



Exploring the link between gender, sensation seeking, and family history of alcoholism in cortisol stress-response dampening[☆]

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ABSTRACT

Many studies have demonstrated an inverse association between cortisol and risk-taking behaviors, with high-sensation seekers (HSS) showing lower cortisol levels. We investigated the potential link between sensation seeking (SS) and stress-induced stress responses, as well as alcohol-induced stress-response-dampening (SRD) effects in cortisol. First, we hypothesized that HSS would show inverse SRD effects in cortisol. Second, we hypothesized that females would display similar SRD effects to males. Third, we hypothesized an independent relationship between SS and family history (FH) with regard to alcohol-induced SRD effects in cortisol. 86 healthy men and women participated in two laboratory sessions, receiving alcohol in one of the two. Experimental stress paradigms were administered and serum cortisol was measured. SRD effects in cortisol developed for both genders in low-sensation seekers (LSS), but not in HSS. This study contributes to current literature by (1) supporting the association between SS and cortisol, (2) demonstrating that SRD effects in cortisol of females is inversely related to SS, and (3) demonstrating an independent relationship between SS and FH with regard to alcohol-induced SRD effects in cortisol.

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1. Introduction

This study investigates the potential link between the high-risk personality trait of sensation seeking (SS) and stress-induced stress response as well as alcohol-induced stress-response-dampening (SRD) effects in the neuroendocrine domain. The personality trait SS, defined as pursuing and taking risks in order to experience a variety of new sensations (Zuckerman, 1979; McCourt et al., 1993), provides a link between risky behavior and biological processes. Risky behavior can be considered a reflection of sensation-seeking tendencies (Newcomb and McGee, 1991; Oetting et al., 1998; Deery and Fildes, 1999), which, in turn, have been associated with testosterone levels in men (e.g., by Daitzman et al., 1978; Gerra et al., 1999; Rosenblitt et al., 2001; Kerschbaum et al., 2006) and

cortisol levels (Netter et al., 1996; Rosenblitt et al., 2001; Harl et al., 2006).

Numerous studies carried out on healthy volunteers and substance abusers have investigated the relationship between SS and the use of drugs and alcohol (e.g., by Brook et al., 1995; Sher et al., 2000; Martin et al., 2004; Dom et al., 2006; Magid et al., 2007; Viken et al., 2007). For otherwise healthy persons, the consistent result is that high-sensation seekers (HSS) showing higher and more manifold drug and alcohol use compared to low-sensation seekers (LSS). Persons who tend to enjoy new and arousing experiences are also more inclined to experiment with alcohol and drugs (Zuckerman, 1983). HSS start consuming alcohol at an earlier age (Zuckerman, 1994) and consume significantly more substances simultaneously (Calafat et al., 2007). Taking these lines of research together, Conrod et al. (2006) and Staiger et al. (2007) have suggested specific interventions to target personality-based behavioral risk strategies.

A widely used scale to measure the personality trait SS is Zuckerman's revised Sensation Seeking Scale SSS-R (Zuckerman, 1994). It consists of four subscales (see Section 2). A meta-analysis specifically found an association between the subscale disinhibition (DIS) and alcohol use (Hittner and Swickert, 2006). Furthermore, related constructs, such as behavioral disinhibition,

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were significantly related to drinking habits (Earleywine et al., 1990). Behavioral disinhibition is said to be related to the SS construct, especially the subscale DIS (Earleywine and Finn, 1991).

Additionally, the prediction of addictive behavior is playing an increasingly larger role in scientific considerations. SS measures have proved to predict later substance consumption behavior very reliably (e.g., by Jaffe and Archer, 1987; Andrew and Cronin, 1997; Brook et al., 1995; Sher et al., 2000; Crawford et al., 2003; Donohew et al., 1999; Ames et al., 2005). These findings suggest that there are strong relationships in the sense of biological mechanisms underlying the association between personality traits of high risk and drinking habits; however, the potential link between personality traits of high risk, biological processes, and addictive behavior is still unclear.

Important findings in the neuroendocrine domain were reported in the 1980s by Schuckit and colleagues, who documented reduced variance in the hormone cortisol following ethanol administration in male family history positive (FH+) participants regarding alcohol (Schuckit et al., 1983, 1987a,b; Schuckit, 1984).

Thereupon, the relationship between cortisol and sensation-seeking behavior also began to receive some attention. This steroid, produced by the adrenal cortices, is released in times of chronic physical or psychological stress and shows a clear circadian rhythm (Kirschbaum and Hellhammer, 1994; Berne and Levy, 1998). Mazur (1995) predicted a negative correlation between cortisol and risk-taking behaviors on the basis of findings from the large sample of veterans he studied. He reasoned that “nervous” individuals with high cortisol would be the least likely to engage in sensation-seeking behaviors, whereas those accustomed to deviant and “norm-breaking” behaviors would exhibit low cortisol levels because their risk-taking behaviors would no longer produce stress. More recent studies have supported this inverse association between cortisol and risk-taking behaviors (Netter et al., 1996; Wang et al., 1997).

Psychological stress experiments were introduced to investigate the autonomous nervous system (ANS) with regard to alcohol-induced SRD effects in FH+ participants, and a certain widely cited alcohol-induced SRD pattern showed up (Finn and Pihl, 1987): When exposed to a psychologically aversive countdown situation in a laboratory experiment, FH+ participants displayed reductions in heart rate, frontal muscle tone, and electrodermal measures after ethanol administration compared to experimental sessions without alcohol. Furthermore, it is remarkable that without alcohol administration their stress responses were significantly higher than those of FH– (family history negative) participants, who generally did not show these psychophysiological changes. This alcohol-induced SRD pattern was replicated in the neuroendocrine domain for cortisol in male FH+ participants (Croissant and Olbrich, 2004). However, the relationship between personality traits of high risk, FH, and gender effects on the one hand and SRD in the neuroendocrine domain on the other hand still has to be investigated.

We build upon the studies mentioned above that have examined the link between cortisol and SS measures. This article deals with the psychometric findings regarding SS. It is not clear from the above-mentioned studies in which way SS and psychological stress might work together in eliciting stress responses and alcohol-induced SRD effects in the neuroendocrine domain, especially with regard to gender. Although it appears that alcoholism risk is associated with greater SRD effects, at least in the ANS domain of FH+ males, it seems highly probable that the contrary is true for HSS in the neuroendocrine domain, especially with regard to the above-cited neurohormone literature (Mazur, 1995; Netter et al., 1996; Wang et al., 1997). This study addresses some of the issues that remain unclear.

A thorough literature review indicated that there have been no studies examining the role of psychological stress applied in experimental paradigms in association with SS personality traits. Reports on SRD effects in females are also rare. The main focus has been on SRD effects and their relationship to a positive FH of alcoholism and their effects in the ANS domain (Sinha et al., 1998; Croissant et al., 2006) or in the neuroendocrine domain (Croissant and Olbrich, 2004); SS measures, however, were scarcely considered, even though both genders produce similar levels of this hormone. Including both men and women in a study sample consisting of participants with SS and with a positive FH regarding alcoholism is a logical step for identifying important linkages between biological variables and sensation-seeking behavior, and may contribute to the understanding and development of new prevention strategies and therapeutic approaches. In the present study we tested three specific hypotheses.

First, we hypothesized that HSS would show inverse SRD effects in the neuroendocrine domain. Second, we hypothesized that females would display similar SRD effects to males. Third, we hypothesized that an independent relationship exists between SS and FH with regard to alcohol-induced SRD effects in the neuroendocrine domain.

2. Method

2.1. Participants

This study used 86 healthy male and female participants: 64 of them FH+ regarding alcoholism: 36 were sons and 28 were daughters of alcohol-dependent fathers. 22 participants were FH–: 10 were sons of nonalcoholics and 12 were daughters of nonalcoholics. This was established by a short, standardized interview (Mini-DIPS Examination, Margraf, 1994). We registered 144 persons. 27 of them did not fulfill the selection criteria, the list of which follows. We could not obtain complete serum data sets from 31 of the remaining 117 participants for technical reasons, thus resulting in 86 participants (characteristics are given in Table 1). With regard to the significant gender differences concerning liver enzymes, we would like to state that the liver enzymes for all participants were within normal range.

We recruited FH+ participants after their fathers had been identified as alcoholics according to ICD-10 criteria (World Health Organization, 2000) by using several psychiatric inpatient facilities as a recruitment source and after receiving the fathers' permission to contact their offspring. We recruited FH– participants by advertising in local companies and local newspapers.

Only healthy persons (verified by a standardized clinical interview and the Mini-DIPS), age from 18 to 65 years, were eligible. Participants needed to be a German native speaker to be able to fill out the given questionnaires and were required to have a fixed abode. We excluded persons who had participated in other clinical trials within the previous 4 weeks. Further, we excluded persons taking any form of medication.

We assessed the FH of potential participants with regard to psychiatric diseases including substance dependence on the basis of given information and rejected all persons with first-degree (siblings and parents) and second-degree (grandparents, aunts, and uncles) psychiatric diseases to avoid family history clashing with biological factors other than alcohol addiction. With respect to an FH+ regarding alcohol, only persons whose alcoholic fathers had no other psychiatric ICD-10 diagnoses were considered for this study.

The study was approved by the Medical Ethical Committee of the University of Heidelberg and was carried out in accordance with the Declaration of Helsinki. All participants gave written informed consent.

2.2. Procedure

Each participant underwent two laboratory sessions on 2 days, including an alcohol challenge on 1 day, which targeted a blood alcohol level (BAL) of 0.7 mg/ml. The alcohol session was conducted in counterbalanced fashion. In addition, the participants underwent a laboratory stress experiment to assess the stress-response-dampening (SRD) hypothesis. The laboratory sessions started at 2 p.m. and lasted approximately 160 min. Participants were seated in a comfortable chair and filled out several psychometric questionnaires without haste. We applied electrodes, and inserted a venous catheter. Participants drank the beverage before the actual laboratory stress experiment started.

In alcohol sessions, female participants received 0.56 g/kg and males 0.66 g/kg of 100% ethanol. These dosages were derived from the Widmark formula (Gullberg and Jones, 1994) and targeted a BAL of 0.7 mg/ml. Alcoholic drinks were administered as a 25%-by-volume solution of ethanol mixed with a bitter lemon beverage and were consumed over a 15-min period. On laboratory days without

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