

SUPPLEMENTARY INFORMATION

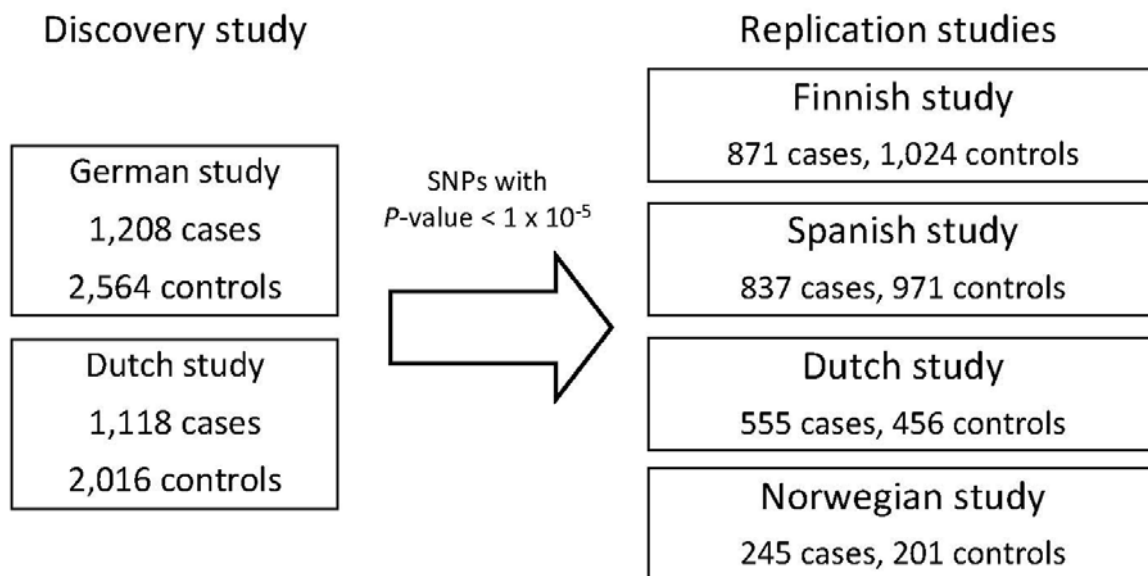
Genome-wide association analysis identifies susceptibility loci for migraine without aura

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Supplementary Figures and Tables

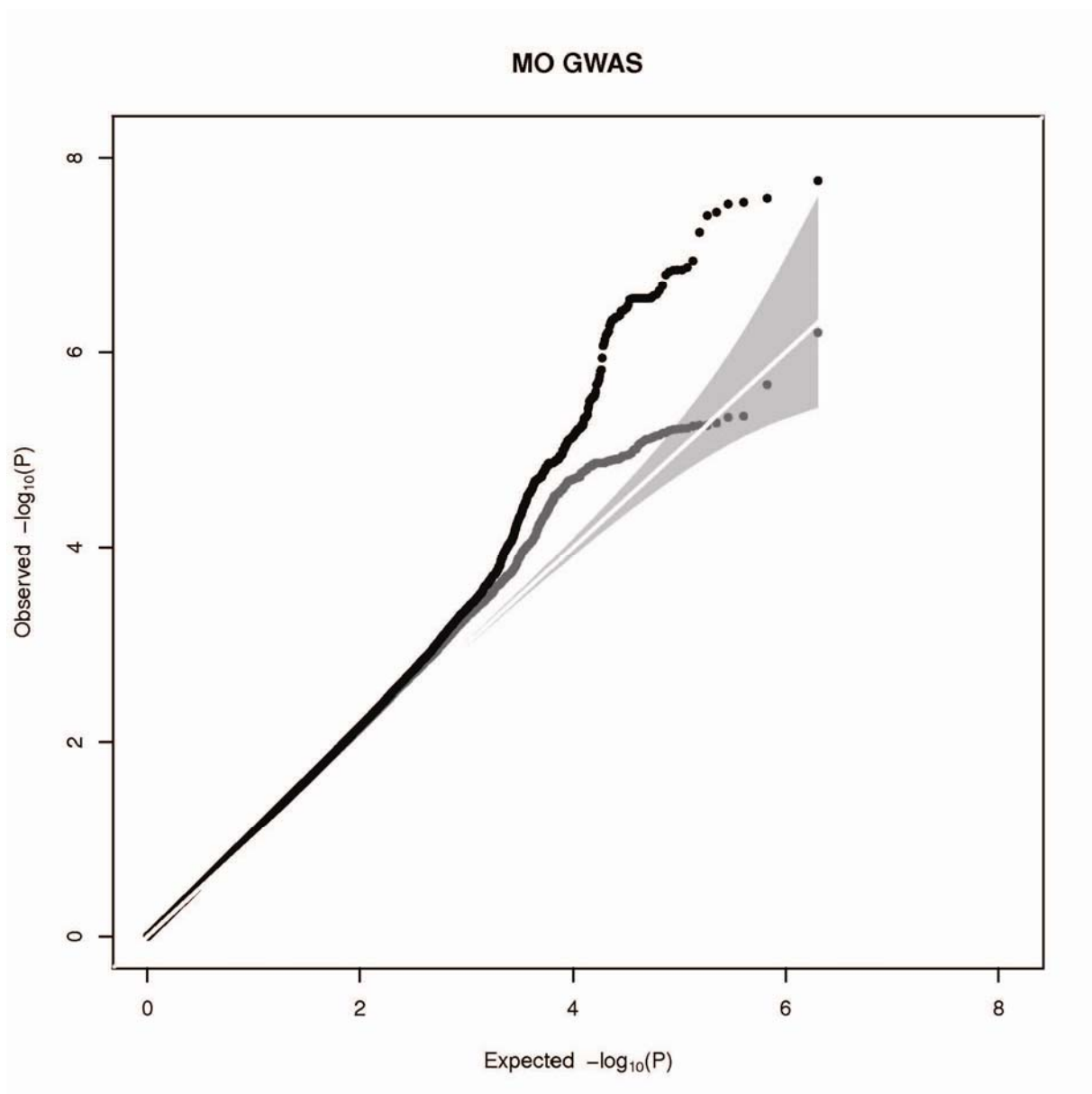
Supplementary Figure 1. Study design

In the discovery stage of the study, migraine without aura (MO) patients from two clinic-based collections were analyzed in a joint genome-wide association analysis. The most significant association signals ($P < 1.0 \times 10^{-5}$) were followed up in independent clinic-based samples from Finland, Spain, the Netherlands, and Norway.



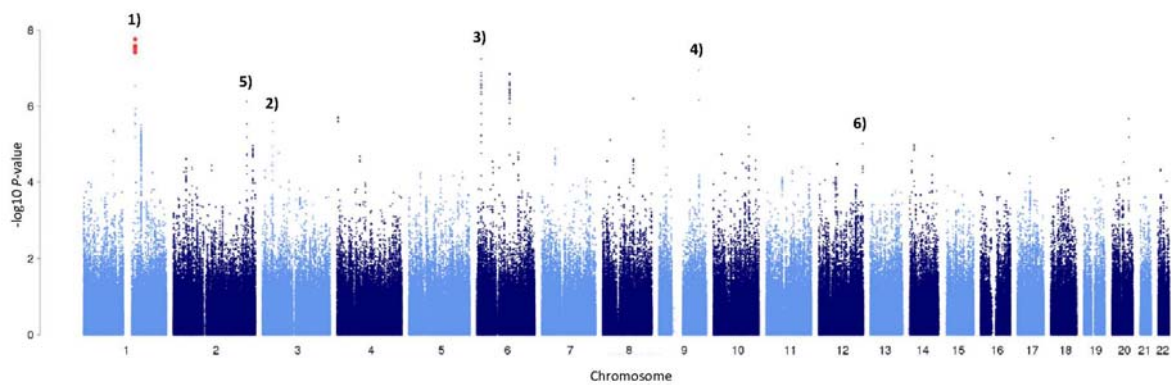
Supplementary Figure 2. Quantile-quantile plot of the meta-analysis

Quantile-Quantile (Q-Q) plot of P -values from the study. The analysis shows the expected (X-axis) and observed (Y-axis) distribution of $-\log_{10} P$ -values of the joint analysis of the two discovery cohorts (in black). The grey line shows the same distribution, but with SNPs at the loci taken to replication excluded. The genomic inflation factor (λ_{1000}) was 1.03.



Supplementary Figure 3. Manhattan plot

Genome-wide association plot showing $-\log_{10} P$ -values per chromosome of the analysis of the discovery sample. SNPs indicated in red are below the threshold for genome-wide significance ($P < 5 \times 10^{-8}$). The six reported MO loci are indicated in the graph with numbers: 1) 1q22, 2) 3p24, 3) 6p24, 4) 9q33, 5) 2q37, 6) 12q13.



Supplementary Table 1. Replication results for loci containing at least two SNPs with P -values $<10^{-5}$ and SNPs from previously reported migraine loci (*MTDH*, *TRPM8*, *LRP1* and *PRDM16*)^{3,4}

General SNP information						Discovery samples			Replication samples P -values				All replications			Overall meta-analysis				
Chr	SNP	Position ^a	Minor allele	minor allele frequency	Gene ^b	P -Value	OR (95% CI)	I^2	Spain (n=837)	Finland (n=871)	Norway (n=245)	Netherlands (n=555)	P -value	OR (95% CI)	I^2	P -value	OR (95% CI)	I^2	Q-stat. p-value	REM P -value
1	rs10733092	91.399.137	G	0.27	<i>HFM1</i>	4.38x10 ⁻⁶	1.21 [1.11-1.31]	0.23	0.48	0.75	-	0.02	0.12	0.94 [0.87-1.02]	0.42	0.032	1.06 [1.01-1.13]	0.83	9.55x10 ⁻⁵	0.56
1	rs1050316	154.701.327	G	0.34	<i>MEF2D</i>	2.59x10⁻⁸	1.24 [1.15-1.33]	0.00	0.04	0.047	0.18	0.34	1.15x10 ⁻³	1.14 [1.06-1.24]	0.00	3.21x10⁻¹⁰	1.19 [1.13-1.26]	0.00	0.79	3.2x10 ⁻¹⁰
1	rs3790455	154.722.925	C	0.34	<i>MEF2D</i>	1.71x10⁻⁸	1.24 [1.15-1.34]	0.00	0.06	0.04	0.17	0.09	4.85x10 ⁻⁴	1.16 [1.07-1.26]	0.00	7.06x10⁻¹¹	1.20 [1.14-1.27]	0.00	0.89	7.1x10 ⁻¹¹
1	rs17350991	172.350.091	T	0.26	<i>RABGAP1L</i>	3.17x10 ⁻⁶	0.83 [0.76-0.90]	0.00	0.32	0.11	-	0.48	0.92	1.00 [0.92-1.08]	0.50	8.03x10 ⁻⁴	0.91 [0.86-0.96]	0.73	0.01	0.14
2	rs6756590	216.916.816	T	0.44	<i>MARCH4</i>	7.52x10 ⁻⁷	0.84 [0.78-0.90]	0.00	0.30	0.10	0.24	1.13x10⁻⁴	0.08	0.93 [0.86-1.01]	0.82	1.21x10 ⁻⁶	0.88 [0.83-0.93]	0.76	4.57x10 ⁻³	5.3x10 ⁻²
3	rs7640543	30.437.407	A	0.32	<i>TGFBR2</i>	2.72x10 ⁻⁶	1.20 [1.11-1.30]	0.00	0.46	4.01x10⁻³	3.33x10⁻³	0.05	1.02x10 ⁻⁰⁴	1.18 [1.09-1.29]	0.51	1.17x10⁻⁹⁹	1.19 [1.13-1.26]	0.19	0.29	8.4x10 ⁻⁸
4	rs12641989	3.389.638	A	0.10	<i>RGS12</i>	2.03x10 ⁻⁶	1.33 [1.18-1.50]	0.36	0.97	0.73	-	0.29	0.39	0.94 [0.83-1.08]	0.00	3.10x10 ⁻³	1.14 [1.05-1.25]	0.76	2.28x10 ⁻³	0.31
6	rs9381462	12.981.761	G	0.48	<i>PHACTR1</i>	3.06x10 ⁻⁶	0.84 [0.78-0.91]	0.00	0.12	0.69	0.09	0.06	0.04	1.09 [1.00-1.18]	0.39	1.44x10 ⁻⁶	1.14 [1.08-1.20]	0.35	0.17	2.7 x10 ⁻⁴
6	rs1332847	13.000.740	T	0.29	<i>PHACTR1</i>	5.95x10 ⁻⁶	1.20 [1.11-1.30]	0.00	0.04	0.81	0.65	0.98	0.12	1.07 [0.98-1.16]	0.00	1.25x10 ⁻⁵	1.14 [1.07-1.21]	0.15	0.32	1.3 x10 ⁻⁴
6	rs9349379	13.011.943	G	0.38	<i>PHACTR1</i>	2.06x10 ⁻⁷	0.82 [0.77-0.89]	0.00	0.01	0.67	0.33	0.31	0.01	0.90 [0.83-0.98]	0.00	3.15x10⁻⁸	0.86 [0.81-0.91]	0.10	0.36	3.0 x10 ⁻⁷
6	rs2327621	13.030.675	G	0.36	<i>PHACTR1</i>	5.79x10 ⁻⁸	1.23 [1.14-1.32]	0.00	0.02	0.46	-	0.99	0.07	1.07 [1.00-1.16]	0.21	2.62x10 ⁻⁷	1.15 [1.09-1.21]	0.55	0.07	1.4 x10 ⁻³
6	rs7739181	13.042.673	A	0.35	<i>PHACTR1</i>	1.33x10 ⁻⁷	1.22 [1.14-1.32]	0.00	0.05	0.48	0.75	0.76	0.06	1.08 [1.00-1.17]	0.00	2.50x10 ⁻⁷	1.16 [1.09-1.22]	0.16	0.31	7.6 x10 ⁻⁶
6	rs2499797	96.955.390	G	0.18	<i>FHL5</i>	1.41x10 ⁻⁷	1.29 [1.17-1.41]	0.00	0.29	0.98	0.92	0.01	0.06	1.11 [0.99-1.23]	0.30	2.25x10 ⁻⁷	1.20 [1.12-1.29]	0.43	0.12	5.7x10 ⁻⁴
6	rs11757063	96.991.607	A	0.21	<i>FHL5</i>	2.32x10 ⁻⁷	1.26 [1.16-1.38]	0.00	0.03	0.79	0.61	0.03	0.02	1.13 [1.02-1.25]	0.24	5.68x10 ⁻⁸	1.20 [1.12-1.28]	0.26	0.24	1.9 x10 ⁻³
8	rs11777116	24.100.246	T	0.08	<i>ADAM28</i>	7.85x10 ⁻⁶	1.35 [1.19-1.55]	0.47	0.50	0.19	-	0.47	0.43	1.06 [0.92-1.23]	0.03	1.26x10 ⁻⁴	1.21 [1.10-1.34]	0.59	0.04	3.1 x10 ⁻²
9	rs6478241	118.292.450	A	0.38	<i>ASTN2</i>	1.14x10 ⁻⁷	1.22 [1.13-1.31]	0.00	0.15	2.82x10⁻³	0.57	0.28	0.02	1.10 [1.02-1.19]	0.57	3.86x10⁻⁸	1.16 [1.10-1.23]	0.56	0.04	2.6 x10 ⁻³
10	rs1712517	105.023.005	T	0.48	<i>INA</i>	3.59x10 ⁻⁶	1.18 [1.10-1.27]	0.00	0.60	0.83	0.07	0.07	0.13	0.94 [0.87-1.02]	0.34	8.82x10 ⁻⁶	1.13 [1.07-1.17]	0.46	0.10	2.3 x10 ⁻³
12	rs6598163	130.891.192	G	0.48	<i>MMP17</i>	9.97x10 ⁻⁶	1.19 [1.10-1.28]	0.00	0.50	2.05x10⁻³	0.19	0.91	7.00x10 ⁻³	0.90 [0.83-0.97]	0.32	4.67x10 ⁻⁷	1.15 [1.09-1.21]	0.14	0.32	6.1x10 ⁻⁶
1	rs2651899	3073572	C	0.45	<i>PRDM16</i>	3.15x10 ⁻³	1.11 [1.04-1.19]	0.00	0.73	0.03	0.11	0.93	0.046	1.08 [1.00-1.17]	0.10	4.18x10 ⁻⁴	1.10 [1.04-1.16]	0.00	0.56	4.2x10 ⁻⁴
2	rs10166942	234.489.832	C	0.18	<i>TRPM8</i>	1.32x10 ⁻⁵	0.82 [0.74-0.89]	0.00	1.08x10⁻⁵	3.76x10⁻³	0.21	0.02	5.62x10 ⁻⁹	0.74 [0.67-0.82]	0.00	9.83x10⁻¹³	0.78 [0.73-0.84]	0.00	0.61	9.8x10 ⁻¹³
2	rs17862920	234.492.734	T	0.10	<i>TRPM8</i>	2.19x10 ⁻³	0.78 [0.69-0.87]	0.00	1.46x10⁻³	0.22	0.30	0.04	6.44x10 ⁻⁵	0.75 [0.66-0.87]	0.00	5.97x10⁻⁹	0.77 [0.70-0.84]	0.00	0.93	6.0x10 ⁻⁹
8	rs1835740	98.236.089	T	0.22	<i>MTDH</i>	0.70	1.02 [0.93-1.11]	0.00	0.09	0.12	0.18	4.79x10⁻³	0.69	0.98 [0.90-1.08]	0.80	0.986	1.00 [0.94-1.07]	0.68	0.01	0.70
12	rs11172113	55.813.550	C	0.39	<i>LRP1</i>	3.38x10 ⁻⁵	0.86 [0.80-0.92]	0.00	2.50x10⁻³	0.86	0.62	2.74x10⁻⁴	2.33x10 ⁻⁴	0.86 [0.79-0.93]	0.67	2.97x10⁻⁸	0.86 [0.81-0.91]	0.45	0.10	7.7x10 ⁻⁵

Genome-wide significant P -values and successful replications are shown in boldface. ^aChromosomal positions are based on NCBI build 36. ^bFor intragenic SNPs, the gene in which the SNP is located is listed, whereas for intergenic SNPs the nearest gene is listed. REM = random effects model.

Supplementary Table 2. Effect of gender on the associations of the genotyped top SNPs within *MEF2D* (locus 1), *TGFBR2* (locus 2) and *PHACTR1* (locus 3)

				Additive regression			Additive regression with gender interaction						Women only			Men only		
				ADD			ADD			ADD x GENDER			ADD			ADD		
Locus	Chr	SNP	Minor allele	OR	SE	P-value	OR	SE	P-value	OR	SE	P-value	OR	SE	P-value	OR	SE	P-value
1	1	rs2274316	C	1.229	0.038	4.53x10 ⁻⁸	1.195	0.045	6.41x10 ⁻⁵	1.163	0.099	0.127	1.195	0.045	6.44x10 ⁻⁵	1.391	0.088	1.90x10 ⁻⁴
1	1	rs3790455	C	1.236	0.038	2.17x10 ⁻⁸	1.210	0.045	1.98x10 ⁻⁵	1.145	0.099	0.171	1.210	0.045	1.99x10 ⁻⁵	1.387	0.088	2.12x10 ⁻⁴
2	3	rs7640543	A	1.202	0.039	2.27 x10 ⁻⁶	1.180	0.046	3.23x10 ⁻⁴	1.005	0.103	0.962	1.181	0.046	3.08x10 ⁻⁴	1.186	0.092	0.0627
3	6	rs9349379	G	0.821	0.038	1.75 x10 ⁻⁷	0.826	0.045	1.93x10 ⁻⁵	0.976	0.099	0.808	0.826	0.045	1.89x10 ⁻⁵	0.806	0.089	0.0149
4	9	rs6478241	A	1.217	0.037	9.79 x10 ⁻⁸	1.179	0.044	1.60x10 ⁻⁴	1.078	0.096	0.435	1.178	0.044	1.61x10 ⁻⁴	1.271	0.086	5.38x10 ⁻³
5	2	rs10166942	C	0.812	0.048	1.41 x10 ⁻⁵	0.795	0.056	4.45x10 ⁻⁵	1.051	0.128	0.699	0.795	0.056	4.54x10 ⁻⁵	0.835	0.115	0.116
5	2	rs17862920	T	0.772	0.062	2.54 x10 ⁻⁵	0.761	0.071	1.29x10 ⁻⁴	1.080	0.163	0.638	0.761	0.071	1.30x10 ⁻⁴	0.822	0.147	0.182
6	12	rs11172113	C	0.855	0.038	2.89 x10 ⁻⁵	0.866	0.044	1.13x10 ⁻³	1.007	0.100	0.948	0.867	0.044	1.17x10 ⁻³	0.873	0.089	0.126

ADD = Additive model, ADD x GENDER indicates the interaction term

Supplementary Table 3. *In silico* replication of the top SNPs of the MO GWAS in the previously published MA GWA study³

SNP	Chr	Position	Minor allele	minor allele frequency	Gene ^a	<i>P</i> -value in current MO study (n=2,324/4,580) ^b	OR in MO	<i>P</i> -value in previous MA study (n=2,731/10,747) ^b	OR in MA
Locus 1:									
rs1050316	1	154.701.327	G	0.34	<i>MEF2D</i>	2.59x10 ⁻⁸	1.24 [1.15-1.33]	0.02	1.09 [1.02-1.16]
rs2274316	1	154.712.866	C	0.35	<i>MEF2D</i>	3.60x10 ⁻⁸	1.23 [1.14-1.33]	0.01	1.09 [1.02-1.17]
rs1925950	1	154.717.364	G	0.35	<i>MEF2D</i>	2.97x10 ⁻⁸	1.24 [1.15-1.33]	0.02	1.09 [1.02-1.16]
rs3790455	1	154.722.925	C	0.34	<i>MEF2D</i>	1.71x10 ⁻⁸	1.24 [1.15-1.34]	0.01	1.09 [1.02-1.16]
rs3790459	1	154.728.331	A	0.35	<i>MEF2D</i>	2.85x10 ⁻⁸	1.24 [1.15-1.33]	0.01	1.09 [1.02-1.17]
rs12136856	1	154.739.738	C	0.34	<i>MEF2D</i>	3.90x10 ⁻⁸	1.23 [1.15-1.33]	0.01	1.09 [1.02-1.17]
Locus 2:									
rs7640543	3	30.437.407	A	0.32	<i>TGFBR2</i>	2.72x10 ⁻⁶	1.20 [1.11-1.30]	0.10	1.06 [0.90-1.14]
Locus 3:									
rs9349379	6	13.011.943	G	0.38	<i>PHACTR1</i>	2.06x10 ⁻⁷	0.82 [0.77-0.89]	0.02	0.93 [0.87-0.99]
Locus 4:									
rs6478241	9	118.292.450	A	0.38	<i>ASTN2</i>	1.14x10 ⁻⁷	1.22 [1.13-1.31]	2.63x10 ⁻⁴	1.13 [1.06-1.21]
Locus 5:									
rs10166942	2	234.489.832	C	0.18	<i>TRPM8</i>	1.32x10 ⁻⁵	0.82 [0.74-0.89]	3.50x10 ⁻⁵	0.83 [0.77-0.91]
rs17862920	2	234.492.734	T	0.10	<i>TRPM8</i>	2.19x10 ⁻⁵	0.78 [0.69-0.87]	3.70x10 ⁻⁵	0.79 [0.70-0.88]
Locus 6:									
rs11172113	12	55.813.550	C	0.39	<i>LRP1</i>	3.38x10 ⁻⁵	0.86 [0.80-0.92]	5.00x10 ⁻⁵	0.87 [0.82-0.93]

ORs are reported for the minor allele. ^aFor intragenic SNPs, the gene in which the SNP is located is listed, whereas for intergenic SNPs the nearest gene is listed. ^bNumber of cases/number of controls.

Supplementary note

Clinical subject ascertainment and control samples

Discovery study

The German sample of 1,229 MO cases was recruited in Munich and Kiel and data were examined by a headache specialist at the Klinikum Großhadern of the Ludwig-Maximilians-University, Munich, and the Kiel Pain and Headache Center, Kiel. Phenotyping was based on a German translation of the FMSQ_{FS}¹. Particular emphasis was put on reliable exclusion of aura symptoms. In case of insufficient or conflicting information, an additional telephone interview was performed. Information was obtained on all aspects of the ICHD-II² criteria as well as on other aspects (such as age at onset, prodromal symptoms, triggers, acute and prophylactic medication, family history, general past medical history, co-morbidity and place of birth). There is no overlap between the cases of the discovery stage of the present GWA MO study, those of the previous clinic-based IHGC MA GWA study³, or those of the population-based migraine Women's Genome Health Study (WGHS)⁴. Some 837 of the 1,229 German MO cases in this study were also genotyped in the replication stage of the clinic-based IHGC MA GWA study for SNP rs1835740 (*MTDH*).

The Dutch sample contains 1,235 Dutch MO patients that were available from the clinic-based Leiden University Migraine Neuro Analysis (LUMINA) study. Self-reported migraineurs were recruited via the project's website. A set of screening questions validated previously in a population-based study⁵ was used. Participants fulfilling the screening criteria then completed an extended questionnaire that focuses on signs and symptoms of migraine headache and aura as outlined in ICHD-II². Individual diagnoses were made using an algorithm based on these criteria and that was validated by a semi-structured telephone interview performed by

experienced study physicians or by well-trained medical students⁶. A subset of the patients was asked to participate upon visiting the outpatient clinic. None of the patient DNAs were used in previous GWA studies (neither in the initial stage nor in the replication stage).

Control samples

Population-matched controls were obtained from pre-existing previously genotyped studies. German controls were available from the KORA S4/F4 (n = 840) as well as from the GSK (n = 861), the MPIPSYKL (n = 490) and the HNR (n = 380) studies. Of these, 1,220 controls (from KORA S4/F4 and HNR) were previously part of the discovery stage of the IHGC MA study³ and 1,351 controls (from GSK and MPIPSYKL) were used in the replication stage of the IHGC MA study, but were analysed for SNP rs1835740 (*MTDH*). Control samples (n = 2,040) for the Dutch sample were obtained from the Rotterdam Study (RSII)⁷. None of the Dutch controls were used in the clinic-based IHGC MA study and neither the Dutch or and the German controls were part of the discovery stage of the population-based migraine Women's Genome Health Study (WGHS)⁴.

Replication studies

The replication stage of the study consisted of four separately recruited clinic-based migraine without aura patient samples from Finland, Spain, the Netherlands, and Norway. None of the cases or controls used in the replication stage of the study were previously used in the clinic-based IHGC MA study³ or the migraine WGHS⁴.

The Finnish replication sample consisted of unrelated 871 migraine without aura cases and 1,024 controls. Each migraine patient belongs to a multigenerational Finnish family with at least three affected family members. Patients were examined by a neurologist, and fulfilled the validated Finnish Migraine Specific Questionnaire for Family studies (FMSQ_{FS})¹. When

necessary a follow-up telephone interview was performed. All patients were diagnosed by the same headache specialist (M. Kallela) according to the current International Headache Society diagnostic criteria (ICHD-II)². The 1,024 control samples were obtained from the Young Finns study⁸.

The Spanish replication sample was recruited between 2002 and 2010 from Hospital Universitari Vall d'Hebron (HUVH) Barcelona, and consisted of 837 unrelated migraineurs and 971 unrelated migraine-free controls matched for ethnicity (Caucasian Spanish) and sex frequency (75% women). All patients were diagnosed by clinical neurologists in the HUVH team as having MO based on the International Criteria for Headache Disorders 2nd edition (ICHD-II)² after administration of a structured questionnaire and direct interview and examination. The control samples were recruited between 2006 and 2010 and consisted of Caucasian Spanish unrelated adults subjects (blood donors, individuals that underwent surgery unrelated to migraine or unaffected partners of migraine patients) that were matched for sex with patients and recruited in the same geographical area. Migraine and positive family history of migraine or any type of severe or recurrent headache in first-degree relatives were excluded in control subjects through personal interview.

The Dutch replication cohort included 555 MO cases that were recently recruited via the clinic-based Leiden University Migraine Neuro Analysis (LUMINA) study. The diagnosis and classification followed the same procedure as in the initial Dutch sample. Control samples (n = 456) came from volunteers that donated blood to the Leiden blood bank.

The Norwegian replication sample consisted of 245 MO subjects that were consecutively recruited from the outpatient clinic of the Norwegian Headache Center and diagnosed by a neurologist according to the ICHD-II criteria². Control samples (201 subjects) were recruited

from blood-donors in collaboration with the Department of Immunology and Transfusion Medicine in Trondheim Norway. No direct interview was made, but participants filled out a questionnaire to determine eligibility for participation. Criteria for inclusion were age > 40 years (since status as "non-migraineur" cannot be determined with relative certainty before this age), no present or former history of migraine or other types of chronic headaches, and on average less than one headache day per month. Subjects had to be of Caucasian origin.

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