Development of automatic and controlled processes in relation to addictive behaviours in youth

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

Summary

ID

NL-OMON31210

Source
ToetsingOnline

Brief title
Development of addictive behaviours in youth

Condition

- Other condition

Synonym
addiction

Health condition

verslaving

Research involving
Human

Sponsors and support

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

Intervention

Keyword: alcohol, implicit cognition, smoking, youth

Outcome measures

Primary outcome

- frequency of alcohol use

- frequency of smoking

- amount of alcohol

- amount of smoking

- binge drinking

- scores on executive functions computer

- scores on implicit cognition computer

- expectations and attitudes

- motivations

- social relationships

- polymorphisms

Secondary outcome

nvt

Study description

Background summary

Addictive behaviors are a prime cause of death and economic loss in the Western

world. Recently, scientist from the W.H.O. estimated that addictive behaviors account for 12.4% of deaths worldwide (Goldman, Oroszi & Ducci, 2005). Alcohol is the primary drug of choice among adolescents, both in the U.S. and in The Netherlands. In fact, Dutch 15-16 year-olds are the heaviest-drinking of European youths (Hibell et al., 2004). The second major addictive behavior among adolescents concerns smoking. Smoking is a major preventable cause of premature death and disability throughout the world. In the Netherlands around 28% of the adult population are smokers; 38% are considered heavy smokers (defined as smoking more then 18 tobacco units a day). Although prevalence of smoking decreases in adults, this is not the case in adolescents (Stivoro, 2006). During the past decade, neurobiological research has made great progress in delineating the neural adaptations that play a role in different phases of addictive behaviors. Although there is discussion about the relative importance of different processes in different phases of addictions (Everitt & Robbins, 2005; Robinson & Berridge, 1993; 2003), there is broad consensus about different neuro-adaptations:
- sensitization (i.e. a progressively stronger psychomotor stimulant reaction with repeated use of a drug, mediated by the mesolimbic dopaminergic system, Robinson & Berridge, 1993; 2003);
- increasing automatization of addictive behaviors (Everitt & Robbins, 2005);
- decreasing controlled processing ability as a result of prolonged alcohol or drug use (e.g., Giancola, 2000),
particularly inhibitory control (e.g., Fillmore & Vogel-Sprott, 1999, 2000).
- decreases in motivation to perform alternative behaviors than alcohol-use as a result of prolonged alcohol-use (e.g., Kalivas, & Volkow, 2005).
In addition, recent animal studies consistently reveal that alcohol exposure (especially at intoxicating doses) impairs cognitive abilities more in adolescents than in adults (e.g., White et al., 2000; White & Swartzwelder, 2004) and that this impairment is long-lasting. Moreover, chronic alcohol involvement in adolescence can have negative consequences for the maturation of a number of brain regions, including structures with extensive projections to prefrontal cortical areas that support executive control functions (e.g., DeBellis et al., 2000; White & Swartzwelder, 2004).
Note that most of the neurobiological research in addiction is primarily based on animal models and that it is unclear to what extent this translates to humans. For example, there are indications that alcohol abuse during adolescence in humans also results in deficits in executive abilities (e.g., Brown et al., 2000) and/or abnormal patterns of brain activity during the performance of executive cognitive tasks (Tapert et al., 2004), but the problem of the interpretation of these studies is that there is also evidence that deficits in executive functions can be a premorbid characteristic of risk for alcoholism or other addictions (Peterson et al., 1992; Wiers et al., 1998).
Note further that in addition to deteriorative effects of prolonged alcohol abuse on executive functions, there is also evidence that the acute effects of alcohol and many other drugs are a deterioration of executive control functions, while implicit associative processes are left unaffected and can even be primed by alcohol use (Fillmore & Vogel-Sprott, 2006). In particular

prospective research can provide insight into these mechanisms as this allows the investigation of transitions in alcohol and cigarette use, controlling for previous use and all kinds of confounders and third variables (for instance family history of drinking).
Recently, Wiers and colleagues (Wiers, Bartholow, et. al., 2006) reviewed the literature on human development and proposed an integrative model for the development of alcohol and drug use during adolescence (see also Wiers & Stacy, in press). Based on a number of recent studies involving adolescents and young adults, it is proposed that sensitization in humans results in a strengthening of largely automatic appetitive responses, which results in an attentional bias for the substance involved (e.g. Franken, 2003), automatic associations between the substance and positive-arousal (e.g. Houben & Wiers, 2006; Wiers et al., 2002; 2005) and in automatically triggered approach-action tendencies (Palfai & Ostafin, 2003). Meanwhile, the executive functions are expected to be negatively affected by prolonged alcohol/drug use.
Almost all research involving implicit cognitive processes in addictive behaviors so far has been cross-sectional or used short-term follow-ups (typically one month, e.g., Stacy, 1997; Wiers et al., 2002). In these studies it was found that both implicit and explicit cognitions uniquely contributed to the prediction of later alcohol/drug use.
Some recently performed studies also found evidence for the proposed moderating role of executive functions on the effect of automatic associations on (concurrent) alcohol and cigarette use (Grenard et al., 2006): automatic associations positively predicted alcohol use and smoking in adolescents scoring low on a working memory task, but not in adolescents scoring high on a working memory task. We recently found a similar pattern of results using a different measure of automatic associations (unpublished data). In another recent study in young adolescents, we found that automatic alcohol-arousal associations and explicit negative expectancies predicted alcohol involvement in boys a year later (Thush & Wiers, resubmitted). Both findings are in line with the model outlined above: we hypothesized that moderation of alcohol use is a function of motivation to moderate drinking, which is predicted by negative drinking experiences that translate into explicit negative expectancies (cf. Jones & McMahon, 1998).
The model is primarily focused on the development of alcohol misuse in adolescents, but given the partially overlapping risk-factors and interactions between smoking and alcohol misuse, we also assess smoking associations and smoking behavior. In the animal literature, there is much evidence for cross-sensitization, i.e. once the mesolimibic dopaminergic system gets sensitized by one substance (alcohol, nicotine, other substances), sensitization develops more rapidly for the second substance (alcohol, nicotine, other substances, Robinson & Berridge, 2003; Schoffelmeer et al., 2002). Hence, we expect that the sensitized response to alcohol and smoking develops more rapidly for individuals who smoke and (binge-) drink. The negative effects on the ability to self-regulate the sensitized action-tendencies are thought to be primarily related to alcohol misuse (there is evidence for negative effects of

alcohol misuse on the development of executive functions, we are not aware of such data for nicotine).

Study objective

In the research project proposed here, we assess all important elements of the model in a longitudinal set-up, with four assessments, separated by 6 months. This approach will allow us to study the different hypothesized mechanisms in the development of addictive behaviors in human adolescents, with a focus on alcohol use and smoking. Specifically, the research will generate answers to the following main hypotheses:
H.1 - premorbid differences in executive functioning predict later alcohol involvement and smoking
H.2 - after initiation of binge-drinking/smoking automatic appetitive processes increase in strength
H.3 - we expect this to happen especially fast in individuals who develop a pattern of smoking and bingedrinking
due to cross-sensitization
H.4 - after initiation of binge-drinking executive functions development is impaired (that is, people who do not
start to binge-drink are expected to show a stronger increase in scores on EFs). Given the absence of research
findings, we do not have specific hypotheses concerning the effects of nicotine on EFs, after controlling for the
effects of alcohol involvement.
H. 5 - We expect that in individuals scoring low on Executive Functions, the associations between alcohol
and/or smoking with arousal are stronger predictors of prospective drinking/smoking than in individuals scoring
high on Executive Functions (Moderation, cf. Grenard et al., 2006)
H.6 - negative experiences with alcohol are hypothesized to predict negative explicit expectancies and
motivation to change drinking.
H.7 - heavy alcohol-involvement is hypothesized to decrease motivation for alternative behaviors (e.g., sports,
hobbies) and on social relationships.

Study design

The general approach taken is a cohort-sequential design. Based on a power-analysis, we include 120 12-yearolds, 120 13-year-olds, 120 14-year-olds, and 120 15-year-olds at T0. We aim for an approximately equal representation of boys and girls. The two youngest cohorts will be most informative regarding initiation of addictive behaviors, the two oldest samples for investigating the escalation of addictive behaviors. Participants are requested to take part in the study which will take four times approximately one hour of testing (one

hour extra of background variables at T0), separated by 6 months intervals.

Study burden and risks

Like previously described, there is no risk associated and only a minor burden. Participants only fill in questionnaires and participate in computertasks every session and donate some saliva once. This is done by spitting in oragene boxes.

Contacts

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Trial sites

Listed location countries

Netherlands

Eligibility criteria

Age
Adolescents (12-15 years)
Adolescents (16-17 years)

Inclusion criteria

boys and girls 12, 13, 14 and 15 years old

Exclusion criteria

nvt (jongeren niet 12-15 jaar)

Study design

Design

Study type: Observational non invasive

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

Recruitment

NL

Recruitment status:	Recruitment stopped
Start date (anticipated):	01-10-2007
Enrollment:	480
Type:	Actual

Ethics review

Approved WMO

Date:	17-09-2007
Application type:	First submission
Review commission:	METIGG: Medisch Ethische Toetsingscommissie Instellingen Geestelijke Gezondheidszorg (Utrecht)

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	NL16827.097.07