Serotonin-transporter polymorphism, early life events and emotional information processing

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Ethical review	Approved WMO
Status	Recruitment stopped
Health condition type	Mood disorders and disturbances NEC
Study type	Observational invasive

Summary

ID

NL-OMON32069

Source
ToetsingOnline

Brief title
SERT and depression vulnerability

Condition

- Mood disorders and disturbances NEC

Synonym
depression, sadness

Research involving
Human

Sponsors and support

Primary sponsor: Universiteit Leiden
Source(s) of monetary or material Support: Ministerie van OC&W,NWO- VICI 453-06-005

Intervention

Keyword: depression, genotype, serotonin, vulnerability

Outcome measures

Primary outcome

Performance on MFRT

Secondary outcome

none

Study description

Background summary

The serotonin transporter (5-HTT) is known to be the key regulator of serotonergic neurotransmission, which in turn affects a wide range of behaviors (Lesch et al, 2003). A common polymorphism in the linked promoter region of the 5-HTT gene results in variants that involve a homozygous short (ss) allele or a long (ll) allele or a heterozygous (sl) allele. Individuals who carry one or two copies of the s allele are found to have increased levels of extracellular serotonin compared to those with two copies of the l allele, which may render them vulnerable to depression (Heils et al., 1996; Lesch et al., 1996).
Recent research has tried to examine the association between the 5HTT polymorphism and cognitive functioning. Hariri and colleagues (2002, 2005) assessed neural activation in a relatively small number of s allele carriers during perceptual processing of fearful and angry human facial expressions. This simple perceptual task is known to reliably engage the amygdala (Hariri et al., 2000), a brain region that is central to the neural circuitry mediating emotional arousal and vigilance across species (Davis & Whalen, 2001). During the task, s allele carriers exhibited nearly fivefold greater amygdala activity than ll homozygotes. Using a facial emotion recognition task Marsh et al (2006) found that effects of acute tryptophan depletion on the processing of emotional expressions varies as a function of genotype at the 5-HTT. Depletion impaired the recognition of fear in s carriers but not in ll homozygotes.

Study objective

The aim of this study is to further investigate the relationship between 5-HTTLPR genotype and emotional processing. Recent literature suggests that s allele carriers of the 5HTT have a greater risk of developing depression in

response to stressful life events (Caspi et al, 2003; Wilhelm et al, 2006). The s allele may facilitate processing of negative emotional information, and this association may be moderated by adverse life experiences. Further, it seems likely that mood induction procedures can enable latent differences in emotional processing to become manifest (Mannie et al.,2007; Beevers et al., 2007); thus, a mood induction will be included in the study.

Study design

Correlational study.

Sequence:
-Psychiatric interview
-Questionnaires
-Inf proc test
-Sad mood induction
-Inf proc test

Study burden and risks

Burden: sad mood induction. Effects are small and short-lasting (few minutes).

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

Age 18 or older

Exclusion criteria

Current diagnosis of:
- depression
- bipolar disorder
- posttraumatic stress disorder
Lifetime diagnosis of:
- psychotic disorder

Study design

Design

Study type: Observational invasive

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

Recruitment

NL

Recruitment status:	Recruitment stopped
Start date (anticipated):	01-09-2010
Enrollment:	64
Type:	Actual

Ethics review

Approved WMO
Application type: First submission

Review commission: METC Leids Universitair Medisch Centrum (Leiden)

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	NL21865.058.08