



Moderate alcohol consumption after a mental stressor attenuates the endocrine stress response



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ABSTRACT

Alcohol is often consumed to reduce tension and improve mood when exposed to stressful situations. Previous studies showed that moderate alcohol consumption may reduce stress when alcohol is consumed prior to a stressor, but data on the effect of alcohol consumption after a mental stressor is limited. Therefore, our objective was to study whether moderate alcohol consumption immediately after a mental stressor attenuates the stress response. Twenty-four healthy men (age 21–40 y, BMI 18–27 kg/m²) participated in a placebo-controlled trial. They randomly consumed 2 cans (660 mL, ~26 g alcohol) of beer or alcohol-free beer immediately after a mental stressor (Stroop task and Trier Social Stress Test). Physiological and immunological stress response was measured by monitoring heart rate and repeated measures of the hypothalamic-pituitary-adrenal axis (HPA-axis), white blood cells and a set of cytokines. After a mental stressor, cortisol and adrenocorticotrophic hormone (ACTH) concentrations were 100% and 176% more reduced at 60 min ($P = 0.012$ and $P = 0.001$, respectively) and 92% and 60% more reduced at 90 min ($P < 0.001$ and $P = 0.056$, respectively) after beer consumption as compared to alcohol-free beer consumption. Heart rate and dehydroepiandrosterone (DHEA) were not influenced by alcohol consumption. Plasma IL-8 concentrations remained lower during the stress recovery period after beer consumption than after alcohol-free beer consumption ($P < 0.001$). In conclusion, consumption of a moderate dose of alcohol after a mental stressor may facilitate recovery of the endocrine stress response as reflected by decreasing plasma ACTH and cortisol.

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1. Introduction

Humans have to respond adequately to physiological and behavioral challenges occurring in a dynamically changing environment in order to survive. Physiological reactions to stress generally comprise changes in neuroendocrine, hormonal and immune functions (Glaser & Kiecolt-Glaser, 2005).

Alcohol consumption has been part of most cultures for thousands of years and is commonly consumed for its mood enhancing and stress reducing effects. Several studies have shown that alcohol consumption shortly before a mental stressor blunts the stress

response (Balodis, Wynne-Edwards, & Olmstead, 2011; Dai, Thavundayil, & Gianoulakis, 2002; Sher, Bartholow, Peuser, Erickson, & Wood, 2007). Such a stress-response dampening effect of alcohol has been investigated and extensively described by Levenson, Sher, Grossman, Newman, and Newlin (1980). They showed in male students that alcohol consumption prior to a stressor (either speech or electric shock) reduced anxiety and heart rate during the stress response as compared to the placebo condition. Similarly, a reduced hypothalamic-pituitary-adrenal axis (HPA axis) activity, evaluated as adrenocorticotrophic hormone (ACTH) and cortisol has been reported (Balodis et al., 2011; Dai et al., 2002). However, an increased activity of the HPA axis by alcohol consumption has also been reported (Schuckit, Gold, & Risch, 1987). This difference in hormonal responses may be related to the dosage used in the studies. Although there is some evidence for a stress-response dampening effect of alcohol when consumed before a stressor, data on the

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effect of alcohol during stress response recovery is scarce. This is surprising, because alcohol is frequently used to reduce the stress after a working day or stressful event. Childs, O'Connor, and de Wit (2011) compared the influence of an intravenous alcohol infusion immediately or 30 min after a mental stressor on the stress response. They showed that infusion of alcohol immediately after the stressor blocked the cortisol response. Another study by de Wit, Söderpalm, Nikolayev, and Young (2003) showed no effect of moderate alcohol consumption on cortisol levels immediately after mental stress. However, they measured cortisol only once after the stressor, possibly leaving an effect of alcohol consumption on cortisol recovery undetected. Moderate alcohol consumption may also shortly induce arousal and increase heart rate (Schrieks et al., 2014). This might interfere with the stress-response dampening effects of alcohol when consumed after the stressor.

The neuroendocrine and immune systems are highly interrelated (Cohen et al., 2000). The acute immune response after a mental stressor has been well described. A meta-analysis has shown robust effects, such as increased levels of circulating IL-6 and IL-1 β , and marginal effects on C-reactive protein (Steptoe, Hamer, & Chida, 2007). Additionally, Kimura, Ohira, Isowa, Matsunaga, and Murashima (2007) have documented changes in the number of circulating T and B cells and natural killer cells following acute mental stress. Moderate alcohol consumption has been suggested to have immuno-modulatory and anti-inflammatory effects (Romeo et al., 2007). For example, Mandrekar, Catalano, White, and Szabo (2006) observed an attenuated monocyte inflammatory response and an augmented response of anti-inflammatory cytokine IL-10 after alcohol consumption. However, the influence of moderate alcohol consumption on the immune response to a mental stressor has not been investigated.

Therefore, our primary aim was to investigate whether moderate alcohol consumption immediately after a mental stressor attenuates the physiological stress response. Our secondary aim was to investigate whether the stress-induced immune response was also affected by moderate alcohol consumption. We hypothesized that both the physiological stress response and the stress-induced immune response would be lower after moderate alcohol consumption as compared to after no alcohol consumption.

2. Materials and methods

2.1. Participants

Twenty-four healthy men (age 21–40 years, BMI 18–27 kg/m²) participated in the study. The participants were recruited from a pool of volunteers at TNO (The Netherlands Organization for Applied Scientific Research) in Zeist. Eligible participants did not use any medication, habitually consumed alcohol (5–27 standard units/week), were non-smokers and had no (family) history of alcoholism. Only men were included in the study to exclude the influence of hormonal changes associated with the menstrual cycle. The study was performed according to the International Conference on Harmonisation Guidelines for Good Clinical Practice. The study also complied with the Declaration of Helsinki and was approved by an independent Medical Ethics Committee (METOPP, Tilburg, The Netherlands). Written informed consent was obtained from all participants.

2.2. Experimental protocol

The study was originally set up as a randomized, open-label, crossover design. Participants consumed either 2 cans of beer

(660 mL, ~26 g alcohol) or alcohol-free beer (<0.5 g alcohol) on two different occasions (one week apart) in a randomized order shortly after they performed a mental stress test. Because of a strongly attenuated cortisol and ACTH stress response after the second mental stress test indicating a strong learning effect, we decided to use data from the first mental stress test only (effect of occasion $P = 0.002$ for cortisol and $P = 0.023$ for ACTH; 6% increase in cortisol concentrations and 2% decrease in ACTH concentrations immediately after the stress response during the second occasion).

Participants were instructed to refrain from eating or drinking anything except water 2.5 h before testing (participants arrived at 11:30 AM). The mental stress test was performed after initial blood samples were taken and heart rate had been recorded. Immediately after the stress test, 2 cans of beer (either beer or alcohol-free beer) were consumed within 30 min. Participants were instructed to drink the first can during the first 15 min, and the second can during the remaining 15 min. During the following 3 h subjects were kept in a fasted state. During this period blood samples were obtained at regular intervals and heart rate was continuously monitored (Table 1).

2.3. Mental stress test

The mental stress test comprised of two stress protocols: the Stroop task and the Trier Social Stress Test (TSST). These tests were performed in approximately 30 min. Both the Stroop task and the TSST have been shown to induce a physiological stress-response (Kirschbaum, Ebrecht, & Hellhammer, 2001).

The Stroop task is a computerized color-word interference task, involving the successive presentation of target color words printed in an incongruous color. Participants had to press the computer key that corresponded to the name of the color in which the word was printed.

The TSST is a standardized laboratory stress test that was performed following the procedure of Kirschbaum, Pirke, and Hellhammer (1993). The TSST consists of two tasks: a mental arithmetic task (10 min) and a public speaking exercise (10 min). In the mental arithmetic task participants had to serially subtract the number 13 from 1022 as fast and accurate as possible. In case of miscalculation they had to start over. For the public speaking exercise, participants got 5 min to prepare a talk about their personal characteristics, e.g. their strengths and weaknesses. Afterwards, they went to a room where they had 5 min to present their talk in front of two actors who criticized their talk non-verbally.

2.4. Physiological measures

Blood samples were collected at 7 time points for the measurement of ACTH, cortisol and dehydroepiandrosterone (DHEA) and at 8 time points for the measurement of cytokines and total and differential white blood cell count (i.e. leucocytes, lymphocytes, neutrophils, monocytes, eosinophils and basophils) (Table 1). Blood was obtained from the antecubital vein of the forearm and collected in pre-chilled tubes containing EDTA for plasma (Vacutainer Systems, Becton Dickinson, Plymouth, UK). After the tubes had been centrifuged, the plasma samples were stored at -80°C until assayed.

Plasma ACTH concentrations were measured by an enzyme-linked immunosorbent assay (ELISA) (AlpcoDiagnostics, Salem, NH). The intra-assay coefficient of variation was 6.7%. Plasma cortisol concentrations were determined using Olympus analytical equipment and reagents. Plasma DHEA concentrations were measured with an ELISA (Enzo Life Sciences, Lausen, Switzerland) with an intra-assay coefficient of variation of 4.8%.

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