

1 Distinct genetic pathways to music enjoyment

2 Giacomo Bignardi ^{1,2*}, Laura W. Wesseldijk ^{3,4,5}, Ernest Mas-Herrero ^{6,7,8}, Robert. J. Zatorre ^{9,10}, Fredrik
3 Ullén ^{5,11,†}, Simon E. Fisher ^{1,12,†}, and Miriam A. Mosing ^{5,11,13,14,†}

4
5 1 Language and Genetics Department, Max Planck Institute for Psycholinguistics, Nijmegen, the
6 Netherlands

7 2 Max Planck School of Cognition, Stephanstrasse 1a, Leipzig, Germany

8 3 Department of Neuroscience, Karolinska Institutet, 171 65 Solna, Stockholm, Sweden.

9 4 Department of Psychiatry, Amsterdam UMC, University of Amsterdam, 1105 AZ Amsterdam, the
10 Netherlands

11 5 Department of Cognitive Neuropsychology, Max Planck Institute for Empirical Aesthetics, 60322
12 Frankfurt am Main, Germany

13 6 Department of Cognition, Development and Educational Psychology, Universitat de Barcelona,
14 Barcelona 08035, Spain

15 7 Institute of Neurosciences, Universitat de Barcelona, Barcelona 08035, Spain

16 8 Cognition and Brain Plasticity Group, Institut d'Investigació Biomèdica de Bellvitge (IDIBELL),
17 Hospitalet de Llobregat 08907, Spain

18 9 Montreal Neurological Institute, McGill University, 3801 Rue University, Montreal, QC H3A 2B4,
19 Canada

20 10 International Laboratory for Brain, Music, and Sound Research (BRAMS), Montreal, QC Canada

21 11 Department of Neuroscience, Karolinska Institutet, SE-171 77 Solna, Sweden.

22 12 Donders Institute for Brain, Cognition and Behaviour, Radboud University, Nijmegen, The
23 Netherlands

24 13 Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, SE-171 77 Solna,
25 Sweden.

26 14 Melbourne School of Psychological Sciences, Faculty of Medicine, Dentistry, and Health Sciences,
27 University of Melbourne, 3010, Melbourne, Australia

28
29
30
31
32
33
34 * Corresponding author: Giacomo Bignardi (Giacomo.bignardi@mpi.nl).

35 † These authors share last authorship

36

37 Abstract

38 Humans engage with music for various reasons that range from emotional regulation and
39 relaxation to social bonding. While there are large inter-individual differences in how much
40 humans enjoy music, little is known about the origins of those differences. Here, we
41 disentangled the genetic factors underlying such variation. We collected behavioural data on
42 several facets of music reward sensitivity, music perceptual ability, and general reward
43 sensitivity from a large sample of Swedish twins ($N = 9,169$). We found that genetic factors
44 substantially explain variance in music reward sensitivity above and beyond genetic
45 influences shared with music perception and general reward sensitivity. Furthermore,
46 multivariate analyses showed that genetic influences on the different facets of music reward
47 sensitivity are partly distinct, uncovering distinct pathways to music enjoyment and different
48 patterns of genetic associations with objectively assessed music perceptual abilities. These
49 results paint a complex picture in which partially distinct sources of genetic variation
50 contribute to different aspects of musical enjoyment and open up new possibilities for using
51 inter-individual differences to gain insights into the biology of a key aspect of human
52 behaviour.

54 Introduction

55 Music can evoke intense pleasure and induce various emotions ¹⁻⁴, leading individuals from
56 different cultures ⁵ to actively seek out and engage with it. This human attraction to music
57 has always been considered somewhat baffling ⁶ and mysterious ⁷, leading many to ask why
58 music has such power over humans ^{8,9}. Oliver Sacks highlighted this conundrum in the opening
59 of his beautifully written commentary, *The Power of Music*: “What an odd thing it is”, he wrote
60 “, to see an entire species—billions of people—playing with listening to meaningless tonal
61 patterns, occupied and preoccupied for much of their time by what they call ‘music’” ⁹.
62 Despite the widespread power of music, however, it should also be noted that many people
63 do not occupy themselves with music. Within human populations, there is indeed ample
64 evidence that music-related cognition, from perceptual to affective-related processes, varies
65 from one person to another ¹⁰⁻¹³.

66 Over the last decade, several studies have explored such differences between individuals in
67 music-related traits and states to better understand the basis of human musicality ¹⁴. These
68 studies show that differences in the ways individuals perceive, produce, or enjoy music
69 correlate with neurobiological differences ¹⁵⁻¹⁷. For example, the study of individuals with
70 lifelong musical pitch deficits underscores the relevance of brain connectivity patterns in
71 distributed neural networks for conscious perception of music ¹⁷. Similarly, studies of
72 differences in musical enjoyment highlight how interactions between cortical and subcortical
73 brain regions support perceptual and affective processes that are fundamental for the
74 experience of musical pleasure ^{15,16,18-20}. Moreover, recent studies have started to uncover
75 the roles of genetic factors in perceptual-motor processing of music ²¹ (e.g., the ability to
76 synchronise with an external beat or recognise a melody) as well as in music production, such

77 as levels of musical achievement ^{22,23}. In general, these studies highlight complex gene-
78 environment interplay ^{24,25} and the involvement of many DNA variants ²¹, each with a small
79 effect (see ²⁶).

80 Despite the many studies that have examined differences in music-related traits, still little is
81 known about the genetic sources of differences in affective aspects of music processing and,
82 in particular, the ability to enjoy music ^{27,28}. A better understanding of such genetic effects
83 will allow us to highlight how the ability to enjoy music is passed from one generation to the
84 other and clarify the mechanisms linking genotypes, brains, and affect, providing a needed
85 complementary perspective to resolve the conundrum of how “meaningless tonal patterns”
86 can have such powerful effects on humans.

87 Here, we study individual differences in musical enjoyment, focusing on music reward
88 sensitivity, a phenotype capturing how much individuals derive pleasure from music, as
89 measured by the Barcelona Music Reward Questionnaire (BMRQ) ^{12,16}. We used the BMRQ as
90 it is a validated and reliable (e.g., one-year test-retest reliability, $R_{xx}(25) = .94$, see ¹²)
91 instrument that provides a fine-grained characterisation of individual differences in emotion
92 evocation, mood regulation, music seeking, sensory-motor, and social reward facets of music
93 enjoyment ¹¹. Furthermore, it is a well-established psychometric tool in the music science
94 literature, showing robust associations with affective experiences ^{29–31}, cognition ^{32–34},
95 physiology ¹², and neurobiology ^{15,16,35,36}. More specifically, we addressed the following three
96 research questions:

- 97 1. To what extent are differences in music reward sensitivity explained by genetic variation?
- 98 2. To what extent do genetic effects influence music reward sensitivity above and beyond
99 genetic effects shared with music perceptual ability and general reward sensitivity?
- 100 3. To what extent are genetic effects shared between the different facets of music reward
101 sensitivity?

102 To address these questions, we utilised a large sample of deeply phenotyped monozygotic
103 (MZ) and dizygotic (DZ) twins with available musicality data. We addressed the first question
104 by estimating the heritability of music reward sensitivity using the classical twin design. We
105 addressed the second question by applying multivariate twin modelling to estimate the
106 genetic overlap between music reward sensitivity (BMRQ), music perceptual abilities based
107 on a composite score of the melody, pitch, and rhythm scales of the Swedish Musical
108 Discrimination Test (SMDT) ¹³, and general reward sensitivity, measured with the Behavioral
109 Approach System Reward Responsiveness (BAS-RR) sub-scale ³⁷, which has previously been
110 shown to correlate with the BMRQ ^{11,12,38}. The third question was assessed by testing if
111 genetic effects are shared across facets of music reward sensitivity, consistent with a common
112 genetic factor of music enjoyment, or whether, alternatively, genetic influences are distinct
113 for each facet. Finally, we further extended the multivariate analyses at the facet level to
114 explore associations with music perceptual abilities and general reward sensitivity.

115

116 Results

117 **Sample and BMRQ descriptives.** We utilised self-reported BMRQ data in a sample of 9,169
 118 monozygotic (MZ) and same-sex and opposite-sex dizygotic (DZ) Swedish twins, with a mean
 119 (M) age of 51 years (standard deviation (σ) = 8 years, range from 37 to 64 years; see Table 1
 120 for sample size split by sex and zygosity; see Methods for details on the cohort and zygosity
 121 identification). BMRQ total scores ranged from 20 to 100, with $M = 71.20$ and $\sigma = 13.95$. In
 122 line with previous studies, the BMRQ distribution was negatively skewed (skew = - 0.58; i.e.,
 123 long tail of individuals with lower BMRQ total scores; see Supplementary Fig. 1). A
 124 confirmatory factor model showed acceptable fit for a model with a single latent music
 125 reward sensitivity factor capturing correlations between the five facets (CFI = .96, SRMR =
 126 .035).

127 **Table 1.** Numbers of monozygotic (MZ) and dizygotic (DZ) twin pairs for each trait.

| Trait | Measure | | MZ women | MZ men | DZ wom en | DZ men | DZ os | Total twins |
|---|--|--------------|-------------|-----------|-----------------|-----------|----------|----------------|
| Music perceptual abilities ⁺ | Swedish Musical | n | 1012 | 632 | 705 | 525 | 1162 | 4036 |
| | Discrimination Test (SMDT) | (n pairs) | (357) | (200) | (201) | (128) | (280) | (716) |
| General reward sensitivity ⁺ | Behavioral Approach | n | 1954 | 1383 | 1510 | 1192 | 2680 | 8719 |
| | System Reward Responsiveness (BAS-RR) | (n pairs) | (629) | (379) | (363) | (244) | (556) | (2171) |
| Music reward sensitivity | Barcelona Music Reward Questionnaire (BMRQ) | n | 2025 | 1459 | 1595 | 1258 | 2832 | 9169 |
| | | (n pairs) | (659) | (400) | (386) | (268) | (592) | (2305) |

128
 129 Note. We collected the two measures for general perceptual-affective phenotypes (top two rows) and one
 130 measure for music reward sensitivity (bottom rows) in a sample of twins from the Swedish twin registry. The
 131 number of pairs with data available for both twins (n pairs) is shown in parenthesis. The measures used to
 132 quantify each trait are shown in the second column. n : number of individual twins; MZ: Monozygotic, DZ:
 133 Dizygotic; os: opposite-sex; ⁺ The total sample size for these traits is shown only for twins in which music reward
 134 sensitivity data were available.

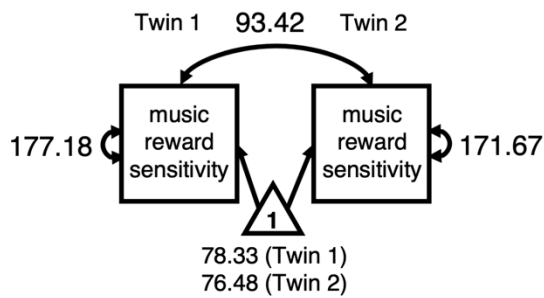
135 **Genetic factors play a substantial role in music reward sensitivity.** To estimate to what
 136 extent genetic effects (A: additive; D: dominance), the family environment shared between
 137 members of a family (C: common environment), and residual experiences unique to each
 138 individual (E: non-shared environment, including measurement error) influence music reward
 139 sensitivity, we use Structural Equation Modeling (SEM), informed by the Classical Twin Design
 140 (CTD). First, as a baseline for further model comparisons, we fit a univariate model to
 141 individuals' BMRQ total scores (Fig. 1A; age and sex were accounted for). Assumptions of
 142 equality of means and variance across zygositys, twins within a pair, and sex were met (see
 143 Supplementary Table 1), except for the equality of means across sex: Consistent with previous
 144 literature³⁹, BMRQ scores were higher in women ($M = 76.26$) than in men ($M = 71.20$) (sex-

145 constrained $\sigma = 13.72$; $\chi^2(30)_{\Delta df} = 300.54$, $p < 0.001$). We, therefore, did not constrain means
146 in subsequent models. Also consistent with previous results^{11,40}, age was negatively
147 associated with overall BMRQ scores, although the effect was small, $\beta_{\text{age}} = -0.03$, (95% CI [-
148 .05, -.01]), $p = 0.004$. Since the skewness of BMRQ scores was below 2 (see⁴¹), all SEM
149 analyses used the full-information maximum likelihood estimator. Analyses using alternative
150 estimators, robust to departures from multivariate normality, did not change the findings; the
151 results of these analyses are provided in Supplementary Note 1.

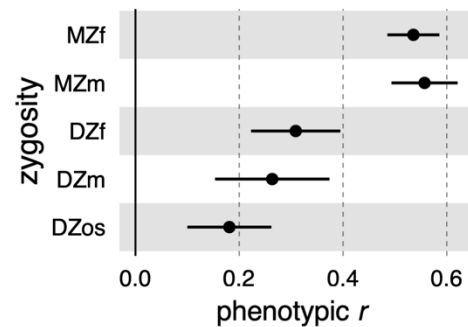
152 By comparing within-pair MZ and DZ correlations of BMRQ scores, we estimated the narrow-
153 sense heritability (h^2_{twin}) of music reward sensitivity, i.e. the proportion of phenotypic
154 variance in this trait which is explained by genetic variation⁴². Twin correlations for music
155 reward sensitivity were higher for MZ ($r_{\text{MZ}} = .55$, 95% CI [.51, .59]) than DZ ($r_{\text{DZ}} = .24$, 95% CI
156 [.19, .29]) twins (Fig. 1B, see Supplementary Fig. 2). As the r_{MZ} was more than twice the r_{DZ} , a
157 model with additive and dominance genetics components (ADE) was fit (Fig. 1C). The ADE
158 model reasonably fitted the data, as indicated by comparison against the baseline model
159 ($\chi^2(33) = 41.13$, $p = .16$). However, a more parsimonious AE model, from which the D
160 component was dropped, showed a better fit to the data ($\chi^2(1)_{\Delta df} = 1.63$, $p = .20$). Therefore,
161 the AE model was deemed the best fit for the data. The heritability for the BMRQ total score
162 was substantial: $h^2_{\text{twin}} = .54$ (95% CI [.51, .58]; Fig. 1D; see Supplementary Table 2 for details).

163

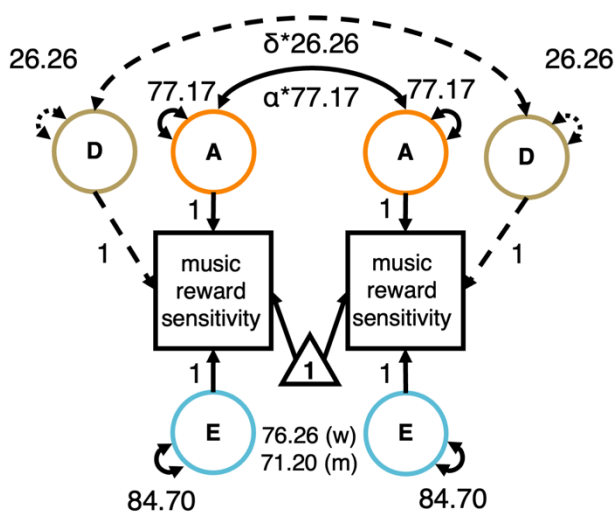
A Baseline model



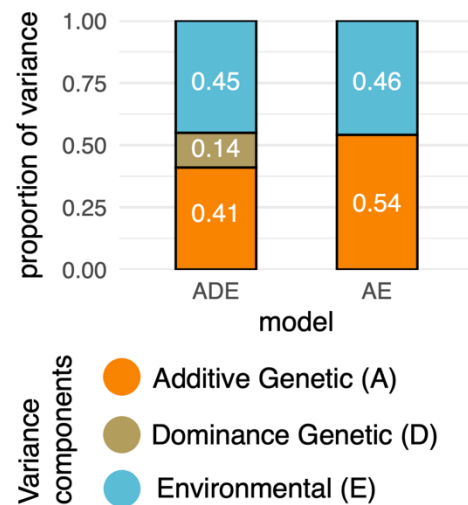
B Twin correlations



C ADE model (DZ group)



D Variance components



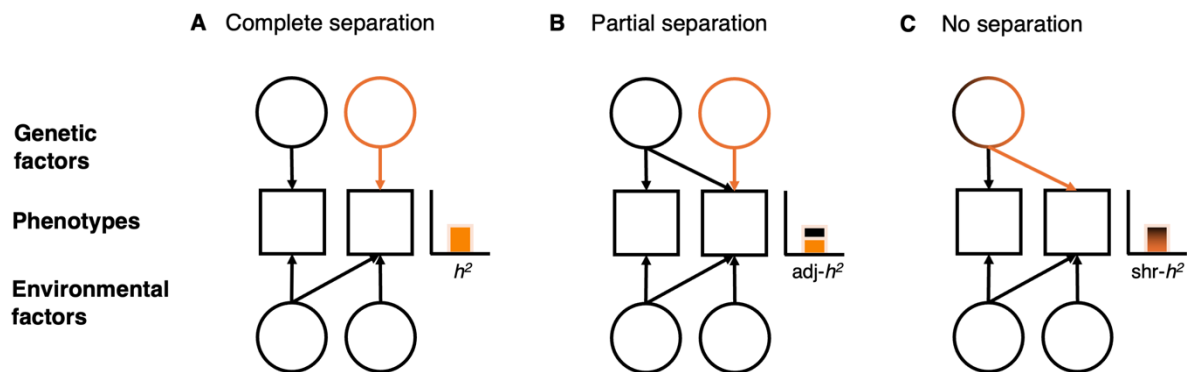
164

165 **Fig. 1. Music reward sensitivity is substantially heritable.** (A) Baseline SEM to test for assumptions
 166 and further compare CTD-informed models fit; for simplicity only one group (MZ women) is shown.
 167 (B) Twin pair correlations grouped by zygosity and sex (women, w; men, m) extracted from the
 168 saturated model; note that MZ twin pairs are more than twice as similar in their music reward
 169 sensitivity as DZ twin pairs. The error bars represent 95% confidence intervals (CI). (C) The ADE model;
 170 note that we identified only A and E components as significant contributors to music reward
 171 variability. α is the expected additive genetic relationship, and δ is the expected dominant genetic
 172 relationship between pairs (i.e., $\alpha = 1$ or $.5$ $\delta = 1$ or $.25$, for MZ and DZ, respectively). (D) Estimated
 173 variance components from the final AE model indicated substantial heritability for music reward
 174 sensitivity. The left bar plot shows the estimates obtained from the full ADE model. *Notes on structural*
 175 *equation models: For simplicity, age is not included in the graphical representation of the model but is*
 176 *included as a covariate; Squares represent the measured phenotypes; Circles are the latent component;*
 177 *Double-headed arrows within circles, the variances associated with the latent components; double-*
 178 *headed arrows between circles covariances; the triangle, the phenotypic mean grouped by twin order*
 179 *(baseline model) and sex (ADE model) already adjusted for age; dashed elements, the component*
 180 *dropped after model comparison.*

181

182 **Music reward sensitivity is influenced by genetic factors above and beyond genetic**
 183 **influences shared with music perceptual abilities and general reward sensitivity.** To better
 184 understand the nature of genetic effects contributing to music reward sensitivity, we tested
 185 whether the genetic influences on BMRQ were partly shared with other related traits, such

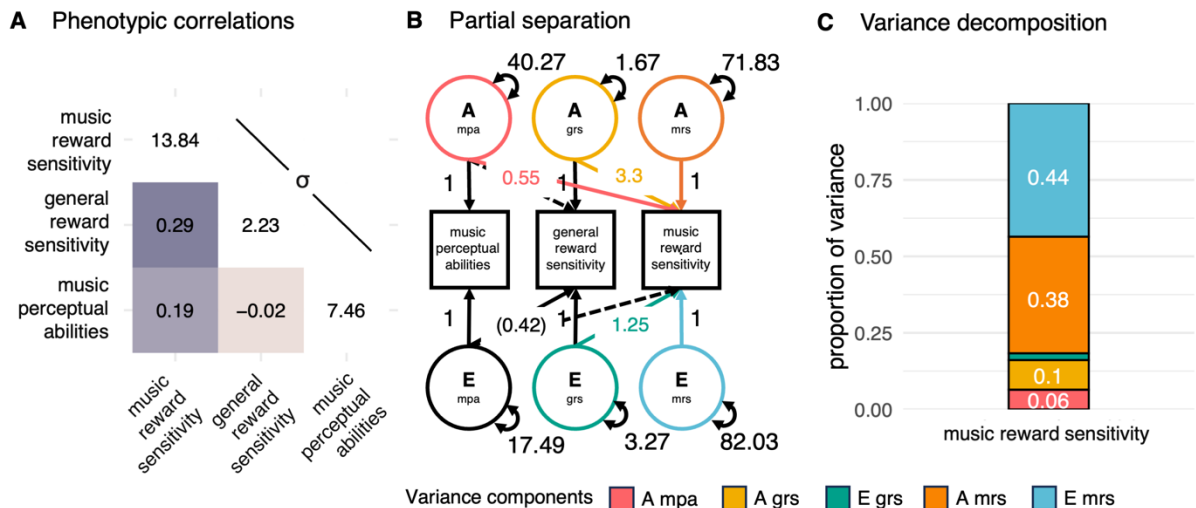
186 as music perceptual abilities and general reward sensitivity. For this purpose, we used a
187 multivariate sequential decomposition approach, which allowed us to discriminate between
188 three possible outcomes, as illustrated in Figure 2. Genetic effects on music reward sensitivity
189 could be either fully (Fig. 2A) or partly (Fig. 2B), separate from genetic effects on music
190 perceptual abilities or general reward sensitivity. Alternatively, they could be fully shared (Fig.
191 2C) and hence entirely accounted for.
192



193
194

195 **Fig. 2. Schematic illustration of the sequential decomposition approach.** The sequential
196 decomposition of phenotypic associations employed to study unique and shared genetic influences.
197 (A) The heritability (h^2) of the second phenotype (orange bar) is fully separate from the genetic effect
198 shared with the first. (B) In this case, after controlling for the h^2 explained by the genetic effect shared
199 with the first phenotype (black bar), an adjusted estimate ($adj-h^2$, remaining orange bar) is still
200 substantial. (C) Here, h^2 is completely shared ($shr-h^2$) between the two phenotypes. For simplicity,
201 only two traits are shown.
202

203 First, we revealed and confirmed that there were significant phenotypic correlations between
204 music reward sensitivity and music perceptual abilities and general reward sensitivity^{11,12,38},
205 respectively ($p < .001$; Fig. 3A; correlations were estimated from a sample of only one twin
206 per pair, to avoid sample dependence; estimates were similar in the other twins, see
207 Supplementary Fig. 3 for details). To simultaneously accommodate the three phenotypes, we
208 employed a tri-variate sequential decomposition. This analysis indicated partial separation of
209 genetic (and environmental) factors influencing the three variables (Fig. 3B; see
210 Supplementary Table 3 for coefficient estimates). The h^2_{twin} of music reward sensitivity
211 adjusted for music perceptual abilities and general reward sensitivity was $adj-h^2_{twin} = .38$ (95%
212 CI [.33,.43], Fig. 3C). Thus, of the total variance in music reward sensitivity explained by
213 genetic factors ($h^2_{twin} = .54$), around 70% (95% CI $\sigma^2_{Au:At} = [.63,.78]$) was unique to this trait.
214 Only the remaining 30% was shared with genetic effects on music perceptual abilities and
215 general reward sensitivity, explaining 12% and 18% of the total genetic variance in music
216 reward sensitivity, respectively. Environmental influences shared across phenotypes, which
217 reached significance only for general reward sensitivity ($p < .001$), explained only 2% of the
218 total variance in music reward sensitivity (see Supplementary Note 2).
219



220

221

222

223

224

225

226

227

228

229

230

231

232

233

234

235

236

237

238

239

240

241

242

243

244

245

246

247

248

249

250

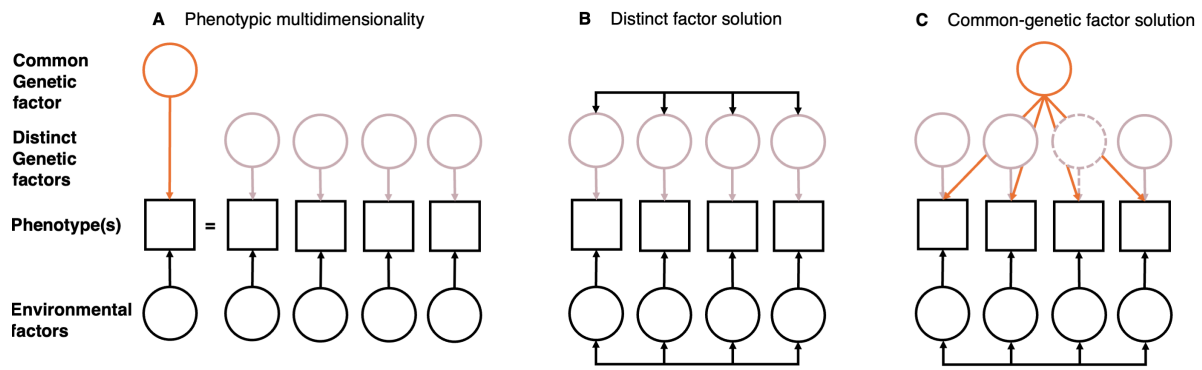
251

252

253

Fig. 3. Genetic effects on music reward sensitivity are partly separate from music perceptual abilities and general reward sensitivity. (A) All cross-phenotypic correlations with music reward sensitivity were significant (all $p < .001$). On the diagonal, the standard deviations (σ). (B) Sequential decomposition of the significant contributions to music reward sensitivity; note that the environmental path from music perceptual abilities to music reward sensitivity is not significant, indicating only common genetic causes. Between parentheses, the significant path from the E component to general reward sensitivity ($p = .03$) (C) Variance decomposition shows that genetic factors explain individual differences in music reward sensitivity (in orange) well beyond shared genetic factors associated with known general perceptual and affective processes (in red and yellow, respectively). The variance components here indicate the proportion of variance explained by the respective components. mpa: music perceptual abilities; grs: general reward sensitivity; mrs: music reward sensitivity. Notes on structural equation models: one-headed arrow represents regression paths partitioned in additive genetics and unique environmental paths; dashed one-headed arrows represent non-significant paths. Other abbreviations and symbols are as in Fig. 1.

Genetic pathways to the different facets of music-reward sensitivity are partly distinct. Having shown that music reward sensitivity has substantial heritability and is partly genetically separate from relevant general perceptual-affective processes, we went on to test whether the pattern of genetic correlations across facets is consistent with an overarching one-genetic-factor solution for music reward sensitivity (Fig. 4 A-C). If largely distinct genetic pathways influence the different facets of music enjoyment, a one-genetic-factor solution would not be supported. This scenario can be modelled as a multivariate correlated factor solution, which solely allows for genetic and environmental pairwise correlations (Fig. 4B). If, on the other hand, there is a common genetic source of different aspects of musical enjoyment, we would expect underlying genetic sources of variability to be mostly shared across different facets (Fig. 4C, see ⁴³). This latter scenario can be instead modelled as a multivariate hybrid independent pathway model (see ⁴⁴). Here, along with distinct genetic effects over single facets, an extra additive genetic common factor is modelled to capture shared genetic effects across all facets. For ease of interpretation, we will hereafter refer to the model depicted in Fig. 4B as the distinct factor solution and the model depicted in Fig. 4C as the common-genetic factor solution.

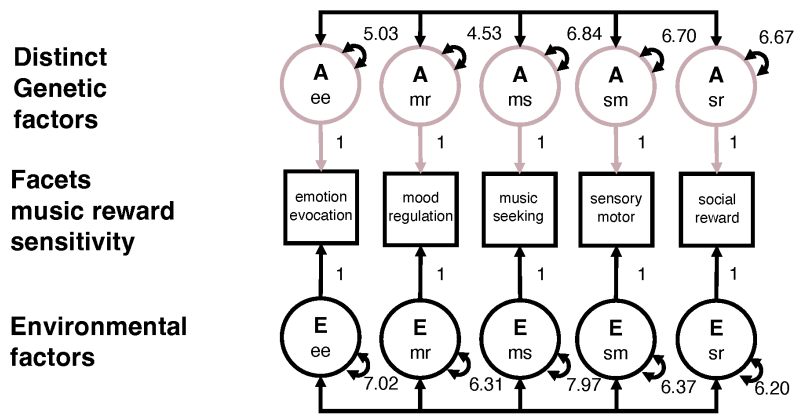


254
255

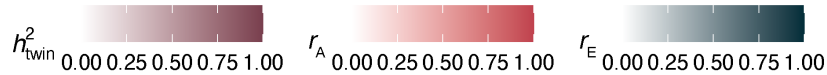
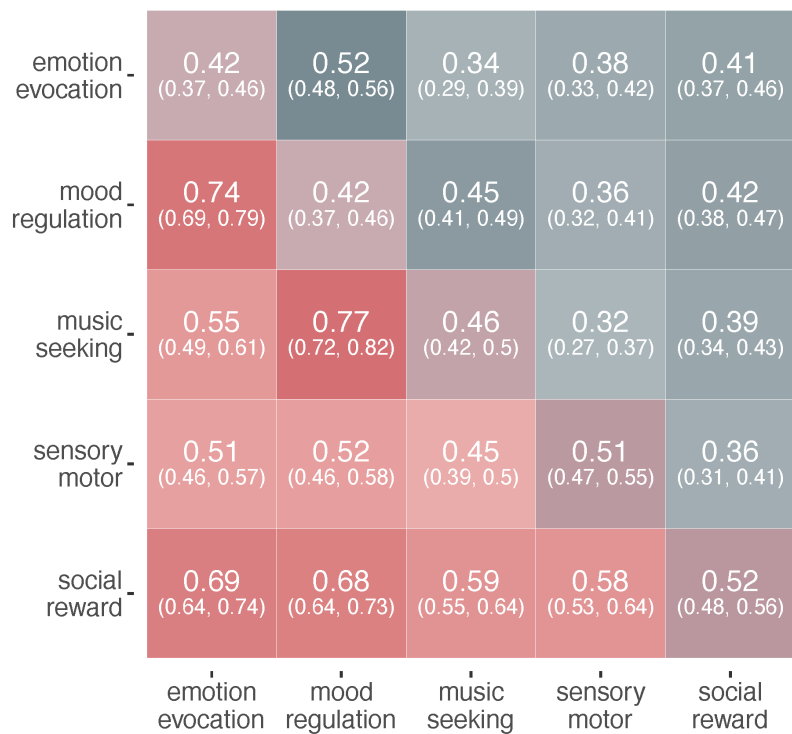
256 **Fig. 4 Schematic illustration of multivariate models employed to quantify distinct and common**
257 **genetic factors.** (A) The phenotype is decomposed into its constituent facets. (B) The first solution
258 includes distinct genetic factors with a simple description of all possible genetic and environmental
259 covariances. (C) A common-genetic factor solution is applied by assuming a genetic factor that
260 captures the genetic covariances across facets. The common latent genetic factor (in orange) could
261 explain all the genetic variance associated with one facet (e.g., dashed circle). (Double-headed arrows
262 are compressed to avoid cluttering.) Figure inspired by⁴⁵.
263

264 Since the common-genetic factor solution is a constrained version of the distinct factor
265 solution, model comparisons can be used to test whether a common-genetic factor of music
266 reward sensitivity facets shows a better fit to the data. While both models fit the data well
267 (CFI = .988, SRMR = .048, and CFI = .981, SRMR = .061, respectively; See Supplementary Table
268 4), the common-genetic factor worsened the fit of the distinct factor solution ($\chi^2(5)_{\Delta df} =$
269 129.61, $p < 0.001$;). This implies that the distinct factor solution is a more appropriate
270 description of the structure of the genetic effects compared to the common-genetic factor
271 solution. (Fig. 5A-B; See Supplementary Note 3 for more details).

A Multivariate model comparison favours distinct factor solution



B Genetic and environmental correlations

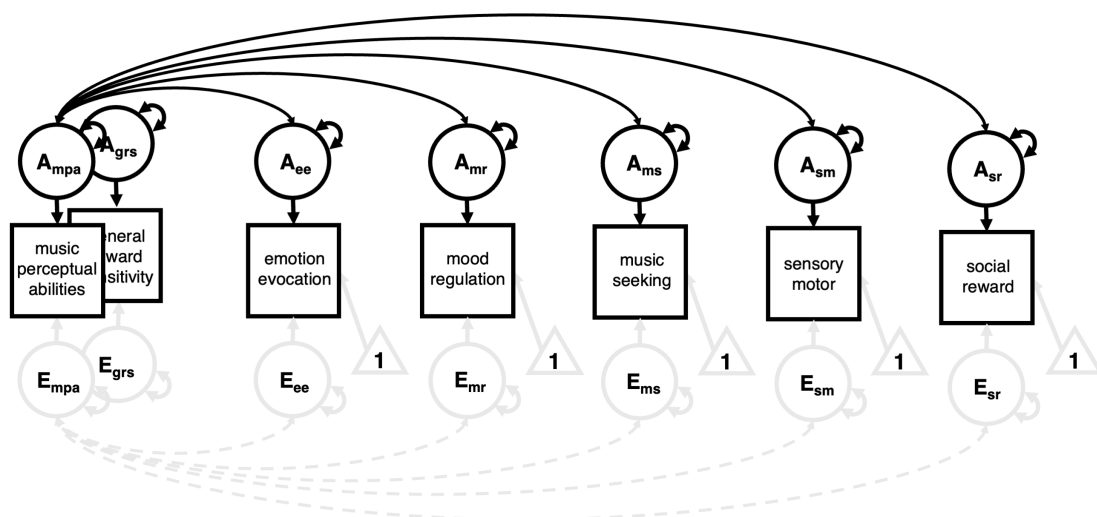


272
273

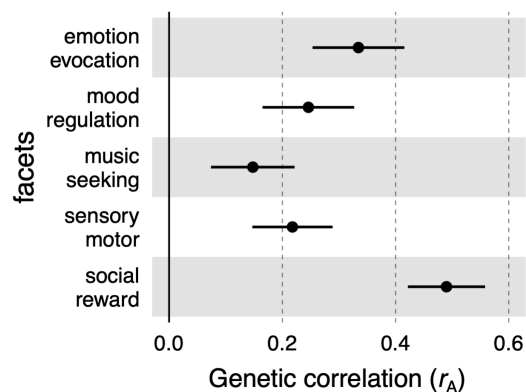
274 **Fig. 5. Genetic heterogeneity between distinct musical affective phenotypes.** (A) Simplified distinct
275 factor solution of music reward sensitivity facets. (B) Genetic effects on music reward sensitivity are
276 partially heterogeneous. Matrix extracted from the correlated factor model. Additive genetic (r_A) and
277 environmental correlations (r_E) are shown below (red) and above (blue) the diagonal, respectively;
278 numbers on the diagonal show heritability estimates. Numbers in parentheses are 95% confidence
279 intervals. Note that genetic correlations are far from 1, suggesting that music reward sensitivity has
280 multiple genetic sources. Phenotypic correlations can be found in Supplementary Fig. 4. *Notes on*
281 *structural equation models: double-headed arrows between circles represent A and E covariance*
282 *between facets. Other abbreviations and symbols are as in Fig. 1 and 3.*

283 **Exploratory analyses reveal that social reward shares substantially more genetic variance**
 284 **with music perceptual abilities than the other facets.** Having shown that genetic influences
 285 are partially distinct between music-reward sensitivity facets, we further explored such
 286 genetic heterogeneity by fitting two additional multivariate distinct factor solutions to data
 287 on music reward sensitivity facets, with music perceptual abilities and general reward
 288 sensitivity added to the models (Fig. 6A). Additive genetic correlations (r_A) between music
 289 reward sensitivity facets and music perceptual abilities varied widely (range $r_A = .15$ to $r_A = .49$;
 290 Fig. 6B), with differences between the r_A values (Δr_A) being significant (Supplementary Table
 291 5). Specifically, the Δr_A estimates were significantly higher for the social-reward facet of music
 292 reward ($r_A = .49$, 95% CI [.42; .56]) than for any other facet (range Δr_A from .19 to .39, all $p <$
 293 .001). In comparison, r_A obtained from the model fit to general reward sensitivity data were
 294 similar across facets (range $r_A = .29$ to $r_A = .36$; Fig. 6C) and did not significantly differ (all $p >$
 295 .05). These observations further strengthen the evidence that different aspects of music
 296 reward show functionally relevant genetic heterogeneity.
 297

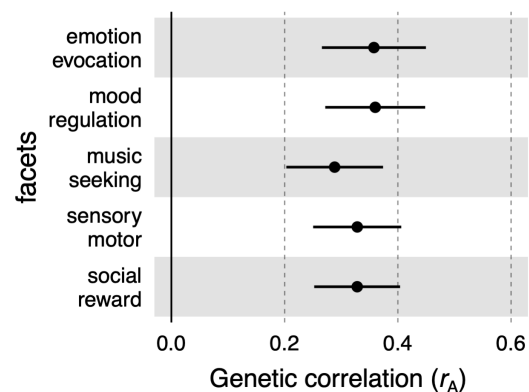
A Extended correlated factor model



B Music perceptual abilities



C General reward sensitivity



298
 299
 300

301 **Fig. 6. Genetic heterogeneity between distinct musical affective phenotypes.** (A) The correlated
302 factor model extended to estimate genetic correlations with music perceptual abilities and general
303 reward sensitivity. (B-C) Magnitude of the genetic correlations (r_A) between facets of music reward
304 sensitivity and music perceptual abilities (B) and general reward sensitivity (C). Error bar represents
305 95% CI. *Notes on structural equation models: For simplicity, all pairwise covariances are not included*
306 *but are present in the model—other abbreviations and symbols are as in previous Figures.*
307

308 Discussion

309 Our understanding of why “meaningless tonal patterns”⁹ have such powerful effects on
310 humans can benefit tremendously from the study of inter-individual differences. Here, by
311 exploiting a large and deeply phenotyped Swedish twin sample, we found that music reward
312 sensitivity has substantial heritability. Most of this genetic variance influences music reward
313 sensitivity independently of music perceptual abilities and general reward sensitivity,
314 suggesting genetic variations influence music reward sensitivity not only via other general
315 perceptual-affective processes. Furthermore, our findings reveal considerable genetic
316 heterogeneity behind different facets of music reward sensitivity. Although all facets show
317 heritability estimates of a similar magnitude (between 42% and 52%) and are genetically
318 correlated (between .45 and .77), the results do not support a single (genetic) dimension of
319 musical enjoyment. Instead, these findings are consistent with musical enjoyment being built
320 upon genetically interconnected yet partly distinct parts. Extended multivariate analyses
321 further strengthened these results by showing that music perception shows stronger genetic
322 correlations with social bonding than other facets of music reward, indicating functionally
323 relevant genetic heterogeneity.

324 Despite answering a long-standing question^{27,28}, the finding that music reward sensitivity is
325 to some extent heritable is not surprising in light of the fact that virtually every human trait is
326 at least partly genetically influenced^{46,47}. Yet, the finding of notably high heritability for music
327 reward sensitivity gives hope for molecular genetic studies to answer questions about genetic
328 underpinnings of musicality in general and musical affect in particular. Prior studies of
329 individual differences in music reward sensitivity^{12,15,16,48} have had far-reaching implications
330 for our knowledge of biological pathways implicated in perceptual-affective processes^{18–20}.
331 These studies have shown that individual differences in music reward sensitivity are
332 associated with variation in functional and structural connections between two systems. The
333 first includes the auditory cortex and its pathways involved in perceptual analysis, feature
334 encoding, and working memory. The second, the reward system, encompasses the striatum,
335 orbitofrontal cortex, and ventral tegmental area and is involved in pleasure, salience, and
336 learning^{1,2,16,19,20,49}. These neurobiological mechanisms could provide a potential substrate
337 for the genetic influences identified in the present study. Therefore, an important question
338 for future studies is to investigate whether variability in structural and functional properties
339 of the relevant brain networks, and their interactions, may mediate the genetic effects on the
340 ability to enjoy music, thus furthering our overall mechanistic understanding of a key aspect
341 of human affect.

342 A complementary genetic perspective on music reward sensitivity could be a particularly
343 fruitful strategy to better understand human musicality and affect because we found genetic
344 influences to be primarily separate from other relevant perceptual-affective processes, such
345 as music perceptual abilities and general reward sensitivity. The dissociation between the
346 genetics of music reward sensitivity and general perceptual and reward processing mirrors
347 the finding that specific musical anhedonia, i.e., blunted or absent hedonic responses from
348 music stimuli, exists in the absence of any perceptual or generalised reward deficit^{12,16}, yet
349 contrasts other findings suggesting sensitivity to intrinsic rewards to be domain general⁵⁰.
350 This implies that genetic variance associated with music reward sensitivity, beyond perceptual
351 and general reward processing, can be used to better disentangle and understand the
352 mechanisms involved in sensory-specific experiences of enjoyment.

353 The partial separation between genetic effects on perception and enjoyment also opens up
354 the possibility that genes influencing music perception and enjoyment may have been a
355 distinct target for natural selection during evolution⁵¹. The finding implies that genetic
356 variation between people may be used to dissect the evolutionary trajectories of different
357 aspects of human musicality. Along these lines, a further question of interest becomes
358 whether genetic variants, which are more specifically associated with music enjoyment, are
359 also enriched in genomic regions of evolutionary interest^{52,53}.

360 Here, we did not find support for a single overarching genetic factor of music reward
361 sensitivity. On the contrary, we found several distinct genetic pathways to music enjoyment.
362 This result aligns with general views of musicality as “built upon a suite of interconnected
363 capacities, of which none is primary”⁵⁴. Our results demonstrate that such heterogeneity is
364 seen even when zeroing in on one hypothesised core feature of musicality: enjoyment. We
365 show that music reward sensitivity is itself not a monolith and that different facets of this trait
366 are influenced by partly different genetic pathways; these facets range from the ability to
367 experience emotion and get chills to the rewarding aspects of social bonding through music.
368 Our results thus may challenge the epistemological status of music reward sensitivity as a
369 latent causal factor^{43,55,56}, as a latent factor is unlikely to hold unless a common-genetic factor
370 solution holds (for additional conditions, see⁴³).

371 Our final exploratory analysis provides a direct example of the implications such a shift in
372 perspective might have on the study of human behaviour and affect. When dissecting the
373 genetic effects at the level of the facets of music reward sensitivity, novel insights emerge.
374 Our findings indicate that music perceptual abilities are genetically more strongly correlated
375 with rewards of social bonding through music. This could be seen as in line with the social
376 bonding hypothesis, which states that “core biological components of human musicality
377 evolved as mechanisms supporting social bonding”⁵⁷. This was not the case for the
378 association between music reward and general reward sensitivity, which were relatively
379 similar across different facets. Furthermore, shared additive genetic variation entirely
380 explained the association between music perceptual abilities and social reward, suggesting
381 shared biological components to be at play. These results highlight how acknowledging the

382 genetic heterogeneity of music reward sensitivity might reveal associations that might
383 have been otherwise unnoticed. (For a detailed discussion on consequences for other well-
384 studied conditions, such as musical anhedonia^{12,15,16}, we refer to Supplementary Note 4.)

385 Notwithstanding such functionally relevant genetic heterogeneity, we also found genetic
386 overlap between the facets, suggesting genetic effects over music reward sensitivity are also
387 partially shared. This finding is important as some degree of genetic overlap across facets of
388 music reward sensitivity is needed to better understand the biology of music enjoyment as a
389 whole. Further studies could test whether these genetic effects underlie other auditory
390 phenomena, such as pleasure derived by timbre in sounds, which has been shown to correlate
391 homogeneously across facets of music reward²⁹ or other broader aspects related to human
392 affect, such as aesthetic sensitivity⁵⁸.

393 Finally, the absence of shared environmental effects on music reward sensitivity (at least
394 under the assumption of the classical twin design, see below) aligns with many other complex
395 traits, including those related to musicality^{26,46}. Yet, it contrasts with findings on some
396 musicality traits, such as musical achievement^{23,24} or singing abilities⁵⁹, for which modest
397 effects of shared environment have been found using similar designs. The lack of shared
398 environmental effects for some traits but not others suggests that different aspects of
399 musicality, namely producing music and enjoying music, might follow different patterns of
400 intergenerational transmission. The likely absence of shared environmental effects may imply
401 only a small, if present, passive gene-environment correlation (e.g., genotypes associated
402 with music reward sensitivity in the parents influence the children via the environment the
403 parents provide and the genes they pass on to their children, see⁶⁰). This is crucial because
404 passive gene-environment correlations would complicate future efforts to detect direct
405 genetic effects on music reward sensitivity by, e.g. confounding direct genetic effects with
406 indirect effects caused by the environment that the parents provide to their children (see^{60,61}
407 for a detailed discussion). Recent efforts to better understand the genetic architecture of
408 complex traits focus on deconstructing indirect sources of heritability, which inflate estimates
409 of genetic effects and confound the possible inferences that can be obtained from
410 downstream analysis of genome-wide-derived estimates⁶¹⁻⁶³. Our findings suggest that music
411 reward sensitivity, or rather its constituent facets, may be especially promising for facilitating
412 discoveries of direct molecular genetic effects on music enjoyment.

413 As with every other twin-informed study⁴², our work depends on a number of assumptions.
414 In the Methods section, we highlight these assumptions and what violation of each entails.
415 One critical assumption is the equal environment assumption, which states that
416 environmentally caused differences between twins within a pair are the same across
417 zygositys. An additional assumption is the lack of gene-by-shared environment interaction,
418 which could lead to an underestimation of the variance of the C component. For example,
419 additive genetic effects associated with music reward sensitivity might vary within different
420 musically enriched environments. However, we also note that the equal environment
421 assumption is not violated if different zygositys experience more similar or dissimilar

422 environments due to genetic differences. On the contrary, this is to be expected if evocative
423 and active gene-environment correlations are at play, which seems likely for traits related to
424 music enjoyment. Such gene-environment correlations would not inflate h^2 estimates. Still,
425 they would change their interpretation as they could reflect, for example, a more complex
426 causal chain that leads individuals to seek or be exposed to environmental changes that, in
427 turn, influence the phenotype, resulting in processes such as niche picking^{64,65}.

428 At the same time, our study also exploits one of the fundamental strengths of the CTD —the
429 possibility to estimate genetic effects on deep phenotypes, such as objectively assessed music
430 perceptual abilities and the full BMRQ, which are notoriously difficult to obtain in large
431 genetically informative samples⁶⁶. In light of the limitations and the strengths of the CTD, our
432 h^2_{twin} can be considered both an upper bound for the h^2 (within an environment, a population,
433 and at a given time) and provide valuable benchmarks for the total effect of DNA variation
434^{42,64,67} of music reward sensitivity and facets, above and beyond perceptual-affective
435 processes. These findings, as discussed in length above, generate novel insights and pave the
436 way for future research on the genetics of music enjoyment and human affect.

437

438 **Conclusions**

439 Musicality is the capacity that allows individuals of a species to perceive, generate, and enjoy
440 music^{14,54}. Much has been said about the sources of the considerable inter-individual
441 variation in music perception, production, participation, and achievement. Yet, relatively little
442 has been written on the genetic contribution to what makes individuals differ in their capacity
443 to enjoy music. Here, we add a new piece to the puzzle of why music has such powerful effects
444 on humans. We show that genes influencing our ability to enjoy music are largely distinct
445 from genes involved in other, more general aspects of perceptual and affective processing.
446 Further, we reveal that genetic pathways to music enjoyment are partially distinct and that
447 the genetic overlap between music perceptual abilities differs between different facets of
448 music reward. In summary, the findings highlight the complex and multifaceted nature of
449 music enjoyment and its genetic underpinnings, paving the way for further studies of the
450 evolutionary origins and genetic and neural mechanisms for a key aspect of human affect.

451

452 **Methods**

453 **Sample**

454 *Swedish Twin Registry: Screening Twin Adults Genes and Environment (STAGE)*. Participants
455 were twins recruited from the Swedish Twin Registry⁶⁸. Twin zygosity was determined by
456 questionnaire data, which, when compared to genotypes, has been shown to be 99% accurate
457 in the Swedish Twin Registry⁶⁹. The twins included in this study took part in two large recent
458 waves of online data collection on music, art and cultural engagement. In 2011 and then again
459 in 2022, a total of 32,000 adult twin individuals were invited from the STAGE cohort born
460 between 1959 and 1985, of which around 11,500 participated in the first wave and then

461 around 9,500 in the latest wave. More details on the survey can be found in Ullén et al. ¹³.
462 Participants took the Swedish Musical Discrimination Test (see below) in the first wave and
463 responded to the Behavioral Approach System and Barcelona Music Reward Questionnaire in
464 the second wave of data collection. A full description of the twin sample across waves of data
465 collection can be found in Table 1, including *n* of twins for which we had both data available,
466 stratified by the zygosity and the sex of the twins; for both waves of data collection, informed
467 consent was given by each participant before data gathering began. Both studies were
468 approved by the Regional Ethical Review Board in Stockholm (Dnrs 2011/570-31/5,
469 2012/1107/32, 2021-02014, 2022-00109-02, 2020-02575).

470 **Primary measure**

471 *Barcelona Music Reward Questionnaire (BMRQ)*. The Barcelona Music Reward
472 Questionnaire (BMRQ) is a psychometric tool used to assess musical anhedonia ^{12,16} and,
473 more generally, music reward sensitivity ¹¹, which has previously been validated across many
474 cultures ^{11,70–72}. It comprises 20 self-report items, with five response options, ranging from
475 completely disagree to completely agree. After recoding response items to numeric options
476 (1 to 5), with two out of 20 items being reverse coded, we used the sum score of the BMRQ
477 as a measure of music reward sensitivity (score range from 20 to 100). Following the original
478 five-factor structure ¹¹, we also created sum scores of the five known facets of music reward
479 sensitivity ²⁸: (1) Emotion-evocation - the degree to which individuals get emotional,
480 experience chills, and even cry when listening to music; (2) Mood regulation - the degree to
481 which individuals experience rewards from relaxing when listening to music; (3) Musical
482 seeking – the pleasure associated with the discovery of novel music-related information; (4)
483 Sensory motor – the rewards obtained from synchronising to an external beat or dancing; (5)
484 Social reward – the rewards of social bonding through music. Additional details are given in
485 Supplementary Note 5.

486 **Secondary measures**

487 *Behavioral Approach System Reward Responsiveness (BAS-RR)*. The Behavioral Approach
488 System (BAS) scale is included in the Behavioral Inhibition System (BIS)/BAS questionnaire, a
489 validated psychometric tool to assess inter-individual differences in two general motivational
490 systems ^{37,73}. The BAS-Reward Responsiveness (BAS-RR) scale, in particular, assesses inter-
491 individual differences in the ability to experience pleasure in the anticipation and presence of
492 reward-related stimuli and predicts general psychological adaptive functioning ⁷⁴. It
493 comprises five items, with four response options for each. BAS-RR is obtained by the sum
494 score of the five items after the numerical conversion of the responses (1-4). Additional
495 details are given in Supplementary Note 6.

496 *Swedish Musical Discrimination Test (SMDT)*. The Swedish Musical Discrimination Test
497 (SMDT) is a test that has good psychometric qualities for individual abilities in auditory
498 perceptual discrimination of musical stimuli ¹³. It comprises three subtests: melody, rhythm,
499 and pitch. A brief description of each test is given below (see ¹³ for more details).

500 *Melody*: This subtest used isochronous sequences of piano tones as stimuli. Tones ranged
501 from C4 to A#5, played at 650 ms intervals (American standard pitch; 262–932 Hz). The
502 number of tones increased from four to nine during the subtest progression. For each of the
503 six stimulus lengths, there were three items. The two stimuli in an item were separated by 1.3
504 s of silence. The pitch of one tone in the melody was always different in the second stimulus.
505 Participants had to identify which tone was different.

506 *Rhythm*: In this subtest, each item included two brief rhythmic sequences of 5-7 sine tones,
507 lasting 60 ms each. The inter-onset intervals between tones in a sequence were 150, 300, 450,
508 or 600 ms. The two sequences in an item were either identical or different, and separated by
509 1 s of silence. The participant had to determine whether the two sequences were the same
510 or not.

511 *Pitch*: The pitch subtest used sine tones with a 590 ms duration as stimuli. In each item, two
512 tones were presented, one of which always had a frequency of 500 Hz. The frequency of the
513 other tone was set between 501 and 517 Hz. The order of the two tones varied randomly,
514 with tones separated by a 1 s silence gap. Participants had to identify whether the first or the
515 second tone had the highest pitch. The item difficulty was increased progressively by
516 gradually making the pitch differences between the tones smaller.

517 **Analyses**

518 *Factor Analysis*. To confirm the BMRQ's sum score as an appropriate measure of music
519 reward sensitivity in the Swedish sample, we ran a one-factor Confirmatory Factor Analysis
520 (CFA) on the five facets of the Swedish version of the BMRQ. CFA was run on one twin per
521 pair, using the `lavaan::cfa()` function, to avoid sample dependence.

522 *Classical twin design (CTD)*. The CTD allows the estimation of additive (A) or dominance (D)
523 genetics, shared environmental (C), and residual source (E) of phenotypic variance (σ_A^2 , σ_D^2 ,
524 σ_C^2 , and σ_E^2 , respectively). This is possible given the expected phenotypic resemblance of
525 monozygotic (MZ) and dizygotic (DZ) twins. MZ arise from the same fertilised egg and thus
526 are ~100% genetically similar (with minimal deviations from expected genetic similarity, see
527 ⁷⁵); DZ arise from separate egg cells and thus, as ordinary siblings, share on average 50% of
528 their segregating genes. Furthermore, when both twins of a pair are raised in the same
529 household, MZ and DZ share 100% of their common environment. Finally, by definition,
530 remaining deviations from the expected values inferred by additive, dominant, and shared
531 environmental effects represent unique environmental influences and measurement errors.
532 Therefore, E is not shared between twins within a family. Under a set of assumptions,
533 including no epistasis (gene-by-gene interaction, see ⁷⁶), the covariance of MZ twin pairs is
534 then equal to:

535

536

$$\sigma_{MZ, MZ} = \sigma_A^2 + \sigma_D^2 + \sigma_C^2$$

537

538 While the covariance of DZ twin pairs is equal to:

539

$$540 \quad \sigma_{DZ,DZ} = .5*\sigma_A^2 + .25*\sigma_D^2 + \sigma_C^2$$

541 Given that the variance and covariance are measured between twins within families, it is
542 possible to specify a multigroup structural equation model and estimate three out of four
543 variance components. The decision of which parameters to include in the model (e.g., A, C, E,
544 or A, D, E) is purely based on twin covariances, which are extracted from the baseline
545 phenotypic model (for details on the baseline model, see below), and biological plausibility.
546 If $\sigma_{MZ, MZ} > 2*\sigma_{DZ,DZ}$, then D is expected to contribute to the phenotypic variance, and,
547 therefore, an ADE model is specified (note that DE models are not biologically plausible).
548 Otherwise, an ACE model is fit to the data.

549 *CTD assumptions.* The estimates from the CTD are unbiased under a set of assumptions. First,
550 the CTD assumes equal environments between the twins. In other words, it assumes that
551 similarities between twins caused by the environment are the same for both zygositys.
552 Suppose, instead, MZ experiences their environment more similarly than DZ due to
553 environmental, not genetic, causes. In that case, the estimate for the genetic variance will be
554 upwardly biased (i.e., $\widehat{\sigma}_A^2 > \sigma_A^2$). Note that the equal environment assumption is not violated
555 if MZ experiences their environment more similarly than DZ due to genetic differences. The
556 latter case would instead result in active gene-environment correlations that are still
557 consistent with the estimate of the variance components. The second assumption is that the
558 phenotypes of the parents of the twins' are uncorrelated (i.e., random mating, also known
559 as panmixia⁷⁷). If the covariance between two parental phenotypes, p_1 and p_2 , is different
560 from 0, $\sigma_{p_1,p_2} \neq 0$, then the shared environmental variance might be upwardly biased (i.e.,
561 $\widehat{\sigma}_C^2 > \sigma_C^2$). The third assumption is that there are no gene-environment interactions or gene-
562 environment passive correlations. Based on the gene-environment interaction, different
563 sources of bias are expected. If AxC is present, then $\widehat{\sigma}_A^2 > \sigma_A^2$ is expected. If AxE is present
564 instead, $\widehat{\sigma}_E^2 > \sigma_E^2$. If passive $r_{G,E}$ is present, then $\widehat{\sigma}_C^2 > \sigma_C^2$ is expected. An additional set of
565 assumptions introduced when estimating parameters via SEM is that means and variances
566 are equal across zygosity group, twin order (i.e., 1 and 2), and sex. Details on the latter set of
567 assumptions are given below. Complex sources of upward or downward biases in CTD-
568 informed models (e.g., heterogeneity) are discussed elsewhere⁷⁸.

569 *Baseline model.* We first fit multigroup SEM models to create a baseline against which to
570 compare the fit of univariate and multivariate models and test for the assumptions of the
571 equality of mean and variances. The models freely estimated all the observed variance and
572 covariances and included the age of the twins as a covariate. For the univariate model,
573 equality of means and variances was tested by sequentially constraining parameters and
574 comparing the Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) of
575 the model to the baseline model, where $AIC = 2k - 2\ln(\widehat{L})$ and $BIC = k\ln(k) - 2\ln(\widehat{L})$, with k
576 being the number of parameters estimated in the model and \widehat{L} the maximised value of the

577 likelihood function. Models with smaller AIC and BIC than the baseline model were deemed
578 a good fit. Additional comparisons are provided by the likelihood-ratio test (LRT), using the
579 `lavaan::lavTestLRT()` function from the `lavaan` R package ⁷⁹. All models were specified
580 following `lavaan` notation and fitted with the `lavaan::sem()` function.

581 *Univariate variance decomposition.* The SEM specification was informed by the CTD,
582 following the pattern of twin pairs correlations extracted from the baseline model and
583 baseline model comparison results. Twin pairs correlations were extracted using the most
584 parsimonious constrained baseline model using the `lavaan::standardizedSolutions()` function.
585 Precisely, we fit a five-group ADE sem model, where the five groups were formed by either
586 full or incomplete MZ female, MZ male, DZ female, DZ male, and DZ opposite-sex pairs. Means
587 for women and men were estimated freely across sex, but not across zygosity or twin order.
588 We fit the model via the direct symmetric approach by directly estimating the variances, as it
589 can derive asymptotically unbiased parameter estimates and is, therefore, less prone to type
590 I errors ⁸⁰. We then decomposed the variance-covariance matrix **T** of twin pairs into the **T = A**
591 **+ D + E** variance covariances, which was predicted as follows:

592

593

$$\mathbf{T} = \begin{bmatrix} \sigma_A^2 + \sigma_D^2 + \sigma_E^2 & \alpha * \sigma_A^2 + \delta * \sigma_D^2 \\ \alpha * \sigma_A^2 + \delta * \sigma_D^2 & \sigma_A^2 + \sigma_D^2 + \sigma_E^2 \end{bmatrix}$$

594

595 Where α is the expected additive genetic relationship, and δ is the expected dominant genetic
596 relationship between pairs (i.e., $\alpha = 1$ or $.5$ $\delta = 1$ or $.25$, for MZ and DZ, respectively). Note
597 that for simplicity, here we exclude the contribution of age to **T**, which was instead included
598 in the model. To test for the significance of the variance components A and D, we additionally
599 fit two models where D and AD variances were constrained to 0. Significance was inferred by
600 model comparison, as above. We fit the model to the raw sum score of the BMRQ using the
601 `lavaan::sem()` function. Assuming data within pairs were missing at random, we used the
602 recommended estimator for twin data analysis, the full information maximum likelihood
603 (FIML; argument estimator = "ML"). We used the following estimator for the narrow-sense
604 heritability:

605

606

$$h_{twin}^2 = \frac{\sigma_A^2}{\sigma_A^2 + \sigma_E^2}$$

607

608 Here we note the detail that $\sigma_A^2 + \sigma_E^2 \neq \sigma_P^2$, as $\sigma_P^2 = \sigma_A^2 + \sigma_E^2 + B^2 * \sigma_{Age}^2$. We also note that,
609 since the E component includes residual deviation, $\sigma_E^2 = \text{inter-}\sigma_E^2 + \text{intra-}\sigma_E^2$, where $\text{inter-}\sigma_E^2$
610 is the inter-individual variance, and $\text{intra-}\sigma_E^2$ is the intra-individual variance ⁷⁷. Comparisons
611 with standard OpenMX protocols are given in Supplementary Note 7 (note that the small
612 differences in test statistics did not lead to different conclusions). A graphical representation
613 of the full univariate multigroup model can be found in Supplementary Fig. 5.

614 *Sequential multivariate model.* The sequential multivariate modelling of SMDT, BAS-RR, and
615 BMRQ twin data was inspired by the classical multivariate Cholesky decomposition of additive
616 genetic (A) and environmental (E) matrices⁸¹. Following the CTD, we specified a multivariate
617 model to estimate variance components and between-components between-trait path
618 coefficients, λ_A and λ_E , based on the between-trait between-twin (also referred to as cross-
619 trait cross-twin) covariances. However, since variance components are directly estimated, it
620 is important to note that the sequential multivariate model is not exactly a Cholesky
621 decomposition. In fact, the predicted A and E variance-covariance matrices are not obtained
622 as $\mathbf{A} = \mathbf{X}\mathbf{X}^T$ or $\mathbf{E} = \mathbf{Z}\mathbf{Z}^T$, as in a Cholesky decomposition, where \mathbf{X} and \mathbf{Z} are the lower triangular
623 matrices with the path coefficients for the additive genetic and environmental components.
624 Instead, the 6x6 variance-covariance matrix \mathbf{S} was decomposed into symmetric matrices as \mathbf{S}
625 = $\mathbf{A} + \mathbf{E}$. As for the univariate case, the 6x6 symmetric matrices \mathbf{A} and \mathbf{E} include the predictions
626 for the phenotypic variances and the twin pair phenotypic covariances. For comparison, we
627 provide parameter estimates derived from the standardised solution, which is equivalent to
628 a Cholesky decomposition, in Supplementary Fig. 6. Additionally, the \mathbf{S} matrix also included
629 the predictions for the within-twin and the between-twin between-trait covariances. One
630 important consequence of our model specification is that we do not impose an implicit lower
631 bound of zero on the variance components, which can cause bias when comparing different
632 models. The sequence of variables was purely chosen to regress out A_1 and A_2 , respectively,
633 implied from SMDT and BAS-RR observed scores, from the BMRQ. To estimate an adjusted
634 heritability (here, for simplicity, $adj-h^2_{twin}$), we calculated the proportion of variance of the
635 BMRQ covarying with the component A over the total BMRQ variance (minus the variance in
636 BMRQ covarying with age):

637

$$638 \quad adj - h^2_{twin} = \frac{\sigma_{A3}^2}{\sigma_{A3}^2 + \sigma_{E3}^2 + \gamma_{A13}^2 * \sigma_{A1}^2 + \gamma_{E13}^2 * \sigma_{E1}^2 + \gamma_{A23}^2 * \sigma_{A2}^2 + \gamma_{E23}^2 * \sigma_{E2}^2}$$

639

640 Where the numerical subscripts simply indicate the order of phenotype in the model (e.g., 3
641 is the BMRQ). To calculate the amount of additive genetic variance unique and associated
642 with BMRQ beyond SMDT and BAS-RR ($\sigma_{Au:At}^2$, u=unique, t=total) we computed the
643 proportion of genetic variance over the total BMRQ additive genetic variance as follows:

644

$$645 \quad \sigma_{Au:At}^2 = \frac{\sigma_{A3}^2}{\sigma_{A3}^2 + \gamma_{A13}^2 * \sigma_{A1}^2 + \gamma_{A23}^2 * \sigma_{A2}^2}$$

646

647 A graphical representation of the full multivariate model can be found in Supplementary Fig.
648 7. Similar to what was reported above, we fit the models using the `lavaan::sem()` function
649 (estimator “ML”).

650 *Distinct factor solution.* To estimate the genetic and environmental correlations between
 651 facets of music reward, we applied a correlated factor model via direct symmetric approach
 652 ⁸⁰ (referred to as distinct factor solution). The direct symmetric approach is conceptually
 653 similar to a correlated factor solution. In the correlated factor solution, the multivariate
 654 phenotypic variance-covariance matrix \mathbf{M} is obtained as $\mathbf{M} = \mathbf{A} + \mathbf{E}$ (in the simplest case of an
 655 AE model), with $\mathbf{A} = \mathbf{X}\mathbf{R}_A\mathbf{X}^T$ and $\mathbf{E} = \mathbf{Z}\mathbf{R}_E\mathbf{Z}^T$, where \mathbf{X} and \mathbf{Z} are the diagonal matrix of the standard
 656 deviation σ_A and σ_E and \mathbf{R}_A is the genetic correlation matrix. Within a direct symmetric
 657 approach, instead, a different parametrisation is specified to directly estimate the \mathbf{M} 10x10
 658 symmetric matrix as $\mathbf{M} = \mathbf{A} + \mathbf{E}$:

659

$$660 \quad \mathbf{M} = \begin{bmatrix} \sigma_{A1}^2 + \sigma_{E1}^2 & \sigma_{A1,A2} + \sigma_{E1,E2} & \cdots & \alpha * \sigma_{A4}^2 & \alpha * \sigma_{A4,A5} \\ \sigma_{A1,A2} + \sigma_{E1,E2} & \sigma_{A2}^2 + \sigma_{E2}^2 & \vdots & \alpha * \sigma_{A4,A5} & \alpha * \sigma_{A5}^2 \\ \vdots & \vdots & \ddots & \vdots & \vdots \\ \alpha * \sigma_{A1}^2 & \alpha * \sigma_{A1,A2} & \vdots & \sigma_{A4}^2 + \sigma_{E4}^2 & \vdots \\ \alpha * \sigma_{A1,A2} & \alpha * \sigma_{A2}^2 & \cdots & \alpha * \sigma_{A4,A5} + \sigma_{E4,E5} & \sigma_{A5}^2 + \sigma_{E5}^2 \end{bmatrix}$$

661

662 Where the $\mathbf{M}_{1:5,1:5}$ and $\mathbf{M}_{5:10,5:10}$ elements include the within-twin variance and between-traits
 663 covariances and are constrained to equal across zygosity, and the $\mathbf{M}_{5:10,1:5}$ and $\mathbf{M}_{1:5,5:10}$
 664 elements include the between-twin additive genetic within- and between-trait covariances
 665 and the expected additive genetic relationship α , which is fixed to either 1 or .5 in MZ and DZ
 666 groups, respectively. While this approach may return out-of-bound values, the absence of
 667 boundaries has been shown to yield asymptotically unbiased parameter estimates and
 668 correct type I and type II error rates ⁸⁰. A graphical representation of the full multivariate
 669 model can be found in Supplementary Fig. 8. Model syntax was written following lavaan
 670 specifications. Model fitting was done via the lavaan: sem() function (estimator “ML”). In sum,
 671 the distinct factor solution provides a multivariate model for the decomposition of phenotypic
 672 variances and covariances in genetic and environmental components. Comparison of this
 673 model with more parsimonious independent pathway models allows us to test for the
 674 presence of a common genetic (or environmental) component shared across facets.

675

676 *Common-genetic factor solution.* The hybrid independent pathway model (referred to as
 677 common-genetic factor solution) is a multivariate approach similar to the correlated factor
 678 solution, except with an additional restriction on the genetic covariances between traits ($\sigma_{A,A}$;
 679 hence hybrid or genetic, as environmental covariances are modelled in a distinct factor
 680 solution fashion). Consider a 5x5 phenotypic variance covariance matrix \mathbf{P} . Under a HIPM AE
 681 model, \mathbf{P} can be written as $\mathbf{P} = \mathbf{A}_c + \mathbf{A}_u + \mathbf{E}$, where $\mathbf{A}_c = \mathbf{X}_c\mathbf{X}_c^T$, with \mathbf{X}_c being a 5x1 vector of the
 682 additive genetic path coefficients of a common additive genetic factor (A_c) loading across all
 683 phenotypes, and \mathbf{A}_u is a 5x5 diagonal matrix including the residual unique genetic variance
 684 for each phenotype, $\sigma_{A_u}^2$. The full additive genetic variance-covariance matrix can be then as
 685 follows:

$$686 \quad \mathbf{A}_t = \mathbf{X}_c \mathbf{X}_c^T + \mathbf{A}_u = \begin{bmatrix} \lambda_{A1}^2 + \sigma_{Au1}^2 & \lambda_{A1} * \lambda_{A2} & \lambda_{A1} * \lambda_{A3} & \lambda_{A1} * \lambda_{A4} & \lambda_{A1} * \lambda_{A5} \\ \lambda_{A1} * \lambda_{A2} & \lambda_{A2}^2 + \sigma_{Au2}^2 & \lambda_{A2} * \lambda_{A3} & \lambda_{A2} * \lambda_{A4} & \lambda_{A2} * \lambda_{A5} \\ \lambda_{A1} * \lambda_{A3} & \lambda_{A2} * \lambda_{A3} & \lambda_{A3}^2 + \sigma_{Au3}^2 & \lambda_{A3} * \lambda_{A4} & \lambda_{A3} * \lambda_{A5} \\ \lambda_{A1} * \lambda_{A4} & \lambda_{A2} * \lambda_{A4} & \lambda_{A3} * \lambda_{A4} & \lambda_{A4}^2 + \sigma_{Au4}^2 & \lambda_{A4} * \lambda_{A5} \\ \lambda_{A1} * \lambda_{A5} & \lambda_{A2} * \lambda_{A5} & \lambda_{A3} * \lambda_{A5} & \lambda_{A4} * \lambda_{A5} & \lambda_{A5}^2 + \sigma_{Au5}^2 \end{bmatrix}$$

687

688 The 5X5 residual environmental covariance \mathbf{E} simply contains the unconstrained residual
 689 environmental variances and covariances σ_E^2 and $\sigma_{E,E}$. The 10X10 between-facet between-
 690 twin matrix \mathbf{M} can then be written as follows:

691

692

$$\mathbf{M} = \begin{bmatrix} \mathbf{A}_t + \mathbf{E} & \alpha * \mathbf{A}_t \\ \alpha * \mathbf{A}_t & \mathbf{A}_t + \mathbf{E} \end{bmatrix}$$

693

694 Where α is the expected additive genetic relationship between twins and is fixed to either 1
 695 or .5 across MZ and DZ groups, respectively. A graphical representation of the full multivariate
 696 model can be found in Supplementary Fig. 9. Model syntax was written in lavaan. Model
 697 fitting was done via the lavaan:sem() function. Model comparison between distinct and
 698 common-genetic factor solutions was carried out via the lavaan::lavTestLRT() function. Here,
 699 we additionally note that the common-genetic factor solution is a less parsimonious
 700 version of the more commonly used independent pathway model and, therefore, provides a
 701 less restrictive and more specific test for a genetic common factor when compared to the
 702 distinct factor solution.

703 *Structural equation modeling assumptions.* SEM-based estimates obtained from the full
 704 information maximum likelihood (FIML) estimator are unbiased under the assumption that
 705 observations follow a multivariate normal distribution⁴¹. Violation of the assumption of
 706 multivariate normality has been found to have little impact on parameter estimates but can
 707 have severe consequences for both the χ^2 test statistics and the standard error of the
 708 estimates for the parameters. An alternative estimator that is less sensitive or robust to
 709 violation of multivariate normality is the maximum likelihood with robust standard error and
 710 scaled test statistics (MLR). Although this estimator assumes missingness to be completely at
 711 random, it has been shown to provide quite reliable estimates of data missing at random⁸².
 712 Relevant comparisons between the two estimators are given in Supplementary Note 1.

713

714 Data availability

715 The datasets generated during the current study cannot be made public as registry data were
 716 used. However, researchers are able to apply online at the Swedish Twin Registry to access
 717 the twin data used in this study (see <https://ki.se/en/research/swedish-twin-registry-for-researchers>).
 718

719

720 **Code availability**

721 All scripts and code used to analyse the data can be found at:

722 https://github.com/giacomobignardi/h2_BMRQ.

723

724 **Acknowledgements**

725 We thank the Swedish twins for their participation. The Swedish Twin Registry is managed by
726 Karolinska Institutet and receives funding through the Swedish Research Council under the
727 grant no 2017-00641. We further thank Anirudh D. Patel for his critical feedback and Cristina
728 Gonzalez-Liencre for her input on earlier versions of the figures and figures' captions. We
729 also would like to thank the participants of the 2023 Nijmegen *Musicality Genomics*
730 *Consortium* meeting (<https://www.mcg.uva.nl/musicgens/program.html>) for insightful
731 discussions that have led to an improved version of the current work. G.B. was supported by
732 the German Federal Ministry of Education and Research (BMBF); G.B. and S.E.F. were
733 supported by the Max Planck Society.

734

735 **Contributions**

736 G.B. conceived the study, analysed and visualised the data; G.B. and M.M. drafted the
737 manuscript; F.U., S.E.F., and M.M. supervised the research; L.W.W. validated the work; S.E.F,
738 M.M., R.J.Z., L.W.W., and F.U. conceptually validated the work; all authors revised and
739 reviewed the last version of this manuscript.

740

741 **References**

- 742 1. Zatorre, R. J. & Salimpoor, V. N. From perception to pleasure: Music and its neural
743 substrates. *Proc. Natl. Acad. Sci.* **110**, 10430–10437 (2013).
- 744 2. Zatorre, R. *From Perception to Pleasure: The Neuroscience of Music and Why We Love It*.
745 (Oxford University Press, 2023). doi:10.1093/oso/9780197558287.001.0001.
- 746 3. Koelsch, S. Brain correlates of music-evoked emotions. *Nat. Rev. Neurosci.* **15**, 170–180
747 (2014).
- 748 4. Cowen, A. S., Fang, X., Sauter, D. & Keltner, D. What music makes us feel: At least 13
749 dimensions organize subjective experiences associated with music across different
750 cultures. *Proc. Natl. Acad. Sci.* **117**, 1924–1934 (2020).
- 751 5. Mehr, S. A. *et al.* Universality and diversity in human song. *Science* **366**, (2019).
- 752 6. McDermott, J. The evolution of music. *Nature* **453**, 287–288 (2008).
- 753 7. Darwin, C. *The Descent of Man*. (D. Appleton and Company, 1871).
- 754 8. Bountiful noise. *Nature* **453**, 134–134 (2008).
- 755 9. Sacks, O. The power of music. *Brain* **129**, 2528–2532 (2006).

- 756 10. Müllensiefen, D., Gingras, B., Musil, J. & Stewart, L. Measuring the facets of musicality:
757 The Goldsmiths Musical Sophistication Index (Gold-MSI). *Personal. Individ. Differ.* **60**,
758 S35 (2014).
- 759 11. Mas-Herrero, E., Marco-Pallares, J., Lorenzo-Seva, U., Zatorre, R. J. & Rodriguez-Fornells,
760 A. Individual Differences in Music Reward Experiences. *Music Percept.* **31**, 118–138
761 (2013).
- 762 12. Mas-Herrero, E., Zatorre, R. J., Rodriguez-Fornells, A. & Marco-Pallarés, J. Dissociation
763 between Musical and Monetary Reward Responses in Specific Musical Anhedonia. *Curr.*
764 *Biol.* **24**, 699–704 (2014).
- 765 13. Ullén, F., Mosing, M. A., Holm, L., Eriksson, H. & Madison, G. Psychometric properties
766 and heritability of a new online test for musicality, the Swedish Musical Discrimination
767 Test. *Personal. Individ. Differ.* **63**, 87–93 (2014).
- 768 14. Honing, H., ten Cate, C., Peretz, I. & Trehub, S. E. Without it no music: cognition, biology
769 and evolution of musicality. *Philos. Trans. R. Soc. B Biol. Sci.* **370**, 20140088 (2015).
- 770 15. Martínez-Molina, N., Mas-Herrero, E., Rodríguez-Fornells, A., Zatorre, R. J. & Marco-
771 Pallarés, J. White Matter Microstructure Reflects Individual Differences in Music Reward
772 Sensitivity. *J. Neurosci.* **39**, 5018–5027 (2019).
- 773 16. Martínez-Molina, N., Mas-Herrero, E., Rodríguez-Fornells, A., Zatorre, R. J. & Marco-
774 Pallarés, J. Neural correlates of specific musical anhedonia. *Proc. Natl. Acad. Sci.* **113**,
775 E7337–E7345 (2016).
- 776 17. Peretz, I. Neurobiology of Congenital Amusia. *Trends Cogn. Sci.* **20**, 857–867 (2016).
- 777 18. Blood, A. J. & Zatorre, R. J. Intensely pleasurable responses to music correlate with
778 activity in brain regions implicated in reward and emotion. *Proc. Natl. Acad. Sci.* **98**,
779 11818–11823 (2001).
- 780 19. Salimpoor, V. N., Benovoy, M., Larcher, K., Dagher, A. & Zatorre, R. J. Anatomically
781 distinct dopamine release during anticipation and experience of peak emotion to music.
782 *Nat. Neurosci.* **14**, 257–262 (2011).
- 783 20. Salimpoor, V. N. *et al.* Interactions Between the Nucleus Accumbens and Auditory
784 Cortices Predict Music Reward Value. *Science* **340**, 216–219 (2013).
- 785 21. Niarchou, M. *et al.* Genome-wide association study of musical beat synchronization
786 demonstrates high polygenicity. *Nat. Hum. Behav.* **6**, 1292–1309 (2022).
- 787 22. Vinkhuyzen, A. A. E., van der Sluis, S., Posthuma, D. & Boomsma, D. I. The Heritability of
788 Aptitude and Exceptional Talent Across Different Domains in Adolescents and Young
789 Adults. *Behav. Genet.* **39**, 380–392 (2009).
- 790 23. Wesseldijk, L. W., Mosing, M. A. & Ullén, F. Why Is an Early Start of Training Related to
791 Musical Skills in Adulthood? A Genetically Informative Study. *Psychol. Sci.* **32**, 3–13
792 (2021).
- 793 24. Hambrick, D. Z. & Tucker-Drob, E. M. The genetics of music accomplishment: Evidence
794 for gene–environment correlation and interaction. *Psychon. Bull. Rev.* **22**, 112–120
795 (2015).

- 796 25. Wesseldijk, L. W., Mosing, M. A. & Ullén, F. Gene-environment interaction in expertise:
797 The importance of childhood environment for musical achievement. *Dev. Psychol.* **55**,
798 1473–1479 (2019).
- 799 26. Wesseldijk, L. W., Ullén, F. & Mosing, M. A. Music and Genetics. *Neurosci. Biobehav. Rev.*
800 **152**, 105302 (2023).
- 801 27. Belfi, A. M. & Loui, P. Musical anhedonia and rewards of music listening: current
802 advances and a proposed model. *Ann. N. Y. Acad. Sci.* **1464**, 99–114 (2020).
- 803 28. Mas-Herrero, E. Auditory pleasure elicited by music. in *The Routledge International*
804 *Handbook of Neuroaesthetics* (Routledge, 2022).
- 805 29. Kathios, N., Patel, A. D. & Loui, P. Musical anhedonia, timbre, and the rewards of music
806 listening. *Cognition* **243**, 105672 (2024).
- 807 30. Kathios, N., Sachs, M. E., Zhang, E., Ou, Y. & Loui, P. Generating New Musical
808 Preferences From Multilevel Mapping of Predictions to Reward. *Psychol. Sci.* **35**, 34–54
809 (2024).
- 810 31. Mas-Herrero, E., Karhulahti, M., Marco-Pallares, J., Zatorre, R. J. & Rodriguez-Fornells, A.
811 The impact of visual art and emotional sounds in specific musical anhedonia. *Prog. Brain*
812 *Res.* **237**, 399–413 (2018).
- 813 32. Cardona, G., Rodriguez-Fornells, A., Nye, H., Rifà-Ros, X. & Ferreri, L. The impact of
814 musical pleasure and musical hedonia on verbal episodic memory. *Sci. Rep.* **10**, 16113
815 (2020).
- 816 33. Ferreri, L. & Rodriguez-Fornells, A. Music-related reward responses predict episodic
817 memory performance. *Exp. Brain Res.* **235**, 3721–3731 (2017).
- 818 34. Ferreri, L. *et al.* Dopamine modulations of reward-driven music memory consolidation.
819 *Ann. N. Y. Acad. Sci.* **1502**, 85–98 (2021).
- 820 35. Hernández, M. *et al.* Separate Contribution of Striatum Volume and Pitch Discrimination
821 to Individual Differences in Music Reward. *Psychol. Sci.* **30**, 1352–1361 (2019).
- 822 36. Gold, B. P. *et al.* Musical reward prediction errors engage the nucleus accumbens and
823 motivate learning. *Proc. Natl. Acad. Sci.* **116**, 3310–3315 (2019).
- 824 37. Carver, C. S. & White, T. L. Behavioral inhibition, behavioral activation, and affective
825 responses to impending reward and punishment: The BIS/BAS Scales. *J. Pers. Soc.*
826 *Psychol.* **67**, 319–333 (1994).
- 827 38. Schlotz, W. *et al.* The Aesthetic Responsiveness Assessment (AReA): A screening tool to
828 assess individual differences in responsiveness to art in English and German. *Psychol.*
829 *Aesthet. Creat. Arts* **15**, 682–696 (2021).
- 830 39. Fuentes-Sánchez, N., Pastor, M. C., Eerola, T. & Pastor, R. Individual differences in music
831 reward sensitivity influence the perception of emotions represented by music. *Music.*
832 *Sci.* **27**, 313–331 (2023).
- 833 40. Belfi, A. M., Moreno, G. L., Gugliano, M. & Neill, C. Musical reward across the lifespan.
834 *Aging Ment. Health* **26**, 932–939 (2022).

- 835 41. Finney, S. J. & DiStefano, C. Nonnormal and categorical data in structural equation
836 modeling. in *Structural equation modeling: A second course, 2nd ed* 439–492 (IAP
837 Information Age Publishing, Charlotte, NC, US, 2013).
- 838 42. Willoughby, E. A., Polderman, T. J. C. & Boutwell, B. B. Behavioural genetics methods.
839 *Nat. Rev. Methods Primer* **3**, 1–16 (2023).
- 840 43. Franić, S. *et al.* Can genetics help psychometrics? Improving dimensionality assessment
841 through genetic factor modeling. *Psychol. Methods* **18**, 406–433 (2013).
- 842 44. Shapland, C. Y. *et al.* Multivariate genome-wide covariance analyses of literacy, language
843 and working memory skills reveal distinct etiologies. *Npj Sci. Learn.* **6**, 1–12 (2021).
- 844 45. Mazancieux, A. *et al.* Towards a common conceptual space for metacognition in
845 perception and memory. *Nat. Rev. Psychol.* **2**, 751–766 (2023).
- 846 46. Polderman, T. J. C. *et al.* Meta-analysis of the heritability of human traits based on fifty
847 years of twin studies. *Nat. Genet.* **47**, 702–709 (2015).
- 848 47. Turkheimer, E. Three Laws of Behavior Genetics and What They Mean. *Curr. Dir. Psychol.*
849 *Sci.* **9**, 160–164 (2000).
- 850 48. Mas-Herrero, E., Dagher, A. & Zatorre, R. J. Modulating musical reward sensitivity up
851 and down with transcranial magnetic stimulation. *Nat. Hum. Behav.* **2**, 27–32 (2018).
- 852 49. Mas-Herrero, E., Maini, L., Sescousse, G. & Zatorre, R. J. Common and distinct neural
853 correlates of music and food-induced pleasure: A coordinate-based meta-analysis of
854 neuroimaging studies. *Neurosci. Biobehav. Rev.* **123**, 61–71 (2021).
- 855 50. Blain, B., Pinhorn, I. & Sharot, T. Sensitivity to intrinsic rewards is domain general and
856 related to mental health. *Nat. Ment. Health* **1**, 679–691 (2023).
- 857 51. Patel, A. D. Human Musicality and Gene-Culture Coevolution: Ten Concepts to Guide
858 Productive Exploration. (2023) doi:10.7551/mitpress/14186.003.0006.
- 859 52. Alagöz, G. *et al.* Using neuroimaging genomics to investigate the evolution of human
860 brain structure. *Proc. Natl. Acad. Sci.* **119**, e2200638119 (2022).
- 861 53. Alagöz, G. *et al.* The shared genetic architecture and evolution of human language and
862 musical rhythm. 2023.11.01.564908 Preprint at
863 <https://doi.org/10.1101/2023.11.01.564908> (2023).
- 864 54. Fitch, W. T. Four principles of bio-musicology. *Philos. Trans. R. Soc. B Biol. Sci.* **370**,
865 20140091 (2015).
- 866 55. Borsboom, D., Mellenbergh, G. J. & Van Heerden, J. The theoretical status of latent
867 variables. *Psychol. Rev.* **110**, 203–219 (2003).
- 868 56. Clapp Sullivan, M. L. *et al.* Beyond the factor indeterminacy problem using genome-wide
869 association data. *Nat. Hum. Behav.* 1–14 (2024) doi:10.1038/s41562-023-01789-1.
- 870 57. Savage, P. E. *et al.* Music as a coevolved system for social bonding. *Behav. Brain Sci.* **44**,
871 e59 (2021).
- 872 58. Bignardi, G., Chamberlain, R., Kevenaar, S. T., Tamimy, Z. & Boomsma, D. I. On the
873 etiology of aesthetic chills: a behavioral genetic study. *Sci. Rep.* **12**, 3247 (2022).
- 874 59. Yeom, D. *et al.* Genetic factors and shared environment contribute equally to objective
875 singing ability. *iScience* **25**, 104360 (2022).

- 876 60. Hart, S. A., Little, C. & van Bergen, E. Nurture might be nature: cautionary tales and
877 proposed solutions. *Npj Sci. Learn.* **6**, 1–12 (2021).
- 878 61. Morris, T. T., Davies, N. M., Hemani, G. & Smith, G. D. Population phenomena inflate
879 genetic associations of complex social traits. *Sci. Adv.* **6**, eaay0328 (2020).
- 880 62. Young, A. I., Benonisdottir, S., Przeworski, M. & Kong, A. Deconstructing the sources of
881 genotype-phenotype associations in humans. *Science* **365**, 1396–1400 (2019).
- 882 63. Howe, L. J. *et al.* Within-sibship genome-wide association analyses decrease bias in
883 estimates of direct genetic effects. *Nat. Genet.* **54**, 581–592 (2022).
- 884 64. Hagenbeek, F. A. *et al.* Maximizing the value of twin studies in health and behaviour.
885 *Nat. Hum. Behav.* **7**, 849–860 (2023).
- 886 65. Eaves, L., Foley, D. & Silberg, J. Has the “Equal Environments” Assumption Been Tested
887 in Twin Studies? *Twin Res. Hum. Genet.* **6**, 486–489 (2003).
- 888 66. Friedman, N. P., Banich, M. T. & Keller, M. C. Twin studies to GWAS: there and back
889 again. *Trends Cogn. Sci.* **25**, 855–869 (2021).
- 890 67. Cheesman, R., Ayorech, Z., Eilertsen, E. M. & Ystrom, E. Why we need families in
891 genomic research on developmental psychopathology. *JCPP Adv.* **3**, e12138 (2023).
- 892 68. Zagai, U., Lichtenstein, P., Pedersen, N. L. & Magnusson, P. K. E. The Swedish Twin
893 Registry: Content and Management as a Research Infrastructure. *Twin Res. Hum. Genet.*
894 **22**, 672–680 (2019).
- 895 69. Lichtenstein, P. *et al.* The Swedish Twin Registry: a unique resource for clinical,
896 epidemiological and genetic studies. *J. Intern. Med.* **252**, 184–205 (2002).
- 897 70. Wang, J. *et al.* The Chinese version of the Barcelona Music Reward Questionnaire
898 (BMRQ): Associations with personality traits and gender. *Music. Sci.* **27**, 218–232 (2023).
- 899 71. Saliba, J. *et al.* French validation of the Barcelona Music Reward Questionnaire. *PeerJ* **4**,
900 e1760 (2016).
- 901 72. Honda, S. *et al.* The Japanese Version of the Barcelona Music Reward Questionnaire (J-
902 BMRQ) Confirms the Cross-Cultural Generalizability of the “Five-Factor” Model. (2024)
903 doi:10.31234/osf.io/7qp45.
- 904 73. Eriksson, L. J. K., Jansson, B. & Sundin, Ö. Psychometric properties of a Swedish version
905 of the reinforcement sensitivity theory of personality questionnaire. *Nord. Psychol.* **71**,
906 134–145 (2019).
- 907 74. Taubitz, L. E., Pedersen, W. S. & Larson, C. L. BAS Reward Responsiveness: A unique
908 predictor of positive psychological functioning. *Personal. Individ. Differ.* **80**, 107–112
909 (2015).
- 910 75. Jonsson, H. *et al.* Differences between germline genomes of monozygotic twins. *Nat.*
911 *Genet.* **53**, 27–34 (2021).
- 912 76. Keller, M. C. & Coventry, W. L. Quantifying and addressing parameter indeterminacy in
913 the classical twin design. *Twin Res. Hum. Genet. Off. J. Int. Soc. Twin Stud.* **8**, 201–213
914 (2005).
- 915 77. Falconer, D. S. Introduction to quantitative genetics. *Introd. Quant. Genet.* (1960).

- 916 78. Wray, N. R. & Maier, R. Genetic Basis of Complex Genetic Disease: The Contribution of
917 Disease Heterogeneity to Missing Heritability. *Curr. Epidemiol. Rep.* **1**, 220–227 (2014).
918 79. Rosseel, Y. lavaan: An R Package for Structural Equation Modeling. *J. Stat. Softw.* **48**, 1–
919 36 (2012).
920 80. Verhulst, B., Prom-Wormley, E., Keller, M., Medland, S. & Neale, M. C. Type I Error Rates
921 and Parameter Bias in Multivariate Behavioral Genetic Models. *Behav. Genet.* **49**, 99–
922 111 (2019).
923 81. Neale, M. C. & Cardon, L. R. Multivariate Analysis. in *Methodology for Genetic Studies of*
924 *Twins and Families* (eds. Neale, M. C. & Cardon, L. R.) 231–259 (Springer Netherlands,
925 Dordrecht, 1992). doi:10.1007/978-94-015-8018-2_12.
926 82. Yuan, K.-H. & Bentler, P. M. 5. Three Likelihood-Based Methods for Mean and
927 Covariance Structure Analysis with Nonnormal Missing Data. *Sociol. Methodol.* **30**, 165–
928 200 (2000).
929