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Research article

Neuroticism related differences in the functional neuroanatomical correlates of multitasking. An fMRI study

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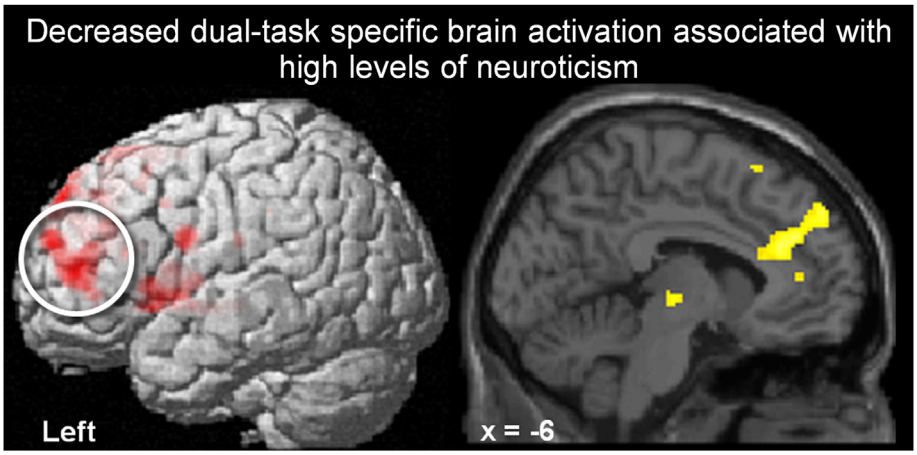
HIGHLIGHTS

- High neurotics show higher behavioural multitasking costs than low neurotics.
- High neurotics show less dual-task specific brain activation than low neurotics.
- Affected brain areas are lateral- and medial-prefrontal cortices.

GRAPHICAL ABSTRACT



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ABSTRACT

It is known that neuroticism impairs cognitive performance mostly in difficult tasks, but not so much in easier tasks. One pervasive situation of this type is multitasking, in which the combination of two simple tasks creates a highly demanding dual-task, and consequently high neurotics show higher dual-task costs than low neurotics. However, the functional neuroanatomical correlates of these additional performance impairments in high neurotics are unknown. To test for this, we assessed brain activity by means of functional magnetic resonance imaging (fMRI) in 17 low and 15 high neurotics while they were performing a demanding dual-task and the less demanding component tasks as single-tasks. Behavioural results showed that performance (response times and error rates) was lower in the dual-task than in the single-tasks (dual-task costs), and that these dual-task costs were significantly higher in high neurotics. Imaging data showed that high neurotics showed less dual-task specific activation in lateral (mainly middle frontal gyrus) and medial prefrontal cortices. We conclude that high levels of neuroticism impair behavioural performance in demanding tasks, and that this impairment is accompanied by reduced activation of the task-associated brain areas.

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1. Introduction

Neuroticism is a personality trait characterized by an inclination towards negative emotional states and high levels of anxiety [1,2], and is widely considered a risk factor for developing psychiatric disorders [1,3,4]. Furthermore, a number of studies have shown that neuroticism impairs cognitive performance [4,5].

H. J. Eysenck [6] proposed that the detrimental effects of neuroticism on cognitive performance could be explained using the well-known finding that arousal and performance are linked by an inverted U-shaped function, i.e. performance is optimal at an intermediate level of arousal and deteriorates if arousal becomes too high or too low [7,8]. H. J. Eysenck and M.W. Eysenck suggested that people with high levels of neuroticism respond more strongly to potential stressors, such as difficult tasks [6,8,9] or uncertain feedback [10]. A key feature of this stronger response is a heightened level of arousal [6,9], which means that high neurotics experience on average higher arousal levels to a potential stressor than low neurotics [6,8]. In the context of experimental tasks, it means that neuroticism would have, at most, only a small effect on easy tasks which are unlikely to be perceived as stressors by low as well as high neurotics [4,6]. However, neuroticism potentially has a detrimental effect on more demanding tasks which are likely to be perceived as potential stressors. In high neurotics, such demanding tasks may increase the arousal beyond the optimum-performance peak of the U-shaped function, effectively impairing their performance. In low neurotics, on the other hand, a more demanding task may increase arousal to a lesser extent so that performance is at or close to its maximum [4,6]. This proposal is supported by previous research showing that differences in cognitive performance between high and low neurotics are more pronounced for hard tasks than for easy tasks [4,5].

One domain where this situation frequently occurs is multitasking, or dual-task performance. Here, the individual tasks are often easy, and so neuroticism has no major effects on their separate performance as single tasks. However, in a multitasking situation, i.e. when two or more tasks have to be performed at the same time, the tasks become much more demanding even if the single-tasks are very easy and basic [11,12]. In line with this, previous research on multitasking showed that high levels of neuroticism negatively affects multitasking performance more than single-task performance [4,12].

While these mechanisms are well understood in terms of cognitive models and behavioural findings, knowledge about their functional neuroanatomical correlates is sparse. More generally, it has been shown that high levels of neuroticism are associated with decreased activation of the fronto-parietal executive control network, e.g. during a working memory N-back task [13]. In addition, in people with high levels of neuroticism gray matter volume in prefrontal areas is decreased [14,15], and their prefrontal cortices are less strongly coupled with other areas by means of functional connectivity [15]. These lateral-prefrontal areas have frequently been associated with executive functions [16], and so it seems likely that high levels of neuroticism should be associated with a deficit in executive functions [17].

The present study aimed at assessing the effects of neuroticism on the functional neuroanatomical correlates of multitasking. We compared the brain activation patterns of participants with low and high levels of neuroticism during single- and dual-task performance. More specifically, we employed the dual-task paradigm of the psychological refractory period, because it has been proven to allow for a high level of experimental control [11]. In our study, this paradigm consisted of two forced-choice response tasks (one auditory-manual, one visual-manual) which were performed either separately as single-tasks or concurrently as dual-tasks. Previous research has shown that when both tasks have to be processed together, certain processing stages, such as the response selection which maps the presented stimuli onto the required button presses, can work only serially, i.e. they constitute a processing bottleneck. This bottleneck results in demands for additional executive control functions to coordinate the processing of the tasks, e.g. by sequencing the task order, by inhibiting the second task which has to wait while the bottleneck processes the first task, and by switching the bottleneck mechanism towards the second task once first task processing has finished [18–21]. These demands all arise due to the presence of a processing bottleneck, so they are dual-task specific and not present during the performance of the single tasks. Based on the arguments made above, we propose that due to the difficulty and nature of the additional processes people with high levels of neuroticism will show performance impairments in the dual-task condition, but these will be less evident for single-task performance. This can be assessed by so-called behavioural dual-task costs, i.e. by the performance difference between performing tasks under dual-task and single-task conditions.

With respect to the functional neuroanatomical correlates, it is of particular interest to identify brain activation specific to dual-task processing, i.e. activation which cannot simply be explained by performance of the single tasks. Studies using the PRP paradigm have reported such dual-task specific activation in lateral-prefrontal cortices [19,22]. Based on previous evidence of functional neuroanatomical correlates of executive functions in highly neurotic participants, we expected high neurotic participants to show decreased dual-task specific activation in lateral prefrontal cortices as compared to low neurotic participants.

2. Methods

2.1. Subjects

To create groups of high and low neurotics (High-N and Low-N, respectively), we screened 700 participants using the 24-item neuroticism scale of the Eysenck Personality Questionnaire [2], of which 32 took part in the MRI study: 15 (6 f) were in the high-N group (mean EPQ score = 18, range = 16–24) and 17 (6 f) in the low-N group (mean EPQ score = 3.89, range = 0–6) [23]. The pre-defined cutoff values (6 and 16) were based on previous research [23–25]. The two groups were closely matched for age (high-N = 23.17, SD 3.88 and low-N = 23.50, SD 3.32) and gender. Each participant gave written informed consent and was paid £20 for 1 h participation. The study was approved by the Department of Life Sciences Ethics committee at Brunel University.

We employed the following exclusion criteria: presence of any past or current major medical, neurological or psychiatric illness that might have diminished cognitive functioning; use of psychoactive medication; consumption of alcohol; consumption of $\geq$8 cups or $\geq$ 900 mg caffeine; scoring over 15 in the Beck depression inventory (BDI) [26]; colour blindness [27].

2.2. Tasks and procedure

Participants lay supine in the MRI scanner holding two MRI compatible response pads, wearing MRI compatible in-ear headphones, and viewing a screen via a mirror system. There were three conditions relevant to the current report presented in an fMRI blocked design.

A trial in the auditory single-task started with the presentation of the target stimulus for 345 ms. Participants heard either the syllables /haha/ (requiring a speeded button-press response with the left middle finger) or /yaya/ (left index finger). Each syllable was randomly drawn from a set of 30 different syllables (15 /haha/, 15 /yaya/) recorded from several different speakers. From start of

stimulus presentation, they had 3075 ms to respond. Afterwards, either an error feedback ('Error') or a fixation cross was presented for 250 ms. A trial in the visual single-task was identical except that the stimulus was either a male face (requiring a speeded button-press response with the right index finger) or a female face (right middle finger). Each face stimulus was randomly drawn from a set of 120 different faces (60 male, 60 female; black-and-white images with an oval mask covering most hair except for the fringe). A trial in the dual-task condition was identical except that both stimuli were presented at the same time at the start of the trial (i.e. before any response), requiring two responses. In half of the blocks, participants had to respond to the auditory stimulus first, and in the other half to the visual stimulus first (for the analyses, both response orders were combined). A trial lasted 3325 ms in total and selection of stimuli was fully random.

Tasks were presented in blocks of 8 trials, lasting 26.6s. Before each block the task of the upcoming block (for the dual-task blocks this included information about the required response order) was presented for 5.9s. Each condition was presented 8 times in an individually randomized order. There were 2 further conditions not relevant to the current report.

2.3. MRI procedure

Imaging was carried out at CUBIC (http://www.cubic.rhul.ac.uk/) using a 3T scanner (Trio, Siemens, Erlangen, Germany) equipped with a 12-channel array head coil. Participants were supine on the scanner bed and cushions were used to reduce head motion. 35 axial slices (192 × 192 mm FOV, 64 × 64 matrix, 3 × 3 mm in-plane resolution, 3 mm thickness, no gap, interleaved slice acquisition) were acquired using a BOLD-sensitive gradient echo EPI sequence (TR 2.5 s, TE 31 ms, 85° flip angle). High-resolution whole-brain images were acquired from each participant using a T1-weighted MPRAGE sequence (TR 1900 ms, TE 3.03 ms, 11° flip angle, 176 slices, 256 × 256 mm FOV, 1 × 1 × 1 mm voxel size). Two functional runs with 364 vols each were acquired, with each volume sampling all 35 slices.

2.4. Data analysis

MRI data were analysed using SPM 12. First, the origin of the structural as well as functional images was manually aligned with the anterior commissure. Next, head motion was corrected (Realign & Unwarp). Anatomical and functional images were normalized to MNI space using unified segmentation. Finally, functional data were spatially smoothed using a Gaussian kernel with a FWHM of 8 mm. Normalization and registration success was validated by visual inspection.

Statistical analysis was based on a voxel-wise least-squares estimation using the general linear model for serially autocorrelated observations [20,28]. Because the current study used a blocked fMRI design, a boxcar function, convolved with a canonical HRF without derivatives, was used to model the BOLD response. A temporal high-pass filter with a cut-off frequency of 1/170 Hz was applied. Individual contrast maps were calculated for all contrasts of interests (see "Results" section), and the second-level analysis was based on independent-sample *t*-tests. All resulting *t*-maps were thresholded at $p < 0.005$ (uncorrected) and only clusters significant with $p < 0.05$ (FWE corrected) were considered. A recent study has shown that cluster level inference using these parameters, i.e. rather strict thresholding for generating the cluster map, is generally accurate [29]. Anatomical locations and Brodmann's areas were determined using the Automated Anantomical Labeling toolbox [20,30,31].

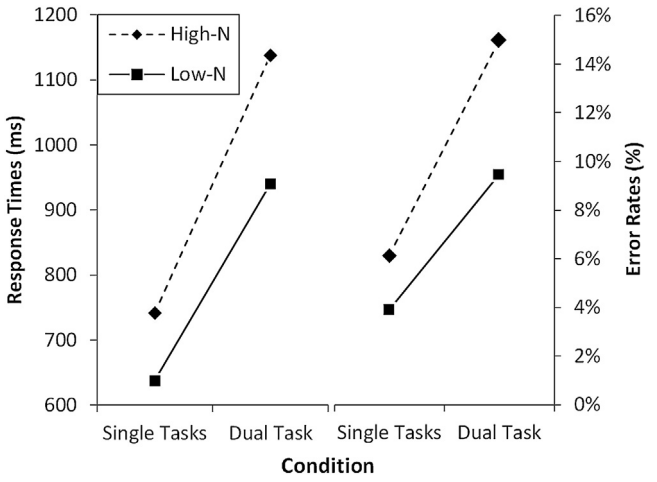

Fig. 1. Behavioural data. Average response times (left panel/axis) and error rates (right panel/axis) for high neurotic (High-N, dashed line) and low neurotic (Low-N, solid line) participants. Single Tasks is the average of both single tasks. Dual Tasks is the average of both dual-task response orders (response times were taken from the first task in the dual-task).

3. Results

3.1. Behavioural results

To test whether the level of neuroticism affected behavioural performance in the dual-task situation, we calculated two 2 × 2-factorial ANOVAs (one for response times (RTs) and one for error rates) with the within-subject factor task (single tasks vs. dual task) and the between-subject factor neuroticism (Low-N vs. High-N). For the RT analysis, we averaged the RTs of the two single tasks, and we also averaged the RTs of the first task (RT1s) of the two dual-task orders. Results (Fig. 1, left panel) showed that RTs in the dual-task were significantly slower than in the single-tasks (main effect task; $F(1,30) = 325.8$. $p < 0.001$), and that RTs were significantly slower for the High-N than for the Low-N group (main effect neuroticism; $F(1, 30) = 15.6$, $p < 0.001$). Importantly, the interaction between task and neuroticism was also significant, indicating that the dual-task costs were higher for the High-N group than the Low-N group ($F(1, 30) = 5.9$, $p < 0.05$). Error rates (Fig. 1, right panel) showed the same pattern, but some effects did not reach statistical significance (main effect task: $F(1, 30) = 65.6$, $p < 0.001$; main effect neuroticism: $F(1,30) = 1.9$, $p = 0.18$; interaction: $F(1,30) = 3.1$, $p = 0.087$).

3.2. Neuroimaging results

To assess dual-task specific activation, we calculated the contrast Dual-Task – Auditory Single-Task – Visual Single Task, i.e. [1–1–1] [32], individually for each participant during first level statistics. If this contrast reveals activation, it is dual-task specific, i.e. it cannot be explained by the summed activation of the single-tasks. During second level statistics, we tested for group differences by comparing the contrast images of the above contrast of the High-N participants with those of the Low-N participants using an independent samples *t*-test. Thus, this contrast tests whether high and low neurotic participants differ in their dual-task specific activation.

Results showed higher activation in lateral and medial prefrontal cortices in low neurotics as compared to high neurotics (Fig. 2, Table 1). In more detail, the cluster in the lateral prefrontal cortex extended mainly along the mid-to-anterior middle frontal gyrus (BA 46), extending into the inferior frontal sulcus/gyrus (BA 47) and superior frontal sulcus/gyrus (BA 10). The cluster in the

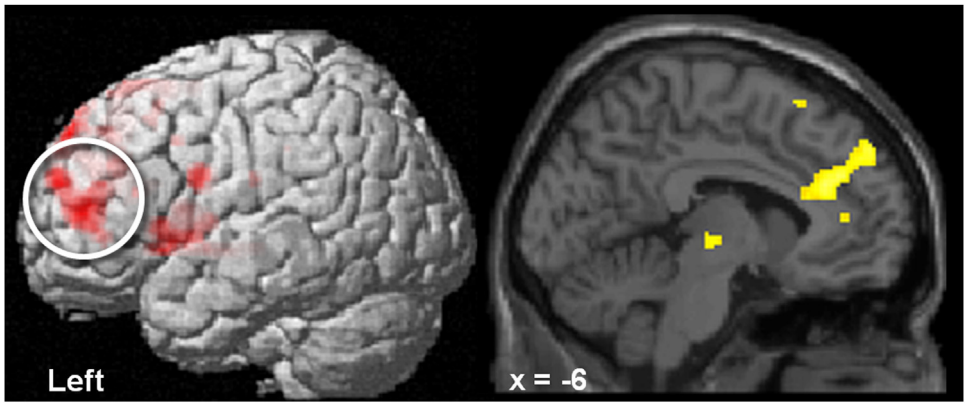

Fig. 2. Imaging data. Higher dual-task specific activation in the left lateral prefrontal cortex (left panel, circled area) and medial prefrontal cortex (right panel) for low as compared to high neurotics as assessed by the contrast $[DT - ST_{Auditory} - ST_{Visual}]_{LowNeurotics} - [DT - ST_{Auditory} - ST_{Visual}]_{HighNeurotics}$. Maps thresholded at $p < 0.005$ (uncorrected), clusters significant at $p < 0.05$ (FWE corrected).

Table 1
Areas exhibiting significantly stronger dual-task specific activation in low neurotics as compared to high neurotics (contrast $[DT - ST_{Auditory} - ST_{Visual}]_{LowNeurotics} - [DT - ST_{Auditory} - ST_{Visual}]_{HighNeurotics}$).

Anatomical area	BA	x, y, z	t/p(uncorr)	Cluster-level p(FWE)	Cluster volume (mm^3)
Cluster 1				0.049	5656
middle frontal gyrus	46	−32, 46, 12	4.52/.00004		
superior frontal gyrus	10	−28, 56, 24	4.17/.00012		
inferior frontal gyrus	46	−44, 44, 6	3.85/.00029		
Cluster 2				0.004	10096
medial superior frontal gyrus	8/9	2, 52, 44	4.45/.00005		
anterior cingulate cortex	32	4, 38, 20	4.25/.00012		
medial superior frontal gyrus	9	−10, 42, 28	3.88/.00027		

Notes. *BA* Brodmann's area. *x, y, z* MNI coordinates of local peaks. *t/p(uncorr)* voxel-level t-value and uncorrected *p*-value of local peaks.

medial prefrontal cortex extended from the medial superior frontal gyrus (BA 8/9) inferiorly into the anterior cingulate cortex (ACC, BA 32).

The reversed contrast (i.e. higher dual-task specific activation in High-N as compared to Low-N) did not reveal any significant voxels in frontal brain regions, even at a lowered voxel-level threshold of $p < 0.05$ (uncorrected).

4. Discussion

Our behavioural findings demonstrated that while all participants, low and high neurotics, were slower in the dual-task condition as compared to the single-task condition, this slowing down was significantly more pronounced in the high neurotics compared with the low neurotics. These higher behavioural dual-task costs were accompanied by lower dual-task specific activation in prefrontal cortices in high as compared to low neurotics. In more detail, neuroticism related group differences showed in the left lateral prefrontal cortex mainly along the middle frontal gyrus, extending into the inferior and superior frontal gyri, as well as medial prefrontal areas reaching from the anterior cingulate gyrus into the medial superior frontal gyrus.

These anatomical areas have been repeatedly reported to be involved in the performance of PRP dual-tasks before [19,22,33]. It has been suggested that these areas are involved in executive functions which resolve interference and coordinate the processing of the tasks at the stage of a processing bottleneck [18,20]. The dual-task activation identified in each group separately is usually taken as indicator of these additional executive functions [19,32]. The observation that high neurotics show lower dual-task specific activation might therefore been taken as evidence that the dual-task specific executive functions are involved to a lesser extent.

The finding that we observed a similar pattern of dual-task specific activation in low and high neurotics, which differed only in the strength of activation, suggests that the underlying mental operations in both groups are qualitatively similar. It is interesting to note that we observed a very similar pattern, i.e. increased behavioural dual-task costs associated with decreased dual-task specific activation, in a recent paper also in normal healthy controls [20]. Although we did not control for neuroticism level in the previous study, it seems likely that the 17 participants are more clustered around the mean of the EPQ than the sample of the present study, in which we formed two extreme groups (total N = 32) based on an initial screening of more than 700 people. Combining these two findings, i.e. same pattern of dual-task related brain areas in high and low neurotics and the same gradual relationship between performance and brain activation in normal controls and neurotics suggests that neuroticism does not alter the neuro-cognitive processing of a dual-task qualitatively, but instead gradually. In other words, our findings suggest that highly neurotic participants multitask rather comparably to low-performing controls.

This interpretation is consistent with the presumed role of these areas for multitasking. We mentioned above that these areas resolve interference and coordinate task processing. It has been argued that lower activity in these areas indicates less efficient mental processing, so that consequently behavioural performance suffers [20,34]. Eysenck's model of neuroticism [6] predicts exactly this: Highly neurotic participants are likely to perceive a demanding task (i.e. dual-task) as considerably more stressful than low neurotic ones so that their arousal levels are increased [4,6,12]. Importantly, this high level of arousal impairs performance, i.e. the mental processes are working less efficiently [4,35].

To summarize the above two paragraphs, we propose that lower dual-task specific activation is associated with higher behavioural

costs. The reason for this association in normal participants is likely to be due to interindividual differences in the amount of mental effort invested into the task [34]. The reason in highly neurotic participants is likely to be due to neuroticism leading to stress related increased arousal, which then in turn limits the efficiency of task processing [4,6,12].

To conclude, this is to our knowledge the first study investigating the effect of neuroticism on the functional neuroanatomical correlates of multitasking. We found that high neurotics showed higher behavioural dual-task costs and at the same time lower dual-task specific brain activation. We interpret the finding as evidence that neuroticism impairs in particular higher cognitive functions, such as executive functions in multitasking, located in lateral and medial prefrontal cortices. The impairment may be caused by an overly increased level of arousal in high neurotics in response to potential stressors such as difficult experimental tasks.

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