Personality and Social Psychology

The relationship between the Five-Factor Model personality traits and peptic ulcer disease in a large population-based adult sample

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The current study examined the relationship between the Five-Factor Model personality traits and physician-confirmed peptic ulcer disease (PUD) diagnosis in a large population-based adult sample, controlling for the relevant behavioral and sociodemographic factors. Personality traits were assessed by participants themselves and by knowledgeable informants using the NEO Personality Inventory-3 (NEO PI-3). When controlling for age, sex, education, and cigarette smoking, only one of the five NEO PI-3 domain scales – higher Neuroticism – and two facet scales – lower A1: Trust and higher C1: Competence – made a small, yet significant contribution \( p < 0.01 \) to predicting PUD in logistic regression analyses. In the light of these relatively modest associations, our findings imply that it is certain behavior (such as smoking) and sociodemographic variables (such as age, gender, and education) rather than personality traits that are associated with the diagnosis of PUD at a particular point in time. Further prospective studies with a longitudinal design and multiple assessments would be needed to fully understand if the FFM personality traits serve as risk factors for the development of PUD.

Key words: Peptic ulcer disease, Five-Factor Model of personality, Neuroticism.

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INTRODUCTION

In recent years, there has been an increasing amount of literature showing that personality has an important impact on people’s lives (e.g., Roberts, Kuncel, Shiner, Caspi & Goldberg, 2007) and most importantly, on health and health-related behavior (Goodwin & Friedman, 2006; Smith & MacKenzie, 2006; Weston, Hill & Jackson, 2015). In the current paper, we examine the associations between the Five-Factor Model (FFM) personality traits and peptic ulcer disease, which has been related to mood and anxiety disorders in the past, but with controversial outcomes.

Peptic ulcer disease (PUD)

Gastric and duodenal ulcer disease (also known as peptic ulcer disease, PUD), that refers to sores or raw areas in the lining of the stomach or duodenum, is the main disease that affects the digestive system. The most common symptom of PUD is a burning or gnawing epigastric pain in the upper abdomen, which may be accompanied by other dyspeptic symptoms such as bloating, fullness, early satiety, nausea, and heartburn (Malfertheiner, Chan & McColl, 2009). Although the management and treatment of PUD has considerably improved during the past decades, PUD still significantly reduces people’s health-related quality of life and general feeling of well-being (Barkun & Leontiadis, 2010). PUD appears more frequently in people of lower socioeconomic status (Goodwin, Keyes, Stein & Talley, 2009) and of older age (Sonnenberg & Everhart, 1996). The differing prevalence of PUD among men and women is less clear; some studies have shown a higher tendency for women (e.g., Goodwin & Stein, 2002) and others for men (e.g., Salih, Abasiyanik, Bayyurt & Sander, 2007) to have PUD, and in some studies no significant gender differences have been found (Goodwin & Stein, 2003).

Ever since the beginning of 1980s, a Helicobacter pylori infection (\( H. pylori \); Warren & Marshall, 1983; Fennerty, 2005) has been considered as the most important cause of PUD (Yuan, Padol & Hunt, 2006). However, recently, this focus has moved away from \( H. pylori \) as the single principal cause of PUD as the proportion of peptic ulcers that are \( H. pylori \) negative is increasing all around the world (Quan & Talley, 2002). The intake of nonsteroidal anti-inflammatory drugs (NSAIDs) has been identified as the most frequent possible cause of PUD in people who are not infected with \( H. pylori \) (Huang, Sridhar & Hunt, 2002; Yuan & Hunt, 2006). Yet, there is solid evidence that 20–40% of peptic ulcers (usually called ‘idiopathic ulcers’ in the literature) are neither associated with \( H. pylori \) infection nor the use of NSAIDs (Chow & Sung, 2009). While the exact mechanism causing the development of idiopathic ulcers is unknown, it is proposed that in addition to a genetic predisposition, acid hypersecretion, and other diseases, also behavioral factors such as smoking as well as psychosocial factors, such as the experience of psychological stress or depression may play a role in ulcer formation (Jones, 2006; Levenstein, Kaplan & Smith, 1997; Quan & Talley, 2002).

Personality and PUD

Within the framework of the FFM of personality, which proposes Neuroticism, Extraversion, Openness to Experience (Openness), Agreeableness, and Conscientiousness as the main factors of personality differences, higher Neuroticism (at both the trait and
facet levels) has been most frequently associated with the development and occurrence of PUD. Many studies have shown that people with PUD seem to be more anxious (Ghosh & Sharma, 2006; Goodwin & Stein, 2002), more impulsive (Griñó, Puttonen, Elovaario et al., 2006) as well as generally more neurotic (Goodwin & Stein, 2003; Jess, 1994), than people without PUD. Depression, maladjustment, and hostility (all seen as facets of Neuroticism, according to the FFM) have also been found to be prospectively associated with peptic ulcer development (Levenstein et al., 1997).

Whereas the relationship between Neuroticism and PUD has been quite well studied, the other Big Five personality traits have received much less attention. In one of the very few such studies, the association between the Big Five personality traits and PUD was examined in a representative household survey of the United States adult population (Goodwin & Stein, 2003). The results showed that none of the other four personality traits (that is, except for Neuroticism) was associated with significantly increased odds of self-reported PUD. Yet, the Big Five personality traits in the abovementioned study were measured by a relatively short personality inventory (i.e., The Midlife Development Inventory Personality Scales; Lachman & Weaver, 1997), the psychometric properties and convergent validity of which have not been fully demonstrated. Another limitation of the abovementioned study (Goodwin & Stein, 2003) is that PUD diagnoses were obtained through lay-administered telephone interviews and were not confirmed by physicians or medical records. Therefore, the findings still remain somewhat inconclusive and need to be replicated in further studies.

The aim of the present study

To overcome the lack of previous empirical research, the current study examined the associations between FFM personality traits and self-reported PUD diagnosis in a large population-based adult sample, while also controlling for the relevant behavioral and sociodemographic factors such as gender, age, socioeconomic status, intake of NSAIDs, and smoking. Due to the relative lack of research on the associations between the FFM personality traits and PUD, the study is largely exploratory in its nature.

Our study went beyond earlier research in several important aspects. First, we examined the relationship between personality and PUD, not only at the broad domain level, but also at the more specific facet level of personality. Second, in addition to self-reports of personality, we employed informant-reports of personality by knowledgeable others to minimize the common method bias (i.e., response tendencies within raters) and thereby increase both the reliability and validity of our findings (see Chang, Connelly & Geeza, 2012 for a review). Third, when examining the associations between the FFM personality traits and PUD, we controlled for the relevant sociodemographic and behavioral factors, including the regular intake of NSAIDs, which, to our knowledge, has not been done in earlier research. Fourth, we combined participants' self-reports of PUD with objective medical records to increase the reliability of their diagnoses. Although earlier studies have shown that self-ratings of health can be reliably used to assess people’s actual health status (e.g., Idler, Hudson & Leventhal, 1999), there is also some evidence showing that self-reported health data are also subject to different biases (e.g., Bauldoff, 2011). Thus, all PUD diagnoses in our study were confirmed by review of participant’s medical records or by the relevant medical data from the national health insurance fund. Finally, we used a large-scale adult sample, which allowed us to generalize our findings to the general population.

METHOD

Participants

Participants for the present study came from the Estonian Biobank cohort (approximately 52,000 individuals) that is a volunteer-based sample of the Estonian resident adult population (see Leitsalu, Häller, Esko et al., 2014, for the cohort profile). The participants were recruited randomly by general practitioners (GPs), physicians, or other medical personnel in private practices and hospitals or in the recruitment offices of the Estonian Genome Centre of the University of Tartu (EGCUT). Each participant signed an informed consent form (available at www.biobank.ee) and the GPs or physicians performed a standardized health examination of the participants. Participants also gave blood samples as well as completed a Computer Assisted Personal Interview (CAPI) on health-related topics such as lifestyle, diet and clinical diagnoses described in WHO ICD-10 (Leitsalu et al., 2014). A part of the Estonian Biobank cohort has been followed up longitudinally but the data we are reporting in this paper is cross-sectional in nature.

Our sample for the current study includes 2,663 people (1,453 women, 54.6%), with a mean age of 46.3 years (SD = 17.3, ranging from 18 to 90 years) who joined the Estonian Biobank cohort during the years of 2002–2012 and who also volunteered to fill out a personality inventory. About 40% of the respondents (n = 1,040) had higher education. In later analyses, we use education as a proxy to estimate respondents’ socioeconomic status. For 2,591 respondents, we also had personality data provided by knowledgeable others. On average, the informants had known the targets for 23 years. The mean age of informants (1,845 women, 69.3%) was 42.4 (SD = 16.1) years.

Measures

Personality: The Estonian version of the NEO Personality Inventory-3 (NEO PI-3; McCrae, Costa & Martin, 2005), which is a slightly modified version of the Revised NEO Personality Inventory (NEO PI-R; Costa & McCrae, 1992) was used to assess personality. Like the original NEO PI-R, the NEO PI-3 has 240 items that measure 30 personality facets, which are grouped into the five FFM domains, such that each domain score is a composite of six facet scores. The NEO PI-R/NEO PI-3 has excellent psychometric properties in a wide range of countries (De Fruyt, De Bolle, McCrae et al., 2009) including Estonia. In the current study, the Cronbach alphas of the five domain personality scales ranged from .79 (Agreeableness) to .93 (Extraversion) in self-ratings and from .89 (Openness) to .94 (Conscientiousness) in informant-ratings of personality.

Participants completed the self-report form and informants the observer-report form of the NEO PI-3; the mean scores of the domain and facet scales are shown in Supporting Online Material (Table S1). In accord with other findings (Connolly, Kavanagh & Viswesvaran, 2007), the correlations between the respective scores based on self- and informant-ratings were 0.53, 0.66, 0.62, 0.48, and 0.53 for Neuroticism, Extraversion, Openness, Agreeableness, and Conscientiousness, respectively, and ranged from 0.39 to 0.63 (median = 0.46) for the 30 facets (all correlations significant at p < 0.001, please see Table S1 in Supporting Online Material). For all subsequent analyses, the mean score of self- and informant-ratings across the five domain and 30 facet scales was used to minimize the common method bias due to individual response biases. As argued by Chang and colleagues (2012, p. 423), “When personality ratings are averaged across multiple raters, the variance in ratings shared across raters (i.e., trait factors) increases, whereas the
variance idiiosyncratic to individual raters (i.e., method factors) declines.” Furthermore, we also calculated the self-other profile agreement at the level of individual participants (see Borkenau & Zaltauskas, 2009) based on the 30 NEO PI-3 facet scales in order to examine whether there are any differences in self-other agreement between people with and without PUD. The average self-other profile agreement was \( r = 0.61 \) (SD = 0.23) and a simple t-test showed that the two groups \((n=2,596)\) were not in the EHIF due to the fact that the diagnosis was made before 2003), or both types of peptic ulcers \((n = 14)\). In our current research we consider duodenal and gastric ulcer patients together as peptic ulcer patients.

During the CAPI, participants’ self-reports were combined with objective medical records to increase the reliability of their diagnoses. Reliability analyses of patients diagnoses showed that for 29% of respondents \((n = 59)\), there were medical records confirming their PUD diagnoses; 57% of respondents claimed that they have PUD but had no medical documents to support their claim (yet, the doctor conducting the interview considered the evidence provided sufficient to establish a PUD diagnosis), whereas in 14% of cases the reliability of diagnosis remained uncertain. In order to further analyse the reliability of the participants’ diagnoses, the participants’ data were linked with relevant medical data in the Estonian Health Insurance Fund (EHIF) which covers the costs of health services required by the person in case of illness. For 78 respondents out of 207 (37.7%) who self-reported PUD during the interview upon joining the Estonian Biobank, their PUD diagnosis was also confirmed by the data in the EHIF, including 45 participants who had no medical records to confirm their diagnosis during the interview. (The data available in the EHIF is only available from 2003 until 2013, so if the person was diagnosed with PUD earlier than 2003 or later than 2013, the data is not available for confirmation).

Taken together, the PUD diagnosis was firmly established for 199 respondents, that is including 78 participants who self-reported PUD \((K25, n = 22; K26, n = 56)\) during the interview at the doctor’s office upon joining the Estonian Biobank and whose diagnoses were also confirmed by the EHIF data; 26 participants who self-reported PUD \((K25, n = 7; K26, n = 19)\) during the interview and who had medical records confirming their diagnoses at the time of the interview (yet their data is not in the EHIF due to the fact that the diagnosis was made before 2003), and 95 participants who did not report having PUD during the interview upon joining the Estonian Biobank but who were diagnosed with either gastric \((K25, n = 42)\) or duodenal ulcer \((K26, n = 53)\) in later years.

NSAIDs. Intake of different drugs was coded according to the Anatomical Therapeutic Chemical (ATC) classification system. The only exception we made was for acetylsalicylic acid, which we also classified as an NSAID although, in the ATC, it belongs to a group of analgesics and antiinfectives. Aspirin has a well-known tendency to cause gastric ulceration (e.g., Hochain, Capet & Colin, 2000) and, in many studies, it is also classified in the group of NSAIDs (Huang et al., 2002). In our sample, 331 (12.4%) respondents claimed that they use NSAIDs on a regular basis.

Cigarette smoking. Sixty-one per cent of the respondents \((n = 1,625)\) claimed that they have never smoked, whereas the rest of the sample was comprised of either former \((n = 471)\) or current smokers \((n = 566)\). In our analyses, we used a dichotomous variable which contrasted those respondents who have never smoked with those who have formerly smoked or are currently smoking.

RESULTS

As mentioned earlier, the total number of people with physician-confirmed PUD diagnosis was 199 which is 7.4% of the total sample in our study. The prevalence of PUD in our study falls within the range of 4 and 15 per cent which, according to the Ministry of Social Affairs of Estonia (www.sm.ee), is the prevalence rate of PUD in Estonia (see also Sung, Kuipers & El-Sera, 2009, for a systematic review of the current global incidence and prevalence of PUD). An analysis of variance showed that people with PUD were older \((M = 56.4, SD = 14.0)\) than those without PUD \((M = 45.5, SD = 17.3)\), \(F = 75.4, p < 0.001\). A chi-square test showed that respondents with PUD were also predominantly men, had a lower level of education, were more likely to be current or former smokers as well as more likely to use NSAIDs on a regular basis compared with people without PUD (see Table 1).

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Personality and PUD

An analysis of variance showed that people with PUD had significantly higher scores of Neuroticism \((F = 8.2, p < 0.004)\) but lower scores of Extraversion \((F = 20.6, p < 0.001)\) and Openness \((F = 37.2, p < 0.001)\) than people without PUD. There were no differences between respondents with and without PUD with regard to the other two personality traits (Agreeableness and Conscientiousness, respectively).

An analysis of variance using PUD as an independent variable and the 30 NEO PI-3 facet scales as dependent variables showed that people with PUD had a significantly \((p < 0.01)\) higher scores on all Extraversion and Openness facet scales except for E1: Warmth and E4: Activity than people without PUD. People with PUD had also lower scores on A1: Trust and higher scores on A5: Modesty than people without PUD (all differences between the two groups being statistically significant at \(p < 0.01\)).

Logistic regression analyses

Next, a binary logistic regression analysis was conducted with the aim of examining the contribution of different sociodemographic and behavioral variables in predicting PUD. The five variables –

<table>
<thead>
<tr>
<th>Gender (%)</th>
<th>No PUD ((n = 2,464))</th>
<th>PUD ((n = 199))</th>
<th>(\chi^2)</th>
<th>(p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>44.1</td>
<td>61.8</td>
<td>23.3</td>
<td>0.000</td>
</tr>
<tr>
<td>Women</td>
<td>55.9</td>
<td>38.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elementary</td>
<td>8.1</td>
<td>15.8</td>
<td>32.2</td>
<td>0.000</td>
</tr>
<tr>
<td>Secondary</td>
<td>24.0</td>
<td>28.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Secondary special</td>
<td>27.4</td>
<td>34.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Higher</td>
<td>40.5</td>
<td>21.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smokes currently or has formerly smoked</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Has never smoked</td>
<td>62.7</td>
<td>40.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intake of NSAIDs (%)</td>
<td>11.9</td>
<td>19.6</td>
<td>10.2</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Notes: PUD = peptic ulcer disease; \(\chi^2\) = Chi-square statistic; SD = standard deviation; NSAIDs = Nonsteroidal anti-inflammatory drugs.
age, sex, education, smoking, and the intake of NSAIDs – explained 13% of the variation (here and below – as indicated by the Nagelkerke pseudo $R^2$) in the dependent variable (i.e., PUD diagnosis). As would be expected, higher age, lower level of education, being a male and a former or a current smoker were all associated with increased odds of PUD ($p < 0.01$). When controlling for other behavioral and sociodemographic variables, the use of NSAIDs was not a significant predictor of PUD in our sample. Therefore, only age, sex, education, and smoking status were included in the models when testing the relationship between personality traits and PUD. The four variables explained 12.8% of the variation in the occurrence of PUD.

The results of the logistic regression analysis, which included both the abovementioned four variables and the five NEO PI-3 personality domain scales showed that none of the personality traits, except for Neuroticism ($\beta = 0.01$, Wald $\chi^2 = 6.88$, $p = 0.009$, OR = 1.01), was any longer significantly associated with PUD (see Table 2). Adding personality traits to the model increased the percentage of the explained variance only by a marginal 1.0%.

At the level of 30 personality facets, only two facet scales – A1: Trust ($\beta = -0.07$, Wald $\chi^2 = 8.21$, $p = 0.004$, OR = 0.93), and C1: Competence ($\beta = 0.11$, Wald $\chi^2 = 7.16$, $p = 0.007$, OR = 1.11) – were significantly associated with PUD diagnosis at $p < 0.01$ then controlling for age, sex, education, and smoking status. When adding all 30 facets to the model (in addition to the four sociodemographic variables), the percentage of explained variance increased by 3.8% (i.e., Nagelkerke pseudo $R^2 = 16.6$%).

**DISCUSSION**

The aim of the present study was to examine the associations between the FFM personality traits and PUD diagnosis. Our study has several important strengths, including the use of a large population-based adult sample and the fact that personality traits were assessed not only by participants themselves but also by knowledgeable informants. Most importantly, though, all PUD diagnoses in our study were confirmed by review of participant’s medical records or by the relevant medical data from the national health insurance fund. These factors strongly support the reliability and validity of our findings.

Overall, our results add evidence for previous findings (e.g., Saliah et al., 2007; Sonnenberg & Everhart, 1996) indicating that PUD occurs more frequently in men, in older people, and in people with lower education (or with lower socioeconomic status). The results of the current study also showed that people with a PUD diagnosis in our sample were more likely to be current or former smokers. Goodwin and colleagues (2009) argue that there are several explanations for the link between smoking and PUD – for example, smoking reduces the amount of bicarbonate in the duodenum, which causes problems in the neutralization of acid. Furthermore, smoking decreases the gastroduodenal mucosal synthesis of prostaglandin, which is an important factor in the regulation of mucosal defence, including the stimulation of duodenal mucosal bicarbonate secretion. As for the intake of NSAIDs, which has been found to be a significant independent risk factors for the occurrence of PUD in previous research (e.g., Chou & Sung, 2009; Huang et al., 2002), it was no longer a significant predictor of PUD when other behavioral and sociodemographic variables were controlled for in the logistic regression analysis. As a result, only age, sex, education, and smoking status were included in the models when testing the relationship between personality traits and PUD.

When controlling for age, sex, education, and cigarette smoking, only one of the five NEO PI-3 domain scales – Neuroticism – made a significant contribution ($p < 0.01$) to predicting PUD in logistic regression analyses. Thus, broadly speaking, the results of our study support the earlier findings of Goodwin and Stein (2003) who found an independent association between Neuroticism and self-reported PUD among adults in the general population. A reason for the effect of Neuroticism on diagnosis of PUD might be that experiencing stress and negative emotions is related to increased secretion of acids in the digestive system, which could lead to gastric or duodenal ulcers. Furthermore, there are studies showing that stress can cause peptic ulcer formation in the absence of *H. pylori* infection (Fink, 2011). An alternative explanation, however, comes from Jones (2006, p. 407), who argues that “at present, there is no definitive study proving a causal relationship between psychological stress and the development of ulcer disease.” In other words, as long as there are no methodologically sound studies that have examined the relationship between stress, autonomic activity, and gastric acid secretion, it is also possible that people higher in Neuroticism are simply more perceptive and attentive to the symptoms of PUD, and, as a result, seek advice from doctors more frequently than people who score lower in Neuroticism. Finally, it cannot be completely ruled out that persistent gastrointestinal and digestive problems associated with PUD are in fact behind the elevated level of Neuroticism (Jess, 1994; Jess & Eldrup, 1994).

It is also noteworthy that none of the Neuroticism facet scales measuring anxiety (N1), hostility (N2), depression (N3), or impulsiveness (N5), that all have been identified as putative risk factors for PUD in previous research, has a significant contribution to the prediction of PUD. However, the results are consistent with the findings of other studies, in which only one area from the facet scales of the personality domain Neuroticism is associated with the development of PUD (see for example, A1: Trust; B1: Temperance; C1: Competence).

**Table 2.** The results of the logistic regression analysis of examining the contribution of the NEO PI-3 domain scales (the mean score of self and informant ratings) in predicting PUD when controlling for age, sex, education, and smoking status

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\beta$</th>
<th>Wald</th>
<th>df</th>
<th>$p$</th>
<th>OR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sociodemographics</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.038</td>
<td>54.00</td>
<td>1</td>
<td>.000</td>
<td>1.039</td>
</tr>
<tr>
<td>Sex (male = 1; female = 0)</td>
<td>.518</td>
<td>8.54</td>
<td>1</td>
<td>.003</td>
<td>0.596</td>
</tr>
<tr>
<td>Education</td>
<td>12.46</td>
<td>3</td>
<td>.006</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking status</td>
<td>.780</td>
<td>22.70</td>
<td>1</td>
<td>.000</td>
<td>0.459</td>
</tr>
<tr>
<td>or has formerly smoked = 1; has never smoked = 0)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEO PI-3 domain scales</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neuroticism</td>
<td>.012</td>
<td>6.88</td>
<td>1</td>
<td>.009</td>
<td>1.012</td>
</tr>
<tr>
<td>Extraversion</td>
<td>.001</td>
<td>0.03</td>
<td>1</td>
<td>.860</td>
<td>1.001</td>
</tr>
<tr>
<td>Openness</td>
<td>-.007</td>
<td>1.98</td>
<td>1</td>
<td>.160</td>
<td>0.993</td>
</tr>
<tr>
<td>Agreeableness</td>
<td>-.003</td>
<td>0.39</td>
<td>1</td>
<td>.535</td>
<td>0.997</td>
</tr>
<tr>
<td>Conscientiousness</td>
<td>.005</td>
<td>1.25</td>
<td>1</td>
<td>.263</td>
<td>1.005</td>
</tr>
</tbody>
</table>

Notes: PUD = peptic ulcer disease (coded so that 1 = respondents with PUD diagnosis and 0 = respondents without PUD diagnosis); $\beta$ = partial logistic regression coefficient; Wald = Wald chi-square statistic; df = degree of freedom; OR = odds ratio.
factors for PUD (Ghosh & Sharma, 2006; Goodwin & Stein, 2002; Gränö et al., 2006; Levenstein et al., 2004), were not associated with PUD in our study after controlling for differences in age, sex, education, and cigarette smoking. Thus, it may be, as argued by Suls and Bunde (2005, p. 260) that a general disposition toward negative affectivity “is more important for disease risk than any specific negative affect.”

At the level of facet scales, only two facet scales – A1: Trust and C1: Competence – remained significant at \( p < 0.01 \) when controlling for abovementioned variables in logistic regression analyses. People who score low on A1: Trust tend to be skeptical, cynical, and pessimistic and believe that others are dishonest and cannot be trusted. As we will also argue later, our study design does not allow us to say exactly what the mechanism relating personality traits to PUD is, but we can speculate that, in this specific case, it has something to do with people low in A1: Trust being negatively predisposed towards social interaction, including health care relationships, which may mediate important health-related behaviors and outcomes (Hall, Camacho, Dugan & Balkrishnan, 2002). In other words, it is possible that high skepticism about the effectiveness of health-related behavior and general distrust of people (including physicians) increases risk of PUD by decreasing knowledge about its risk factors and about the availability of effective treatments for PUD.

We also found that higher C1: Competence was significantly associated with PUD diagnosis. Previous research has shown that conscientious people live longer and are less likely to be engaged in health-risk behaviors, such as eating unhealthy food, smoking, and excessive use of alcohol (Bogg & Roberts, 2004). However, as argued by Friedman and colleagues (2014), the causal links between conscientiousness and health are rather complex and not yet fully understood. Furthermore, different facet scales of Conscientiousness have been found to relate differently to external criteria, both at the level of individuals and national cultures (Möttus, Allik & Realo, 2010). At the cultural level, for instance, other-ratings of C1: Competence were significantly positively related to both prevalence of tobacco use and obesity among adults aged more than 15 years. Thus, although at first sight, the positive relationship between C1: Competence and PUD diagnosis seems counterintuitive, there might be mechanisms behind this association which are not yet fully known and deserve future investigation.

Limitations of the current study

Despite the strengths of the current study, there are also a number of limitations that should be considered when interpreting the results of our study. First, we had no data on the presence of \( H. \) pylori infection in our respondents, the causative role of which in PUD has been confirmed by a number of studies (Huang et al., 2002). It may be that personality traits are associated with PUD primarily or more strongly in those people who are not infected with \( H. \) pylori.

Second, we also did not have data on the level of psychological or environmental stress in our respondents’ everyday lives, which is often believed to play a role in the development of PUD (see Fink, 2011; Jones, 2006, for controversial findings in the literature). Further studies should examine whether the association between Neuroticism and PUD remains significant also after controlling for differences in everyday stress.

Third, since our study was cross-sectional in nature, we cannot say anything about whether Neuroticism (or other personality trait) predicts the onset of PUD (cf Weston et al., 2015). Thus, further prospective studies with a longitudinal design and multiple assessments would be needed to fully understand if the FFM personality traits are associated with the risk of developing PUD (as well as other health problems). As recently argued by Friedman and Kern (2014, p. 735), “the field is ready for longitudinal studies of mediators and moderators, and for intervention studies of how, when, and why changes in individual character affect health and well-being.”

CONCLUSIONS

Although there is an increasing number of studies which show that personality traits are meaningfully related to health and health-related behavior (e.g., Goodwin & Friedman, 2006; Smith & Mackenzie, 2006; Weston et al., 2015), our results show that the associations between the FFM personality traits and the diagnosis of PUD are relatively modest when controlling for relevant behavioral and sociodemographic factors. Even though Neuroticism and two of the NEO PI-3 facet scales (A1: Trust and C1: Competence) made a small significant contribution (\( p < 0.01 \)) to predicting PUD in logistic regression analyses, our findings imply that it is certain behavior (such as smoking) and sociodemographic variables (such as age, gender, and education) rather than personality traits that are associated with the diagnosis of PUD at a particular point in time.

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NOTES

1 We conducted the logistic regression analyses also separately for self- and informant-ratings of personality. The results – which are shown in Supporting Online Material (Tables S2 and S3, respectively) – were all highly similar regardless the type of personality ratings (i.e., self-, informant-, or the mean score of self- and informant-ratings) was used. Therefore, for brevity’s sake, only the results using the mean scores of the self- and informant-ratings of personality are reported throughout the paper.

2 When logistic regression analyses were conducted separately for self- and informant-ratings of personality at the level of facet scales, only self-rated C1: Competence was significantly associated with PUD at \( p < .01 \) then controlling for age, gender, education, and smoking status (\( \beta = 0.10, \text{Wald} \chi^2 = 9.69, p = 0.002, \text{OR} = 1.10 \)).
REFERENCES


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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article at the publisher’s web site:

**Table S1.** Mean scores and standard deviations as well as the correlations of the self and informant ratings of the NEO PI-3 domain and facet scales.

**Table S2.** The results of the logistic regression analysis of examining the contribution of the NEO PI-3 domain scales (self ratings) in predicting PUD when controlling for age, sex, education, and smoking status

**Table S3.** The results of the logistic regression analysis of examining the contribution of the NEO PI-3 domain scales (informant ratings) in predicting PUD when controlling for age, sex, and smoking status