An investigation of the role of testosterone, cortisol and serotonin in aggression

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Primary Objective: To investigate the role of testosterone, cortisol and serotonin in aggression (experimentally induced aggression and self-reported aggression over the last six month).Secondary Objective(s): the role of approach and avoidance...

Ethical review	Approved WMO
Status	Pending
Health condition type	Other condition
Study type	Observational non invasive

Summary

ID

NL-OMON37187

Source
ToetsingOnline

Brief title
DNA, hormones and aggression

Condition

- Other condition

Synonym
Aggression

Health condition

agressie

Research involving
Human

Sponsors and support

Primary sponsor: Radboud Universiteit Nijmegen
Source(s) of monetary or material Support: Ministerie van OC&W

Intervention

Keyword: 5HT, Aggression, Cortisol, Testosteron

Outcome measures

Primary outcome

Hormonal levels of testosterone and cortisol, 5HT polimorphisms and aggression

on the Taylor aggression paradigm.

Secondary outcome

questionnaires (mood, psychopathy, social fear, etc) and emotion recognition

ability, approach avoidance tendencies.

Study description

Background summary

The hormones testosterone, cortisol and the neurotransmitter serotonin (5-HT) have been suggested to influence aggressive behavior. For example, the triple Imbalance hypothesis suggests that individuals that show reactive aggression in social situations have altered levels of testosterone and cortisol. High levels of endogenous testosterone and low levels of endogenous cortisol together with low levels of cortical 5-HT predispose individuals to reactive aggression (van Honk, et al., 2010). Evidence for this hypothesis is mainly based on animal research. The research that has been conducted in humans shows that testosterone is related to antisocial personality disorders and socially deviant behavior (Stalenheim, Eriksson, von Knorring, & Wide, 1998), and correlates positively with aggressive acts in male offenders with a personality disorder (Dolan, Anderson, & Deakin, 2001). High testosterone and also low cortisol are related to aggressive and approach-related behavior in response to threat (angry faces; (van Honk, et al., 2010)). Furthermore, psychopathy scores are associated with a high testosterone (baseline) to cortisol stress response ratio, but not to these values independently or baseline cortisol (Glenn, Raine, Schug, Gao, & Granger, 2011). Finally, aggressive behavior in males is

hypothesized to be related to high testosterone combined with low levels of 5-HT (Birger et al., 2003). Serotonergic signaling is affected by a serotonin transporter polymorphism (5-HTTLPR), of which human short allele carriers (s-carriers; homozygous and heterozygous) show reduced serotonin transporter availability and serotonin reuptake (Lesch et al., 1996). This genetic variation leads to an increased risk for development of social psychopathologies (Caspi, Hariri, Holmes, Uher, & Moffitt, 2010), such as aggression-related disorders (Garcia, Aluja, Fibla, Cuevas, & Garcia, 2010). These studies give some insight in the proposed mechanisms, but a crucial test of its interactions is lacking. The aim of this study is to test whether endogenous testosterone and cortisol, and 5-HT interact leading to increased aggression.

Aggression can be subdivided in instrumental and reactive aggression. Reactive aggression can be described as premeditated or planned violence, and is typically not related to impulsive responding to a threat or provocation. Reactive aggression on the other hand is characterized by such an impulsive, but not premeditated response (van Honk, et al., 2010). To reliably assess these facets, several questionnaires related to instrumental and reactive aggression will be used, next to a set of cognitive experiments. The Taylor aggression paradigm measures reactive aggression (REF, and can I say this?). There is no experimental measure of instrumental aggression, but the approach-avoidance (AA) task has previously been found to be modulated by instrumental aggression (Von Borries et al., 2012). During the AA task participants push and pull pictures of angry (and happy) faces using a joystick, leading to a relative size decrease or increase. This way the automatic tendency of a participant to avoid or approach that stimulus can be measured (Von Borries, et al., 2012). If a violent prone participant scores high on instrumental aggression, he shows an increased relative approach tendency of angry faces. To control for possible differences in recognition of facial emotions, an emotion recognition task will also be completed by the participants.

1. We hypothesize that 5-HTTLPR s-carriers with a high testosterone to cortisol ratio show increased reactive aggression during the Taylor aggression paradigm. Homozygous long-allele carriers should not show this effect, and it should also not be present during the instrumental aggression measures. This shows that especially the group that is more vulnerable for the development of psychopathologies (s-carriers) combined with a predisposing hormonal state show aggression when provoked.
2. As a second, more exploratory step, we will test the influence of other polymorphisms related to aggression on this hormonal balance, namely SPTLC3 gene; genes encoding estrogen and androgen receptors, MAOA-VNTR, dopamine transporter and receptor polymorphisms (eg DAT1) (Pavlov, Chistiakov, & Chekhonin, 2012). We will also explore the effects of testosterone and cortisol reactivity (reflecting the difference in hormonal levels before and after the aggression tasks).

Study objective

Primary Objective: To investigate the role of testosterone, cortisol and serotonin in aggression (experimentally induced aggression and self-reported aggression over the last six month).
Secondary Objective(s): the role of approach and avoidance behaviour and emotion recognition abilities in aggression. Furthermore, questionnaire measures of social fear, psychopathy, life time events, and mood will be included to investigate the role of these factors in aggression.

Study design

STUDY DESIGN
We will include 200 female students in this study. The study has a cross-sectional design.
Participation includes filling in questionnaires at home via internet and a test session at the BSI laboratory of the Radboud University (duration of test session circa 1,5 hours).

Study burden and risks

none as it is not an invasive study.

Contacts

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Trial sites

Listed location countries

Netherlands

Eligibility criteria

Age
Adults (18-64 years)
Elderly (65 years and older)

Inclusion criteria

Healthy, right-handed female students, aged 18-35.

Exclusion criteria

- male
All subjects will be pre-screened for drug abuse, head trauma, neurological or psychiatric illness, tricyclic antidepressants, neuroleptics, family history of psychiatric illness.

Study design

Design

Study type: Observational non invasive

Masking:	Open (masking not used)
Control:	Uncontrolled
Primary purpose:	Diagnostic

Recruitment

NL

Recruitment status:	Pending
Start date (anticipated):	01-12-2012
Enrollment:	200
Type:	Anticipated

Ethics review

Approved WMO Date:	05-03-2013
Application type:	First submission
Review commission:	CMO regio Arnhem-Nijmegen (Nijmegen)
Approved WMO Date:	17-01-2014
Application type:	Amendment
Review commission:	CMO regio Arnhem-Nijmegen (Nijmegen)

Study registrations

Followed up by the following (possibly more current) registration

No registrations found.

Other (possibly less up-to-date) registrations in this register

No registrations found.

In other registers

Register	ID
CCMO	NL42229.091.12