Supplementary information for *Distinct genetic pathways to music enjoyment*

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Supplementary Notes

Supplementary Note 1. *Test statistics for alternative estimators.* Here, we provide robust test statistics for model comparison or parameters for which we used the maximum likelihood estimator with robust ("MLR") sandwich (Huber-white) standard error and test statistic [1]. More information on how lavaan [2] handles robust test statistics can be found at https://users.ugent.be/~yrosseel/lavaan/lavaan2.pdf. Below, we contrast the estimates obtained from the MLR estimator with the results obtained from the full-information maximum likelihood (ML) estimator used in the main analyses.

- *Baseline univariate model:*
  Contrast between comparison between models with and without constrained means across sex. Results for the estimator = "ML": $\chi^2(30)_{\Delta df} = 297.5$, $p < 0.001$. Results for the estimator = MLR: $\chi^2(30)_{\Delta df} = 288.6$, $p < 0.001$. *Contrast between standard error (se) and significance of the age as a manifest covariate*. Results for the estimator = "ML": *se* = .011; $p = 0.004$; Results for the estimator = "MLR": *se* = .011, $p = 0.003$. Therefore, conclusions that BMRQ means differ across sex, and that age significantly predicts BMRQ differences are unaltered when using the MLR estimator.
- *ADE univariate model. Contrast between model comparison between models with and without the A component.* Results for the estimator = "ML": $\chi^2(1) = 458.49$, $p < .001$; Results for the estimator = "MLR": $\chi^2(1)_{\Delta df} = 534.51$, $p < 0.001$. Therefore, the conclusion that dropping the A component significantly deteriorates model fit remains unaltered when using the MLR estimator.
- *Sequential approach. Contrast between significance of the path coefficients*. Results for the estimator = "ML" for the path coefficients are provided in Supplementary Table 4. Similarly, Results for the estimator = "MLR" for the path coefficients are provided in Supplementary Table 4. Notably, all genetic and environmental correlations reported as significant in the main remain significant using the MLR estimator.
- *Distinct factors solution vs common-genetic factor solution. Contrast between model comparison test statistics to infer the absence of a single common genetic pathway.* Results for the estimator = ML: $\chi^2(5)_{\Delta df} = 129.61$, $p < 0.001$. Results for the estimator = MLR: $\chi^2(5)_{\Delta df} = 129.51$, $p < 0.001$. Therefore, the main finding remains unaltered when using different estimators.

Supplementary Note 2. *Alternative causal models for the sequential decomposition.* In the main text, we note that the variance shared between music perceptual abilities and music reward sensitivity could be mainly due to shared genetic factors acting as common causes. This can be drawn as the direct acyclic graph [3] depicted in Supplementary Fig. 10. However, there are more alternative models that could better describe the data-generating process behind the covariances observed (and implied) in the main text. It is possible for the genetic effects shared between the two traits to not act as a common cause. Instead, one trait could mediate genetic effects on one of the two phenotypes. Suppose we ask: What generates the observed association between music perceptual abilities and music reward sensitivity? Then Supplementary Fig. 10 could guide future research, in which alternative causal models, which we could not appropriately assess in this study, could be put to the test (e.g., model Fig. 10 D). Nevertheless, we note that, as our main interest was to quantify the magnitude of heritability for music reward sensitivity beyond shared genetic effects with music perceptual abilities, our main conclusion holds, regardless of the actual causal structure. The only exception would be under reverse causal models depicted in Supplementary Fig. 10 E-G, as the heritability of music perceptual abilities would not explain the heritability of music reward sensitivity but vice versa. However, we would like to note that under this latter scenario, our estimates are still conservative estimates of the adjusted heritability of music reward sensitivity.

Supplementary Note 3. *Genetic heterogeneity between facets of music reward is robust.* Despite no evidence for a common factor model, the genetic correlations between the facets suggest the possibility of multiple common-genetic factors. To explore this possibility, following a similar protocol applied by others [4], we applied a principal component analysis of the additive genetic correlation matrix extracted from the correlated factor solution (hereafter referred to as PCA_A). To get an alternative data-driven estimate of the dissimilarity between the genetic factors, we decomposed the 5x5 additive genetic correlation matrix **A** into its eigenvalues and eigenvectors (i.e., spectral decomposition). In Supplementary Fig. 11, following standard PCA practices [5], we plot the two eigenvectors explaining the majority of genetic variance, $PC1_A$ and $PC2_A$. As can be seen, the sensorimotor facet is maximally distinct from every other facet. This indicated that genetic variances for the sensory-motor are mainly differentiated from emotion-evocation, mood-regulation, and social-rewards facets. Or, in other words the genetic variance associated with the sensory-motor component differs notably from the other facets. Based on the results of the PCA_A, we respecified multivariate analysis, excluding the sensory-motor facet from the analysis. We fit a correlated factor model via a direct symmetric approach and a hybrid independent pathway model (Note these two models are identical to the distinct factor and common-genetic factor solutions described in the Methods) to emotion-evocation, mood regulation, music seeking, and social reward facets data. The comparison between re-fitted multivariate models, excluding the sensory-motor facet, still indicated some genetic heterogeneity across emotion-evocation, mood regulation, musical seeking and social reward facets (AIC = 230465 and BIC = 230725, AIC = 230503 and BIC =230749, respectively; $\chi^2(2)_{\Delta df} = 42.14$, $p < 0.001$). This conclusion did not change by using the different "MLR" estimator ($\chi^2(2)_{\Delta df} = 45.38$, $p < 0.001$). This confirms that a single common-genetic factor model of music reward sensitivity facets is a worse solution than the distinct factor model, even when excluding maximally genetically distinct facets.

Supplementary Note 4. Although we found substantial genetic heterogeneity across facets (see Supplementary Note 3), the dissimilarity of the genetic effects exerted across facets is moderate. As such, our results are also consistent with moderate to large genetic pleiotropy across different facets of music reward sensitivity. These results could explain why using music reward sensitivity as a tool to, for example, cluster individuals in different categories of musical an- or hyper-hedonics has been successful as a strategy to link individual differences in music enjoyment to physiological and neurobiological differences [6–9]. Musical anhedonia is a well-studied condition that is "diagnosed" in individuals with a BMRQ total score less than a certain threshold, but that have no deficit in music perception or general hedonic processes. Previous studies indicate that this condition relates to physiological and neurobiological differences, notably indicating that individuals with musical anhedonia tend to show altered structural and functional connectivity in cortico-subcortical loops along the auditory ventral stream [7,8,10]. However, our findings suggest that two musical an- or hyper-hedonics might not be sensitive to music alike. Suppose genetic pathways to music reward sensitivity are partly distinct between different facets of music reward. In that case, different individuals coexisting in the same musical anhedonia group are likely to carry different DNA variants. This might, therefore, mask some neurobiological differences associated with within-facet (e.g., sensory-motor) differences. For example, genetic variants influencing facets of music reward that are more tightly genetically correlated (e.g., genetic variants associated with emotion evocation, mood regulation and social reward facets) could enhance the detection of neurobiological associations that are either preferentially or strongly covarying with such facets, while blunting detection of neurobiological pathways associated with other facets. This genetic heterogeneity could explain why previous studies found specific associations between alteration in cortico-subcortical loops along the ventral but not the dorsal stream [7,8]. The finding of a lower genetic correlation estimated for the sensory-motor facet could be also interpreted in light of the differentiation of the two streams' (ventral and dorsal) role in different aspects of music cognition, from perception to pleasure [11]. Based on previous models [10–12], we indeed speculate that genetic variants associated with structural and functional connectivity differences along these two streams can partially capture the genetic dissociation found in this study. This speculation can be further formalised into testable hypotheses that can be falsified. For example, further study could test whether genetic correlations between different facets of music reward sensitivity and functional or structural connectivity between selected areas along the ventral and dorsal stream vary.

Supplementary Note 5. *Barcelona music reward questionnaire* [13]. The English version of the questionnaire is outlined below. For simplicity, reversed-scored items (that is, items for which a lower rating in Likert-items indicates the opposite score, e.g., *completely disagree = 5)* are indicated by the superscript r. The facets to which items belong are indicated between parentheses.

Instructions:

Each item of this questionnaire is a statement that a person may either agree with or disagree with. For each item, indicate how much you agree or disagree with what the item says. Please respond to all the items; do not leave any blank. Choose only one response to each statement. Please be as accurate and honest as you can be. Respond to each item as if it were the only item. That is, do not worry about being consistent in your responses. Choose from completely disagree (left) to completely agree (right) one of the five options.

Response options:

1;2;3;4;5

completely disagree; disagree; neither agree nor disagree; agree; completely agree.

Items:

1. When I share music with someone I feel a special connection with that person. (Social reward)

2. In my free time I hardly listen to music.[r] (Music seeking)

3. I like listen to music that contains emotion. (Emotion evocation)

4. Music keeps me company when I'm alone. (Mood regulation)

5. I don't like to dance, not even with music I like.[r] (Sensory motor)

6. Music makes me bond with other people. (Social reward)

7. I inform myself about music I like. (Music seeking)

8. I get emotional listening to certain pieces of music. (Emotion evocation)

9. Music calms and relaxes me. (Mood regulation)

10. Music often makes me dance. (Sensory motor)

11. I'm always looking for new music. (Music seeking)

12. I can become tearful or cry when I listen to a melody that I like very much. (Emotion evocation)

13. I like to sing or play an instrument with other people. (Social reward)

14. Music helps me chill out. (Mood regulation)

15. I can't help humming or singing along to music that I like. (Sensory motor)

16. At a concert I feel connected to the performers and the audience. (Social reward)

17. I spend quite a bit of money on music and related items. (Music seeking)

18. I sometimes feel chills when I hear a melody that I like. (Emotion evocation)

19. Music comforts me. (Mood regulation)

20. When I hear a tune I like a lot I can't help tapping or moving to its beat. (Sensory motor)

The Swedish version of the questionnaire is outlined below. Please refer to the English version above for details on reversed items and facets.

Instructions:

Varje påstående i detta avsnitt är något man antingen kan hålla med om eller inte hålla med om. För varje påstående vill vi att du indikerar hur mycket du håller med om det. Försök att vara så ärlig du kan när du svarar. Svara på varje påstående som om det vore det enda påståendet, dvs oroa dig inte för att vara inkonsekvent i dina svar.

Response options:

1;2;3;4;5

Håller inte alls med; Håller delvis inte med; Varken eller; Håller delvis med; Håller helt med.

Items:

1. När jag delar en musikupplevelse med någon känner jag ett särskilt band till den personen.

2. På min fritid lyssnar jag sällan på musik.

3. Jag tycker om att lyssna till känslosam musik.

4. Musik håller mig sällskap när jag är ensam.

5. Jag tycker inte om att dansa, inte ens till musik som jag gillar.

6. Musik får mig att knyta an till andra personer.

7. Jag håller mig uppdaterad kring musik jag tycker om.

8. ag blir känslosam när jag lyssnar till vissa musikstycken.

9. Musik gör mig lugn och avslappnad.

10. Musik får mig ofta att dansa.

11. Jag håller alltid utkik efter ny musik.

12. Jag kan få tårar i ögonen eller börja gråta när jag lyssnar till en melodi som jag tycker mycket om.

13. Jag tycker om att sjunga eller spela instrument tillsammans med andra.

14. Musik hjälper mig att ta det lugnt.

15. Jag kan inte låta bli att nynna eller sjunga med i musik som jag gillar.

16. Vid konserter känner jag samhörighet med artisterna och publiken.

17. Jag spenderar en hel del pengar på musik och musikrelaterade saker.

18. Ibland kan jag få rysningar när jag hör en melodi som jag tycker om.

19. Musik tröstar mig.

20. När jag hör en låt som jag tycker mycket om så kan jag inte låta bli att trumma med eller röra mig till takten.

Supplementary Note 6. *Behavioural approach system reward responsiveness* [14,15]. The English and Swedish versions of the questionnaire are outlined below. Note that here only the items comprising the reward responsiveness scale (i.e., 4, 7, 14, 18, and 23) are given. Reversed-scored items are indicated by the superscript r.

English:

Instructions:

Each item of this questionnaire is a statement that a person may either agree with or disagree with. For each item, indicate how much you agree or disagree with what the item says. Please respond to all the items; do not leave any blank. Choose only one response to each statement. Please be as accurate and honest as you can be. Respond to each item as if it were the only item. That is, don't worry about being "consistent" in your responses. Choose from the following four response options:

Response options:

1;2;3;4

very true for me; somewhat true for me; somewhat false for me; very false for me

Items:

4. When I'm doing well at something I love to keep at it.[r]

7. When I get something I want, I feel excited and energized.[r]

14. When I see an opportunity for something I like I get excited right away.[r]

18. When good things happen to me, it affects me strongly.[r]

23. It would excite me to win a contest.[r]

Swedish:

Instructions:

Hur jag är Varje påstående i detta avsnitt är något man antingen kan hålla med om eller inte hålla med om. För varje påstående vill vi att du anger hur mycket du håller med om det. Var vänlig svara på alla påståenden och försök att vara så ärlig du kan när du svarar.

Response options:

1;2;3;4

Mycket sant för mig ;Något sant för mig; Något falskt för mig; Mycket falskt för mig)

Items:

4. När jag är bra på något älskar jag att hålla på med det

7. När jag får något jag vill ha, blir jag upprymd och fylld av energi

14. När jag ser en möjlighet att göra/få något jag gillar, blir jag uppspelt på direkten

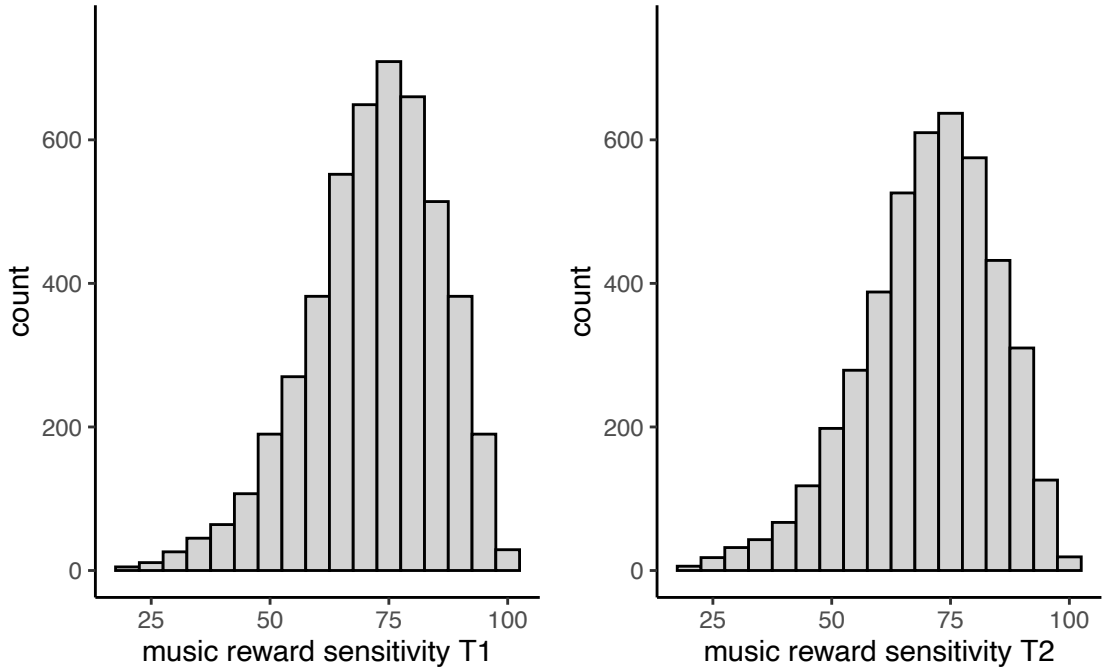
18. När bra saker händer påverkar det mig mycket starkt

23. Jag skulle bli uppspelt av att vinna en tävling

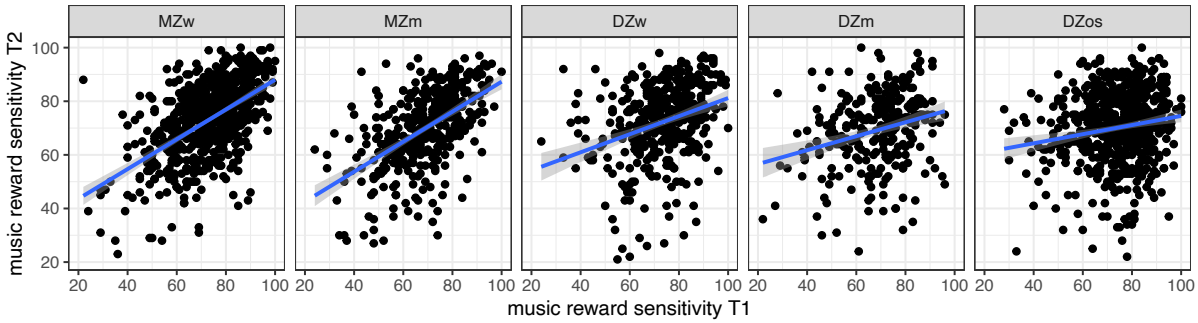
Supplementary Note 7. Comparison with results obtained using OpenMx. Below is a contrast between estimates obtained using lavaan, a five-groups model (MZ women, MZ men, DZ women, DZ men, DZ opposite-sex), with the inclusion of age as a manifest covariate and standard protocols in behavioural genetics using OpenMx, a two-groups (MZ and DZ only) with the inclusion of age as a definition covariate (scripts available at: https://hermine-maes.squarespace.com/s/oneADEvca-s8hc.pdf).

- *AE versus ADE model comparison.* Results obtained from lavaan, manifest covariate, five groups: $\chi^2(1)_{\Delta df}$ = 1.63, p = .20. Results obtained from OpenMx, definition covariate, four groups: $\chi^2(1)_{\Delta df}$ = 0.31, p = .57.
- *AE versus E model comparison.* Results obtained from lavaan $\chi^2(1)$ = 458.49, p <.001**.** Results obtained from OpenMx $\chi^2(1)_{\Delta df}$ = 464.90, p <.001**.**
- *Additive genetic variance and heritability.* Results obtained from lavaan, h^2_{twin} = .54 (95% CI [.51, .58]); σ_A^2 = 101.98 (95% CI [93.73, 110.23]) Results obtained from OpenMx h^2_{twin} = .56; σ_A^2 = 110.03 (95% CI [100.93, 119.18]).

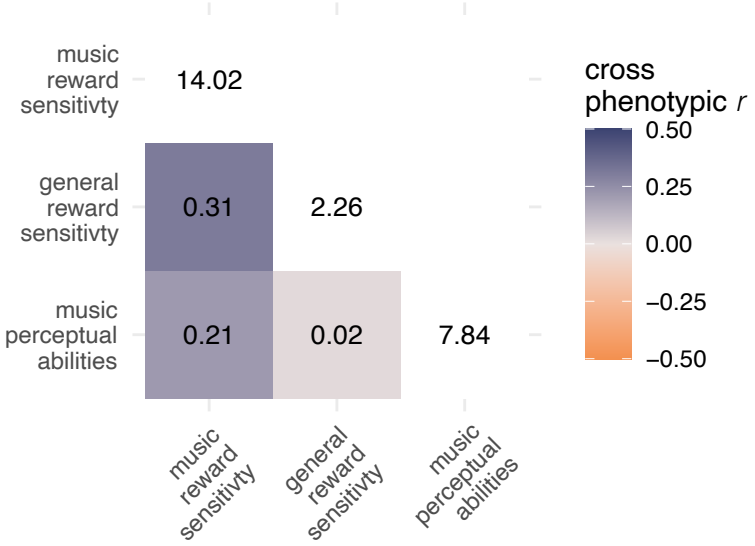
Supplementary Figures



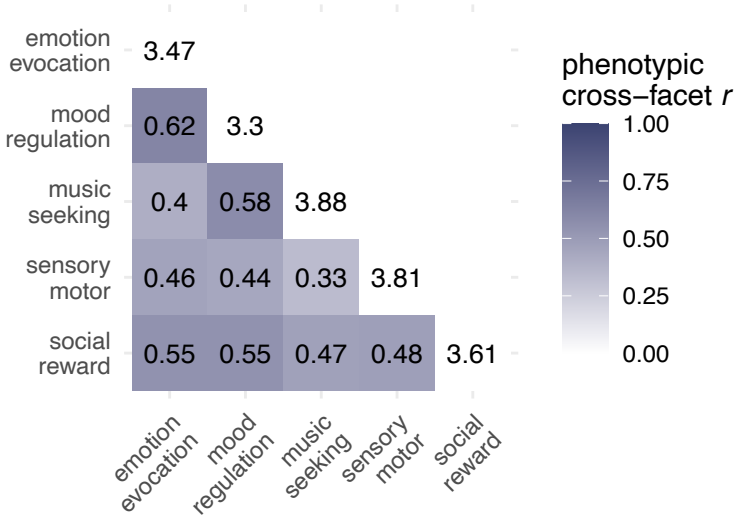
Supplementary Figure 1. *Distribution of BMRQ total scores.* Histogram of music reward sensitivity measured by the BMRQ total score. Each bin spans five BMRQ points on the scale. T1: twin 1; T2 twin 2. Note twin order is randomised within pair but for opposite-sex Dyzigotic twins, for which T1 is always a woman.



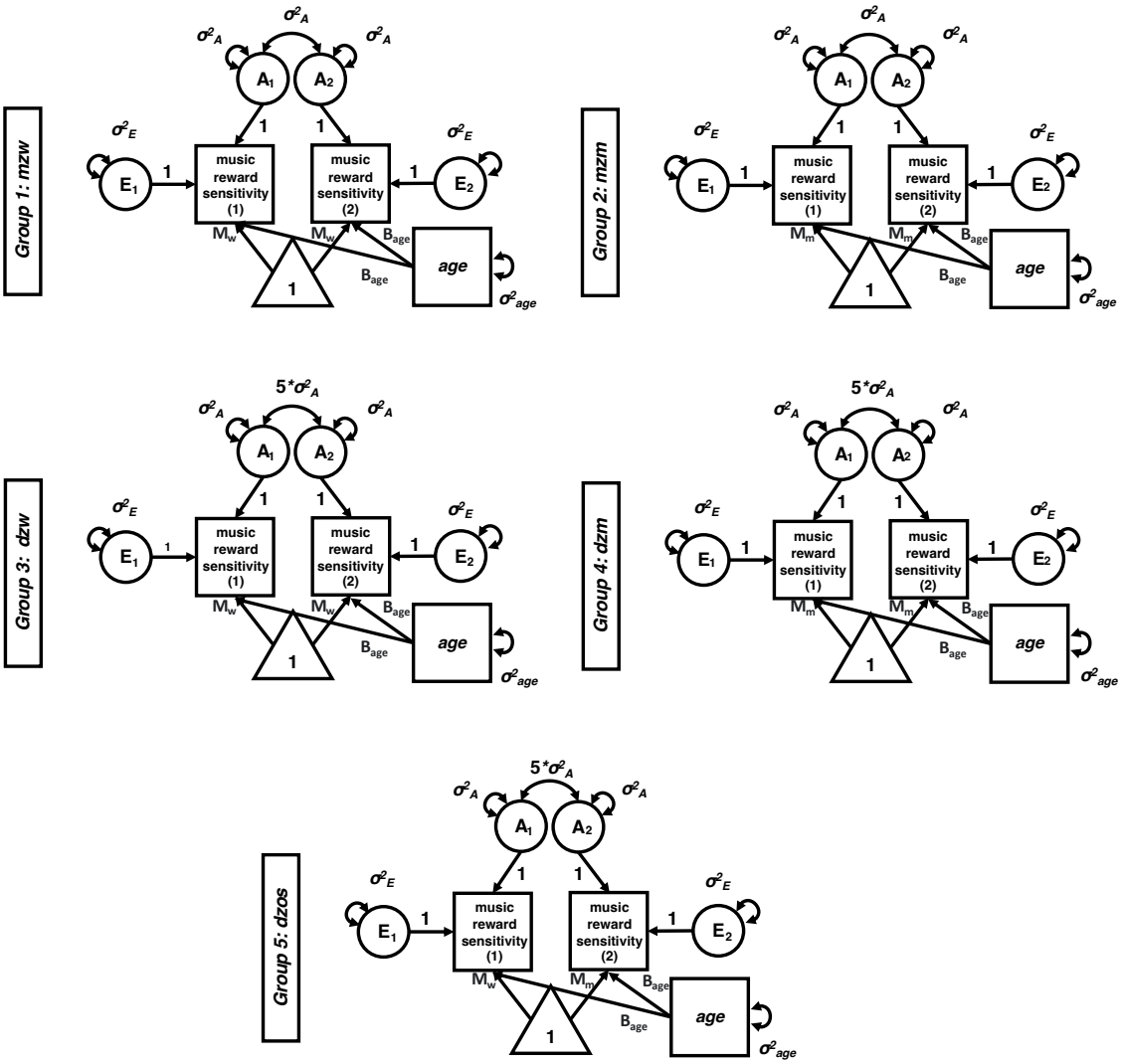
Supplementary Figure 2. *Scatter plot of the between-twin within-pair BMRQ correlations stratified across zygosity group.* Correlations between twins for music reward sensitivity as measured by the BMRQ (Twin 1, T1 and Twin 2, T2, note order is randomised but for DZos, for which T1 is always a woman). In blue, line of best linear fit. Each dot represents a twin pair. MZw: Monozygotic women; MZm: Monozygotic male; DZw: Dizygotic women; DZm: Dizygotic male, DZos: Dizygotic opposite-sex.



Supplementary Figure 3. *Phenotypic correlations matrix for facets of music reward sensitivity.* Phenotypic correlations and standard deviation (on the diagonal) for the correlation matrix depicted in Fig. 2 (main manuscript) obtained instead from the pair's other twins. All cross-phenotypic correlations with music reward sensitivity are significant (all p<.001).

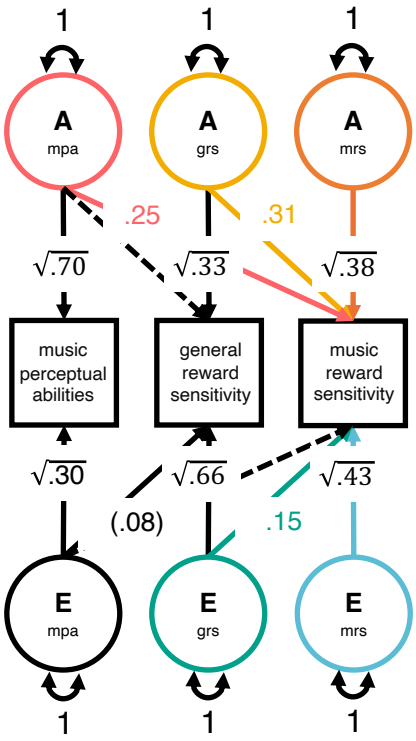


Supplementary Figure 4. *Cross-facet phenotypic correlations.* Correlations between facets of music reward sensitivity are all moderate. To avoid sample dependence, correlations were obtained from samples of only one twin per pair. Standard deviations are on the diagonal.

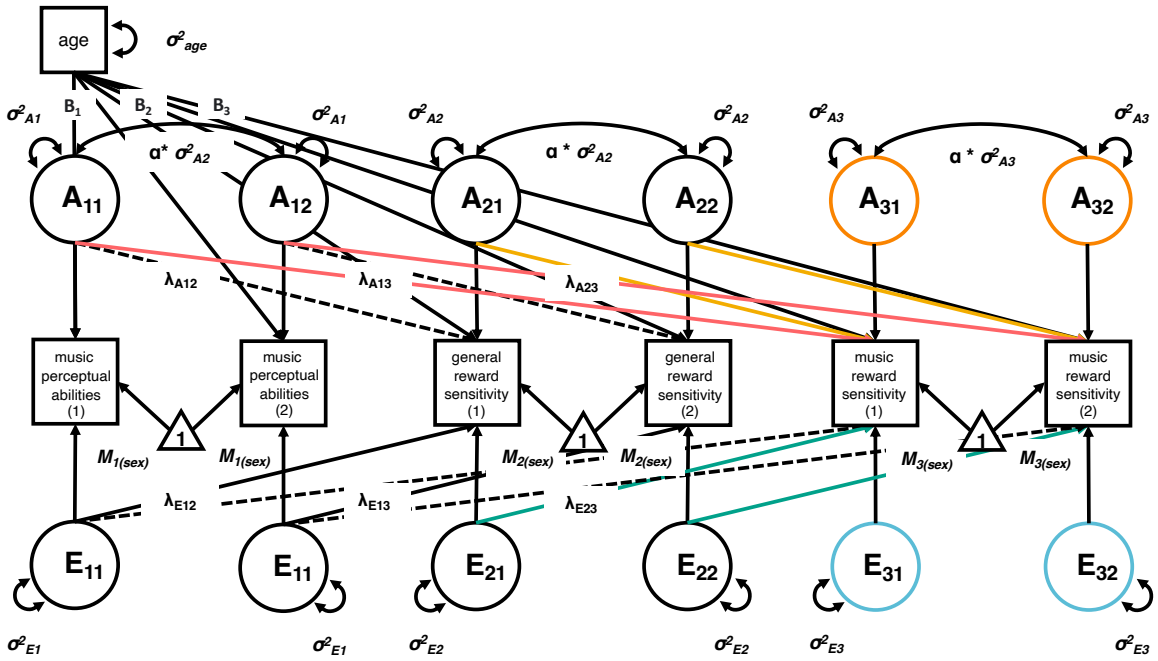


Supplementary Figure 5. *Full AE SEM model.* The final univariate structural equation model is informed by the classical twin design (CTD) and estimated via a direct symmetric approach. Each group represents a zygosity. Parameters are constrained to be equal across groups, except for means, which are kept unconstrained across sex. Parameter estimates can be found in Supplementary File 1.

Notes on structural equation models: Squares represent the measured phenotypes; Circles are the latent component; Double-headed arrows within circles represent the variances associated with the latent components or measured phenotypes; double-headed arrows between circles covariances; the triangle represents the phenotypic mean grouped sex. σ^2: variance; A: additive genetic component; E: residual environmental component; M: mean.

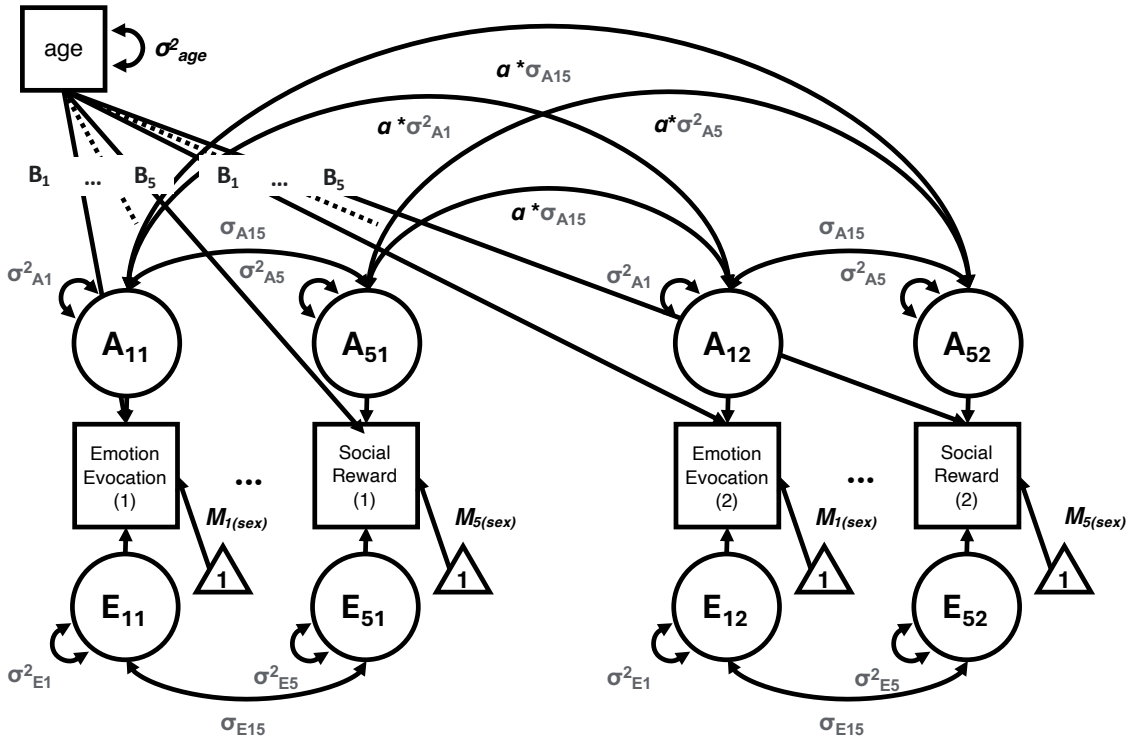


Supplementary Figure 6. *Standardised sequential decomposition.* Solution obtained via the lavaan::standardizedSolotion() function, equivalent to a Cholesky decomposition. Squared-rooted path coefficients represent the estimates for the heritability of the trait. Note that colours and abbreviations match the colour in the main manuscript (Fig. 3) *Notes on structural equation models: one-headed arrow represents regression paths partitioned in additive genetics and unique environmental paths; dashed one-headed arrows, nonsignificant paths. For simplicity, duplicated path parameters. Other abbreviations and symbols are as in previous Supplementary Figures.*



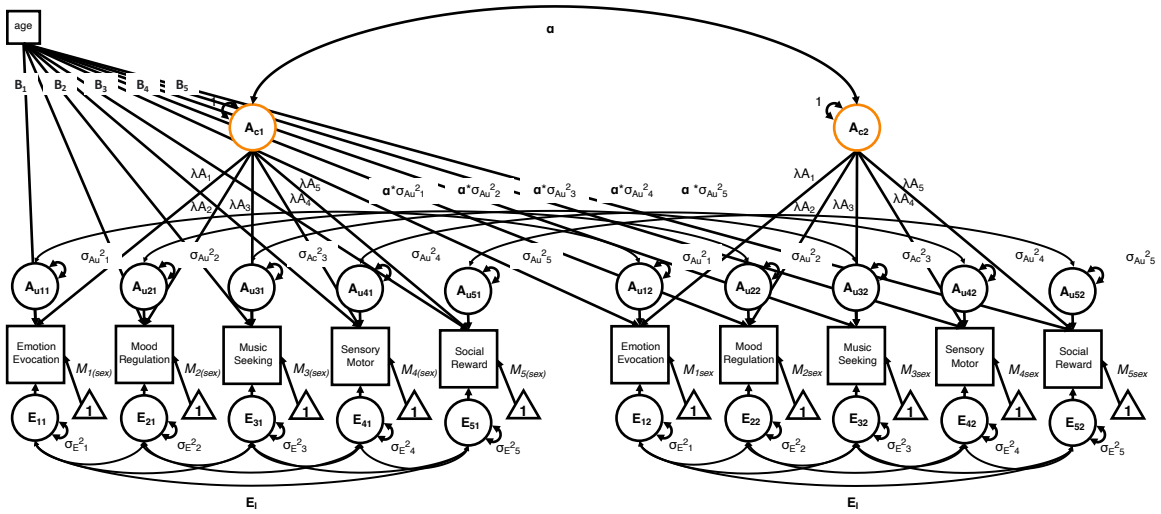
Supplementary Figure 7. *Full AE SEM model.* Sequential structural equation decomposition model. For simplicity, only one zygosity group is shown. Observed variables in the two twins are indicated by number in parentheses (1 or 2, respectively). Parameters are constrained to be equal across groups, except for means across sex. Note that colours match the colour in the main manuscript (Fig. 3). The 22 parameter estimates can be found in Supplementary File 2. (Note that given estimates are standardised, including age as a manifest covariate.)

Notes on structural equation models: one-headed arrow represents regression paths partitioned in additive genetics (λA) and unique environmental (λE) paths; dashed one-headed arrows, nonsignificant paths. For simplicity, duplicated path parameters (i.e., parameters set to be equal) are omitted (e.g., we report only one λA_{13}**).** *Other abbreviations and symbols are as in previous Supplementary Figures.*



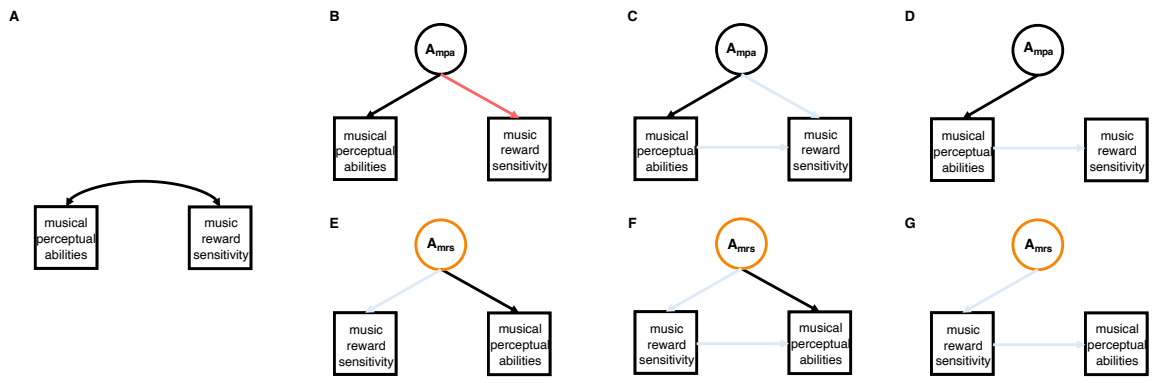
Supplementary Figure 8. *Correlated factor model via direct symmetric approach (distinct factor solution).* To avoid cluttering, only one zygosity group and two phenotypes are shown. Observed variables for the two twins are on the left and right. All path coefficients are set to 1, except for the coefficients from the covariate age. Variances or covariances are directly estimated. Observations on the left (1) and the right (2) correspond to within-twin between-trait variance covariances. Additive genetic covariances fully capture between-twin within- and between-trait covariances (top). The 46 parameter estimates can be found in Supplementary File 3. (Note that given estimates are standardised, including age as a manifest covariate.)

Notes on structural equation models: σ_{Aij} represents the additive genetic covariance between A component i and j. Other abbreviations, symbols, and notes are as in previous Supplementary Figures, except for dashed paths, which indicate omitted paths to avoid cluttering but do not indicate non-significance.

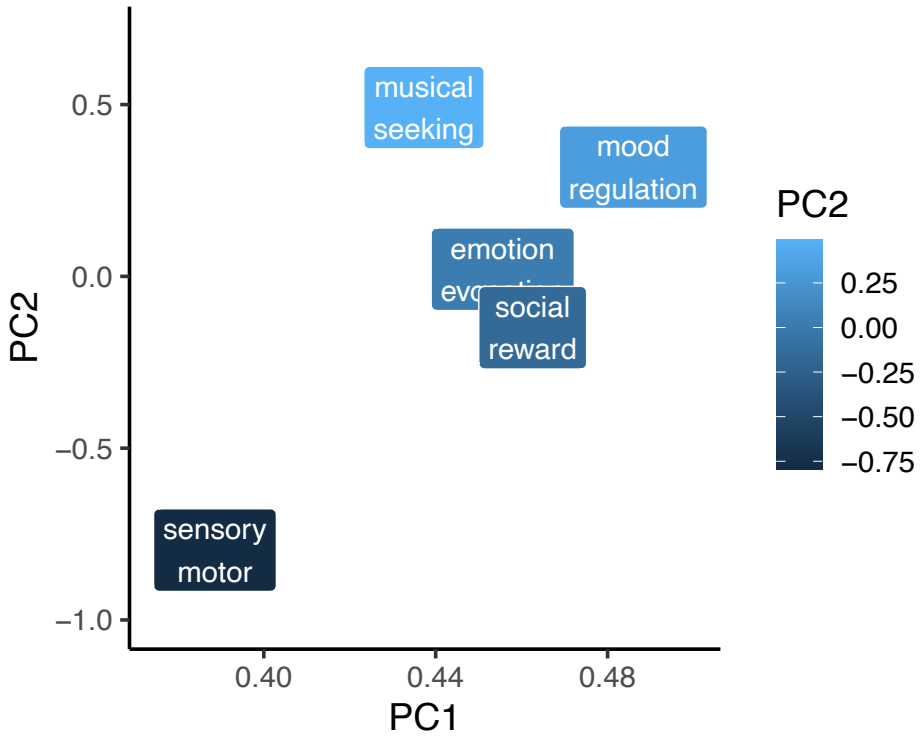


Supplementary Figure 9. *Hybrid Independent pathway model (common-genetic factor solution).* To avoid cluttering, only one group is shown (as in Supplementary Figs. 3 and 4). Observed variables for the two twins are on the left and right. All path coefficients are set to 1, with exceptions of path coefficients from the covariate age (for simplicity, constrained duplicates are not reported) and the common additive genetic factor (A_{c1} and A_{c2}), which are instead directly estimated. Besides the variances of the A_c components, which are fixed to 1, variances or covariances are also directly estimated. Note that this model is nested within the model depicted in Supplementary Figure 4. Parameter estimates can be found in Supplementary File 4.

Notes on structural equation models: λ represents the path coefficients from the additive genetic common factor. Other abbreviations, symbols, and notes are as in previous Supplementary Figures.



Supplementary Figure 10. *Alternative causal modes for the sequential decomposition.* The observed covariance between musical auditory discrimination and music reward sensitivity (**A**) can be caused by different data-generating mechanisms. **(B-G)** represents some of the possible causal structures**. (B)** Genetic common cause (note this is equivalent to the specification of the sequential decomposition in the main). The colour matches the colour used in the main manuscript. **(C)** Partial mediation; colour represents paths for which we have no estimates. **(D)** Full mediation; **(E)** Genetic common (reverse) cause. Note that path estimates would differ from those obtained from the model depicted in (B). **(F)** and **(G)**, as (C) and (D), but reverse. *Notes on structural equation models: Abbreviations, symbols, and notes are as in previous Supplementary Figures.*



Supplementary Figure 11. *Principal component analysis of the additive genetic correlation matrix.* First and second principal component (PC) coordinates (i.e., Eigenvector elements 1 and 2) of the facets' additive genetic variances.

Supplementary Tables

Supplementary Table 1. Assumptions of equality of means and variances of the BMRQ sum score (see Fig. 1A in the main text).

Model	χ^2(*df*)	AIC	BIC	$\Delta\chi^2$;Δdf	*p*
Baseline model	3.04 (4)	120967	121247		
Birth order	14.03 (21)	120944	121108	10.99 (17)	.857
Zygosity	25.78 (29)	120940	121049	22.74 (25)	.593
Sex					
Same mean	300.54 (30)	121212	121315	297.50 (26)	<.001
Same variance	36.18 (30)	120948	121050	33.13 (26)	.158
Quantitative	37.37 (32)	120945	121034	34.33 (28)	.190
Qualitative	41.13 (33)	121029	121029	38.088 (29)	.120

Note. Model comparison test statistics for the assumption of equality of means and variances across zygosity and sex. Models are tested against the first baseline model (i.e., χ^2(*df*) are obtained in reference to the fully saturated model). Note that the baseline model does not worsen the model fit of a fully saturated model in which age is not constrained to be equal across groups (χ^2(*df*)=; *p* =.55). Models are recursively nested, except for sex models, for which parameters are selectively constrained (e.g., 'Same variance' is not nested in the 'Same means' model, since removing the covariate results in a deterioration of the overall fit).

Supplementary Table 2. Univariate ADE model comparison (see Fig. 1C and 1D in the main text)

Model	χ^2(*df*)	AIC	BIC	$\Delta\chi^2$;Δdf	*p*
Baseline model	3.04 (4)	120967	121247		
ADE	41.13 (33)	120947	121022	38.09 (29)	.120
AE	42.76 (34)	120947	121022	22.74 (1)	.593
E	501.25 (35)	121471	121471	458.48(1)	<.001

Note. Model comparison test statistics across univariate ADE models. Models are recursively tested against the model appearing in the above row (i.e., χ^2(*df*) are obtained in reference to the above models). The AE model is the most parsimonious model.

Supplementary Table 3. Estimates for the path coefficient computed using the sequential approach (see Fig. 3B in the main text).

Path	*est*	*est.std*	*se*	*se* (robust)	*p*	*p* (robust)
λ_{A12}	-0.02	-.05	.03	.03	.095	.114
λ_{A13}	0.55	.25	.02	.02	<.001	<.001
λ_{A23}	3.03	.31	.03	.03	<.001	<.001
λ_{E12}	0.04	.08	.04	.04	.03	.044
λ_{E13}	-0.01	-.002	.03	.03	.95	.949
λ_{E23}	1.13	.15	.02	.02	<.001	<.001

Note. Path coefficients unstandardised (*est*) and standardised (*est.std*) estimates are reported in the second and third columns, respectively. Note that coefficients are standardised over the total phenotypic variance, including effect of age. Standard errors (*se*) are reported for the standardised path coefficients. Robust statistics are obtained from the "MLR" estimators.

Supplementary Table 4. Test statistic for multivariate model comparison (see Fig. 5A in the main text).

Model	χ^2(*df*)	AIC	BIC	$\Delta\chi^2$;Δdf	*p*
Baseline model	41.36 (49)	276615	278911		
AE distinct factor solution	537.01 (334)	276540	276889	495.65 (285)	<.001
AE common-genetic factor solution	666.62 (339)	276660	276974	129.61 (5)	<.001

Note. Model comparison test statistics across multivariate models. Models are recursively tested against the model appearing in the above rows. Note that the AIC is lower for the distinct factor solution.

Supplementary Table 5. Significant differences between additive genetic correlations (see Fig. 6B in the main text).

Pairwise Δr_A	Δr_A	p_{FIML}	p_{MLR}
EE-MR	.10	.019	.020
EE-MS	.20	<.001	<.001
EE-SM	.13	.006	.007
MR-MS	.10	.010	.014
EE-SR	-.19	<.001	<.001
MR-SR	-.28	<.001	<.001
MS-SR	-.39	<.001	<.001
SM-SR	-.31	<.001	<.001

Note. The differences between additive genetic correlations were obtained by subtracting Fisher-z transformed r_A values. For ease of interpretation, r back-transformed values of the differences are reported (Δr_{AA}). *p:* p-value; FIML: Full information maximum likelihood; ML: Maximum likelihood with sandwich (Huber-White) standard error; EE: emotion evocation; MR: mood regulation; MS: musical seeking; SM: sensory-motor; SR: social reward.

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