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Intelligence and neuroticism in relation to depression and psychological distress: Evidence from two large population cohorts



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ABSTRACT

Background: Neuroticism is a risk factor for selected mental and physical illnesses and is inversely associated with intelligence. Intelligence appears to interact with neuroticism and mitigate its detrimental effects on physical health and mortality. However, the inter-relationships of neuroticism and intelligence for major depressive disorder (MDD) and psychological distress has not been well examined.

Methods: Associations and interactions between neuroticism and general intelligence (*g*) on MDD, self-reported depression, and psychological distress were examined in two population-based cohorts: Generation Scotland: Scottish Family Health Study (GS:SFHS, *n* = 19,200) and UK Biobank (*n* = 90,529). The Eysenck Personality Scale Short Form-Revised measured neuroticism and *g* was extracted from multiple cognitive ability tests in each cohort. Family structure was adjusted for in GS:SFHS.

Results: Neuroticism was strongly associated with increased risk for depression and higher psychological distress in both samples. Although intelligence conferred no consistent independent effects on depression, it did increase the risk for depression across samples once neuroticism was adjusted for. Results suggest that higher intelligence may ameliorate the association between neuroticism and self-reported depression although no significant interaction was found for clinical MDD. Intelligence was inversely associated with psychological distress across cohorts. A small interaction was found across samples such that lower psychological distress associates with higher intelligence and lower neuroticism, although effect sizes were small.

Conclusions: From two large cohort studies, our findings suggest intelligence acts a protective factor in mitigating the effects of neuroticism on psychological distress. Intelligence does not confer protection against diagnosis of depression in those high in neuroticism.

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1. Introduction

Major depressive disorder (MDD) is a leading cause of disease burden worldwide [1]. Although MDD aetiology remains elusive, a

large proportion of its genetic covariance is attributable to neuroticism [2,3], suggesting a causal relationship. Neuroticism is a partially-heritable personality trait representing high emotionality and stress sensitivity [4], which correlates highly with MDD [5]. Cross-sectional studies suggest a strong positive association between neuroticism and MDD [6–8], whilst higher neuroticism prospectively associates with depression longitudinally [2,9–12], even when controlling for overlapping criteria [13–15] and

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demographics [16,17]. Whilst the public health impacts of neuroticism are wide-ranging (for a comprehensive review see Lahey [18]), neuroticism may be an indirect measure of later MDD risk, rather than the causative risk factor itself. Whereas MDD is often recurrent [19], neuroticism is a stable trait [20] suggesting that their correlation is unlikely to be substantially attributable to an effect of MDD on neuroticism.

General intelligence (*g*) is a latent construct theorized to explain the common observation that people who excel in one type of cognitive task tend to excel in others [21]. When reduced to a single factor (*g*) these correlations explain approximately 50% of the covariance between tests. Lower intelligence in early life has been found to be a risk factor for poor physical health [22] and early mortality in adulthood [23,24]. Although research specifically regarding MDD is relatively sparse [25], there is evidence to suggest that *g* is impaired in depression [26,27] with longitudinal studies suggesting lower *g* in childhood or adolescence confers vulnerability to psychopathology in adulthood [28–31].

Psychological distress represents a cluster of emotional symptoms linked to depression [32–34]. Although symptoms of distress are common in population samples [35,36], they indicate only subthreshold mental health problems. With self-report measures of distress [37,38] freely available in epidemiological research, their measurement provides greater detective power to make distinctions between syndrome and subthreshold symptoms. Longitudinal research suggests neuroticism has a strong, direct effect on psychological distress [39]. Low childhood intelligence strongly associates with increased psychological distress in adulthood [28,40], which may precede MDD onset [41]. However, this is not a universal observation, particularly in studies accounting for socioeconomic status (SES).

Intelligence and neuroticism may interact to influence indices of health. A longitudinal study of war veterans [42] found high neuroticism and low cognitive ability were separate risk factors for mortality. Specifically, a 1-standard deviation increase in neuroticism resulted in a 33% increase in mortality; a 1-standard deviation decrease in intelligence associated with a 27% increase in mortality. An interaction (hazards ratio of 0.89) suggested that high neuroticism with low cognitive ability associates with high risk of poor health and reduced lifespan. Furthermore, high cognitive ability moderates the adverse effects of neuroticism on adjustment [43]. Whether similar interactions exist with regard to their effects on depression remains unknown. No investigation has yet examined how intelligence and neuroticism influence risk for MDD and how they may moderate each other's associations in

depression and psychological distress. Such an analysis may serve to clarify the mechanisms underlying MDD.

In this study, two large population-based cohorts were examined – Generation Scotland: Scottish Family Health Study (GS: SFHS) [44,45] and UK Biobank [46,47]. As previous studies suggest strong associations of neuroticism with risk of MDD [2,5], the same effect was hypothesised here. We hypothesised that higher intelligence may reduce MDD risk by mitigating the adverse effects of neuroticism, similarly to the interaction identified for mortality [42]. This reasoning transfers to psychological distress, hypothesising a positive association between neuroticism and psychological distress would be ameliorated by higher intelligence.

2. Method

2.1. GS:SFHS Overview

GS:SFHS is a family and population-based cohort recruited throughout Scotland between 2006 and 2011 [44]. During clinic assessment, participants aged 18–98 ($n = 24,084$) provided clinical, cognitive and biological data. Full details are provided elsewhere [44,45]. The GS:SFHS sample is predominately female (59%), and generally healthier and wealthier than the Scottish population [44]. This study includes 19,200 individuals with complete data of interest. Demographic information from this cohort is provided in Table 1 and within the Supplementary materials.

Study assessments: during clinic assessment, participants were screened for lifetime history of MDD using a structured clinical interview [48]. Diagnosis of MDD follows DSM-IV criteria; if either symptoms of depressive mood or anhedonia are endorsed, a minimum of four further symptoms must also be endorsed. Clinical significance must be endorsed, too (ie., symptoms lasting nearly all day, every day for a minimum of two weeks). This study includes 2481 individuals meeting criteria for lifetime history of MDD (13%), and 16,719 non-MDD cases (87%).

Four cognitive tests measuring intelligence were administered during clinic assessment [44,45]. The Wechsler Digit Symbol Substitution Task [49] measured processing speed. One paragraph from The Wechsler Logical Memory Test I & II [50] measured verbal declarative memory. The Verbal Fluency Test measured executive function [49] using phonemic lists of C, F and L. Vocabulary was measured with The Mill-Hill Vocabulary Test [51], using combined junior and senior synonyms. General intelligence (*g*) was extracted

Table 1
Demographic, clinical, and cognitive characteristics of GS:SFHS and UK Biobank individuals in the current study.

	GS:SFHS			UK Biobank		
	Total ($n = 19,200$)	Control ($n = 16,719$)	Lifetime MDD ($n = 2481$)	Total ($n = 90,529$)	Control ($n = 60,402$)	Lifetime MDD ($n = 30,127$)
Age	47.16 (14.97)	47.23 (15.27)	46.39 (12.89) [*]	56.64 (8.13)	57.15 (8.16)	55.60 (7.98) [*]
Sex (% female)	59	57	72 [*]	52	46	65 [*]
Neuroticism	3.84 (3.16)	3.45 (2.94)	6.45 (3.32) [*]	3.46 (2.86)	2.65 (2.43)	5.09 (2.96) [*]
GHQ score	15.93 (8.81)	14.93 (7.56)	22.70 (12.77) [*]	–	–	–
PHQ score	–	–	–	1.36 (1.91)	0.89 (1.33)	2.30 (2.47) [*]
Wechsler Digit Symbol Substitution Task	72.31 (17.09)	72.45 (17.23)	71.44 (16.06) [*]	–	–	–
Mill-Hill Vocabulary Test	30.06 (4.76)	30.05 (4.75)	30.15 (4.84)	–	–	–
Wechsler Logical Memory Test I & II	31.01 (8.04)	30.99 (8.09)	31.02 (7.68) [*]	–	–	–
Verbal Fluency Test	25.68 (8.10)	25.60 (8.11)	26.21 (8.05) [*]	–	–	–
Reaction time	–	–	–	564.00 (119.87)	564.70 (119.98)	562.58 (119.66) [*]
Visual memory	–	–	–	4.04 (3.21)	4.04 (3.23)	4.04 (3.17)
Verbal-numerical reasoning	–	–	–	6.09 (2.14)	6.07 (2.16)	6.12 (2.11) [*]
SIMD	3903.82 (1851.91)	3957.58 (1832.28)	3541.51 (1941.03) [*]	–	–	–
Townsend Score	–	–	–	–1.37 (2.84)	–1.47 (2.77)	–1.06 (2.94) [*]

GS:SFHS: Generation Scotland: the Scottish Family Health Study; MDD: Major Depressive Disorder; GHQ: General Health Questionnaire; PHQ: Patient Health Questionnaire; SIMD: the Scottish Index of Multiple Deprivation. With the exception of sex, values represent Mean (SD).

^{*} Significantly different from controls at $P < 0.05$.

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