Supporting Information

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SI Materials and Methods

Behavioral Genetic Study, Hypothesis-Testing Sample. A total of n = 1,802 healthy, young Swiss university students or age-matched employees/trainees participated (1,211 females and 591 males). Age was 22.4 ± 0.1 y (mean ± SE). After a complete description of the study to the subjects, written informed consent was obtained. The local ethics committee approved the study protocol.

Picture task, version A (1): this version was performed by 1,436 participants: subjects were presented with 24 neutral, 24 positive, and 24 aversive photographs in a random order. The photographs were taken from the international affective picture system (IAPS) and were presented for 2.5 s each. Immediately following the presentation of each photograph, subjects were asked to rate it for valence and arousal using the IAPS rating scales. Free recall was tested 10 min after presentation of all photographs. To document recall performance, subjects had to describe in writing each picture with a few words. A picture was judged as correctly recalled if the rater could identify the presented picture based on the subject's description. Two blinded investigators independently rated the descriptions for recall success (interrater reliability >99%). For the pictures, which were judged differently by the two raters (i.e., a particular picture was judged as correctly recalled by one rater but not the other), a third independent and blinded rater made a final decision with regard to whether the particular picture could be considered as successfully recalled. Picture task, version B (2): this version was performed by 366 participants. Subjects were presented 10 neutral, 10 positive, and 10 aversive pictures selected from the IAPS and presented for 4 s each. Immediately following the presentation of each picture, subjects were asked to rate it for valence and arousal using the IAPS rating scales. Delayed free recall was tested 10 min after presentation and rated as described for version A. Aversive memory performance (i.e., enhanced free recall performance for previously shown pictures with negative emotional valence) was the trait of interest and was analyzed as a continuous variable. To increase sample size, we combined phenotypic and genotypic data from both groups. Before data pooling, aversive memory performance (dependent variable) was z-transformed in each group.

Behavioral Genetic Study, Replication Sample. A total of n = 781 healthy, young Swiss university students or age-matched employees/trainees participated (484 females and 297 males). Age was 22.4 ± 0.1 y (mean ± SE). After complete description of the study to the subjects, written informed consent was obtained. The local ethics committee approved the study protocol. Participants of this sample performed the identical picture task (version A) as those of the hypothesis-testing sample. As in the hypothesis-testing sample, aversive memory performance (i.e., enhanced free recall performance for previously shown pictures with negative emotional valence) was the trait of interest and was analyzed as a continuous variable.

Array-Based SNP Genotyping. Samples were processed as described in the Genome-Wide Human SNP Nsp/Sty 6.0 User Guide (Affymetrix). Briefly, genomic DNA concentration was determined by fluorometry (Qubit dsDNA BR Assay Kit; Invitrogen) in a Qubit 1.0 fluorometer and adjusted to 50 ng/μL in water. Two hundred fifty nanograms of DNA was digested in parallel with 10 units of StyI and NspI restriction enzymes (New England Biolabs) for 2 h at 37 °C. Enzyme-specific adaptor oligonucleotides were then ligated onto the digested ends with T4 DNA Ligase for 3 h at 16 °C. After adjustment to 100 μL with water, 10 μL of the diluted ligation reactions were subjected to PCR. Three PCR reactions of 100 μL were performed for Sty digested products and four PCR reactions for Nsp. PCR was performed with Titanium Taq DNA Polymerase (Clontech) in the presence of 4.5 μM PCR primer 002 (Affymetrix), 350 μM each dNTP (Clontech), 1 M G-C Melt (Clontech), and 1× Titanium Taq PCR Buffer (Clontech). Cycling parameters were as follows: initial denaturation at 94 °C for 3 min, amplification at 94 °C for 30 s, 60 °C for 45 s, and extension at 68 °C for 15 s repeated a total of 30 times, and a final extension at 68 °C for 7 min. Reactions were then verified to migrate at an average size between 200 and 1,100 bp using 2% (wt/vol) Tris-borate-EDTA gel electrophoresis. PCR products were combined and purified with the Filter Bottom Plate (Millipore, P/N MDRLN0410) using Agencourt AMPure XP Beads (Beckman Coulter). Purified PCR products were quantified on a Zenith 200rt microplate reader (Anthos-Labtec). Four to five micrograms per microliter were obtained on average for each sample. From this stage on, the SNP Nsp/Sty 5.0/6.0 Assay Kit (Affymetrix) was used. Around 250 μg of purified PCR products was fragmented using 0.5 units of DNase I at 37 °C for 35 min. Fragmentation of the products to an average size less than 180 bp was verified using 4% TBE gel electrophoresis. Following fragmentation, the DNA was end-labeled with 105 units of terminal deoxynucleotidyl transferase at 37 °C for 4 h. The labeled DNA was then hybridized onto Genome-Wide Human SNP 6.0 Array at 50 °C for 18 h at 60 rpm (i.e., 0.241488 × g). The hybridized array was washed, stained, and scanned according to the manufacturer's (Affymetrix) instructions using Affymetrix GeneChip Command Console (AGCC, version 3.2.0.1515). Generation of SNP calls and Array quality control were performed using the command line programs of the Affymetrix Power Tools package (version: apt-1.14). According to the manufacturer's recommendation, Contrast QC was chosen as QC metric, using the default value of greater or equal than 0.4. Mean Call Rate for all samples averaged >98.5%. All samples passing QC criteria were subsequently genotyped using the Birdseed (v2) algorithm.

Statistics. ***Gene set analysis (hypothesis-testing sample).*** Genetic associations were run under the assumption of two genetic models (i.e., additive and dominant). After initial quality control (QC), we used following SNP inclusion criteria: nonsignificant deviation from Hardy–Weinberg equilibrium [HWE; P(HWE) > 0.01], minor allele frequency (MAF) > 0.01, and a genotype call rate >95%. SNP P values of association with aversive memory were calculated with Golden Helix SNP and Variation Suite 7 (SVS7, version 7.6.4; Golden Helix). These P values (one P value list per genetic model, resulting in two lists) served as input for the gene set analysis, which was performed on the i-GSEA4GWAS [improved GSEA for genomewide association studies (GWASs)] web server (3). Only intragenic SNPs (i.e., exonic, intronic, 5′- and 3′-UTR SNPs) were included to minimize multiple SNP-to-gene assignment, especially in gene-rich regions. Gene-set significance is calculated by following procedure: the maximum statistics [−log(P value)] of a gene's SNPs is used to represent the gene; then, the ranked genomewide gene list with corresponding representing maximum values is used to calculate each gene set's enrichment score (ES). ES is a Kolmogorov–Smirnov statistic with weight 1 and reflects the trend that genes of a gene set tend to be located at the top of the entire ranked genomewide gene list. SNP label permutation is performed to an-

alyze SNP *P* values and to correct for gene variation (i.e., different genes with different number of SNPs mapped will result in identification of gene sets containing genes with more SNPs mapped, instead of genes with functional correlation) and gene set variation (i.e., different gene sets contain different number of genes). k/K is multiplied to the ES to get the significance proportion based enrichment score (SPES), where k is the proportion of significant genes of the gene set and K is the proportion of significant genes of the total genes in the GWAS. Significant genes are defined as the genes mapped with at least one of the top 5% of all nominally significant ($P < 0.05$) SNPs. Instead of ES, which focuses on the total significance coming from either a few or many significant genes, SPES emphasizes on total significance coming from high proportion of significant genes. Consequently, the gene set enrichment analysis used in this study tends to select gene sets including a high proportion of significant genes and is more appropriate for the study of the combined effects of possibly modest SNPs/genes in complex traits. Based on all distributions of SPESs generated by permutation, the false discovery rate (FDR) is used for multiple testing correction. Per default, the method considers FDR < 0.25 as the threshold for possible association with the trait, whereas FDR < 0.05 is considered as a high confidence threshold. For the purposes of this study, we used the FDR < 0.05 threshold. The used gene sets are extracted and curated from the MSigDB v2.5 database (4) (www.broadinstitute.org/gsea/msigdb), which includes sets from different online databases (KEGG, Gene Ontology GO, and BioCarta.com) (5, 6). The gene consisted of MSigDB's canonical pathways (1,452 gene sets) and the curated gene ontology (GO) gene sets biological process (825 gene sets), molecular function (396 gene sets), and cellular component (233 gene sets). We used a gene set size ranging between 20 and 200 genes to avoid both too narrow and too broad categories. Gene and SNP annotations were derived from the Ensemble Biomart database (www.ensembl.org/biomart/martview), and each gene was represented by the maximum SNP *P* value within ±0 kb of the annotated gene (i.e., we used intragenic SNPs only).

Genetic heterogeneity. Population stratification was assessed with EIGENSTRAT (7) by analyzing all genomewide, array-based autosomal SNPs passing QC criteria. Principal component analysis (PCA) was first applied to reduce genetic variation to a few dimensions. For PCA, default parameters were used (i.e., definition of 10 principal components in five iterations, outlier criterion was 6 SDs). We also applied the genomic control (GC) program that is implemented in the EIGENSTRAT package to compute the inflation factor λ (8) before and after removal of the individuals identified as outliers. After outlier removal, λ indicated the absence of population stratification (λ = 1.00).

Replication procedure. In the replication sample, gene set analysis was also performed on the *i*-GSEA4GWAS web server (3) with the identical settings as those used in the hypothesis-testing sample. In particular, only intragenic SNPs were included and the FDR < 0.05 threshold was used to define a gene set as being significant. Gene set size ranged between 20 and 200 genes, and each gene was represented by the maximum SNP *P* value within ±0 kb of the annotated gene. Because for a number of gene sets in the pathway databases (including MSigDB), the proportion of between-set overlapping genes is highly significant, a gene set of the hypothesis-testing sample was considered replicated, if the particular set or at least one highly overlapping set ($P_{overlap} < 10^{-8}$, as defined by MSigDB) also surpassed the FDR < 0.05 threshold in the replication sample. This condition was the case for the neuroactive ligand–receptor interaction gene set (significant gene sets in the replication sample: cellular cation homeostasis, cation homeostasis, substrate-specific channel activity, ion channel activity, gated channel activity, homeostatic process, hsa04020 calcium signaling pathway) and for the long-term depression gene set (significant gene set in the replication sample: hsa04020 calcium signaling pathway; see Table S2 for a list of significant, nonsignificant, and unmapped genes of the replicated sets in the replication sample). This replication criterion was not fulfilled by either the VEGF or the IL-1R gene set. At the gene level, replication was defined as occurrence of a gene in the significant gene category in the sets of the hypothesis-testing sample and the replicated sets in the replication sample (Table S3).

Genetic Study in the Rwandan Sample. We recruited 349 survivors of the Rwandan genocide (185 females and 164 males) living in the Nakivale refugee camp. Age was 34.9 ± 0.3 y (mean ± SE). As the Nakivale refugee camp has grown over the last decade and is spread over a large area, participants were sampled proportionally to the population size from each zone. To exclude genetic relatives in the samples, only one person per household was interviewed. Interviewers had been trained to detect current alcohol abuse and acute psychotic symptoms; candidates exhibiting these signs were excluded. All subjects had experienced highly aversive traumatic situations and were examined in 2006/2007 by trained experts using a structured interview based on the Posttraumatic Diagnostic Scale (PDS) (9) with the help of trained interpreters. Traumatic events were assessed with a checklist of 36 war- and non–war-related traumatic event types, e.g., injury by weapon, rape, accident (2, 10). Traumatic load was estimated by assessing the number of different traumatic event types experienced or witnessed. This measure is considered more reliable than assessing the frequency of traumatic events (10). Depressive symptoms were assessed with the depression section of the Hopkins Symptom Checklist (HSCL-D). A subset of this sample has been analyzed in previous studies (2, 11). The procedures were approved by the Ethics Committees of the University of Konstanz, Germany, and the Mbarara University of Science and Technology (MUST), Mbarara, Uganda.

The PDS and event list were completed in the form of a standardized interview. Interviewers were first trained in a 6-wk course on principles of quantitative data collection and interviewing techniques. Instruments were translated into Kinyarwanda using several steps of translations, blind back-translations, and subsequent corrections by independent groups of translators (12). Following the translations, the psychometric properties of the translated scales were investigated in a validation study including a retest spanning a 2-wk period and a cross-validation with expert rating (13). To avoid known ceiling effects (14), subjects were selected to have experienced no more than 16 traumatic event types. Saliva samples were obtained from each person using the Oragene DNA Self-Collection Kit (DNA Genothek). DNA was extracted from saliva using standard protocols. In this sample (as was the case in the Swiss sample) we performed array-based SNP genotyping (Affymetrix 6.0 genomewide human SNP array). QC procedures were identical between samples.

Rheumatoid Arthritis Study. We used the publicly available data of a large meta-analysis of six GWAS collections for rheumatoid arthritis (RA), counting 5,500 cases and 20,000 controls in total (15). All cases were autoantibody (anticitrullinated peptide antibodies or rheumatoid factor) positive. Association analysis was done with logistic regression, and meta-analysis was done with inverse-variance weighting of logistic regression β coefficients and their SEs (15). For gene set analysis in this sample, the identical software and settings were used as for the Swiss samples.

Pharmacological Intervention Study. ***Population.*** The study population consisted of 40 healthy human subjects (19 females and 21 males) with European ancestry. Age was 23.0 ± 0.8 y. Weight was 70.1 ± 1.9 kg. The ethics committee of the Cantons of Basel-City and Basel-Country, Switzerland, and the Swiss Agency for Therapeutic Products (Swissmedic) approved the study protocol. The study was registered at ClinicalTrials.gov. Inclusion criteria for the

intervention study were as follows: healthy, male or female, aged between 18 and 40 y, native or fluent German-speaking, BMI between 19 and 27 kg/m^2, able and consenting to give written informed consent and comply with the requirements of the study protocol, and consenting to donate saliva sample for DNA analysis. Female participants also needed to consent to perform a pregnancy test at the beginning of the two test visits. Exclusion criteria were as follows: acute or chronic psychiatric or somatic disorder, pathological ECG, known hypersensitivity to diphenhydramine, hypotension (Riva-Rocci < 110/70 mmHg), bradycardia (<50 bpm), pregnancy, breastfeeding, long-term medication within the last 3 mo (oral contraceptives were disregarded), smoking (>3 cigarettes/d), concurrent participation in another study, participation in one of our previous studies using the same memory tests, and inability to read and understand the participant's information. Subjects received 400 Swiss francs compensation for participating in the study.

Study medication. The study medication was diphenhydramine 50 mg (histamine H1 receptor antagonist; Nardyl 50 mg manufactured by Vifor SA, Villars-sur-Glâne formulated for oral administration). For placebo, mannitol 50 mg formulated for oral administration was used. Subjects received a single oral administration of diphenhydramine or placebo in a cross-over design. Diphenhydramine and the placebo were encapsulated in identically looking capsules. The preparation of study medication, blinding, and the randomization list was performed by the pharmacy of the University Hospital Basel according to Good Manufacturing Practice and Good Clinical Practice. Randomization was done in a counterbalanced way according to treatment, sex, and order of medication (i.e., either diphenhydramine or placebo first).

Screening procedures. During the screening visits, the investigator explained to the subject the aims of the study, the study procedures, the drug under investigation, and the potential risks. Written informed consent was obtained from all participants. Subjects who were candidates for enrollment in the study were evaluated for eligibility during the screening visits by the investigator (inclusion and exclusion criteria). Screening visits took place within 4 wk and at least 1 d before the first test visit and consisted of assessment of personal and family history, psychosocial assessment, assessment of medication history, and physical examination. The following questionnaires were used to assess psychiatric or somatic disorders: sociodemographic self-assessment questionnaire and a self-assessment questionnaire covering mental health. Furthermore, a medical examination (including physical examination and ECG) took place.

Procedure on test days. After the administration of the study medication (i.e., active drug or placebo), there was a 3-h waiting period to allow the medication to reach high plasma concentrations. After this time period, emotional and cognitive functions were tested. After 7 ± 3 d, subjects returned for a second test day, this time with the study medication they did not receive the first time (i.e., active drug or placebo). According to the findings of the presented genetic study in healthy humans, the primary outcome variable was recall performance of aversive pictures.

Picture memory task (1). Subjects were presented with 24 neutral, 24 positive, and 24 aversive photographs in a random order. The photographs were taken from the international affective picture system (IAPS) and were presented for 2.5 s each. Immediately following the presentation of each photograph, subjects were asked to rate it for valence and arousal using the IAPS rating scales. Free recall was tested 5 (short delay) and 90 min (long delay) after presentation of all photographs. To document recall performance, subjects had to describe in writing each picture with a few words. A picture was scored as correctly recalled if the rater could identify the presented picture based on the subject's description. Two blinded investigators independently rated the descriptions for recall success (interrater reliability >99%). A third independent and blinded rater decided on pictures, which were rated differently. Scores were calculated by summing the correctly remembered photographs per valence. A parallel version with different photographs was used for the second test day.

Verbal memory task (16). Participants viewed six series of five semantically unrelated nouns presented at a rate of one word per second with the instruction to learn the words for immediate free recall after each series. The number of correctly recalled words (hits) was the relevant output. Total score was calculated by summing the number of correctly recalled words. Delayed recall of all 30 words was tested 15 (short delay) and 115 min (long delay) after presentation. A parallel version with different words was used for the second test day.

Figural memory task, Rey. Free recall of visual material was assessed with the Rey-15-figures test. The figures had to be drawn 5 min and 1 h after learning. Total score was calculated by summing the number of correctly recalled figures. A parallel version was used for the second test day.

d2 cancellation test (17): Selective attention and concentration was assessed with the d2 cancellation test. Performance is defined by the number of correctly crossed signs minus false positives during 5 min.

Digit span task. Working memory was assessed with the digit span task, a subtest of the Wechsler intelligence inventory for adults. Total score was calculated as described in the manual.

Mood state was assessed with the self-rating instrument MDBF, Mehrdimensionale Befindlichkeitsfragebogen (18) consisting of 12 items to be rated in a five-scale mode. The total score is calculated by summing the answers in each of the three dimensions good/bad mood, alertness/sleepiness, and rest/restlessness with four items in each dimension. Version A was used on test day 1 and version B on test day 2.

Montgomery-Asberg Depression Scale. Depressive symptoms were assessed with the self-rating questionnaire Montgomery-Asberg Depression Scale (MADRS). This scale consists of nine items assessing subjects' mood, feelings of unease, sleep, appetite, ability to concentrate, initiative, emotional involvement, pessimism, and zest for life. Each item is scored between 0 and 3, with three intermediate levels (0.5, 1.5, and 2.5). The total score is calculated by summing the answers of the nine items, ranging between 0 and 27 (higher scores indicate increased impairment).

Anxiety was measured with the self-rating instrument State-Trait Anxiety Inventory form ×1 (state). This instrument consists of 20 items that is scored between 1 and 4. The total score is calculated by summing the answers, ranging between 20 and 80.

Sleepiness was rated by the subject using a visual analog scale (VAS). Subjects specify their level of agreement to the statement by indicating a position along a continuous line (10 cm) between the two endpoints wide awake and extremely sleepy. The score ranges between 0 and 10.

Matrix reasoning. We administered the Bochumer Matrizentest (BOMAT) to measure fluid intelligence (Gf), consisting of 29 items. A parallel version was used for the second visit. We used a time-limited version. The total score was calculated by summing the correct solutions, ranging between 0 and 29.

Saliva sampling for DNA analysis. A saliva sample (2 mL) was obtained from each person using the Oragene DNA Self-Collection Kit OG-500, manufactured by DNA Genotek. DNA was extracted from saliva samples using standard procedures.

Statistics. Repeated-measures AN(C)OVAS were used to analyze treatment effects (i.e., diphenhydramine vs. placebo) on outcome measures. Because sleepiness at testing was significantly different between the drug and the placebo condition ($P < 0.001$), diphenhydramine-induced sleepiness was not included as a covariate in the statistical analysis of drug effects (19), but instead as a between-subjects factor (i.e., each participant was assigned to a high and low sleepiness group according to median split of VAS sleepiness in the placebo condition; VAS sleepiness in the diphenhydramine condition). In the high sleepiness group, mean

sleepiness difference (placebo − diphenhydramine) was −4.2 ± 2.0 (mean ± SD; $P < 0.001$, paired t test). In the low sleepiness group, mean sleepiness difference was −0.3 ± 1.5 ($P = 0.3$, paired t test), indicating an almost complete absence of drug-related sleepiness in this group. Sex, age, body weight, and treatment order were considered as covariates. A significance level of $P < 0.05$ (two-sided) was considered as significant. The estimation of $n = 40$ per group was based on a power analysis assuming to detect a medium effect size of a drug with a power of 95% at $\alpha = 0.05$.

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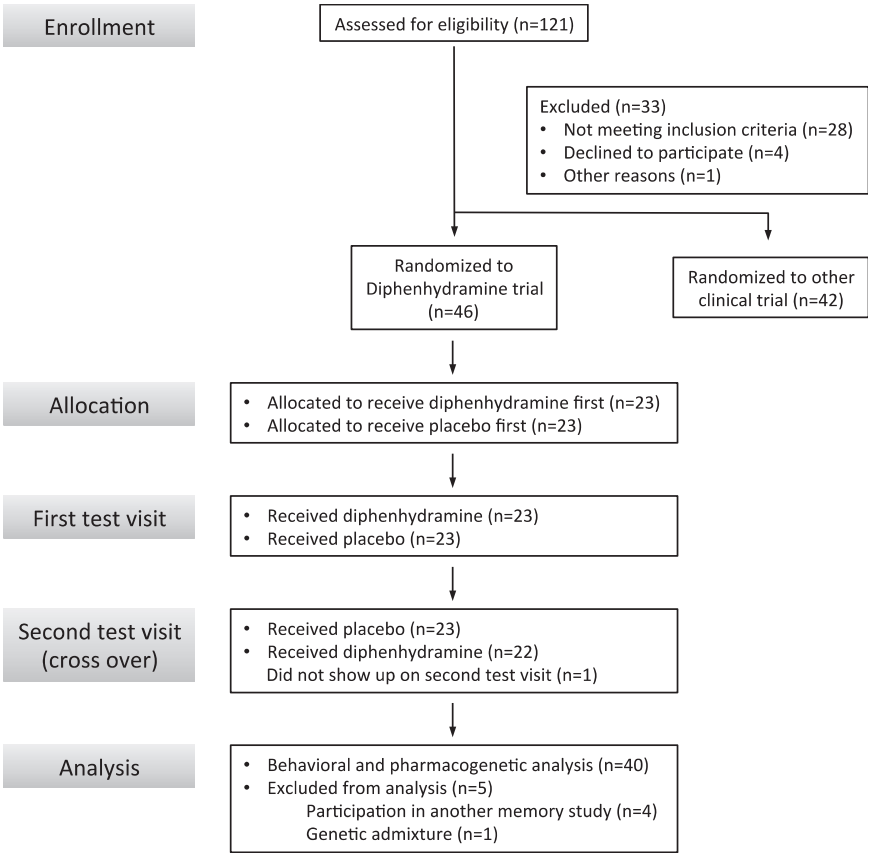

Fig. S1. Flow diagram.

Table S1. Gene set genes (testing and replication sample) and replicated gene set genes

Table S1

Table S2. Therapeutic compounds/compound groups

Table S2

Table S3. Genetic association with traumatic memory

Table S3

Table S4. Drug effects on sleepiness, anxiety, attention, and fluid intelligence

Table S4

Table S5. Diphenhydramine-induced reduction of aversive memory

Table S5

Table S6. Drug effects on short-delay (5 min) free recall of pictures and on valence and arousal ratings

Table S6

Table S7. Gene sets related to rheumatoid arthritis

Table S7

Table S8. Rheumatoid arthritis gene set genes

Table S8

Table S9. Therapeutic compounds/compound groups (rheumatoid arthritis study)

Table S9