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The role of neuroticism and pain in dental anxiety: A twin study

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Abstract

Objectives: Accumulating evidence has revealed that dental anxiety is robustly associated with dental care-related pain and discomfort, but also with the personality trait of neuroticism (i.e. the relatively stable disposition to experience the world as distressing, threatening and unsafe). However, there is a near absence of research on these risk factors in samples for which genetic information is available. With the aim of arriving at a more refined understanding of dental anxiety, this twin cohort study assessed genetic and environmental influences on neuroticism, dental care-related pain and dental anxiety, and the relation between these phenotypes.

Methods: Participants were recruited from the Norwegian Twin Registry, and data collections were carried out in 1992–98 (Time 1) and 2011 (Time 2). Well-validated questionnaires were used to assess the study variables, including Corah's Dental Anxiety Scale, the Numerical Pain Rating Scale, the NEO Personality Inventory Revised (Time 2) and Eysenck's Personality Questionnaire (Time 1). Pearson correlation analysis and generalized estimating equations (GEE) were used to investigate phenotypic associations. Analyses of genetic and environmental influences were performed using Cholesky modelling.

Results: A total of 746 monozygotic (MZ) and 770 dizygotic (DZ) twins in the age group of 50–65 participated in the study. Moderate estimates of heritability for dental anxiety (0.29), treatment-related pain (0.24) and neuroticism (0.45–0.54) were found. Cholesky modelling showed furthermore that neuroticism assessed at Time 1 and Time 2 was related to dental anxiety and pain via both genetic and individual-specific environmental pathways, albeit not very strongly. The particularly high phenotypic correlation observed between dental care-related pain and anxiety (r = .68) was explained by both overlapping genetic and individual-specific environmental influences (the genetic and environmental correlations were .84 and .63 respectively).

Conclusions: The findings provide deeper insight into the aetiology of dental anxiety and confirm that while it is strongly linked to treatment-related pain experiences, this relation is to a considerable degree independent of general negative affectivity/neuroticism.

KEYWORDS
dental anxiety, neuroticism, pain, twins

1 | INTRODUCTION

Dental anxiety and avoidance have been shown to be both common and persistent in the general population, with prevalence estimates for high dental fear ranging from 5% to 10% in recent longitudinal studies.[1,2] Several studies have described a vicious circle of dental anxiety, individual vulnerability and stressful dental treatment experiences that interact over time and produce a rise in anxiety levels and subsequent irregular dental utilization behaviours,[1,3] reduced oral health status[4] and poorer oral health-related quality of life.[5] Given its chronicity and consequences for health and well-being, understanding the aetiology of dental anxiety is imperative and may inform treatment and prevention programmes. In particular, knowledge of the interplay of risk factors, and specifically their underlying genetic and environmental structure, is greatly needed.

Accumulating research has documented that conditioning responses to dental experiences, particularly involving treatment-related pain or fear of pain, may trigger or aggravate dental anxiety.[6] Thus, studies in general population and clinical samples have consistently found substantial correlations between dental anxiety and ratings of pain and discomfort related to dental treatment.[7,8] In a recent review and meta-analysis Lin et al.[6] maintained that dental anxiety has a distinct impact on pain (expected/experienced) through the entire period of dental treatment (i.e. before, during and after), and across different types of dental procedures. It should be emphasized, however, that the possible existence of confounding factors such as genetic susceptibilities make it difficult to establish a clear causal link between dental anxiety and pain. Of significance, a study by Randall et al.[9] showed that both dental fear and fear of pain are about 30% heritable, and that the two phenotypes are genetically related. However, the design of this study did not permit an analysis of environmental influences on the phenotypic association between dental fear and pain, and the participants' pain experiences in dental treatment situations were not assessed. Ray et al.[10] studied dental anxiety in adolescent twins (age range 13–17 years), and showed that heritability estimates were much higher for girls (0.55–0.77) than for boys (0–0.14). The only twin study[11] which has included analyses of environmental influences showed that individual-specific (non-shared) environmental effects were significant and somewhat stronger than genetic effects. So far, however, there is evidently no study of the genetic and environmental relationship between dental anxiety and actual dental care-related pain experiences, which is clearly an essential component of dental anxiety. Moreover, it is not known whether dental anxiety is related to dental care-related pain specifically, or to pain responses generally, such as common musculoskeletal (MS) pain symptoms.

Another important line of research has focused on predisposing temperamental or personality characteristics. The disposition to experience negative affect such as anxiety, depression and anger/irritability is a defining characteristic of the personality trait of neuroticism,[12] and has been shown to be the personality dimension most consistently related to dental anxiety.[3,11] Complicating the picture, people who are higher in neuroticism are also vulnerable to a broad range of somatic and psychological symptoms and disorders,[13] higher levels of comorbidity[14] and chronic pain conditions.[15] Moreover, neuroticism seems to influence pain-modulating states such as pain vigilance and catastrophizing[16] and somatic symptom amplification processes in general.[17] Evidently, the relations between neuroticism and various pain phenomena appear to be rather non-specific. Taken together, existing research indicates that neuroticism is best viewed as a general and probably lasting vulnerability factor,[14] which in this context may possibly explain—fully or partly—the association between dental anxiety and several of its risk factors, including treatment-related pain and fear of pain.

In the present study we sought to extend the existing literature by examining both phenotypic, genetic, and environmental associations between dental anxiety, neuroticism and dental care-related pain in adult twins aged 50–65. More specifically, the objectives of the study were threefold: First, phenotypic associations were investigated, both zero-order correlations and independent effects of neuroticism, pain, and sex on dental anxiety in regression models. In order to examine the specificity of the association between dental care-related anxiety and pain, analyses including non-dental pain, that is MS pain symptoms, were also conducted. Second, to assess the stability and pervasiveness of neuroticism as a risk factor for dental anxiety and pain, measurements of neuroticism obtained concurrently (Time 2) as well as 13–19 years earlier (Time 1) were employed in the analyses. Third, biometric twin modelling was employed to determine to what extent genetic and environmental liability factors contribute to the variance in, and the covariance between, the phenotypes. Specifically, the potential role of neuroticism as a shared risk factor that may explain the association of dental anxiety with treatment-related pain, was examined.

2 | MATERIALS AND METHODS

2.1 | Participants

Twins were recruited from the Norwegian Twin Registry (NTR)[18] in 2011 (Time 2), and the current study is based on a random sample from the cohort born 1945–1960. This twin cohort also participated in surveys in 1992–98 (Time1). The study was approved by the Regional Committee for Medical and Health Research Ethics-South East Norway, and informed consent was obtained from all participants. It should be noted that the previous twin study of dental anxiety by Vassend et al.,[11] mentioned in the introduction, was based on a different sample drawn from a younger twin cohort (born 1967–1979).

2.2 | Assessment instruments

In 1992–98 (Time 1) a short form of the Eysenck Personality Questionnaire (EPQ) neuroticism scale was applied. In 2011 (Time 2), neuroticism was assessed using a Norwegian version of the NEO

Personality Inventory Revised (NEO PI-R).[12,19] This scale is comprised of 48 items (rated on a 5-point scale), whereas the short form of the EPQ neuroticism scale consists of 12 items[20,21] (rated yes/no). Examples of neuroticism items are 'I am easily frightened', 'I often worry about things that might go wrong', and 'Sometimes I feel completely worthless'. Both the EPQ and the NEO PI-R have been used extensively in personality and health related research, including Norwegian twin studies.[11,22] Thus, neuroticism was assessed at both Time 1 and Time 2, whereas dental anxiety and pain was assessed only at Time 2.

Musculoskeletal pain symptoms were measured using 3 items from the MS sub-scale of the Giessen Symptom Checklist (GSCL).[22,23] The participants were asked to rate the degree to which they 'generally' suffered from pain in the (1) joints or limbs (2), back and (3) neck and shoulders, using a 5-point scale. As noted, inclusion of this pain measure makes it possible to examine whether dental anxiety is associated specifically with dental care-related pain or with pain symptoms and responses more generally.

Dental care-related pain was assessed using the Numerical Pain Rating Scale (NPRS) in response to the question 'Generally, how painful is dental treatment to you?' The scale ranges from 0 (no pain at all) to 10 (worst imaginable pain). The NPRS is an easy to understand, reliable and valid pain assessment instrument appropriate for use in both clinical and research settings.[24]

Dental anxiety was measured using Corah's Dental Anxiety Scale (CDAS).[25] The CDAS is a 4-item questionnaire with total scores that can range from 4 (not anxious at all) to 20 (extremely anxious). The respondents are asked to indicate how they would feel if they should 'go to the dentist tomorrow', 'wait in the waiting room', 'sit in the dental chair while the dentist makes the drill ready' and 'get tooth cleaning'. A CDAS score of 13 or higher is commonly judged to indicate high dental anxiety.[25,26] All questionnaires were sent to the twins by mail.

2.3 | Statistical analyses

Bivariate associations were assessed using Pearson correlation analysis, followed by regression analysis to examine the effects of independent variables on total CDAS score. Because regression analysis needs to reflect the paired structure of the data, generalized estimating equations (GEE) was used.[27]

Trivariate Cholesky models[28] were used to estimate the genetic and environmental contributions to variance in, and covariance between, neuroticism, pain and dental anxiety in the best-fitting models. Classical twin analysis is typically aimed at disentangling three sets of influences that may cause individual differences or variation in a given trait: additive genetic (A) effects (the summed effect of the action of genes relevant to the phenotype), common or shared environmental (C) effects (all experiences and environments that twins share), and individual-specific or non-shared (E) effects (all experiences and environments not shared by the twins, in addition to measurement error). The Cholesky model specifies as many latent genetic and environmental factors as variables (phenotypes) in a triangular decomposition. Thus, the genetic factor A1 (Figure 1) influences the neuroticism trait and the two other phenotypes, factor A2 influences dental treatment-related pain and dental anxiety, controlling for A1 (neuroticism), whereas factor A3 influences dental anxiety, controlling for A1 and A2. The same will apply for the latent variables representing the contributions of the environmental influences. Heritability (h^2) measures the fraction of total phenotypic variation that can be attributed to genetic variation (the sum of the A effects on a given phenotype). A high genetic correlation (r_g) between two phenotypes indicates that genetic influences on the first trait also affect the second. On the other hand, an individual-specific environmental correlation (r_e) will be induced by any environmental effect that family members do not have in common and that influences more than one trait.

All models were run with OpenMx.[29] Several nested models were compared in order to identify the best-fitting one according to the minus2LogLikelihood difference test (Δ −2LL) and the Akaike Information Criterion (AIC).[30] Thus, an ACE model was compared with an AE model, and the consequences of constraining the parameters to be equal across sex in a given model were assessed. In investigating potentially sex-limited effects of genetic and environmental factors using the Cholesky model, the approach outlined by Neale et al.[31] was adopted (for a more detailed description of this approach applied to the present sample, see Vassend et al.[22]).

3 | RESULTS

In 2011, questionnaires were sent to a total of 2136 twins, of which 1516 responded (response rate 71%). The cohort comprises only same-sex monozygotic (MZ) and dizygotic (DZ) twins, and the sample consisted of 537 male twins (MZ/DZ: 290/247) and 979 female twins (MZ/DZ: 456/523). Age range of the sample was 50–65 (mean = 57.1, SD = 4.5). The number of participants at Time 2 with complete data varied between 1497 (for NPRS score) and 1514 (for neuroticism). However, several of the participants at Time 2 had not been recruited to the data collection at Time 1. Thus, the number of participants with EPQ neuroticism data was somewhat lower, that is 1374.

Satisfactory to excellent internal consistency reliability (Cronbach's alpha) was found for all the multi-item scales, that is CDAS (0.93), EPQ neuroticism (0.80), NEO PI-R neuroticism (0.84) and the MS pain scale (0.76). Descriptive statistics and intercorrelations of the variables are shown in Table 1 (data for each sex-zygosity group are included in Table S1). Mean CDAS score for the total sample was in the expected range for the middle-aged general population,[7,26] with female participants scoring higher than male participants (7.3 and 6.2, respectively, $p < .001$). The proportion of participants reporting high dental fear (CDAS score ≥ 13) was 5.1%. Average dental care-related pain level (NPRS score) was as anticipated at the lower end of the scale in this non-clinical sample. However, only a minority of the participants (12.9%)

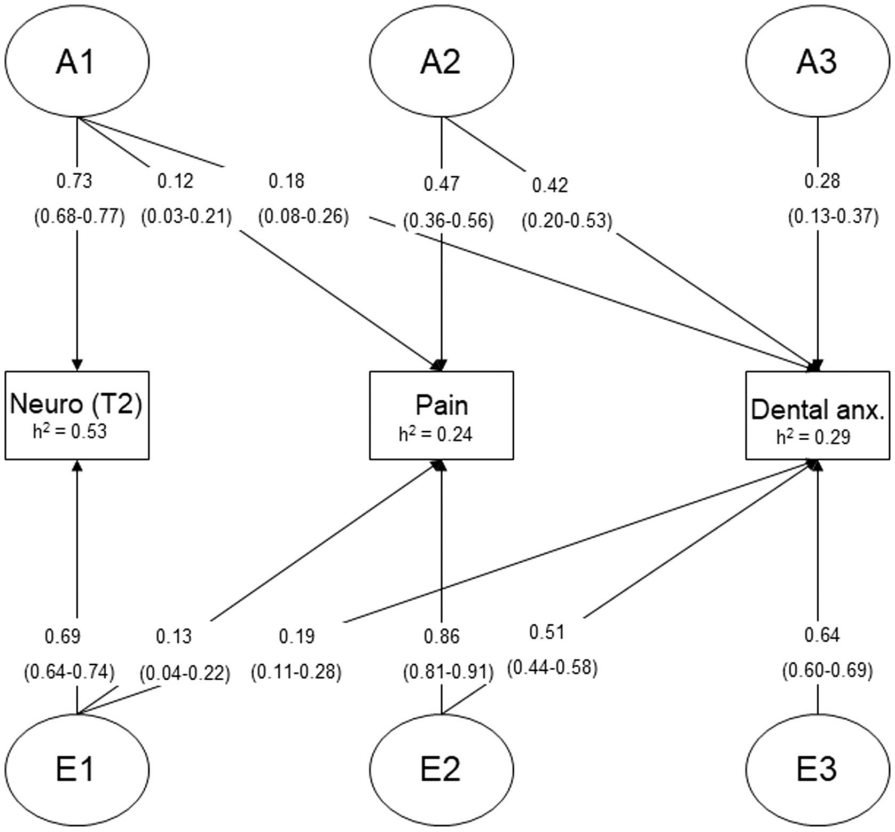

FIGURE 1 Path diagram of the best-fitting AE Cholesky model. The model is depicting genetic (A) and environmental (E) influences on the phenotypes neuroticism (Neuro, assessed at time 2: 2011), dental care-related pain (Pain), and dental anxiety (Dental anx.); 95% CI in parentheses; h^2, heritability coefficient

TABLE 1 Descriptive statistics and phenotypic correlations

	Mean (SD)	Neuroticism (Time 1)	Neuroticism (Time 2)	Dental anxiety	Dental care-related pain
Neuroticism (Time 1)	0.2 (0.2)				
Neuroticism (Time 2)	1.6 (0.4)	0.54 (0.50–0.57)			
Dental anxiety	6.9 (2.9)	0.18 (0.13–0.24)	0.28 (0.24–0.33)		
Dental care-related pain	2.4 (2.0)	0.13 (0.08–0.18)	0.18 (0.13–0.23)	0.68 (0.65–0.71)	
Musculoskeletal pain	2.0 (0.9)	0.26 (0.21–0.31)	0.28 (0.24–0.33)	0.13 (0.08–0.18)	0.10 (0.05–0.15)

Note: Time 1—(1992–1998); Time 2—(2011); Pearson correlation coefficients (with 95% CI in parentheses).

TABLE 2 Predictors of dental anxiety

Variable	Regression coefficients (Neuroticism Time 1)	Regression coefficients (Neuroticism Time 2)
Neuroticism (Time 1/Time 2)	0.28 (0.15–0.40)	0.25 (0.18–0.31)
Dental care-related pain	0.24 (0.22–0.26)	0.23 (0.21–0.25)
Sex	0.18 (0.13–0.24)	0.18 (0.12–0.23)

Note: Mean dental anxiety scores (CDAS) are entered as dependent variables. Coefficients are unstandardized (95% Wald CI in parentheses).

experienced dental visits to be essentially pain free, and 5.3% of the sample rated their dental visits in general to be severely painful (NPRS score ≥ 7).

Neuroticism measured at the two time points correlated significantly with both dental anxiety and dental care-related pain, albeit weakly (Table 1). Furthermore, the correlation between dental

anxiety and MS pain score was much reduced when neuroticism was controlled for in partial correlation analysis ($r = .05$, $p = .04$), whereas the correlation between dental anxiety and dental care-related pain was almost unchanged after this statistical control ($r = .67$, $p < .001$). In multiple regression analysis (GEE), both neuroticism (Time 1 and Time 2), dental care-related pain and sex turned out to be significantly associated with dental anxiety (Table 2).

Table 3 shows the fit of the different Cholesky models including neuroticism and the other study variables assessed simultaneously (i.e. at Time 2). As can be seen, compared with the first two models (sex-specific parameters), model 3 and 4 (equal standardized parameters across sex) resulted in a significant improvement of fit, with model 4 (AE) having lowest AIC and thus designated as the best-fitting model.

Figure 1 shows the parameter estimates for this model. By definition, the A1 genetic factor accounted for all the genetic variance in neuroticism (53% (0.53), i.e. 0.73^2). This factor also accounted for 1% of the variance in dental care-related pain, and 3% in dental anxiety.

A second genetic factor (A2), independent of A1, accounted for an additional 22% of the variance in dental care-related pain and 18% in dental anxiety. The genetic factor A3 accounted for specific genetic variance (8%), indicating that a non-trivial proportion of the genetic influence on dental anxiety is unique to the phenotype and not shared with neuroticism and dental care-related pain. The heritability estimates were moderate for dental anxiety and pain, and high for neuroticism (Figure 1). The individual-specific environmental effects were comparable with or even stronger than the genetic effects. While significant, the cross-effects of E1 were rather modest, accounting for 2% of the variance in dental care-related pain and 4% in dental anxiety. However, the second environmental factor (E2) accounted for 26% of the variance in dental anxiety, which should be regarded as a strong effect. Finally, the total individual-specific environmental effects on each phenotype were substantial, explaining a large amount of the variance in neuroticism (47%), dental care-related pain (76%) and dental anxiety (71%).

Parameter estimates based on the AE Cholesky model including neuroticism assessed at time 1 were broadly similar to estimates based on time 2 assessments, particularly with regard to genetic effects. Thus, the genetic component of neuroticism (A1, $h^2 = 0.45$), measured 13–19 years earlier, accounted for a comparable amount of the total variance in dental care-related pain (2%) and dental anxiety (3%). The cross-effects of the E1 factor in this AE model were somewhat smaller than the corresponding parameters in the first model, however, accounting for less than 0.01% of the variance in dental care-related pain or dental anxiety.

Genetic and individual-specific environmental correlations generated from the model including neuroticism at Time 2 are shown in Table 4. As can be seen, the genetic correlations are moderate to strong and somewhat larger than the environmental correlations. Particularly strong genetic and individual-specific environmental correlations emerged between dental care-related pain and dental anxiety.

4 | DISCUSSION

The findings of the current study indicate significant phenotypic, genetic and individual-specific environmental associations between concurrent/previous neuroticism assessments, dental care-related pain and dental anxiety. There was no evidence of common environmental factors, such as growing up in the same family and socioeconomic situation, in the relationship between the phenotypes.

The findings should be interpreted in view of some limitations, however. First, the study was based on a middle-aged sample, and genetic and environmental effect estimates obtained in this sample are not necessarily valid in younger age groups, especially not in child populations. On the other hand, environmental influences tend to accumulate with age, so that their impact on phenotypic associations will probably be easier to detect in older adults. Second, the pain ratings were based on the participants' retrospective recall, not on pain assessments in real-time clinical conditions. While such reporting delay is likely to cause recall bias,[32] it could be argued that precisely this generalized, global dental care-related pain experience is what is being addressed in this study. It should also be noted that different measures of dental treatment-related pain and discomfort (including 'general' pain during dental treatment) tend to be inter-correlated and may even be conceptualized as indicators of a single, underlying factor.[7] Furthermore, dental anxiety predicts pain experience across treatment stages and dental procedures, ranging from stressful

TABLE 3 Model fitting results for model including neuroticism (Time 2)

Model	−2 log likelihood	*df*	Δ−2LL (Δ*df*)	AIC
1. ACE (sex-specific parameters)	9845.19	4484	—	887.19
2. AE (sex-specific parameters)	9853.14	4496	7.95 (12)	861.14
3. ACE (equal standardized parameters across sex)	9856.37	4493	11.18 (9)	870.37
4. AE (equal standardized parameters across sex)	9857.90	4499	12.71 (15)	**859.90**

Abbreviations: Δ−2LL, −2 log likelihood difference relative to Model 1; A, additive genetic effects; AIC, Akaike's Information Criterion; C, common environmental effects; E, non-shared environmental effects. The AIC value (in bold) is a model fit measure, and a significance value is not relevant/applicable.

TABLE 4 Genetic correlations (above diagonal) and individual-specific environmental correlations (below diagonal) between the phenotypes

	Neuroticism (Time 2)	Dental care-related pain	Dental anxiety
Neuroticism (Time 2)		0.25 (0.07–0.44)	0.33 (0.16–0.49)
Dental care-related pain	0.15 (0.06–0.25)		0.84 (0.70–0.97)
Dental anxiety	0.23 (0.13–0.32)	0.63 (0.57–0.68)	

Note: 95% CI in parentheses.

surgical treatments to dental cleaning (prophylaxis).[6] However, inclusion of a measure of fear of pain would have been desirable to explore its genetic and environmental associations with dental care-related pain and anxiety, to which it is closely related phenotypically.

The mean CDAS score in the present sample (6.9) was slightly lower than the general population mean score (7.5) obtained in comprehensive Scandinavian studies.[7,26] This finding agrees with studies showing that the peak prevalence of dental anxiety often occurs in early adulthood and declines with age, particularly after 50 years of age.[2] Of significance, it has also been amply documented that the neuroticism trait shows a continuous and fairly strong decline across the lifespan,[33] and this phenomenon may at least partly explain the age-related reduction in dental anxiety and other symptoms and disorders associated with neuroticism.[34] Furthermore, the low initial correlation between dental anxiety and MS pain was further reduced when the common effect of neuroticism was partialled out, but this was not the case with the correlation between dental treatment-related pain and anxiety. These findings indicate that the association between dental care-related pain and anxiety is specific and not due to a generally heightened pain symptom level or a neuroticism-related propensity to symptom amplification.

Some authors have suggested that increased general pain sensitivity may at least partially contribute to the development of dental anxiety.[35,36] In a large Finnish cohort sample,[37] lower pressure pain tolerance (but not pain threshold) was associated with moderate or high dental fear in both female and male subsamples. Binkley et al.[35] showed that variants of the melanocortin-1 receptor (*MC1R*) gene were associated with increased dental anxiety, fear of dental pain and avoidance of dental care. Randall et al.[38] confirmed Binkley and coworkers'[35] findings and showed in addition that fear of pain fully mediated the relation between *MC1R* variant status and dental fear. In line with these findings, the authors suggest the hypothesis that *MC1R* variants may affect orofacial pain perception and predispose the individual to develop fear of pain, and in turn dental fear. However, as acknowledged by Randall et al.,[38] their study involved only one gene, and the effect size of *MC1R* variant status was small and accounted for only a minor proportion of variance in dental fear (1%) and fear of pain (5%).

Interestingly, in an early study Klepac et al.[39] showed that patients seeking treatment for high dental fear and avoidance had lower pain tolerance for dental pain (electrical tooth pulp stimulation), but not non-dental pain (electrocutaneous stimulation of the left forearm), compared to low dental fear patients. There were no differences in pain threshold between the two groups. These results concur with a study by Vassend et al.,[11] who found no associations between dental anxiety and experimental pain responsivity (i.e. heat and cold pain measurements performed in standardized and non-dental settings). Generally, pain tolerance measures are more strongly related to personality traits, affective states and situational characteristics than is the case with baseline pain threshold measures.[40] Thus, the strong association of dental anxiety with fear of pain and dental care-related pain (suggesting a lowered dental pain tolerance) may primarily reflect a readiness to respond with fear and withdrawal during dental treatment, rather than a heightened sensitivity to sensory-discriminative aspects of the pain.

The biometric findings of the present study are probably the most interesting in this context and may in several ways contribute to a more nuanced understanding of the dental anxiety-pain relation. First, the common genetic factor (A1) may reflect a general, probably lifelong, susceptibility to psychological and somatic distress,[14] including pain and emotional responding in dental care settings. Second, while significant, its effect appears to be rather weak, in contrast to dental care-related pain that is strongly associated both genetically and environmentally with dental anxiety. Third, dental anxiety is in addition influenced by unique (i.e. independent of neuroticism and pain) genetic and environmental factors. Taken together, these results suggest that there are both general and specific genetic risk factors, affecting distress susceptibility and pain responding, and probably pain tolerance and fear of pain in particular.

Clinicians should be aware that individuals showing high levels of negative affectivity or dental care-related pain are likely to have a stronger genetic predisposition to develop dental anxiety. Importantly, however, the unique individual-specific environmental influences on each phenotype were substantial, and the environmental cross-effects were of the same magnitude as the genetic effects. From an etiological point of view, this indicates that while the phenotypes share similar genetic influences, suggesting genetic confounding, the findings are also consistent with environmental causal interpretations. Moreover, while neuroticism, dental anxiety and pain share some similarities, they are also clearly distinct psychologically, suggesting that different preventative and treatment strategies may be needed to adequately target each of these phenotypes and their interplay.

In conclusion, this study reveals a rather complex genetic and environmental architecture underlying the relation between neuroticism, dental care-related pain and dental anxiety. Of particular significance, the findings highlight the role of dental anxiety as an individual propensity or trait—to a large extent independent of neuroticism—that is strongly related to dental treatment-related pain and distress.

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CONFLICT OF INTEREST

The authors declare no potential conflicts of interest.

DATA AVAILABILITY STATEMENT

All relevant numbers and estimates to this study have been made available to the reader in the manuscript. Individual twin data are unavailable for public deposition due to restrictions from the Norwegian Twin Registry (organized under the Norwegian Institute of Public Health), and data privacy restrictions. Before data can be released, a full application must be submitted and, in most cases, a contract must be signed with the Norwegian Twin Registry. Information about data access is available here: https://www.fhi.no/en/studies/norwegian-twin-registry/.

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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