The relationship between Type D personality and physical health complaints is mediated by perceived stress and anxiety but not diurnal cortisol secretion

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RUNNING HEAD: Type D, stress and cortisol

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Abstract

Type D personality has been associated with minor health complaints in the general population and dysregulation of basal cortisol secretion in coronary patients. The aims of the present study were to investigate i) whether there is an association between Type D personality and basal cortisol secretion in the general population, and ii) whether subjective measures of stress and anxiety, as well as indices of basal cortisol secretion, mediate the relationship between Type D personality and self-reported physical symptoms in this group. Self-report measures of stress, trait anxiety and physical symptoms were provided by 101 individuals aged 18-45 years. Saliva samples were also provided over two consecutive 'typical' days, to enable indices of the cortisol awakening response and diurnal cortisol profile to be determined. There was a significant relationship between Type D personality and self-reported physical symptoms, which was fully mediated by subjective stress and anxiety. However, there were no significant relationships between Type D personality and the basal cortisol indices. These findings suggest that the association between Type D personality and minor health complaints in the general population can be explained by feelings of stress and anxiety, but a precise biological mechanism for this link is yet to be elucidated.

Keywords

Type D personality; stress; anxiety; physical symptoms; distress; cortisol

Lay summary

Type D personality is characterised by the experience of negative emotion in the context of limited expression of emotional thoughts to others, and is associated with a range of physical and psychological health problems. Here, we sought to investigate whether the relationship between Type D personality and self-reported symptoms of physical ill-health could be

explained by the stress hormone cortisol, and self-reported stress and anxiety. Stress and anxiety, but not cortisol, explained the association between Type D personality and physical symptoms, suggesting that the adverse effect of Type D personality on health is explained by feelings of stress and anxiety, but the biological mechanisms remain uncertain.

Introduction

Type D (distressed) personality is characterised by high levels of both negative affectivity (NA) and social inhibition (SI). Type D personality has been associated with the experience of substantial negative emotions, in the context of limited outward expression of these emotions due to avoidance of social contact and/or repression for fear of negative social appraisal. Consequently, Type D personality can increase vulnerability to chronic distress (Denollet, 2005). However, a number of criticisms of the Type D construct have been put forward (Coyne & de Voogd, 2012; Coyne et al., 2011). For example, is has been suggested that Type D personality is merely a reconceptualization of the well-established traits neuroticism and (inverse) extraversion (De Fruyt & Denollet, 2002; Howard & Hughes, 2012), and it has been further suggested that owing to the large correlation between mood measures and Type D personality, that the construct is merely a proxy for depression (Coyne et al., 2011). Despite these criticisms, it is now well established that Type D personality is a risk factor for adverse physical and psychological health outcomes in patients with chronic illness, most notably, cardiovascular disease (Denollet & Brutsaert, 1998; Versteeg, Spek, Pedersen, & Denollet, 2012). On this basis, both aberrant cardiovascular (Howard & Hughes, 2013; Kelly-Hughes, Wetherell, & Smith, 2014; O'Leary, Howard, Hughes, & James, 2013; Williams, O'Carroll, & O'Connor, 2009) and cortisol (Bibbey, Carroll, Ginty, & Phillips, 2015; Habra, Linden, Anderson, & Weinberg, 2003) responses to stress have been investigated as a potential mechanism by which Type D personality may increase the likelihood of cardiovascular ill-health. Further, Type D personality has been associated with elevated physical symptom reporting in ‘healthy’ individuals from the general population who are free from chronic illness (Mols & Denollet, 2010; Stevenson & Williams, 2014; Williams, Abbott, & Kerr, 2016; Williams & Wingate, 2012). These findings imply that Type D personality may have consequences for minor health problems prior to the onset of chronic

ill-health. However, the physiological mechanisms which may account for a link between Type D personality and minor health complaints in the general population remain uncertain.

Cortisol is the hormonal endpoint of the hypothalamic-pituitary-adrenal (HPA) axis. While acute cortisol increases have been observed in response to stress under a range of conditions, cortisol is known to follow a distinct diurnal profile in healthy adults (Dmitrieva, Almeida, Dmitrieva, Loken, & Pieper, 2013; Wetherell, Lovell, & Smith, 2015). In healthy individuals, cortisol levels typically peak within 30-45 minutes of awaking (this is known as the cortisol awakening response; CAR), followed by a decline throughout the day, reaching a nadir in the middle of the night (known as the diurnal cortisol decline). Dysregulation of this basal cortisol profile has been associated with a range of health problems, including burnout (Kudielka, Bellingrath, & Hellhammer, 2006; Pruessner, Hellhammer, & Kirschbaum, 1999), low energy (Harris et al., 2015), depression (Cohen et al., 2006), suicide attempt (O'Connor, Green, Ferguson, O'Carroll, & O'Connor, 2017) and cardiovascular disease (Kumari, Shipley, Stafford, & Kivimaki, 2011).

In relation to Type D personality, it has been observed that in recent sufferers of acute coronary syndrome, Type D personality is associated with a greater CAR magnitude (Whitehead, Perkins-Porras, Strike, Magid, & Steptoe, 2007). Further, it has also been observed that Type D individuals secrete more cortisol throughout the day, relative to non-Type D individuals, in this same patient group (Molloy, Perkins-Porras, Strike, & Steptoe, 2008). These findings suggest that elevated cortisol secretion may be a mechanism underpinning the relationship between Type D personality and cardiovascular ill-health. However, what remains unclear is whether dysregulation of basal cortisol is a feature of Type D personality more generally, and in a related vein, whether basal cortisol dysregulation might precede the onset of chronic health problems in Type D individuals, potentially mediating the increased risk of adverse health outcomes in this group. It has also been

observed that Type D personality is associated with increased background stress (Polman, Borkoles, & Nicholls, 2010), which provides further weight to the assertion that increases in psychological stress and basal cortisol may drive the relationship between Type D personality and physical health complaints in the general population.

On this basis, the aims of the present study were i) to investigate the association between Type D personality and basal cortisol indices in the general population, and ii) to ascertain whether indices of basal cortisol and self-report measures of background stress and anxiety mediate the relationship between Type D personality and physical symptoms in this group who were free from chronic illness. Predicated by previous work which has established a link between Type D personality and physical symptoms in the general population (Mols & Denollet, 2010; Stevenson & Williams, 2014; Williams et al., 2016; Williams & Wingate, 2012), as well as studies which have observed an association between Type D personality and dysregulated basal cortisol in recent acute coronary syndrome sufferers (Molloy et al., 2008; Whitehead et al., 2007), it was hypothesised that Type D personality would be related to i) physical symptoms, ii) basal cortisol and iii) stress and anxiety. It was further hypothesised that cortisol, stress and anxiety would mediate the relationship between Type D personality and physical symptoms.

Method

Participants

In order to take part in the study, it was required that participants were aged between 18 and 45 years, not currently pregnant or breastfeeding, free of any chronic illnesses and not currently taking any steroid-based or anti-depressant medications. Participants were recruited via poster and email advertisements sent to staff and students of a North East University as well as social media posts. Participants received no financial or other remuneration for taking

part. A total of 117 participants were recruited. However, 16 participants were excluded from the final analyses due to non-adherence with the saliva sampling protocol, missing saliva samples or problems with the provided saliva samples (see Analysis section, below). On this basis, 101 participants were included in the final sample for analysis (64 females, M_{age} = 27.7, SD_{age} = 7.5; see Table 1 for characteristics of the final sample). This sample size was adequate for achieving power of 0.8, when observing a medium mediation effect (Fritz & Mackinnon, 2007).

Materials

DS-14. The DS-14 (Denollet, 2005) was employed to measure Type D personality. This 14-item questionnaire comprises two 7-item subscales: NA (e.g. 'I take a gloomy view of things') and SI (e.g. 'I often feel inhibited in social interactions'). Two positively worded items on the SI subscale (e.g. 'I often talk to strangers') were reverse scored. Reponses to each item were made on a five-point scale ranging between 0 and 4, yielding a total score of between 0 and 28 for each subscale. Both subscales have been found do demonstrate good internal consistency (NA: α=.88, SI: α=.86; Denollet, 2005).

Cohen Hoberman Inventory of Physical Symptoms (CHIPS). The CHIPS (Cohen & Hoberman, 1983) was employed as a measure of physical symptoms. Participants indicated how much bother or distress they had experienced, in the past two weeks, as a result of each of 33 common physical symptoms, e.g. 'back pain', 'headache', 'cold or cough'. Participants responded on a five-point scale ranging from 0 (have not been bothered by the problem) to 4 (problem has been an extreme bother.) Responses on each item were summed to provide a total score ranging between 0 and 132.

State Trait Anxiety Inventory. The State-trait anxiety inventory (STAI; Spielberger, 1983) incorporates two 20-item subscales, measuring a) state anxiety and b) trait anxiety. For

the purposes of the present study, only the trait anxiety items of the STAI were administered. The trait anxiety subscale of the STAI required participants to rate how they 'generally feel' with respect to 20 statements on a four-point scale ('almost never', 'sometimes', 'often', 'almost always'). Positively worded items (e.g. 'I feel pleasant') were reverse scored, so that a score of 4 for an individual item represented the highest level of anxiety. A total score was calculated by summing together the scores for each of the items.

Perceived Stress Scale-10 (PSS). The PSS (Cohen, Kamarck, & Mermelstein, 1983) is a 10-item questionnaire which was used in the present study as a measure of perceived background stress. The single-factor scale asked the participant to report the extent to which they experienced various potentially stressful events in the previous month (e.g. 'how often have you found that you could not cope with all the things that you had to do?'). Participants responded on a five-point scale ranging from 'never' (0) to 'very often' (4). Four positively worded items were reverse scored and the score for each item summed to yield a total score ranging between 0 and 40.

Salivary cortisol collection and assay. Saliva was collected using Salivettes (Sarstedt, Germany). Salivettes are sealed plastic tubes which contain a cotton swab. Participants were instructed to remove and chew on the cotton swab for 3 minutes, before replacing it in the tube and re-sealing. Participants were requested to store their collected samples at room temperature. As soon as possible upon completion of the study protocol, participants returned the Salivettes to the researcher (range = 1-4 days) and the samples were subsequently frozen at -20 degrees Celsius. For analysis, samples were defrosted and saliva extracted from the cotton swabs by centrifuging at 3000 rpm for 15 minutes. Salivary cortisol was measured by enzyme immunoassay (Salimetrics Europe, UK), according to the manufacturer's instructions. The maximum detectable limit of the assay is a cortisol concentration of 82.77

nmol/L. Standards, as well as high and low controls were assayed in duplicate. Inter- and intra-assay coefficients of variation were less than 10%.

Procedure

The study procedure was granted ethical approval by the relevant institutional ethics committee. Informed consent was obtained from all individual participants included in the study. Data collection took place during the Spring/Summer months of May, June and July in the UK.

Researchers met with participants, typically on the university campus, to provide a research pack which comprised a booklet containing participant information, consent forms, comprehensive instructions for salivary cortisol collection and the self-report questionnaires, along with eight Salivettes. In a small number of cases where the participant was known to one of the researchers, this meeting took place in participants' homes. The procedure for self-collection of saliva samples and completion of the self-report questionnaires was also explained verbally and any questions answered, prior to informed consent being obtained. Given that the study did not comprise any objective adherence measures to verify the time of waking or saliva sample collection times, the importance of precise adherence to saliva sampling times and accurate reporting of waking and saliva sampling times was emphasised (Wetherell et al., 2015). The researcher was not present during questionnaire completion nor saliva sampling.

The questionnaires were completed at a time and location that was convenient to the participant. Participants were asked to collect saliva samples as instructed on two consecutive 'typical' days of their choice (i.e. days on which they were due to awake and go to bed at times which were approximately normal for them, and days on which they weren't expecting any atypical stressful or demanding events to occur; no restrictions were placed on whether

these should be weekdays, work days or weekend days), at awakening, 30 minutes post-awakening, 45 minutes post-awakening and immediately before going to bed. Pre-labelled Salivettes were provided, that indicated which sample should be collected in which Salivette. Participants were asked to refrain from eating, drinking (other than water) and brushing their teeth in the 30 minutes preceding each sample collection. For each day of sample collection, participants were provided with a diary in which to record time of awakening and times of each sample collection. Additionally, they were asked to record in writing whether the day had been 'typical' for them.

Analysis

Treatment of cortisol data. If any of the following issues were identified with any single sample, data for the entire set for that day was excluded from the analysis, because in such cases analysis of the CAR and/or diurnal decline would be compromised (as informed by Griefahn & Robens, 2011; Smyth, Clow, Thorn, Hucklebridge, & Evans, 2013; Stalder et al., 2016): i) cortisol levels exceeded the maximum detectable limits of the assay, ii) cortisol levels greater than 2.5 standard deviations from the sample mean for that time point (representing substantial outliers which could be indicative of ill-health, protocol non-adherence or technical errors with the assay procedure), iii) reports that any of the samples were obtained more than 10 minutes earlier or later than the time that they should have been, with respect to the reported time of waking (representing protocol non-adherence), or iv) subjective reports that the sampling day did not represent a typical day for that individual. Ten participants reported being non-adherent to the saliva collection protocol (samples obtained more than 10 minutes either side of the required time) on at least one sampling day, but only two participants reported non-adherence on both days. Data from 16 participants was completely excluded from the analysis, due to at least one of the above issues being identified

on both sampling days. Thus, data from a total of 101 participants was analysed. Of these, 75 participants contributed data from two sampling days to the analysis. For these individuals, data for each time point was averaged across the two days. For 26 participants, data from one of the sampling days was excluded due to at least one of the above issues being identified on one of the sampling days. For these individuals, only the data from the non-contaminated day was used in the analysis. This conservative approach was taken to increase the integrity of the cortisol data.

The CAR area under the curve with respect to ground (AUCg), indicative of the amount of free (unbound) cortisol secreted during the CAR period, and CAR area under the curve with respect to increase (AUCi), representing CAR magnitude, were calculated in accordance with the formulas provided by Pruessner and colleagues (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003) using the wake, wake+30 and wake+45 samples. To assess the diurnal cortisol slope, a regression line was fitted separately for each participant that predicted cortisol values from time since waking, and the beta coefficient recorded as an index of diurnal decline. For this analysis, the cortisol value (dependent variable) was regressed against time (in minutes) since waking at which the sample was collected (independent variable). The 'wake' and 'bed' samples were used for the purpose of this analysis. More negative beta values indicated a steeper diurnal cortisol slope. The 'wake' and 'bed' samples were also considered in the analyses as individual time points of interest.

Statistical analyses. Initially, analyses compared the Type D and non-Type D groups with respect to all self-report and cortisol measures using independent samples t-tests. Participants were classified as Type D if their scores on both the NA and SI subscales of the DS14 were ≥10, as suggested by Denollet (2005).

Subsequently, a continuous Type D personality score (Ferguson et al., 2009) was calculated by determining the product of the NA and SI scores (Stevenson & Williams,

2014). This score was used to determine the continuous relationship between Type D and each of the self-report and cortisol measures, as well as to determine whether each of the PSS, trait anxiety and cortisol measures mediated the relationship between Type D personality and CHIPS scores. The continuous analyses were performed using the PROCESS for SPSS macro, version 2. 16. 1 (Hayes, 2013), using the recommended 5,000 bootstrap resamples. Age and sex were entered into each model as covariates. The bootstrap mediation analyses performed are represented diagrammatically in Figure 1. Firstly, this analysis enabled the determination of the direct relationship between Type D personality and CHIPS scores in the absence of mediating variables (path *c*). The relationship between Type D personality and each of the proposed mediators (path *a*) and between each mediator and the CHIPS score (adjusted for variance explained by Type D personality, path *b*) was subsequently established. In order to ascertain whether PSS scores, trait anxiety or any of the basal cortisol indices fully mediated the relationship between Type D personality and CHIPS scores, two conditions needed to be met: i) the confidence intervals relating to the indirect effect between Type D and CHIPS scores, via the relevant mediator, did not overlap with 0, and ii) the direct effect between Type D and CHIPS scores, when the mediator was included in the model (path *c*') needed to become nonsignificant. If only the first of these conditions were met, it could be concluded that partial mediation had occurred.

INSERT FIGURE 1 ABOUT HERE

Results

Cortisol levels were higher for females at the 'wake+45' time point, t (99) = 3.14, p = 0.002. The CAR AUCi was also greater for females, t (99) = 3.28, p = 0.001. Of the 101 participants included in the analysis, 28 met the criteria for Type D personality. The Type D

and non-Type D groups did not differ with respect to age, t (98) = -0.59, p = 0.56. As expected, the two groups differed with respect to NA, t (99) = -6.26, $p < 0.001$, SI, t (99) = 5.84, $p < 0.001$, and continuous Type D, t (99) = 10.86, $p < 0.001$ scores (see Table 1).

Dichotomous analysis

The Type D group scored significantly higher on trait anxiety, t (99) = 6.18, $p < 0.001$, and PSS-10, t (99) = 3.88, $p < 0.001$, relative to the non-Type D group. The between-group difference on the CHIPS did not reach significance, t (99) = 1.80, p = 0.08. There were no significant between-group differences on any of the cortisol indices (all p values ≥ 0.26; see Table 1).

INSERT TABLE 1 ABOUT HERE

Continuous analysis

Type D personality was a significant predictor of CHIPS scores, B = 0.0610, SE = 0.0192, t = 3.18, p = 0.002 (path *c*). Age was also a significant predictor of CHIPS scores, B = -0.5541, SE = 0.2185, t = -2.54, p = 0.01. Sex did not significantly predict CHIPS scores, B = 3.8296, SE = 3.3091, t = 1.16, p = 0.25.

Mediation of relationship between Type D and CHIPS by PSS, Trait Anxiety and basal cortisol indices

With respect to the relationships between the predictor and mediator variables (path *a*), Type D personality was significantly related to PSS scores and Trait Anxiety, but was not significantly related to any of the cortisol measures. Likewise, PSS scores and Trait Anxiety

were significantly related to CHIPS scores (path *b*), but none of the cortisol indices were significantly related to CHIPS scores (see Table 2).

INSERT TABLE 2 ABOUT HERE

With respect to indirect effects of Type D personality on CHIPS scores, the only significant indirect effects observed (whereby the bootstrapped confidence interval for the indirect effect did not include 0) occurred via the mediators PSS score and Trait Anxiety. For both of these models the direct effect between Type D personality and CHIPS score (path *c'*) became nonsignificant when each of these mediators was considered (i.e. both PSS score and Trait Anxiety fully mediate the relationship between Type D personality and CHIPS score). The indirect effects via all of the cortisol indices failed to reach significance, and the direct effect between Type D personality and CHIPS score (path *c'*) remained significant when each indirect pathway via a cortisol measure was considered (i.e. no cortisol indices mediated the relationship between Type D personality and CHIPS score; see Table 3).

INSERT TABLE 3 ABOUT HERE

Discussion

To the best of our knowledge, this was the first study to investigate basal cortisol as a mechanism underpinning the relationship between Type D personality and self-report physical health complaints in the general population. Firstly, we found evidence to support the previously established relationship (Mols & Denollet, 2010; Stevenson & Williams, 2014; Williams et al., 2016; Williams & Wingate, 2012) between Type D personality and physical health in the general population. Further, while self-reported background stress and anxiety

were found to mediate this relationship, we found no support for a link between Type D personality and indices of the i) CAR or ii) diurnal cortisol profile.

On the basis of the study findings, it appears that perceived background stress and feelings of anxiety can explain the relationship between Type D personality and physical health complaints in the general population. These very interesting findings extend previous work which suggests i) that social support and avoidant coping mediate the relationship between Type D personality and physical health (Williams & Wingate, 2012), and ii) that health behaviours partially mediate this relationship (Williams et al., 2016) in the general population.

Given previous work which supported a link between Type D personality and basal cortisol in recent sufferers of acute coronary syndrome, an objective of the present study was to investigate whether such a relationship could be replicated in 'healthy' individuals free of chronic illness. However, we found no evidence to support a relationship between Type D personality and basal cortisol in the general population, and nor did we find that basal cortisol mediated the relationship between Type D personality and physical health. There are several possible interpretations of this null finding. Firstly, from an allostatic load perspective (McEwen, 2000), it may be the case that it takes several years for the Type D personality to induce major and observable physiological changes, including dysregulation of the HPA axis. Our participants were free of chronic illness and relatively young (less than 45 years of age, with a mean age of 27.7 years). The allostatic load hypothesis purports that repeated stress exposure can, over time, impact upon the basal functioning of the HPA axis, which can, in turn, result in chronic illness (McEwen, 2000). However, in younger individuals, such physiological changes are likely to be subtle (Van Cauter, Leproult, & Kupfer, 1996), and may be more likely to manifest as cortisol hypo- or hyper-responses to acute stress. It is noteworthy that cortisol hyper-responding to cognitive (Habra et al., 2003) and social

(Bibbey et al., 2015) stress in Type D individuals has been demonstrated previously. However, in relatively young individuals who are free of chronic illness, it may well be the case that major, chronic physiological changes induced by Type D personality have not yet manifested. Previous studies which have observed a relationship between Type D personality and i) CAR magnitude (mean age = 56.5 years; Whitehead et al., 2007) and ii) diurnal cortisol secretion (mean age = 59.0 years; Molloy et al., 2008) have focussed on a substantially older sample. Thus, further longitudinal research is needed to investigate whether dysregulated basal cortisol is indeed a mechanism which precedes the onset of chronic cardiovascular illness. Such research should focus on middle aged and older individuals who are more at risk for developing cardiovascular disease, given that we were unable to detect a significant relationship between Type D personality and basal cortisol in our sample of relatively young adults. In this regard, it is noteworthy that ageing is associated with increased cortisol levels (Feller et al., 2014), although such relationships appear to be moderated by a range of lifestyle risk factors (Feller et al., 2014; Lupien et al., 1996) and thus prone to inter-individual variation (Lupien et al., 1996).

There are a number of other explanations for the null relationship between Type D personality and the basal cortisol indices. Of course, it is difficult to rule out the possibility of Type 2 error, or the possibility that the study was underpowered. While our sample size substantially exceeded those of the previous studies which have investigated the relationship between Type D personality and basal cortisol (Molloy et al., 2008; Whitehead et al., 2007), as mentioned above, both of these studies were conducted in chronically ill samples of much older participants. On this basis, for reasons outlined above, under these conditions the likelihood of observing a relationship between Type D personality and basal cortisol are increased. While the present study sample size was adequate to observe medium effects with a power of 0.8, the observed effect sizes for the cortisol analyses were small (Fritz &

Mackinnon, 2007), suggesting that the mediation analyses which considered the basal cortisol variables as a mediator were possibly underpowered.

The finding that feelings of stress and anxiety mediated the relationship between Type D personality and physical symptoms is novel and interesting. It has been established previously that Type D personality is related to perceived background stress (Polman et al., 2010). In this study, it was found that the link between Type D personality and perceived stress could be partially explained by the use of maladaptive avoidance coping strategies (Polman et al., 2010). However, to the best of our knowledge, the present study is the first to observe that the link between Type D personality and physical symptoms can be explained indirectly via perceived stress and anxiety. This finding is noteworthy, because it suggests that as a consequence of the greater feelings of psychological stress which are experienced with increases in Type D personality, individuals are more likely to also experience greater physical health complaints. On this basis, it could be argued that Type D personality can lead to decreases in both psychological and physical wellbeing in the general population, which has consequences for later-life psychological and physical health. However, owing to the cross-sectional design employed here, it is difficult to ascertain whether subjective distress causes physical health problems, or whether the experience of physical symptoms leads to heightened psychological distress. Further, missing from the present study was any investigation of psychobiological or subjective reactivity to stressors. Previous studies have reported that Type D personality is associated with increased cortisol (Bibbey et al., 2015) and blunted cardiovascular (Kelly-Hughes et al., 2014) reactivity to acute stress exposure. In this context, we also know from the extant literature that individuals who experience high levels of background stress may respond more sensitively when exposed to an acute stressor, and in turn, this can be a mechanism of ill-health (McEwen, 1998). Speculatively, as an explanation for the present study findings, it could be that Type D individuals, who

experience greater levels of stress and anxiety, are psychobiologically more reactive to acute stress exposure (in the absence of chronically elevated HPA axis activity), which in turn leads to greater subjective ratings of ill-health. Additionally, the symptom perception hypothesis (Watson & Pennebaker, 1989) may provide a further explanation for the self-report findings. This theory posits that individuals who report high levels of subjective stress and NA are also more likely to notice and report physical symptoms. It is of course difficult to determine from self-report measures, particularly in cross-sectional studies, whether elevated symptom reporting in distressed individuals is reflective of actual health problems, or whether such relationships are indicative of the propensity of more distressed individuals to over-report physical symptoms. Future work could overcome this limitation by measuring more objective indicators of ill-health in the general population, such as frequency of GP visits. Further, longitudinal research in this area should consider the development of major or chronic health conditions as an outcome variable. Such objective measures of health are less contaminated by subjective over-reporting of ill-health. One final limitation is that only limited demographic variables were collected in the present study. A range of other demographic and lifestyle factors such as race and ethnicity, diet, exercise and BMI are known to influence the variables under investigation in the present study (Ross & Bird, 1994; Stachowicz & Lebiedzińska, 2016). It is therefore unknown to what extent these unmeasured indices may have impacted the observed findings.

In the present study, we observed that the relationship between Type D personality and distress from physical symptoms in the general population can be explained by feelings of stress and anxiety. In other words, the increased psychological stress and anxiety which occurs as a consequence of Type D personality can in turn lead to an increase in physical health complaints in otherwise healthy, relatively young adults. While this is a novel and interesting finding, the physiological mechanism which links such feelings of stress and

anxiety to physical symptoms remains uncertain. Previous research has suggested that Type D personality is associated with aberrant cardiovascular (Howard & Hughes, 2013; Kelly-Hughes et al., 2014; O'Leary et al., 2013; Williams et al., 2009) and cortisol (Bibbey et al., 2015; Habra et al., 2003) reactivity to stress in the general population, but no studies to date have investigated whether such psychobiological factors mediate the now well established relationship (Mols & Denollet, 2010; Stevenson & Williams, 2014; Williams et al., 2016; Williams & Wingate, 2012) between Type D personality and physical symptom reporting in this group. This presents a potential avenue for future research in this area.

Declaration of Interest

The authors report no conflicts of interest.

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Table 1

Demographic characteristics and mean (SD) values for all participants, Type D and non-Type D groups on all outcome measures.

	All participants	Type D	Non-type D	*p*
N (% male)	101 (36.6)	28 (42.86)	73 (34.25)	-
Age	27.7 (7.5)	27.0 (7.5)	28.0 (7.5)	0.56
NA	9.62 (5.28)	14.14 (3.20)	7.89 (4.88)	< 0.001
SI	9.07 (5.36)	13.43 (3.51)	7.40 (5.00)	< 0.001
Dimensional Type D score (NA x SI)	93.17 (84.65)	193.46 (87.15)	54.70 (41.14)	< 0.001
CHIPS	18.64 (17.25)	23.57 (17.11)	16.75 (17.05)	0.08
Trait anxiety	18.33 (9.89)	26.71 (8.71)	15.11 (8.34)	< 0.001
PSS-10	15.54 (6.96)	19.61 (5.63)	13.99 (6.81)	< 0.001
Salivary cortisol (nmol/L)				
Wake	9.32 (3.82)	8.79 (3.78)	9.52 (3.84)	0.39
Wake+30	13.28 (5.43)	13.49 (5.11)	13.20 (5.58)	0.81
Wake +45	12.32 (5.28)	12.47 (4.33)	12.26 (5.63)	0.86
Bed	1.86 (2.00)	2.06 (2.74)	1.79 (1.65)	0.55

CAR peak	14.28 (5.29)	14.38 (4.66)	14.24 (5.54)	0.91
CAR AUCg	530.99 (198.11)	528.96 (177.61)	531.77 (206.58)	0.95
CAR AUCi	82.03 (90.14)	98.26 (97.08)	75.81 (87.22)	0.26
Diurnal slope	-0.0079 (0.0045)	-0.0071 (0.0047)	-0.0082 (0.0044)	0.81

Table 2

The relationship between i) Type D personality (path *a*) and ii) CHIPS scores (path *b*), and each mediator.

	Type D personality (path *a*)[1]			CHIPS (path *b*)[2]		
Mediator	B	SE	*p*	B	SE	*p*
PSS	0.0403	0.0068	<0.001	1.0118	0.2713	<0.001
Trait Anxiety	0.0783	0.0084	<0.001	0.8733	0.2163	<0.001
Wake	-0.0051	0.0044	0.25	-0.1944	0.4444	0.66
CAR peak	-0.0031	0.0061	0.60	-0.3358	0.3227	0.30
CAR AUCg	-0.1396	0.2282	0.54	-0.0097	0.0086	0.26
CAR AUCi	0.0687	0.1042	0.51	-0.0218	0.0188	0.25
Diurnal slope	0.0000	0.0000	0.29	265.3452	379.1421	0.48
Bed	-0.0008	0.0024	0.73	0.0733	0.8111	0.93

[1]Adjusted for age and sex

[2]Adjusted for age, sex and Type D personality

Table 3

Mediation by PSS, trait anxiety and salivary cortisol measures on the association between Type D personality and CHIPS scores. The direct effect shows the direct relationship between Type D personality and CHIPS scores via path *c'* when each mediator is included in the model. The indirect effect shows the indirect relationship between Type D personality and CHIPS scores via the mediator (i.e. path *a*b*).

	Direct effect					Indirect effect[1]			
Mediator	B	SE	*p*	LLCI	ULCI	B	SE	LLCI	ULCI
PSS	0.0203	0.0211	0.34	-0.0216	0.0621	0.0407	0.0150	0.0190	0.0786
Trait Anxiety	-0.0073	0.0146	0.76	-0.0562	0.0415	0.0683	0.0243	0.0295	0.1235
Wake	0.0600	0.0194	0.003	0.0215	0.0986	0.0010	0.0033	-0.0031	0.0122
CAR peak	0.0599	0.0192	0.002	0.0218	0.0981	0.0011	0.0033	-0.0019	0.0131
CAR AUCg	0.0596	0.0192	0.002	0.0215	0.0978	0.0014	0.0035	-0.018	0.0148
CAR AUCi	0.0625	0.0192	0.002	0.0244	0.1006	-0.0015	0.0032	-0.0123	0.0022
Diurnal slope	0.0595	0.0194	0.003	0.0211	0.0980	0.0015	0.0035	-0.0022	0.0148
Bed	0.0611	0.0193	0.002	0.0227	0.0994	-0.0001	0.0026	-0.0058	0.0045

[1]The indirect (mediation) effect is significant if the bootstrapped confidence intervals do not include 0.

LLCI = Lower Level Confidence Interval; UCLI = Upper Level Confidence Interval

Figure Captions

Figure 1

Non-mediated (A) and mediated (B) pathways between Type D personality and CHIPS scores. Path *c'* represents the direct effect of Type D personality on CHIPS scores with the mediator included in the model. The indirect effect is the product of path *a* and path *b*. Each mediator was considered in a separate statistical model. All models controlled for age and sex.