in 2012 were not significantly different in sex, age, socio-economic status, stress, body fat% or adiponectin values at baseline than those only participating in 2010. On the examination day in 2010 and 2012, fasting blood withdrawal and anthropometry was executed and questionnaires on stress and diet were completed. In the same week as the examination day in 2010, saliva was collected at home for cortisol analysis. The study was conducted according to the guidelines laid down in the Declaration of Helsinki and the project protocol was approved by the Ethics Committee of the Ghent University Hospital. A written informed consent was obtained from the parents and a verbal assent from the children.

2.2. Adiponectin (2010 and 2012)

A venous blood sample in fasting state was taken. All blood samples were centrifuged at 2500*g* for ten minutes and were stored at −80 °C until further analyses of the extracted serum. Serum adiponectin was measured using a Meso Scale Discovery sandwich electrochemiluminescence immunoassay (Rockville, USA). The inter-assay CV was 7.6%, whereas the intra-assay CV was 6.9%.

2.3. Salivary cortisol (2010 only)

Saliva was collected at home via Salivette synthetic swabs specifically designed for cortisol analysis (Sarstedt, Germany). The participants were asked to collect saliva during two consecutive weekdays at four time points: immediately after wake up, 30 min after wake up, 60 min after wake up and in the evening between 7 and 8 PM. Standardized sampling instructions with strict time instructions have been published (Michels, Sioen et al., 2012). As we found that cortisol concentrations differed between parental reported 'time compliers' and 'time non-compliers' (Michels, Sioen et al., 2012), morning samples collected more than 5 min different from the requested time point and evening samples not collected between 7 and 9 PM were excluded (271 of 3290 samples). Furthermore, samples of corticosteroid-users were excluded (N = 5). Salivary cortisol was assayed in the routine laboratory of the Ghent University Hospital on a Modular E 170 immunoanalyser system (Roche Diagnostics, Mannheim, Germany; measuring range 0.49-1748.95 nmol/L; inter-assay CV = 3.9%; intra-assay CV = 1.9%) using the Roche Cobas Cortisol assay, a competitive electrochemiluminescence immunoassay. More details and reference ranges on the cortisol values in this population were published elsewhere (Michels, Sioen et al., 2012). Summary variables have been calculated to represent chronic stress by two independent cortisol patterns over time: the cortisol awakening response and the diurnal pattern (Fekedulegn et al., 2007). To represent the cortisol awakening response, the absolute increase in cortisol (AINC) was calculated as the concentration difference between peak (maximum of 30 and 60 min after wake up) and the first sample. The diurnal pattern was investigated using the total cortisol output (area under the curve over the whole day).

2.4. Stress reports (2010 and 2012)

We have used a broad definition of stress by measuring different aspects of the stress process: negative events, emotions and behavioral problems. After all, stress is an adaptive, dynamic state that is composed of several aspects. The initiating stimulus is the 'stressor' and if the person is unable to handle a persistent situation, the sustained, chronic arousal can trigger emotional and behavioral disturbances that might put a person at risk for psychiatric or physical disease (Ursin & Eriksen, 2004).

Download English Version:

https://daneshyari.com/en/article/7278232

Download Persian Version:

https://daneshyari.com/article/7278232

Daneshyari.com