Impulsivity, health-related behaviour and disease: A prospective study

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CONTENTS

ABSTRACT

In clinical settings impulsivity refers to a symptom of psychiatric disorder, but nonclinically oriented research treats impulsivity as a personality and temperament dimension. This prospective study examined whether impulsivity predicts adverse health-related behaviour and increased risk of health problems in a large, nonclinical sample of 5433 subjects working in 12 Finnish hospitals. The data were collected using two questionnaire surveys at a 2-year interval. After controlling for alcohol use at baseline, higher impulsivity predicted increased alcohol consumption at follow-up in both genders ($p < .01$) and was associated with increased likelihood of becoming a heavy drinker or taking up smoking ($p < .05$). Impulsivity also predicted an increased number of cigarettes smoked per day in the follow-up among women ($p < .001$), but not among men, although adjustment for the number of cigarettes smoked at baseline attenuated these associations ($p = .08$ for women). In men, higher impulsivity was associated with shorter sleep duration and waking up several times per night independent of baseline characteristics ($p < .01$), whereas in women, higher impulsivity predicted difficulty in falling asleep and waking up feeling tired after the usual amount of sleep ($p < .05$). In women, these associations became nonsignificant after adjustment for pre-existing somatic and psychiatric diseases. Finally, higher impulsivity was associated with an increased 2-year incidence of physician-diagnosed peptic ulcer disease (adjusted odds ratio (OR) = 2.42, 95% confidence interval (CI) = 1.21 - 4.82) and onset of depression (OR = 1.95, 95% CI = 1.28 - 2.97) after adjustment for a variety of baseline covariates. In conclusion, this study shows that in a nonclinical population, impulsivity appears to be a risk factor for various unhealthy behaviour and health problems.

TIIVISTELMÄ

Kliinisissä tutkimuksissa impulsiivisuutta on pidetty lähinnä psykiatrisena häiriönä, mutta normaaliväestössä impulsiivisuus kuvaa ulottuvuutta temperamentti- ja persoonallisuus piirteissä. Käsillä oleva seurantatutkimus selvitti impulsiivisuuden yhteyttä haitalliseen terveyskäyttäytymiseen ja lisääntyneeseen sairastumiseen laajassa otoksessa 5433 tutkittavalla, jotka työskentelevät 12 suomalaisessa sairaalassa. Aineisto kerättiin kahdella lomakekyselyllä kahden vuoden aikavälillä. Lähtötason alkoholin kulutuksen vakioimisen jälkeen korkeampi impulsiivisuus ennusti suurempaa alkoholin kulutusta molemmilla sukupuolilla ($p < .01$) ja oli yhteydessä lisääntyneeseen todennäköisyyteen siirtyä alkoholin suurkuluttajaksi tai aloittaa tupakointi ($p < .05$). Impulsiivisuus ennusti myös suurempaa savukkeiden kulutusta naisilla ($p < .001$), mutta ei miehillä, vaikkakin lähtötason savukkeenkulutuksen vakioimisen jälkeen yhteys naisilla ei ollut enää merkitsevä ($p = .08$). Miehillä korkeampi impulsiivisuus ennusti lyhyempää yöunta ja lisääntynyttä yöheräilyä ($p < .01$), kun taas naisilla korkeampi impulsiivisuus oli yhteydessä nukahtamisvaikeuksiin ja virkistämättömään yöuneen ($p < .05$). Naisilla yhteys heikkeni ei-merkitseväksi somaattisten ja psyykkisten sairauksien vakioimisen jälkeen. Korkeampi impulsiivisuus ennusti myös lisääntynyttä riskiä sairastua lääkärin diagnosoimaan vatsahaavaan (OR = 2.42, 95% CI = 1.21 - 4.82) ja masennukseen (OR = 1.95, 95% CI = 1.28 - 2.97) useiden tunnettujen riskitekijöiden vakioimisen jälkeen. Käsillä oleva tutkimus esittää, että impulsiivisuus näyttäisi olevan riskitekijä erilaiselle terveyskäyttäytymiselle ja terveydelle.

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LIST OF ORIGINAL PUBLICATIONS

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II Granö, N., Puttonen, S., Elovainio, M., Virtanen, M., Väänänen, A., Vahtera, J., Keltikangas-Järvinen, L. & Kivimäki, M. (2006). Impulsivity as a predictor of newly-diagnosed peptic ulcer disease: A prospective cohort study. *Personality and Individual Differences,* 41, 1539-1550.

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ABBREVIATIONS

ADHD	attention deficit/hyperactivity disorder
APA	American Psychiatric Association
BIS	Behavioural Inhibition System
BIS-10	Barrat Impulsiveness Scale
BMI	Body Mass Index
CI	confidence interval
DRD4	dopamine D4 receptor gene
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders IV
GHQ-12	General Health Questionnaire
HA	harm avoidance
H. pylori	Helicobacter pylori
IQ	intelligence quantient
IRS	Impulsivity Rating Scale
KSP	Karolinska Scales of Personality
MAO	monoamine oxidase
MET	metabolic equivalent task
NS	novelty seeking
NSAID	nonsteroidal anti-inflammatory drugs
OR	odds ratio
PUD	peptic ulcer disease
RD	reward dependence
SD	standard deviation
STAKES	National Research and Development Centre for Welfare and Health
WHO	World Health Organization
5-HT	5-hydroxytryptamine

1. INTRODUCTION

Impulsivity in clinical populations has often been seen as a symptom of psychiatric disorder, such as bipolar disease and attention deficit/hyperactivity disorder (ADHD) (American Psychiatric Association (APA) 1994). However, impulsivity is also a major personality and temperament dimension in several theoretical models (Cloninger et al. 1993; Evenden 1999; Eysenck, H.J. 1967; Eysenck, S. B. G. 1993; Gray 1987; Jackson 2002; Lecrubier et al. 1995; Patton et al. 1995; Schalling et al. 1987; Whiteside & Lynam 2001). In nonclinical settings, impulsivity typically refers to a tendency to engage in behaviour that involves rashness, a lack of foresight or planning, or that occurs without reflection or careful deliberation (Dawe et al. 2004).

Numerous studies of hostility, trait anxiety and optimism suggest that temperament and personality differences can be important determinants of health-related behaviour, such as smoking and heavy alcohol consumption, and increase the likelihood of developing a disease, such as depression, in general populations (Miller et al. 1996; Scheier et al. 1994; Spielberger & Jacobs 1982). However, little is known of the role of impulsivity as a predictor of such risk behaviour and morbidity or whether impulsivity is associated with sleeping problems, an increasingly prevalent problem in working populations (Lehto & Sutela 2004; Pearson, Johnson & Nahin 2006; Sateia, Doghramji, Hauri & Morin 2000.)

1.1. The concept of impulsivity

The concept of impulsivity covers a wide range of differing behaviour and the specific definitions given in the literature for impulsivity have varied, depending on the broader theoretical framework (Table 1). A seminal work by H. J. Eysenck and M. W. Eysenck (1985) conceptualized personality as three biologically based traits of temperament: extraversion, neuroticism and psychoticism. Extraversion is characterized by being outgoing, talkative, high in positive affect and in need of external stimulation. Neuroticism is characterized by high levels of negative affect such as depression and anxiety, and

psychoticism is characterized by tough-mindedness, nonconformity, hostility and impulsivity. The theory states that impulsivity is a part of one of the three main dimensions. A further study by S. G. B. Eysenck (1993) divided impulsivity into two factors: (1) impulsiveness, referring to unconscious risk taking and (2) venturesomeness, which can be described as conscious sensation seeking.

Gray (1987) proposed a two-dimensional model of personality and motivation in which impulsivity was based on the Behavioural Approach System (BAS) and anxiety on the Behavioural Inhibition System (BIS). Gray´s definition states that impulsivity in BAS means a brain system underlying the tendency to seek out pleasant stimuli. Impulsivity in Gray´s model is related to extraversion rather than psychoticism in Eysenck´s model (Gray 1987).

A more recent psychobiological model of temperament and character by Cloninger (1987a) proposes three temperament dimensions: reward dependence (RD), harm avoidance (HA) and novelty seeking (NS). RD, proposed to be linked with norepinephrine levels, refers to a tendency to respond intensely to signals of reward, in particular to signals of social approval. HA may be associated with variations in 5-hydroxytryptamine (5-HT) (serotonin) levels and is characterized by inhibition and social withdrawal, shyness and slow adaptation to change. NS may be associated with variation in dopamine levels, with a tendency toward exploratory activity, intense excitement in response to novelty, impulsive decision making and active avoidance of monotony or frustration. Thus, in this model impulsivity is seen as a part of NS, which appears to be conceptually similar to Gray´s BAS (Carver & White 1994).

Zuckerman introduced the trait of sensation seeking (Zuckerman 1984; Zuckerman et al. 1978), which overlaps impulsivity as introduced in models by Eysenck and Cloninger (Zuckerman & Cloninger 1996). Sensation seeking can be described as a tendency to seek exciting experiences. Zuckerman showed that sensation seeking appears to be genetically determined and biologically correlated (e.g. with high levels of gonadal hormones, monoamine oxidase (MAO) and augmenting of cortical-evoked potential) (Zuckerman 1984).

Dickmann (1990) emphasized that impulsivity has both dysfunctional and functional

components. Dysfunctional impulsivity is defined as a tendency to act with less forethought than most people of equal ability when this tendency is a source of difficulty. Functional impulsivity is defined as a tendency to act with relatively little forethought when such a style is optimal.

The Big Five Theory is a widely used personality theory and consists of five main factors: Openness, Agreeableness, Extraversion, Neuroticism and Conscientiousness (John et al. 1991). However, trait impulsivity is included as a subtrait in this personality model and some dimensions closely resemble impulsivity, e.g. conscientiousness in a reverse direction (Buss & Plomin 1975).

Finally, several empirical studies have examined the construct of impulsivity with psychometric techniques. Lecrubier et al. (1995) developed the Impulsivity Rating Scale (IRS), where impulsivity is measured by seven components: irritability, patience-impatience, time to make decisions, capacity to pursue an activity, aggressiveness, control of responses and capacity to delay. Patton et al. (1995), in turn, divided impulsivity into three factors using data obtained with the Barrat Impulsiveness Scale (BIS-10): motor factor (acting without thinking), cognitive factor (making quick cognitive decisions) and non-planning factor (present orientation). The Karolinska Scales of Personality (KSP, Schalling, Edman & Åsberg 1983) is a major personality assessment instrument consisting of 15 independent subscales of personality, including the impulsivity subscale. A later view by Schalling et al. (1987) suggests that KSP contains five dimensions, in which impulsivity is one subscale, with other subscales such as sensation seeking and social withdrawal, covering different aspects of extraversion. Four factors were derived from a factor analysis of KSP items by af Klinteberg, Schalling & Magnusson (1986). Impulsivity was loaded on the same factor as monotony avoidance and negative socialization.

There is evidence suggesting that personality traits are reasonably stable over time. For example, personality traits measured with KSP, including impulsivity as a subscale, showed a high mean level stability and rank-order stability after 9 years for these traits (Gustavsson et al. 1997). For impulsivity, the stability coefficients were 0.70 and 0.60. In general, personality trait test-retest reliabilities are also higher in adulthood than in childhood, as indicated by the Big Five personality traits (Hampson & Goldberg 2006).

In summation, impulsivity can be described, defined and measured in several ways and there is no unitary impulsivity or only one type of impulsive behaviour. Evenden (1999) used the term varieties of impulsivity to describe these several related behavioural and cognitive factors, which are often classified together as impulsivity. However, it should be noted that impulsivity is a normal phenomenon and can also have positive outcomes such as boldness, quickness, spontaneity, courageousness or unconventionality, even though impulsivity is usually a term for maladaptive behaviour (Daruna & Barnes 1993).

Table 1. Definitions of impulsivity in different personality theories

Author and terms	Definition
H. J. Eysenck & M. W. Eysenck 1985. Personality theory of Extraversion, Neuroticism and Psychoticism.	Impulsivity is a part of the psychoticism personality trait. Other personality traits include Extraversion and Neuroticism.
Gray 1987. Behavioural Approach System (BAS) and Behavioural Inhibition System (BIS).	Impulsivity in BAS proposed to be a brain system underlying the tendency to seek out pleasant stimuli.
Cloninger 1987a. Psychobiological model of temperament and character.	Impulsivity is a subdimension of the temperament trait Novelty seeking.
Zuckerman et al. 1978, Sensation seeking.	Sensation seeking, a tendency to seek exciting experiences, is similar to the concept of impulsivity
Dickmann 1993. Functional and dysfunctional impulsivity.	Dysfunctional impulsivity is defined as a tendency to act with less forethought than most people of equal ability when this tendency is a source of difficulty. Functional impulsivity is defined as a tendency to act with relatively little forethought when such a style is optimal.
<u>Specification of impulsivity</u>	
S. G. B. Eysenck 1993. Impulsiveness and venturesomeness.	Impulsiveness is defined as unconscious risk taking. and Venturesomeness as conscious sensation seeking.
Lecrubier et al. 1995. Impulsivity rating scale (IRS).	Impulsivity includes seven components: irritability, patience-impatience, time to make decisions, capacity to pursue an activity, aggressiveness, control of responses and capacity to delay.
Patton et al. 1995. Barrat Impulsiveness Scale (BIS-10).	Impulsivity is divided into three factors: motor factor (acting without thinking), cognitive factor (making quick cognitive decisions) and nonplanning factor (present orientation).
Schalling et al. 1987. Karolinska Scales of Personality.	Impulsivity is characterized by a tendency to act on the spur of the moment, without thinking about it so deeply, a tendency to get enthusiastic about new ideas that are dropped and forget to find out if there are any disadvantages and a self-appraisal of impulsivity.

1.2. Stress, impulsivity and health

There is some evidence that strong psychological stress may cause alteration in development of executive cognitive functions and emotional perception, which are adjusted by the brain´s prefrontal cortex and limbic system (Arnsten & Shansky 2004; Moradi et al. 1999; Perry & Pollard 1998). These systems regulate behavioural inhibition, attention control, sensitivity to rewards and penalties, decision making and emotional regulation. Development and functioning of this neural network are sensitive to environmental factors, such as stress, and can lead to difficulties in executive cognitive functions and emotional regulatory skills, which may result in an inability to sustain attention, evaluate consequences and control impulsivity (Bremner 1999; Bremner et al. 1999; Steckler & Holsboer 1999).

Psychological stress is associated with several health outcomes and health behavioural factors. For example, there is some evidence linking stress with impaired sleep (Akerstedt 2006), alcohol drinking (Sillaber & Henniger 2004), smoking (Baker, Brandon & Chassin 2005), peptic ulcer disease (PUD) (Jones 2006), and depression (Kendler, Thornton & Gardner 2000; Kendler et al. 1999; Caspi et al. 2003). On the other hand, impulsivity has also been associated with health and health behavioural factors. Studies have reported associations of impulsivity with depression and mania in bipolar disorder (Swann et al. 2007), PUD (Feldman et al. 1986), smoking (Baker, Brandon & Chassin 2004), alcohol drinking (Hussong & Chassin 1994) and sleep (Ireland & Culpin 2006).

Taken together, stress has been associated with impulsivity, health-related behaviour and diseases, while evidence also suggests associations between impulsivity, health behaviour and diseases. However, systematic research of direct associations between impulsivity and health-related behaviour and diseases is largely lacking.

1.3. Impulsivity, smoking and alcohol consumption: previous findings

Personality and temperament may play a role in behaviour involving risk to health (Barefoot et al. 1989; Caspi et al. 1997; Cloninger 1987; Hoyle 2000). In terms of public health significance, cigarette smoking and heavy alcohol drinking are particularly important. Tobacco use causes an estimated 3 million annual deaths worldwide, of which 1.9 million occur in the developed world (World Health Organization (WHO) 1997). Half of all long-term regular smokers who begin smoking during adolescence can expect to die from tobacco use, and 50% of these die prematurely in middle age, losing 20-25 years of life expectancy compared with nonsmokers (Peto 2004). According to a recent WHO project, the Global Burden of Disease Study, alcohol accounts for a substantial amount of harm in the world (Rehm et al. 2004). Alcohol was estimated to have caused 1.8 million deaths or 3.2% of all deaths worldwide and almost half of these deaths (48%) were due to various alcohol-related diseases.

There is some evidence suggesting a relationship between impulsivity, smoking and alcohol consumption (Bergen & Caporaso 1999; Grau & Ortet 1999; Mitchell 1999; Mulder 2002; Lipcus et al. 1994; Waldeck and Miller 1997). For example, Grau & Ortet (1999) found that impulsivity assessed with the KSP was associated with increased alcohol consumption in a study of 149 nonalcoholics. Waldeck & Miller (1997) studied impulsivity and use of alcohol, nicotine and caffeine in their cross-sectional study of 332 students. They found that men with high impulsivity used alcohol and caffeine more than men with low impulsivity, no differences were seen for smoking. Among women, significant differences between high- and low- impulsivity groups were only found for alcohol intake and smoking. Lipcus et al. (1994), using a large, prospective database that impulsive personality predicted smoking status. Mitchell (1999) reported in her study with 40 nonclinical subjects that smokers were more impulsive than never smokers.

These studies suggest that cigarette smoking and alcohol consumption may be more common among impulsive subjects. However, the existing evidence is typically based on cross-sectional data that cannot exclude the possibility of reversed predictive relationships

(i.e. health risk behaviour may predict increasing impulsivity rather than vice versa) and prior research has been insufficiently controlled for potential third factors, such as socioeconomic status, a strong correlate of health risk behaviour (Adler & Newman 2002). Furthermore, many previous studies were limited to clinical samples with poor generalization to healthy populations.

1.4. Impulsivity, sleep duration and insomnia: previous findings

There is some evidence supporting a link between stress and various sleep problems, and between impulsivity and stress. In considering this, the association between impulsivity and sleep is plausible. Sleep disturbances affect a wide part of the population and are therefore also a major public health issue. It has been estimated that 10-15% of the adult population suffers from chronic sleep disturbances and an additional 25-35 % from transient or occasional insomnia (Roth 2001). The APA (1994) defined insomnia "as having difficulty initiating or maintaining sleep or nonrestorative sleep, causing clinically significant distress or impairment in social, occupational or other important areas of functioning, not occurring exclusively during the course of another sleep disorder, not occurring exclusively during the course of a mental disorder, and not being due to the direct physiological effects of a substance or a general medical condition" (p. 553). The likelihood of insomnia is higher among older people (Ohayon, Zulley, Guilleminault, Smirne & Priest 2001), and other potential confounding factors associated with sleep disturbances has shown to be marital status, gender, socioeconomic status, shift work, obesity, depression, somatic illness, mental disorders and substance use (Fabsitz, Sholinsky & Goldberg 1997; Flemons 2002; Harvey 2001; Härmä et al. 2006; Ohayon et al. 2001; Sateia et al. 2000).

The association between impulsivity and insomnia has previously been examined in children and adolescents, especially in those with ADHD. For example, Ring et al. (1998) found that significantly more children with ADHD demonstrated single or multiple sleep disturbances as well as higher rates of specific sleep disorders such as initial and middle insomnia, compared with their siblings. Furthermore, Owens, Maxim, Nobile, MvGuinn & Msall (2000) reported that children with ADHD had significantly more disturbed sleep than

controls. The average sleep duration as reported by parents was significantly shorter in the ADHD group and children with ADHD also reported their own sleep to be more disturbed than controls. Finally, Ireland & Culpin (2006) found a significant association between increased impulsivity and decreased hours of sleep in a sample of juvenile and young offenders, measured by a self-report with BIS-10. However, research on impulsivity and insomnia in a nonclinical context is largely lacking.

1.5. Impulsivity and peptic ulcers: previous findings

Indirect evidence suggests that higher impulsivity may be associated with a greater risk of PUD, since some studies have reported a positive association between impulsivity and stress and other studies a positive association between stress and PUD. The idea that personality may be associated with ill health and even mortality was already stated in the 15th century (Lipowski 1984). While cardiovascular diseases have been the most intensively studied, PUD, a multifactorial disease, has also been the focus of study. Although Helicobacter pylori (H. pylori) is considered to be the main determinant of PUD, most people having H. pylori infection never develop PUD (Quan & Talley 2002). A number of studies have shown that environmental factors, such as the intake of nonsteroidal anti-inflammatory drugs (NSAIDs)(Kurata & Nogawa 1997), smoking (Martin et al. 1989), heavy alcohol consumption (Levenstein, Kaplan & Smith 1997), and psychosocial stress (Ellard et al. 1990) are also significant predictors of PUD. In mice, long-term stress resulted in gastric mucosal inflammation and erosion and this effect was suggested to occur independently of H. pylori infection (Kim, Lee & Lee 2002).

Previous data show that emotional disturbances and personality-related individual differences may also have an impact on the development of PUD. The results of Goodwin & Stein (2003) showed that neuroticism was associated with significantly increased risk for PUD when differences in sociodemographic characteristics, smoking, perception of poor health, comorbid mental disorders and physical illnesses were statistically controlled for. Other studies have suggested that other personality factors such as, generalized anxiety disorder (Goodwin & Stein 2002), hostility (Levenstein, Kaplan & Smith 1997) and

dependency (Magni et al. 1988) may predispose to PUD.

Few small-scale studies have examined the relationship between impulsivity and PUD. Feldman and co-workers (1986) showed with PUD patients and controls that personality disturbances, such as impulsivity, immaturity, feelings of social isolation and alienation, were more common in PUD patients than in controls. A further study by this group (Feldman et al. 1992) found that serum gastrin concentrations were related to the affective state in normal men and this relationship was altered in PUD. Temperamental impulsivity and social isolation were associated with more labile basal acid secretion rates, possibly predisposing to the development of PUD (Feldman et al. 1992), which is also associated with depression (Levenstein, Kaplan & Smith 1997). However, no evidence is available to test whether impulsivity can actually predict the onset of PUD in nonclinical populations.

1.6. Impulsivity and depression: previous findings

Depression is a severe disease, that may increase the risk of premature death (Carney et al. 2003), sickness absence (Druss et al. 2000) and impaired work capacity (Kessler et al. 2001). According to an estimate of the Global Burden of Disease Study, depression will account for 15 % of the disease burden in the world by 2020 (Murray & Lopez 1996). There is some evidence of factors related to depression, such as increasing age (Hasin, Goodwin, Stinson & Grant 2005), female gender (Fryer et al. 2004), lower socioeconomic status (Paykel 2003), lack of social support (Chen et al. 2005), smoking (Van Gool et al. 2003) and alcohol consumption (Perreira & Sloan 2002). In addition, environmental factors such as stressful life events involving threat, loss, humiliation and defeat, are associated with the incidence of depression (Brown 1998; Kendler et al. 1999; Kessler 1997; Pine et al. 2002).

Shared biological correlates have led to research on the potential relationships between impulsivity and depression. For example, depression was associated with underactivity of central 5-hydroxytryptamine (serotonin, 5-HT) transmission (Malison et al. 1998; Owens & Nemeroff 1998; Spreux-Varoquaux et al. 2001) and impulsivity was related to decreased 5-HT activity (Coccaro et al. 1989; Cremniter et al. 1999; Evenden 1999; Linnoila et al.

1983; Spreux-Varoquaux et al. 2001). Cremniter et al. (1999) reported a correlation between impulsivity and low levels of cerebrospinal fluid 5-hydroxy-indoleacetic acid (CSF-5-HIAA) in a sample of 23 suicide attempters. Spreux-Varoquaux et al. (2001) found in 27 suicide attempters that the plasma 5-HIAA levels were lower in impulsive suicide attempters than in non-impulsive suicide attempters, although these groups did not differ in regard to depression scales. In the same study, platelet 5-HT was associated with the intensity of depression and the lower plasma 5-HIAA concentration was inversely associated with the degree of impulsivity.

The majority of studies that have directly focused on impulsivity and depression have dealt with the effect of impulsivity on suicidality among depressed patients (e.g. Corruble et al. 2003; Corruble et al. 1999; Fawcett et al. 1987; Mann et al. 1999; Pezawas et al. 2002). In a longitudinal study by Pezawas et al. (2002), impulsivity was a major underlying risk factor for suicide in a depressed population. Corruble et al. (1999) found in their longitudinal study with 50 depressed in-patients that suicide attempters had significantly higher impulsivity scores than nonsuicide attempters at baseline and after a 4-week period of treatment. Both impulsivity and depression scores decreased in the 4-week treatment period throughout the sample. A later study by the same research group (Corruble et al. 2003) identified three dimensions of impulsivity in their study of 127 depressed in-patients: behavioural loss of control, nonplanning and cognitive impulsivity. Few studies have examined impulsivity and depressive symptoms in nonclinical populations. For example, Elovainio et al. (2004) showed in their prospective study with a 4-year follow-up that some subtraits of Cloningers psychobiological model, such as impulsiveness, shyness with strangers, fatigability, sentimentality and persistence were associated with increased risk of depressive symptoms independently of a variety of other known risk factors for depression. However, the researchers could not define a clinical cut- off point for depression and the sample was restricted to subjects 20-35 years of age at baseline.

There are phenotypic associations occurring in childhood and adolescence between constructs relating to impulsivity and depression. The association between impulsivity and depression has previously been examined with children and adolescents, especially with those with ADHD and conduct disorders. For example, Brodsky et al. (2001) found an

association of childhood trauma and impulsivity with suicidal behaviour and major depression in adulthood. Cataldo, Nobile, Lorusso, Battaglia & Moltemi (2005) reported that some depressed children and adolescents were rated by their parents as significantly more impulsive or restless than controls. Furthermore, ADHD and conduct disorders include significant problems with impulse control. There are several studies reporting an association between depression and ADHD (Anderson, Williams, McGee & Silva 1987; Velez, Johnson & Cohen 1989) and depression and conduct disorders (McGee et al. 1990; Kashani et al. 1987) in child and adolescent studies (see for review Angold, Costello & Erkanli 1999).

Despite these associations, it remains unclear whether impulsivity is a predictor of the onset of depression in adulthood. Prior research on this issue has typically focused on the relationship between impulsivity and suicide in clinical populations or in samples of children or adolescents with ADHD or conduct disorders. In comparison to research on impulsivity and mental health among children, few studies have reported appropriate data with adults. Since prospective studies on nonclinical populations free of depression are largely lacking, previous research has also failed to address the question of temporal direction between impulsivity and depression. Further limitations in existing evidence include failure to use clinically defined cut offs, limited age range within adulthood and insufficient control for potential confounding factors. In summation, direct evidence for the relationship between impulsivity and depression in nonclinical populations is scarce.

1.7. Aims of the present study

To reduce many of the limitations in previous research on impulsivity, the present longitudinal study examined the relationship of impulsivity to smoking, alcohol consumption, insomnia, sleep duration, PUD and depression in a large, nonclinical sample of employees with a large age range.

The first aim of the study was to investigate the relationships of impulsivity with health-related behaviour, such as smoking status, initiation of smoking, smoking intensity and alcohol consumption, when potential confounding factors such as age, gender, education

and socioeconomic status were adjusted for. We examined the association between impulsivity, insomnia and sleep duration when potential confounding factors were adjusted for. We hypothesized that higher impulsivity could predict higher consumption of alcohol and cigarettes as well as insomnia and short sleep duration after taking into account the baseline characteristics.

The second aim was to examine the association between impulsivity and incidence of newly diagnosed PUD and newly diagnosed depression. We hypothesized that impulsivity could predict the incidence of these diseases when potential confounding factors are controlled for. These include shift work, smoking, alcohol consumption, body mass index (BMI) and physical activity. We hypothesized that higher impulsivity could independently predict a higher risk of incident PUD and a higher risk of new-onset depression.

2. METHODS

2.1. Participants

Twelve hospitals in four of the 23 health districts in Finland volunteered to participate in the project 'Work and Health in Finnish Hospital Personnel' coordinated by the Finnish Institute of Occupational Health (Kivimäki, Elovainio & Vahtera 2000). Based on employers' registers, there were 10 968 employees 18-63 years of age working in these hospitals. They were asked to respond to a questionnaire on personality and health risk behaviour in 1998. The employees were informed of the study before responding to the questionnaire and the voluntariness of the response was highlighted. In all, 8107 employees (74%) responded to the first survey, and of these 6675 were working in the target hospitals two years later at the time of the second survey (81% of the eligible population at follow-up). The male respondents were from the following occupations at follow-up: 29% physicians, 32% nurses, 7% other professionals, 3% laboratory and x-ray staff, 7% administrative staff, and 22% in maintenance, cleaning and other supporting activities. In women, the occupational distribution was as follows: 3% physicians, 58% nurses, 4% other professionals, 7% laboratory and x-ray staff, 12% administrative staff, and 16% in maintenance, cleaning and other supporting activities. The sex distribution of this cohort is similar to that in Finnish hospitals in general (National Research and Development Centre for Welfare and Health (STAKES), 2004). The ethics committee of FIOH approved the Hospital Personnel study.

In the first study the associations between impulsivity, smoking and alcohol consumption were investigated (I) and in the fourth study the associations between impulsivity, insomnia and sleep duration (IV). The total number of subjects was 5433 (601 men, 4832 women) in both studies (I, IV). The respondents of the second study (II) were all those 4636 employees who responded to impulsivity, gender and age questions in the first survey and PUD questions in the first and second surveys and had no diagnosed PUD at baseline. The prevalence of lifetime PUD at baseline was 5.0% (383 cases among 7717

employees). Those subjects who did not work in the target hospitals or respond to the second survey, did not differ in impulsivity (p = .826) or prevalence of physician-diagnosed PUD (p = .278) at baseline from those who responded to both questionnaires. The third study was designed to investigate the associations between impulsivity and depression (III), and in the final cohort included all those 4505 employees (530 men and 3975 women) who had no physician-diagnosed depression and no missing data in questions of age, gender, impulsivity and depression at baseline. The prevalence of lifetime depression at baseline was 8.0% (632 cases among 7743 employees), however, lifetime depression was more common among dropouts (p = .039).

2.2. Assessment of impulsivity

Impulsivity was measured at time 1, using a five-item impulsivity scale derived from the KSP, a measure used both in clinical and nonclinical populations (af Klinteberg et al. 1987; Grau & Ortet 1999; Nordstrom et al. 1996; Schalling et al. 1987). The skewness of the scale was 0.975 and kurtosis 0.629. The scale was highly reliable (Cronbach's alpha = 0.88). Participants gave a self-assessment of their style of reacting in different situations on a four-point scale (1="does not describe me at all" to 4="describes me very well"). The items were as follows: "I have a tendency to act on the spur of the moment, without thinking about it so deeply", "I get very enthusiastic about new ideas and suggestions that I forget to find out if there are any disadvantages", "It often happens that I get involved in things a little too hastily", "I speak before I think what I am saying" and "I consider myself impulsive". We calculated the sum of the item scores and replaced missing values with the mean of the missing item (I, II, III, IV). The impulsivity scale was treated as a continuous variable in the study on smoking and alcohol consumption and in the study on insomnia and sleep duration (I, IV). In the study on PUD and depression (II, III), the impulsivity scale was further divided into quartiles for the analysis and treated as a categorical variable.

2.3. Assessment of smoking and alcohol consumption

In the study on smoking and alcohol consumption (I), smoking was assessed as the number of cigarettes smoked during an average day. In addition, we calculated pack-years based on questions requesting whether the respondent was a "never smoker", "former smoker" or "current smoker" and information on the age at which the respondent had begun smoking and quit smoking, as well as the number of cigarettes smoked in a day, were used to measure the number of cigarettes smoked per day. The number of pack-years was calculated as follows: the number of smoking years × number of cigarettes smoked per day divided by 20. This measurement was used in the studies on smoking and alcohol consumption and insomnia and sleep duration (I, IV). In the study on PUD (II), pack-years was treated as a three-level variable (0, 0.1-5 and > 5 pack-years, Kivimäki et al. 2004). In the study on depression (III), the measure of smoking distinguished smokers from nonsmokers. Smoking was assessed both at baseline and at follow-up.

Alcohol consumption (I, II, III and IV) was assessed with the alcohol questionnaire developed by Kaprio et al. (1987) at baseline and at follow-up. The questionnaire requests information on the frequency and quantity of alcohol consumed during an average week (or month), separately for beer, wine and spirits. A bottle of beer, a glass of wine or a single measure of spirits was taken as equivalent to 13 g of pure alcohol. For each type of beverage, consumption was converted into grams of pure alcohol and summed to yield an estimate of total consumption. From this estimate, we constructed a variable that measures alcohol consumption in grams of pure alcohol per week. This estimate was used as a continuous variable in the study on alcohol consumption and smoking and the study on insomnia and sleep duration (I, IV). In the study on PUD (II), alcohol consumption was divided into four categories (0 - 50, 51 - 100, 101 - 275 and > 275g pure alcohol per week, Vahtera et al. 2002). The cutoff- points for heavy alcohol consumption were 190g/week for women and 270g/week for men (Virtanen et al. 2002) in studies on smoking, alcohol consumption and depression (I, III).

2.4. Assessment of sleep duration and insomnia

Sleep duration (IV) was measured with the question "How many hours do you normally sleep during the day and night?" (1 = 6 hours or less, 2 = 6.5 hours,... 9 = 10 hours or more). The responses were transformed into hours for analysis.

The four-item Jenkins Scale (Jenkins, Stanton, Niemcryk & Rose 1988) was used to indicate whether the participants had experienced sleep disturbances (IV) during the previous 4 weeks. The questions in the Jenkins scale concern "having trouble falling asleep", "waking up several times per night", "having trouble staying asleep" and "waking up after the usual amount of sleep feeling tired and worn out". Each of the items was rated on a six-point response scale (1 = never to 6 = every night). The first question identifies participants with sleep-onset insomnia, the second and third questions identify those with sleep-maintenance insomnia, and the last question determines those with unrefreshing sleep. The scales for sleep duration and insomnia were assessed as continuous variables and therefore cutoff- points were not used.

2.5. Assessment of peptic ulcer

PUD was measured, using a self-administered checklist of common diseases (Kivimäki, Elovainio & Vahtera 2000; Kivimäki et al. 2003; Koskenvuo et al. 1988). For each disease, the respondent was asked to indicate whether or not a physician had diagnosed him or her as having the disease. The following question referred to PUD: "Has a physician told you that you have or have had peptic ulcer disease?". Incident cases of newly diagnosed PUD included respondents who had no missing data on the PUD question in either survey and who reported being diagnosed with this disease at follow-up in 2000 but not at baseline in 1998. Based on physician's interviews and objective medical measurements, the checklist of common chronic diseases is highly valid e.g. for coronary heart disease and asthma (Koskenvuo et al. 1988; Haapanen et al. 1997; Kilpeläinen et al. 2001), but specific validation studies for PUD are lacking.

2.6. Assessment of depression

Depression (III) was measured with a self-administered checklist of common diseases (Kivimäki et al. 2000; Kivimäki et al. 2003; Koskenvuo et al. 1988). For each disease, the respondent was asked to indicate whether or not a doctor had diagnosed him or her as having the disease. Incident cases of newly diagnosed depression included respondents who had no missing data on the depression question and who reported being diagnosed with this disease in 2000 but not in 1998. Based on physician's interview and objective medical measurements, the checklist of common chronic diseases is highly valid for coronary heart disease and asthma (Koskenvuo et al. 1988; Kilpeläinen et al. 2001). However, there are no validity tests available for depression.

2.7. Assessment of covariates at baseline

Demographic variables measured at baseline included gender (I,II, III, IV), age (I,II, III, IV), marital status (single or living with someone) (III, IV), education as indicated by an eight-point scale (1 = no occupational education, 2 = occupational course, 3 = indentured education or vocational school, 4 = college, short form, 5 = college, 6 = vocational high school or polytechnics, 7 = institution of higher education, 8 = university) (I, IV), salary (monthly income in Finnish marks) (I), and shift work, in which participants were dichotomized into those who worked in permanent day shift vs. those who worked in some other shift system (II, IV). In the studies on alcohol PUD and depression age was divided into three categories < 35, 35 - 45, > 45 years and education was divided into four levels (1 = no occupational education, occupational course, indenture education or vocational school, 2 = college, short form, 3 = college, 4 = university or high school) (II, III).

A subsidiary analysis was performed in relation to minor psychiatric morbidity (II, III, IV), a proxy measure for undiagnosed depression and other mental health disorders in nondiagnosed populations, using the well-validated 12-item version of the General Health Questionnaire (GHQ-12) (Goldberg & Williams 1988). In the questionnaire, participants gave a self-assessment of their mood and well-being on a four-point scale, e.g. "Have you

recently felt yourself as unhappy and depressed?", "Have you recently lost your self-confidence?" and " Have you recently felt yourself as worthless?" Individuals scoring ≥ 4 were estimated to have mental distress according to studies validating GHQ-12 against standardized psychiatric interviews (Holi, Marttunen & Aalberg 2003) and this threshold was applied in the present study to identify people with minor psychiatric morbidity at baseline and follow-up.

The BMI was calculated from self-reported information on the participant's weight and height (kg/m^2). In the study on PUD (II), the BMI was divided into three generally used levels: BMI < 25, BMI 25 - 30 and BMI > 30 kg/m^2 (Kopelman 2000; WHO 2002). In the study on sleep duration and insomnia (IV), the BMI was treated as a continuous variable. Physical activity was measured by asking participants to report how many hours they walk, walk fast, jog or run in 1 week. This leisure-time physical activity was transformed into metabolic equivalent task (MET) –hours/week (Tanasescu et al. 2002). The MET score was divided into quartiles in the study on PUD (II).

Social support (III, IV) was measured with Sarason's (Sarason, Shearin, Pierce & Sarason 1987) questionnaire (six items on a 6-point scale, range = 0 - 30) of social network size (Cronbach's alpha 0.82). In the questionnaire, the participants gave a self-assessment of people who could potentially help them when they were in need of support or help in different situations (spouse, relatives, close friends, colleagues, friends, or nobody). For example:"Who can you really trust when you want to forget your trouble when you are stressed?" and "Who can you really trust when you are sad and would like to feel better?" The number of positive answers was summed to describe the size of the social network (IV). The scale was further divided into quartiles in the study on depression (III).

Physician-diagnosed somatic disease, and depression and other psychiatric diseases were measured, using a self-administered checklist of common diseases (Kivimäki, Elovainio & Vahtera 2000; Koskenvuo et al. 1988) (IV). For each disease, the respondent was asked to indicate whether or not a physician had diagnosed him or her as having the disease. The responses were transformed into two dichotomous variables: to a somatic disease and to a depression and other psychiatric disease (IV).

2.8. Statistical analysis

We used linear regression analysis to investigate the relationship between impulsivity, smoking, alcohol consumption, sleep duration and insomnia (I, IV). We adjusted the models for age, gender, education, salary, smoking and baseline alcohol consumption (I). All analyses were performed using the SPSS 11.0 for Windows program.

Linear regression analysis was used to estimate the strength of the association between impulsivity, sleep duration and insomnia. The analysis was performed separately for men and women. Adjustments were made in five steps, for (1) age and marital status, (2) additionally for education and shift work, (3) additionally for life-style factors (smoking, alcohol consumption, physical activity and BMI), (4) additionally for psychosocial factors (minor psychiatric morbidity and social support), and (5) additionally for physician-diagnosed diseases (somatic disease and depression and other psychiatric disease at baseline). All analyses were performed using the SPSS 13.01 statistical program.

Logistic regression analysis was used to estimate the strength of the association between impulsivity and incidence of PUD (II). The odds ratios (ORs) for new physician-diagnosed PUD by levels of impulsivity were calculated. We adjusted ORs and their 95% confidence intervals (CIs) for demographics (age, gender, education and shift work), life-style factors (smoking status, alcohol use, BMI and physical activity) and mental health factors (minor psychiatric morbidity, depression and other psychiatric disease). All analyses were performed using the SPSS 12.0 for Windows program.

Logistic regression analysis was used to estimate the strength of the association between impulsivity and the incidence of physician-diagnosed depression (III). The adjusted ORs and their 95% CIs for new medically diagnosed depression for impulsivity were calculated. Adjustments were made for demographics (age, gender and education), life-style factors (smoking and heavy drinking) and social and psychological factors (social network size and minor psychiatric morbidity) at baseline. We replicated the analysis (III) using GHQ caseness (derived from minor psychiatric morbidity measure) as an outcome measure instead of diagnosed depression. Impulsivity and covariates were entered into logistic models as with the factor of medically diagnosed depression. All analyses were performed

using the SPSS 12.0 for Windows program.

3. RESULTS

3.1. Impulsivity, smoking, and alcohol consumption

The means, standard deviations (SDs), and bivariate correlations among demographic variables, smoking, alcohol consumption, and impulsivity are shown in Table 2 (I). Impulsivity was correlated with younger age, male sex and lower education. Higher impulsivity was also associated with higher numbers of cigarettes smoked and greater alcohol intake at Time 1 and Time 2. Smoking and alcohol consumption were intercorrelated and their Time 1 - Time 2 correlations were high. Smoking was more common and alcohol consumption greater in men, those with lower education, and those with lower salaries.

Table 2.

Means, standard deviations and Pearson correlations among variables.

Variable	Mean	SD	1	2	3	4	5	6	7	8	9
Time 1											
1. Age	43.3	8.4	-	-.02	-.10***	.12***	-.07***	-.02	.05***	-.03	.05***
2. Gender [1)]	1.9	0.3		-	-.14***	-.40***	.07***	-.10***	-.35***	-.09***	-.31***
3. Education[2)]	5.4	1.6			-	.59***	-.05***	-.12***	.03*	-.13***	.03*
4. Salary[3)]	11037	3427				-	-.07***	-.06***	.16***	-.06***	.17***
5. Impulsivity	8.4	3.1					-	.06***	.06***	.06***	.06***
6. Smoking [4)]	1.6	4.5						-	.18***	.89***	.19***
7. Alcohol cons. [5)]	74.9	105.3							-	.17***	.63***
Time 2											
8. Smoking	1.5	4.4								-	.18***
9. Alcohol cons.	74.7	115.6									-

N=4757 after listwise deletion

*p<.05, **<p.01, ***p<.001.

[1)] 1=men, 2=women. [2)] 1=not occupational education to 8=university. [3)] Finnish Marks per month. [4)] Cigarettes smoked per day. [5)] Grams of absolute alcohol in an average week.

Hierarchical linear regression models for smoking at Time 2 are shown in Table 3. Impulsivity at Time 1 was related to smoking at Time 2 in models controlled for the effects

of age, gender, education and salary (Models 1 - 3). After controlling for the strong effect of smoking at Time 1, the predictive relationship between impulsivity at Time 1 and smoking at Time 2 became nonsignificant (Model 4, Adj. R^2 = .695). We replicated these analyses using pack-years as the indicator of smoking instead of the number of cigarettes smoked daily. The results were essentially the same.

Table 3. Hierarchical linear regression models of smoking at Time 2.

Variables	Model 1		Model 2		Model 3		Model 4	
at Time 1	Beta	p	Beta	p	Beta	p	Beta	p
Impulsivity	.06	<.001	.07	<.001	.06	<.001	.01	.134
Age			-.02	.134	-.03	.030	-.01	.240
Gender			-.08	<.001	-.19	<.001	-.01	.360
Education					-.13	<.001	-.02	.070
Salary					-.03	.106	-.00	.685
Smoking							.89	<.001

N=4757

The association between impulsivity and smoking was dependent on gender (p for interaction .021), and therefore we also performed separate analyses for men (Table 4) and women (Table 5). In men, there was no relationship between impulsivity at Time 1 and smoking at Time 2 in any of the four models tested (Model 4, Adj. R^2 = .610). In women, there was a significant relationship between impulsivity at Time 1 and smoking at Time 2 before adjustment for smoking at baseline. After this adjustment, the relationship was almost significant (p = .08, Adj.R^2 = .694).

Table 4. Hierarchical linear regression models for smoking at Time 2 in men.

Variables	Model 1		Model 2		Model 3		Model 4	
Time 1	Beta	p	Beta	p	Beta	p	Beta	p
Impulsivity	-.01	.729	-.02	.729	-.02	.663	-.01	.737
Age			-.01	.896	.07	.145	.02	.387
Education					.03	.650	.00	.997
Salary					-.20	.004	-.02	.525
Smoking							.87	<.001

N=539

Table 5. Hierarchical linear regression models for smoking at Time 2 in women.

Variables	Model 1		Model 2		Model 3		Model 4	
Time 1	Beta	p	Beta	p	Beta	p	Beta	p
Impulsivity	.08	<.001	.08	<.001	.08	<.001	.01	.078
Age			-.02	.102	-.05	.001	-.01	.072
Education					-.16	<.001	- .02	.061
Salary					.01	.793	-.01	.601
Smoking							.89	<.001

N=4538

Hierarchical regression analyses for alcohol consumption are shown in table 6. The relationship between impulsivity at Time 1 and increased alcohol consumption at Time 2 remained statistically significant after controlling for demographic characteristics and alcohol consumption at Time 1 (p = .004, Adj. R^2 = .402). The association between impulsivity and alcohol consumption was not dependent on gender (p for interaction .268).

Table 6. Hierarchical linear regression models of alcohol consumption at Time 2.

Variables	Model 1		Model 2		Model 3		Model 4	
at Time 1	Beta	p	Beta	p	Beta	p	Beta	p
Impulsivity	.04	<.001	.08	<.001	.08	<.001	.03	.004
Age			.05	<.001	.03	.019	.01	.420
Gender			-.32	<.001	-.29	<.001	-.09	<.001
Education					-.05	.006	-.03	.033
Salary					.08	<.001	.06	<.001
Alcohol cons.							.59	<.001

N=4757

During the follow-up, 57 participants took up smoking and 288 became heavy drinkers (as defined by alcohol consumption >280 g of pure alcohol in men and >190 g of pure alcohol in women). Impulsivity was significantly associated with increased likelihood of taking up smoking or becoming a heavy drinker (Beta = .04, p = .024) among participants who were nonsmokers or nonheavy drinkers at entry into the study.

3.2. Impulsivity, sleep duration and insomnia

Means, SDs and bivariate correlations for baseline characteristics, impulsivity and insomnia are shown in Tables 7 and 8. In men, the mean age was 44 years, mean smoking was 5.4 pack-years, alcohol consumption was 179 g pure alcohol per week, physical activity was MET 14.2 hours/week, social support was 10.8 persons and BMI was 25.4. In women, the mean age was 43 years, mean smoking was 2.3 pack-years, alcohol consumption was 62 g pure alcohol per week, physical activity was MET 12.5 hours/week, social support was 12.6 persons and BMI was 24.4 In men (IV), higher impulsivity was correlated with higher alcohol consumption, lower physical activity, somatic disease, shorter sleep duration, difficulties in falling asleep and waking up during the night (Table 7). In women, higher impulsivity was associated with lower age, lower education, higher smoking and greater alcohol consumption, higher rate of minor psychiatric morbidity and depression and other psychiatric disease, and with having trouble falling asleep and waking up feeling tired and worn out after the usual amount of sleep (Table 8).

After controlling for all baseline characteristics, higher impulsivity in men at Time 1 was significantly associated with shorter sleep duration and waking up several times per night at Time 2 (Table 9). In women, higher impulsivity at Time 1 was related to difficulties in falling asleep and waking up after the usual amount of sleep and feeling tired and worn out at Time 2 in steps 1 - 4 (Table 10). After additionally controlling for the effects of somatic and psychiatric diseases in step 5, these associations became nonsignificant.

Table 7.

Means, standard deviations and Pearson correlations among variables in men.

Variable	Mean	SD	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.
Time 1																			
1. Age (years)	43.7	9.0	.16[c]	.20[c]	-.31[c]	.21[c]	.14[b]	-.24[c]	.11[a]	-.22[c]	.07	.20[c]	.08	-.07	-.11[a]	-.03	.13[b]	.12[b]	-.09
2. Marital status	1.8	0.36	-	.18[c]	-.06	-.03	-.07	-.10[a]	.05	.07	.01	.05	.01	-.04	-.05	-.04	.00	-.01	.03
3. Education	6.2	1.8		-	-.28[c]	-.11[a]	-.08	-.08	-.13[b]	.03	-.03	-.04	-.01	-.07	-.08	-.08	-.03	-.00	-.06
4. Shift work	1.36	0.48			-	.10[a]	-.04	.18[c]	.07	.17[c]	.01	-.09	-.03	.01	.08	.13[b]	.04	.03	.05
5. Smoking (pack-years)	5.4	10.3				-	.24[c]	-.06	.16[c]	-.02	.02	.15[b]	.03	.02	-.05	.09[a]	.08	.05	.04
6. Alcohol consumption (g/week)	179	199					-	-.01	.22[c]	-.04	.06	.10[a]	.00	.11[a]	-.01	.17[c]	.11[a]	.06	.06
7. Physical activity (MET-hours)	14.2	15.5						-	-.13[b]	.20[c]	-.07	-.07	-.06	.10[a]	.15[b]	.00	-.05	-.04	-.01
8. Body mass index (kg/m^2)	25.4	3.3							-	-.01	-.02	.19[c]	-.03	.08	-.02	.08	.07	.03	.04
9. Social support	10.8	5								-	-20[c]	-.09[a]	.01	.03	.05	.01	-.11[a]	-.13[b]	-.07
10. Minor psychiatric morbidity	0.2	0.4									-	.09[a]	.08	.04	-.05	.08	.23[c]	.19[c]	.29[c]
11. Somatic disease	1.47	0.5										-	.09	.10[a]	-.05	.10[a]	.12[b]	.15[b]	.14[b]
12. Depression and other psychiatric diseases	1.1	0.3											-	.03	-.00	.09	.13[b]	.13[b]	.15[c]
13. Impulsivity	7.8	2.7												-	-.10[a]	.10[a]	.12[b]	.04	.03
Time 2																			
14. Sleep duration	7.09	0.64													-	-.15[c]	-.19[c]	-.19[c]	-.16[c]
15. Having trouble falling asleep	1.9	1.1														-	.54[c]	.55[c]	.43[c]
16. Waking up several times per night	2.5	1.4															-	.69[c]	.52[c]
17. Having trouble staying asleep	2.2	1.4																-	.54[c]
18. Waking up after the usual amount of sleep and feeling tired and worn out	2.6	1.3																	-

Men, N=520 after listwise deletion. [a]p<0.05, [b]p<0.01, [c]p<0.001.

Table 8.

Means, standard deviations and Pearson correlations among variables in women.

Variable	Mean	SD	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.
Time 1																			
1. Age (years)	42.9	8.4	.02	-.14^{c}	-.16^{c}	.09^{c}	.03^{a}	-.06^{c}	.19^{c}	-.11^{c}	.05^{b}	.23^{c}	.07^{c}	-.08^{c}	-.15^{c}	.10^{c}	.18^{c}	.20^{c}	-.03
2. Marital status	1.8	0.42	-	.04^{a}	.00	-.08^{c}	.01	-.06^{c}	.04^{a}	.12^{c}	-.04^{a}	.01	-.08^{c}	-.02	-.02	-.04^{a}	-.01	-.01	-.03
3. Education	5.4	1.5		-	-.05^{b}	-.15^{c}	.01	-.00	-.13^{c}	.14^{c}	-.00	-.09^{c}	-.05^{b}	-.04^{a}	.04^{a}	-.06^{c}	-.06^{c}	-.05^{b}	.01
4. Shift work	1.58	0.49			-	-.00	-.04^{a}	.05^{b}	.02	.05^{b}	-.02	-.07^{c}	-.03^{a}	.01	.07^{c}	.07^{c}	.02	-.01	.00
5. Smoking (pack-years)	2.3	5.6				-	.16^{c}	-.04^{b}	.06^{b}	-.04^{b}	.01	.08^{c}	.10^{c}	.07^{c}	-.07^{c}	.08^{c}	.01	.01	.05^{b}
6. Alcohol consumption (g/week)	62	79					-	.02	.06^{c}	-.04^{a}	.05^{b}	.02	-.06^{c}	.10^{c}	-.05^{b}	.06^{c}	.06^{b}	.06^{c}	.06^{c}
7. Physical activity (MET-hours)	12.5	12.1						-	-.17^{c}	.07^{c}	-.05^{b}	-.05^{b}	.04^{a}	.03	.02	-.02	-.03	-.03	-.04^{a}
8. Body mass index (kg/m^{2})	24.4	3.9							-	-.02	-.02	.14^{c}	.07^{c}	.02	-.06^{c}	.06^{b}	.09	.09^{c}	.01
9. Social support	12.6	5.2								-	-.12^{c}	-.05^{b}	-.08^{c}	-.03	.03	-.07^{c}	-.05^{b}	-.05^{b}	-.07^{c}
10. Minor psychiatric morbidity	0.2	0.4									-	.09^{c}	.20^{c}	.08^{c}	-.06^{b}	.22^{c}	.18^{c}	.21^{c}	.24^{c}
11. Somatic disease	1.52	0.5										-	.13^{c}	.03	-.09^{c}	.15^{c}	.18^{b}	.18^{c}	.14^{c}
12. Depression and other psychiatric disease	1.1	0.3											-	.06^{c}	-.02	.14^{c}	.12^{c}	.14^{c}	.15^{c}
13. Impulsivity	8.4	3.15												-	.03	.06^{b}	.02	.03	.06^{c}
Time 2																			
14. Sleep duration	7.2	0.71													-	-.18^{c}	-.16^{c}	-.23^{c}	-.18^{c}
15. Having trouble falling asleep	2.0	1.2														-	.51^{c}	.57^{c}	.45^{c}
16. Waking up several times per night	2.7	1.5															-	.68^{c}	.49^{c}
17. Having trouble staying asleep	2.2	1.4																-	.50^{c}
18. Waking up after the usual amount of sleep and feeling tired and worn out	2.7	1.4																	-

N=3808 after listwise deletion. $^{a}p<0.05$, $^{b}p<0.01$, $^{c}p<0.001$.

Table 9.

Hierarchical linear regression analysis of associations between impulsivity at Time 1 and sleep disturbances at Time 2 in men.

Impulsivity at time 1	Sleep duration		Having trouble falling asleep		Waking up several times per night		Having trouble staying asleep		Waking up after the usual amount of sleep and feeling tired and worn out	
	Beta	p	Beta	p	Beta	p	Beta	p	Beta	p
Impulsivity adjusted for age and marital status	-0.09	0.023	0.09	0.034	0.13	0.002	0.05	0.212	0.02	0.601
+ education and shift work	-0.10	0.011	0.09	0.026	0.12	0.003	0.05	0.259	0.02	0.679
+ smoking, alcohol consumption, physical activity and body mass index	-0.12	0.006	0.07	0.099	0.12	0.004	0.03	0.434	0.02	0.734
+ social support, minor psychiatric morbidity	-0.12	0.006	0.07	0.119	0.11	0.006	0.03	0.555	0.00	0.998
+ somatic disease and depression with any other psychiatric disease	-0.12	0.008	0.07	0.092	0.11	0.008	0.03	0.426	-0.01	0.854

After all adjustments: N=535, Adj.R^2=.021 for sleep duration; N=530, Adj.R^2=.062 for having trouble falling asleep; N=529, Adj.R^2=.095 for waking up several times per night; N= 533 Adj.R^2=.068, for having trouble staying asleep; and N=532, Adj.R^2=.106 for waking up after the usual amount of sleep feeling tired and worn out.

Table 10.

Hierarchical linear regression analysis of associations between impulsivity at Time 1 and sleep disturbances at Time 2 in women.

Impulsivity at time 1	Sleep duration		Having trouble falling asleep		Waking up several times per night		Having trouble staying asleep		Waking up after the usual amount of sleep and feeling tired and worn out	
	Beta	p	Beta	p	Beta	p	Beta	p	Beta	p
Impulsivity adjusted for age and marital status	0.00	0.957	0.06	0.000	0.04	0.007	0.05	0.001	0.06	0.000
+ education and shift work	0.01	0.703	0.05	0.000	0.04	0.014	0.05	0.002	0.06	0.000
+ smoking, alcohol consumption, physical activity and body mass index	0.02	0.143	0.05	0.002	0.03	0.052	0.04	0.020	0.06	0.000
+ social support, minor psychiatric morbidity	0.03	0.109	0.03	0.037	0.02	0.209	0.02	0.139	0.04	0.011
+ somatic disease and depression with any other psychiatric disease	0.03	0.080	0.03	0.077	0.01	0.518	0.01	0.482	0.03	0.061

N=4014, Adj.R^2=.032 for sleep duration; N=3977, Adj.R^2=.091 for having trouble falling asleep; N=3980, Adj.R^2=.086 for waking up several times per night; N=3938, Adj.R^2=.104for having trouble staying asleep; N=4002, Adj.R^2=.087 for waking up after the usual amount of sleep feeling tired and worn out.

3.3. Impulsivity and peptic ulcers

Of the 4636 participants (533 men, 4106 women), 89 reported newly diagnosed PUD in the follow-up survey (II). The 2-year incidence for PUD was 1.9%. The mean age was 43.6 (range 19 – 62) years, 55% had college as the basic education and 43% worked in permanent day shift. A total of 9% had smoked 5 pack-years or more, about 4% consumed pure alcohol > 275 g/week, 9% were obese, as indicated by a BMI > 30 kg/m^2, 20% had minor psychiatric morbidity and 7% had a history of physician-diagnosed depression or other psychiatric disorder at baseline.

Table 11 presents the relationships between baseline characteristics and incidence of PUD. Increased risk of PUD was significantly associated with minor psychiatric morbidity and nonsignificantly associated with older age, male gender, higher BMI, smoking and a history of depression or other psychiatric disease. No association was found with education, alcohol consumption or physical activity.

Table 12 shows the results of multiple regression models for the association between impulsivity and incident newly diagnosed PUD. After adjustment for age, gender and education, belonging to the highest impulsivity quartile, compared with belonging to the lowest quartile, was associated with a 2.42- fold increased risk of incident PUD (model 1). Further adjustments for lifestyle factors (model 2) and mental health (model 3) had little effect on this association (Nagelkerke $R^2 = .037$).

Table 11.
Associations between baseline characteristics and incidence of PUD.

Baseline characteristics	N	Odds ratio (95% CI)
Age		
< 35	823	1.00
35 – 45	1885	1.96 (0.95 - 4.06)
> 45	1928	1.92 (0.93 - 3.97)
Gender		
Men	533	1.00
Women	4103	0.69 (0.39 - 1.23)
Education		
Less than college	704	1.00
College, short form	821	0.69 (0.33 - 1.45)
College	2547	0.84 (0.48 - 1.49)
University or high school	512	0.77 (0.34 - 1.76)
Working schedule		
Day work	2006	1.00
Shift work	2611	0.90 (0.59 - 1.37)
Smoking (pack-years)		
0	3101	1.00
0.1-5	702	0.73 (0.37 - 1.44)
>5	644	1.29 (0.74 - 2.26)
Alcohol consumption*		
0-50 g	2216	1.00
51-100 g	1390	1.05 (0.64 - 1.72)
101-275 g	791	1.10 (0.61 - 1.96)
>275 g	191	1.14 (0.40 - 3.20)
Body mass index (kg/m^2)		
<25	2853	1.00
25-30	1306	1.18 (0.74 - 1.90)
>30	409	1.41 (0.71 - 2.79)
Physical activity (MET score)		
Quartile 1 (low)	1038	1.00
Quartile 2	1248	1.21 (0.66 - 2.21)
Quartile 3	1112	1.30 (0.71 - 2.40)
Quartile 4 (high)	1162	0.94 (0.49 - 1.81)

(continued)

Table 11. (continued)	N	Odds Ratio (95% CI)
Minor psychiatric morbidity		
No	3697	1.00
Yes	939	2.25 (1.45 - 3.50)
Diagnosed depression or other psychiatric disorder		
No	4307	1.00
Yes	322	1.15 (0.53 - 2.50)

Respondents with PUD at baseline were excluded from the analysis.
* Pure alcohol in grams consumed during one week.

Table 12.
Odds ratios (95% confidence intervals) of newly diagnosed ulcer disease by levels of baseline characteristics and impulsivity among participants with no diagnosed ulcer disease at baseline.

Characteristics	N	Model 1	Model 2	Model 3
Age				
< 35	760	1.00	1.00	1.00
35 – 45	1730	2.09 (1.01 - 4.36)	2.16 (0.99 - 4.70)	2.18 (1.00 - 4.75)
> 45	1725	1.98 (0.94 - 4.18)	1.84 (0.83 - 4.09)	1.80 (0.81 - 4.01)
Gender				
Men	490	1.00	1.00	1.00
Women	3725	0.56 (0.30 - 1.06)	0.53 (0.26 - 1.06)	0.52 (0.26 - 1.05)
Education				
Less than college	623	1.00	1.00	1.00
College, short form	747	0.70 (0.33 - 1.49)	0.84 (0.38 - 1.83)	0.83 (0.38 - 1.82)
College	2366	0.91 (0.51 - 1.64)	0.94 (0.50 - 1.77)	0.94 (0.50 - 1.77)
University or high school	479	0.63 (0.26 - 1.51)	0.67 (0.27 - 1.69)	0.66 (0.26 - 1.64)
Working schedule				
Day work	1857	1.00	1.00	1.00
Shift work	2358	0.91 (0.58 - 1.44)	0.83 (0.51 - 1.33)	0.83 (0.51 - 1.33)
Smoking (pack-years)				
0	2937		1.00	1.00
0.1-5	670		0.78 (0.40 - 1.56)	0.78 (0.39 - 1.55)
>5	608		1.25 (0.69 - 2.26)	1.24 (0.69 - 2.25)
Alcohol consumption*				
0-50 g	2059		1.00	1.00
51-100 g	1274		1.08 (0.64 - 1.82)	1.08 (0.64 - 1.82)
101-275 g	707		1.08 (0.57 - 2.04)	1.06 (0.56 - 2.00)
>275 g	175		0.88 (0.29 - 2.69)	0.82 (0.27 - 2.53)
Body mass index (kg/m^2)				
<25	2649		1.00	1.00
25-30	1192		0.92 (0.54 - 1.55)	0.94 (0.56 - 1.59)
>30	374		1.39 (0.68 - 2.84)	1.39 (0.68 - 2.84)

(continued)

Table 12. (continued)	N	Model 1	Model 2	Model 3
Physical activity (MET score)				
Quartile 1 (low)	945		1.00	1.00
Quartile 2	1156		0.98 (0.52 - 1.87)	0.99 (0.52 - 1.88)
Quartile 3	1031		1.27 (0.68 - 2.37)	1.32 (0.70 - 2.48)
Quartile 4 (high)	1083		0.88 (0.44 - 1.75)	0.92 (0.46 - 1.82)
Minor psychiatric morbidity				
No	3347			1.00
Yes	868			2.08 (1.29 - 3.35)
Diagnosed depression or other psychiatric disorder				
No	3918			1.00
Yes	297			0.96 (0.43 - 2.16)
Impulsivity				
Low	908	1.00	1.00	1.00
Intermediate	2420	1.54 (0.82 - 2.92)	1.41 (0.74 - 2.69)	1.39 (0.73 - 2.65)
High	887	2.42 (1.21 - 4.82)	2.18 (1.07 - 4.43)	2.06 (1.01 - 4.18)

*Pure alcohol in grams consumed during one week.
Model 1= 87 PUD-incident case (N=4565)
Model 2= 80 PUD-incident case (N=4222)
Model 3= 80 PUD-incident case (N=4215).

3.4. Impulsivity and depression

Of the 4505 participants (530 men, 3975 women), 211 reported newly diagnosed depression in the follow-up survey (III). The 2-year incidence for depression was 4.7%. The mean age was 42.9 (range 19 – 62, SD = 8.4) years, 55% had college as the basic education and 88% of the participants were women. A total of 9% of the participants were heavy drinkers and about 14% were current smokers, 20% had minor psychiatric morbidity at baseline. The mean size of the social network was 12 (SD = 5) persons.

Table 13 presents the relationships between the baseline characteristics and incidence of depression. Increased risk of depression was significantly associated with smoking, heavy drinking and mental distress. A large social network was associated with a lower risk of depression. No association was found with age, sex and education.

Table 13.
Odds ratios (95% confidence intervals) of newly diagnosed depression by baseline characteristics of employees with no history of depression at entry into the study.

Baseline characteristics	N	Odds ratio (95% CI)
Age		
< 35	817	1.00
35 – 45	1838	1.12 (0.75 - 1.68)
> 45	1856	1.20 (0.80 - 1.80)
Gender		
Men	530	1.00
Women	3975	0.76 (0.52 - 1.13)
Education		
Less than college	679	1.00
College, short form	797	1.28 (0.80 - 2.04)
College	2490	0.91 (0.60 - 1.37)
University or high school	484	1.24 (0.73 - 2.10)
Smoking		
Nonsmokers	3718	1.00
Current smokers	615	1.57 (1.10 - 2.24)
Alcohol consumption		
Other drinkers	4076	1.00
Heavy drinkers	385	1.54 (1.01 - 2.36)
Minor psychiatric morbidity		
No mental distress	3658	1.00
Mental distress	847	2.93 (2.20 - 3.91)
Size of social network		
Quartile 1 (low)	1131	1.00
Quartile 2	869	0.74 (0.49 - 1.10)
Quartile 3	1286	0.71 (0.50 - 1.02)
Quartile 4 (high)	1201	0.65 (0.45 - 0.95)

Cut off points for heavy alcohol consumption were 190 g/week for women and 270 g/week for men.

Table 14 presents results regarding the associations between impulsivity and the incidence of depression. After adjustment for age, sex and education (Model 1), the odds of depression were 1.95 (95% CI = 1.28 - 2.97) times higher for the highest quartile of impulsivity than for the lowest quartile. The association remained statistically significant after additional adjustment for smoking, heavy drinking (Model 2), minor psychiatric morbidity and the size of the social network (Model 3, Nagelkerke $R^2 = .051$).

We then repeated the analysis, using GHQ-12 cases rather than medically diagnosed depression as the outcome. Comparison with the previous analysis should indicate the specificity of the association patterns and, hence, the validity of the predictive association between impulsivity and depression. After adjustment for age, sex, education, smoking, heavy drinking, GHQ-12 and the size of social network at baseline, the odds of GHQ-caseness at follow-up were 1.26 (95% CI = 1.02 - 1.56) times higher for the highest quartile of impulsivity than for the lowest quartile. The OR between GHQ-12 caseness and doctor-diagnosed depression was 2.93 (95% CI = 2.20 - 3.91), confirming the link between these conceptually overlapping constructs.

Table 14. Odds ratios (95% confidence intervals) of newly diagnosed depression by levels of impulsivity among participants with no diagnosed depression at baseline.

Characteristics	Model 1	Model 2	Model 3
Age			
< 35	1.00	1.00	1.00
35 – 45	1.14 (0.75 - 1.71)	1.11 (0.73 - 1.69)	1.10 (0.72 - 1.68)
> 45	1.22 (0.81 - 1.84)	1.18 (0.77 - 1.80)	1.11 (0.72 - 1.70)
Gender			
Men	1.00	1.00	1.00
Women	0.76 (0.50 - 1.17)	0.83 (0.53 - 1.30)	0.85 (0.54 - 1.33)
Education			
Less than college	1.00	1.00	1.00
College, short form	1.30 (0.81 - 2.08)	1.25 (0.76 - 2.06)	1.29 (0.78 - 2.14)
College	0.96 (0.63 - 1.46)	1.03 (0.66 - 1.59)	1.06 (0.68 - 1.65)
University or high school	1.23 (0.71 - 2.15)	1.36 (0.76 - 2.43)	1.35 (0.75 - 2.42)
Smoking			
Nonsmokers		1.00	1.00
Current smokers		1.46 (1.01 - 2.12)	1.50 (1.03 - 2.19)
Alcohol consumption			
Other drinkers		1.00	1.00
Heavy drinkers		1.35 (0.86 - 2.11)	1.31 (0.83 - 2.05)
Minor psychiatric morbidity			
No mental distress			1.00
Mental distress			2.62 (1.93 - 3.56)
Size of social network			
Quartile 1 (low)			1.00
Quartile 2			0.82 (0.54 - 1.26)
Quartile 3			0.73 (0.49 - 1.09)
Quartile 4 (high)			0.77 (0.51 - 1.15)
Impulsivity			
Quartile 1 (low)	1.00	1.00	1.00
Quartile 2	0.78 (0.48 - 1.26)	0.82 (0.50 - 1.33)	0.83 (0.51 - 1.35)
Quartile 3	1.51 (1.00 - 2.26)	1.44 (0.94 - 2.19)	1.42 (0.93 - 2.17)
Quartile 4 (high)	1.95 (1.28 - 2.97)	1.82 (1.17 - 2.82)	1.70 (1.09 - 2.65)

Respondents with depression at baseline were excluded from the analysis.
Cut off points for heavy alcohol consumption were 190 g/week for women and 270 g/week for men.

4. DISCUSSION

The present 2-year follow-up study showed that impulsivity was associated with health-related behaviours and physician-diagnosed diseases in a cohort of hospital employees. Impulsivity predicted an increase in smoking in women and increase in drinking in both genders and the likelihood of taking up smoking or becoming a heavy drinker was higher in impulsive individuals than in others (I). Furthermore, higher impulsivity was significantly associated with shorter sleep duration and waking up several times per night in men (IV) but not in women. Finally, impulsivity was predictive of new-onset PUD (II) and an increased likelihood of new-onset depression (III) among employees who were free from these diseases at entry into the study.

4.1. Impulsivity, health behaviour and sleep

The present results on impulsivity, smoking and alcohol consumption are in line with previous studies based on cross-sectional data and smaller sample sizes (Labouvie & McGee 1986; Lipcus et al. 1994; Mulder 2002; Waldeck & Miller 1997) and the results of associations between impulsivity and insomnia support those of previous studies, which in turn were mostly done with children and adolescents (Ireland & Culpin 2006; Owens, Maxim, Nobile, MvGuinn & Msall 2000; Ring et al. 1998). This represents the first time when these associations were shown for large bodies of nonclinical follow-up data.

Gender differences

It is unclear why gender differences regarding impulsivity, health behavior (I) and sleep disturbances exist (IV). For women, there was some evidence suggesting that somatic diseases, depression and comorbid psychiatric conditions may mediate the association between impulsivity and some aspects of insomnia. Firstly, there were significant positive correlations of impulsivity, somatic disease and depression with prevalence of minor

psychiatric morbidity in women. Secondly, the significance level for the association between impulsivity and having trouble in falling asleep and difficulties in waking up after the usual amount of sleep and feeling tired and worn out decreased when the effect of somatic disease, depression and other psychiatric disease was controlled for. A corresponding pattern was not seen in men. Thus, impulsive personality may be more directly associated with insomnia in men while in women some aspects of insomnia may be dependent on general somatic and psychiatric well-being. Voderholzer et al. (2003) suggested that gender differences in insomnia could be related to the prevalence of anxiety and depression, which are more common among women than among men. In addition, HA is correlated with depression (Grucza, Przybeck, Spitznagel & Cloninger 2003), which was associated with insomnia (Saint Hilaire, Straub & Pelissolo 2005), and women tend to score higher on HA than men (Hansenne, Delhez & Cloninger 2005). Furthermore, in women, the significant relationship between impulsivity at baseline and smoking at follow-up became nonsignificant after controlling for smoking at baseline (I). Since the distribution occupations differed between genders, the differences in occupational background may have impacted the results, even though the socioeconomic status in forms of education (I, II, III, IV) and salary (I) were controlled for. For example, the largest occupational group in men was physicians (29%) and in women the largest group was nurses (58%), showing a clear segregation in occupations.

All these results suggest that impulsivity is only one potential factor behind health risk behaviour. Smoking, drinking and insomnia may also cause impulsivity, even though we found that those who became heavy drinkers or took up smoking or had sleep disturbances scored significantly higher in impulsivity.

Potential mechanism linking impulsivity with health behaviour and sleep

It is unclear why impulsivity contributes to health behaviour, such as smoking and alcohol consumption. Cloninger et al. (1991, 1993) reported that trait impulsivity is part of NS, which implies a low threshold for new stimuli in triggering behavioural responses for an impulsive person. Dickman (1990) showed that dysfunctional impulsivity is a type that

causes negative consequences. Compared with other persons, individuals scoring high on dysfunctional impulsivity may think less of the consequences before they act. Such lower thresholds for acting may partially explain our observation of the increased likelihood of smoking and heavy drinking in impulsive individuals. Moreover, there may be personality differences in developing addiction to nicotine and alcohol. Thus, those who score high in impulsivity may not only begin to use addictive substances more easily than others but also continue to use these substances. Moreover, the result that impulsivity predicts sleep disturbances (IV) is also in line with previous findings of the association between 5-HT, insomnia and impulsivity. Impulsivity is related to decreased serotonin levels (Linnoila et al. 1983; Spreux-Varoquaux et al. 2001), and there is also evidence that decreased serotonin could be involved in sleep (Singareddy & Balon 2001; Voderholzer et al. 1998). These previous findings are in accordance with present results and emphasize that the existence of an underlying biological factor may be related to underactivity of serotonin function.

4.2. Impulsivity and the onset of physician-diagnosed PUD and depression

In the present study, impulsivity was predictive of an increased likelihood of newly diagnosed depression among hospital employees who were free from diagnosed PUD (II) and depression (III) at study entry. These relationships could not be explained by other risk factors such as age, education, shift work, smoking, heavy alcohol consumption and psychiatric morbidity. Dropouts did not differ in the prevalence of PUD or in the level of impulsivity at baseline, compared with those who responded in both questionnaires; thus selection bias is an unlikely explanation for these findings. Further analysis using the GHQ as an outcome measure of minor psychiatric morbidity yielded similar findings: the risk of minor psychiatric morbidity was significantly higher among impulsive employees than their nonimpulsive counterparts. The results are in line with previous clinical studies and studies of young adults (Corruble, Benyamina, Bayle, Falissard & Hardy 2003; Corruble et al. 1999; Elovainio et al. 2004).

Few small-scale cross-sectional studies have examined the relationship between impulsivity and PUD. Feldman et al. (1986) reported with male PUD patients and controls that PUD subjects had significantly higher Minnesota Multiphasic Personality Inventory (MMPI) scores for impulsivity. Further study by this research group (Feldman et al. 1992) reported with a small sample of PUD subjects and controls (N = 15) that personality traits including high levels of impulsivity were associated with more labile basal acid secretion rates. The present study is the first large-scale prospective study on the association between impulsivity and PUD. Even though the association between impulsivity and depression has been well documented in children and adolescents with ADHD as well as in small clinical samples of impulsivity and depression, this is apparently the first time these relationships have been reported for a large working population.

Previous studies have found a relationship between depression and PUD (Levenstein, Kaplan & Smith 1997). Levenstein et al. (1996) also reported that depressiveness and life stress were associated with poorer prognosis in PUD. In addition, there is evidence for an association between impulsivity and depression (Steiger et al. 2001; Fergus et al. 2003; Swann et al. 2003), which supports the present results (III). This raises the possibility that increased risk of PUD among impulsive individuals is attributable to the effects of depression on PUD. However, present study shows the results of an association between impulsivity, PUD and depression, suggesting that associations between impulsivity and depression, and impulsivity and PUD, are stronger than the association between depression and PUD, because in the present study the odds for this association did not attain statistical significance (II).

Stress response as the link between impulsivity, PUD, depression, health behaviour and sleep

The mechanisms linking impulsivity with PUD, depression, health behaviour and sleep are unknown, but in all cases stress may play a role. Psychological stress is associated with stress and impaired sleep (Akerstedt 2006), stress and alcohol drinking (Sillaber & Henniger 2004), stress and smoking (Baker, Brandon and Chassin 2005), stress and PUD

(Jones 2006), and stress and depression (Kendler, Thornton and Gardner 2000; Kendler et al. 1999; Caspi et al. 2003). Increased risk-taking among impulsive people is a source of stress and research also reports an association between impulsivity and posttraumatic stress disorder (Kotler et al. 2001). Previous studies suggest that stress may play a role in the development of ulcers. An increase in PUD was found after the Kobe earthquake, (Aoyama, Kinoshita & Fujimoto 1998). Being a prisoner of war results in increased risk for developing PUD (Nice et al. 1996). Concrete life stressors (Levenstein, Kaplan & Smith 1997) and self-perceived stress (Anda et al. 1992) can also predict the incidence of PUD. Helicobacter pylori infection is usually present in PUD and the effect of stress and infection is often considered as additive. However, stress may also interrelate with H. pylori infection as an independent aetiological agent (Lewin & Lewis 1995). In line with this, a recent study showed that poor socioeconomic status, a proxy measure of greater life stress, may increase the risk of PUD independently of H. pylori infection (Rosenstock et al. 2004). The present prospective study suggests that impulsivity may be a risk factor for the development of PUD, and this effect appears to be independent of several other risk factors, such as low level of education, health-risk behaviours and depression.

Environmental factors such as stressful life events involving threat, loss, humiliation and defeat, are associated with the incidence of depression (Brown 1998; Kendler, Karkowski & Prescott 1999; Kessler, 1997; Pine, Cohen, Johnson & Brook 2002). Kendler et al. (1999) showed that stressful life events have a causal relationship with episodes of major depression and Pine et al. (2002) found a relationship between life events in adolescence and the onset of major depression in early adulthood. The diathesis-stress theory of depression (e.g. Kwon & Laurenceau 2002) suggests that people with negative attributional style in stressful situations are at greater risk of depression. Impulsive individuals may also be more prone to run into adverse life events that in turn act as triggers for depression. The link between nature and nurture may partly be explained by temperament (including impulsivity), which has its roots in genetic endowment (Rothbart, Ahadi & Evans 2000), but also affects how individuals adapt to the demands of their environment, e.g. stressful life events. Thus, impulsive and nonadapting temperament style may indirectly increase the vulnerability to depression.

That impulsivity may be a symptom of depression cannot totally be excluded. There is some evidence for an association between impulsivity in suicidality among depressed patients (Fawcett et al. 1987; Mann et al. 1999; Pezawas et al. 2002; Corruble et al. 2003). Corruble et al. (1999) reported that suicide attempters had significantly higher impulsivity scores than nonsuicide attempters both at baseline and after a 4-week period of treatment. Both impulsivity and depression scores decreased in a 4-week period of treatment throughout the sample, suggesting a close association between the two.

Stress and sleep problems were associated in several studies (e.g. see the review by Akerstedt 2006). In particular, the anticipation of high demands or effort during the following day appears to impair sleep. An association between stress and shortened sleep is also a well-known result. Furthermore, heavy alcohol drinking (Sillaber & Henniger 2004), and tobacco smoking (Baker, Brandon & Chassin 2004) are associated with higher levels of stress in some but not in all studies (Siegrist & Rödel 2006). Since stress may lead to difficulties in executive cognitive functions and emotional regulatory skills, causing inability to sustain attention, evaluate consequences and control impulsivity (Bremner 1999; Bremner et al. 1999; Steckler & Holsboer 1999), and since stress was associated with all our outcomes, PUD, depression, health behaviour and sleep, impulsivity may be a correlate of stress, which underlies increased health-related behaviour and diseases. Unfortunately, measurements of stress were not included in this study, thus the possible association between impulsivity and stress could not be controlled in the present study. Further research is needed to examine whether stress may be a factor behind impulsivity, PUD, depression, health behaviour and sleep.

Serotonin underactivity: a potential link between impulsivity, depression and PUD?

A range of other mediating factors may plausibly explain the associations between impulsivity and PUD (II) and depression (III). At the biological level, the monoamine neurotransmitters dopamine and 5-HT are often related to temperament and our study variables (impulsivity, depression and PUD) linked with serotonin underactivity in 5-HT

receptors (Coccaro et al. 1989; Cremniter et al. 1999; Evenden 1999; Linnoila et al. 1983; Malison et al. 1998; Owens & Nemeroff 1998; Spreux- Varoquaux et al. 2001). Furthermore, cortisol reactivity and serotonergic and dopaminergic system activity are other potential explanatory factors, because they are associated with both PUD (Athow et al. 1985; Landeira-Fernandez & Grijalva 2004; Lechin et al. 1990;) and impulsivity (Friedel 2004; Paris et al. 2004; Reist et al. 1996). A common genetic background may also partially underlie the association of impulsivity with depression and PUD. While shared genetic background is a potential explanation for present findings on impulsivity and depression, interactions with the environment as underlying mechanisms should also be considered.

As noted earlier, impulsivity is a subdimension and a strong correlate of NS (Cloninger et al. 1993), a temperament dimension mediated by genetic variability in dopamine transmission (Cloninger 1987b). Indeed, Ekelund et al. (1999) found that the two- and five-repeat alleles of the dopamine D4 receptor gene DRD4, were more common in individuals scoring high on NS scores, and Keltikangas-Järvinen et al. (2003) found an association between NS and DRD4 polymorphism, particularly in those with hostile childhood-rearing environments. On the other hand, Caspi et al. (2002) showed that the association between monoamine oxidase A (MAOA), which metabolizes the neurotransmitter 5-HT) and depression is moderated by the maltreatment of a child. Caspi et al. (2003) further showed that individuals with one or two copies of the short allele of the 5-HT transporter (5-HTT) gene become more easily depressed in responses to environmental stress than individuals homozygous for the long allele. In summation, these findings support the present results and the possibility that underactivity of serotonin functioning could be related to impulsivity, PUD and depression, as an underlying biological factor.

4.3. Strengths and limitations of the study

The specific strengths of this study were large sample size, relatively high response rate both at baseline (74%) and follow-up (81%) and a longitudinal design that allowed adjustment for baseline values (I- IV). This is apparently the first time these relationships

were reported for a large sample of healthy working population at baseline (I, II, III, IV). The sex distribution of this cohort is similar to that in Finnish hospitals in general (STAKES 2004) and KSP impulsivity scale was highly reliable (Crombach's alpha 0.88).

There are also notable limitations in the present studies. As in other survey studies, the response to the questionnaire was voluntary. Bryman (1989) showed that volunteers tend to be individuals with higher academic levels, higher scores in intelligence quantient (IQ) tests, less authoritarian and have a better capacity for adjustment than individuals who do not volunteer. Therefore this factor may be a source of bias limiting the generalization of our results to a wider population. Evenden (1999) suggested in his review that impulsivity is a multidimensional phenomenon and may be made up of several independent factors. Although the KSP assesses the broad dimensions of personality, it covers only a narrow facet of impulsivity. For example, the Eysenck questionnaire for impulsivity (Eysenck, S.G.B. 1993) or BIS-11 (Patton, Stanford & Barratt 1995) were designed to assess the various features of the impulsivity construct (e.g. cognitive, motor and trait). Our brief measure of impulsivity did not allow a more detailed analysis of different impulsivity dimensions and furthermore investigations are needed to study these possible differences. We had no opportunity to investigate the temporal stability of impulsivity, because impulsivity was assessed only at baseline, but there is evidence that impulsivity measured with the KSP is stable over time (Gustavsson et al. 1997). The sample was 90% female, included mainly caucasian people who worked in hospital environments, and most of them were healthcare professionals. Since impulsivity was measured with self-reports, our data are also open to common-method variance bias. However, while common-method variance is a serious source of artificially inflated associations in cross-sectional data, this is less likely to occur in longitudinal data with homogenous populations in terms of the outcome at baseline.

Use of self-reports (instead of a diagnostic instrument) to detect medically diagnosed depression and PUD may have resulted in some misclassification, further reducing the validity of present findings. The sensitivity and specificity of self-reports for PUD (II) and depression (III) may not be as good as for some other diagnoses, such as diabetes and cardiovascular diseases (Haapanen, Miilunpalo, Pasanen, Oja & Vuori 1997; Metzger,

Goldberg, Chastang, Leclerc & Zins 2002). However, there is evidence that self-report depression may be both a valid and reliable measurement compared with Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV) diagnosed depression (Zimmerman et al. 2006). Furthermore, health care employees may be more accurate in reporting diagnoses than those from the general population. Moreover, the participants had regular checkups and other occupational health services offered by their employer. In the present cohort, the mean age was 43 years and the lifetime prevalence of PUD 5.0%. The 2-year incidence was 1.9% and this incidence was lower in younger employees. Although the empirical treatment of dyspepsia with potent antisecretory agents in recent years makes the diagnosis of PUD less frequent and reliable, our findings are well in line with those reported for other cohorts. For example, Ehlin et al. (2003) found a PUD prevalence of 2.3% at 30 years of age and 4.4% at 42 years of age in two British birth cohorts. Rosenstock et al. (2003) reported a PUD prevalence of 2.9% in a population-based sample of Danish adults. Previous studies have also shown associations between PUD and smoking (Martin et al. 1989), high alcohol consumption (Levenstein, Kaplan &, Smith 1997), higher age, male sex and lower social class (Ehlin et al., 2003). Our findings on predictors of newly diagnosed PUD accords with most of these findings, although the ORs did not attain statistical significance. Furthermore, the prevalence of lifetime depression (III) at baseline in the present study was 8.0%, which accords with previous reported figures for doctor-diagnosed depression in primary care patients (men 8.7% and women 13.0%; Wittchen, Höfler & Meister 2001) and also with the 5% 12-month prevalence of major depression obtained from structured diagnostic interviews in a representative sample of an employed population in Finland (Pirkola et al. 2005). Moreover, depression in relation to self-reports of mental distress (GHQ-12 responses) was also assessed (III). Mental distress may serve as a proxy measure for undiagnosed depression and, in this study, strongly predicted the onset of medically diagnosed depression. A subsidiary analysis using GHQ-12 responses as an outcome variable validated the findings of a predictive association between impulsivity and depression. This analysis, when the GHQ is used as an outcome measure of minor psychiatric morbidity, showed a finding similar to that of the main analysis in self-reported medically diagnosed depression, since the risk of minor psychiatric morbidity was

significantly higher among impulsive employees than among nonimpulsive subjects. These results are in accordance with previous studies and suggest that self-reporting can be a reliable and useful way to measure PUD and depression.

Finally, the present results may be useful in relation to screening of larger populations to identify subjects at increased risk of developing unhealthy behaviour and disease. The KSP impulsivity scale is a short and neutral questionnaire instrument that can easily be included in surveys of health risk behaviour, PUD, depression and sleep problems. The information obtained may help both the respondent and occupational health care in planning preventive actions against the excess risk related to high impulsivity.

4.4. Implications for further studies

Our findings raise several issues that deserve further study. First, it is unclear why impulsivity in women but not in men is related to higher use of nicotine (I). The reasons may relate to gender-specific roles, biological differences or some other factors. Further research effort is needed to determine whether the effect of impulsivity on health behaviour is independent of other temperament-related constructs (e.g. Heath et al. 1995; Pomerleau et al. 1992; Sabol et al. 1999). Since our data were based on self-reports and the subjects were caucasian, mainly female, hospital employees, further research with more objective measurements, greater ethnic variability and performed separately for men and women and in other occupational settings is needed.

Second, the insomnia scales (IV) used were based on self-reports, which may also reflect factors other than primary insomnia (van Dongen et al. 2005). Hence, there is a need for further studies in which insomnia is measured by clinical interview and laboratory recordings in addition to self-reports. No data were available for impulsivity at follow-up or insomnia at baseline and thus the temporal order between impulsivity and insomnia could not be confirmed (IV). Further studies with repeated measurements of both constructs are needed to determine the status of impulsivity as a risk factor for insomnia and to confirm that the present study identified the traitlike rather than the reactive aspects of impulsivity.

Third, although a range of possible confounding effects was statistically controlled for in the study on PUD, the possible role of the intake of NSAIDs and H. pylori remains unresolved (II). Since H. pylori is an important risk factor for PUD, it is a potential confounder if it also affects impulsivity, which is not currently known. Thus, more information is needed to confirm the generalizability of these findings and to investigate a larger variety of potential confounding factors that may affect the association between impulsivity and PUD.

Fourth, more information is also needed to determine how the interactions between impulsivity and environmental stressors affect the incidence of depression in nonclinical populations (III). In particular, it would be beneficial to examine the relationship between impulsivity and depression within the context of diathesis-stress theory (i.e. whether the more negative attributions of life events or greater exposure to such events among impulsive individuals explain their increased risk of depression). The follow-up period was relatively short (2 years) and the mean age of the study population was 43 years, a relatively late age of onset for major depressive disorders. If impulsivity is indeed a stable trait and affects depression risk across the life course, then a longer follow-up period with younger participants at baseline may reveal a stronger association between impulsivity and depression, but further research is needed to confirm this (III). An alternative explanation for the present findings is that a shared third factor underlies the association. A shared genetic background for impulsivity and insomnia, PUD and depression (II, III, IV), for example, provides an interesting topic for future studies with genetic data.

5. CONCLUSION

In conclusion, prospective evidence from a large nonclinical population of hospital employees support the possibility that impulsivity increases the tendency towards behaviour involving risk to health (I) and, in men, may be associated with increased risk for insomnia (IV). This study also suggests that impulsivity could be a risk factor for PUD (II) and depression (III).

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