

BMJ Open Association of clinical and genetic risk factors with management of dyslipidaemia: analysis of repeated cross-sectional studies in the general population of Lausanne, Switzerland

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ABSTRACT

Objectives To assess the importance of clinical and genetic factors in management of dyslipidaemia in the general population.

Design Repeated cross-sectional studies (2003–2006; 2009–2012 and 2014–2017) from a population-based cohort.

Setting Single centre in Lausanne, Switzerland.

Participants 617 (42.6% women, mean±SD: 61.6±8.5 years), 844 (48.5% women, 64.5±8.8 years) and 798 (50.3% women, 68.1±9.2) participants of the baseline, first and second follow-ups receiving any type of lipid-lowering drug. Participants were excluded if they had missing information regarding lipid levels, covariates or genetic data.

Primary and secondary outcome

measures Management of dyslipidaemia was assessed according to European or Swiss guidelines. Genetic risk scores (GRSs) for lipid levels were computed based on the existing literature.

Results Prevalence of adequately controlled dyslipidaemia was 52%, 45% and 46% at baseline, first and second follow-ups, respectively. On multivariable analysis, when compared with intermediate or low-risk individuals, participants at very high cardiovascular risk had an OR for dyslipidaemia control of 0.11 (95% CI: 0.06 to 0.18), 0.12 (0.08 to 0.19) and 0.38 (0.25 to 0.59) at baseline, first and second follow-ups, respectively. Use of newer generation or higher potency statins was associated with better control: OR of 1.90 (1.18 to 3.05) and 3.62 (1.65 to 7.92) for second and third generations compared with first in the first follow-up, with the corresponding values in the second follow-up being 1.90 (1.08 to 3.36) and 2.18 (1.05 to 4.51). No differences in GRSs were found between controlled and inadequately controlled subjects. Similar findings were obtained using Swiss guidelines.

Conclusion Management of dyslipidaemia is suboptimal in Switzerland. The effectiveness of high potency statins is hampered by low posology. The use of GRSs in the management of dyslipidaemia is not recommended.

STRENGTHS AND LIMITATIONS OF THIS STUDY

- ⇒ Multiple cross-sectional studies conducted in a population-based cohort.
- ⇒ Three different genetic risk scores and 51 single nucleotide polymorphisms for lipids were tested.
- ⇒ Two criteria to define and treat dyslipidaemia were applied.
- ⇒ Lack of consensus regarding diagnosis and management of dyslipidaemia; results cannot be extrapolated to other settings and populations.
- ⇒ Results based on a single population and hence not forcefully generalisable to other settings and populations.

INTRODUCTION

Adequate management of dyslipidaemia [high Low density lipoprotein (LDL)-cholesterol levels] translates into a reduction in fatal and non-fatal cardiovascular disease (CVD),^{1,2} and guidelines for the management of dyslipidaemia have been issued by the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS).³ Potent hypolipidaemic drugs are available, allowing a considerable reduction in LDL-cholesterol levels.³ Still, management of dyslipidaemia is suboptimal, with a significant percentage of treated patients not reaching target levels.⁴ Likely contributing factors are inadequate perception of risk by physicians,⁵ low compliance by patients⁶ or use of lesser potent drugs.⁴ It has also been suggested that the efficacy of statins, the main hypolipidaemic drugs used, could be modulated by the genetic background of the patients.^{7,8} A recent review suggested that several single nucleotide polymorphisms (SNPs) could be associated with a reduction in the efficacy of statin treatment.⁸ Still, the effect of genetic

markers on the management of dyslipidaemia in the general population has seldom been established.

Thus, we aimed to assess the importance of clinical and genetic factors in the management of dyslipidaemia using data from a population-based cohort.

METHODS

Study population

The CoLausPsyCoLaus (www.colaus-psycolaus.ch) is a prospective cohort study following every 5 years a sample of the inhabitants of the city of Lausanne (Switzerland, population 137 810 in 2017), aged 35–75 years at baseline.⁹ In the present study, data from the baseline (2003–2006), the first (2009–2012) and the second (2014–2017) follow-ups were used.

Inclusion and exclusion criteria

Participants were eligible if they received any type of lipid-lowering drug. Participants were initially excluded if they had missing information regarding lipid levels, covariates, or genetic data.

Lipid-lowering treatment and control of dyslipidaemia

At each survey, participants reported which drugs they were taking. Based on the Anatomical Therapeutic Chemical classification system of the WHO, participants were considered as being treated for dyslipidaemia if they were taking one drug coded C10 ('lipid modifying agents'). Lipid-lowering drugs were further classified into statins, fibrates and other lipid-lowering drugs. For statins, a further classification regarding the generation and potency was performed in the first and second follow-ups (online supplemental table 1). Such classification could not be achieved in the baseline survey due to limited coding. Two approaches regarding statin potency were conducted: (1) not taking into account and (2) taking into account posology as defined by US guidelines.¹⁰ This last approach is similar to another study conducted in Poland.¹¹

As there is no consensus regarding CVD risk assessment in Switzerland, two approaches were applied. The first approach used the ESC/EAS guidelines³ by applying the SCORE equation recalibrated for Switzerland¹² (online supplemental table 2). Three CVD categories were defined: very high, high and other. The second approach used the Swiss Group for Lipids and Atherosclerosis (GSLA) criteria¹³ (online supplemental table 3). Depending on the risk category, the threshold to define adequate control changed (online supplemental tables 2 and 3).

Genetic analysis and genetic scores

Genome-wide genotyping was performed using the Affymetrix 500K SNP array. Subjects were excluded from the analysis in case of inconsistency between sex and genetic data, a genotype call rate of <90%, or inconsistencies of genotyping results in duplicate samples. Quality control

for SNPs was performed using the following criteria: monomorphic (or with minor allele frequency <1%), call rates <90%, deviation from the Hardy-Weinberg equilibrium ($p < 1 \times 10^{-6}$). Phased haplotypes were generated using SHAPEIT2.¹⁴ Imputation was performed using minimac3 and the Haplotype Reference Consortium V.r1.1. Fifty-one SNPs associated with lipid-lowering drug efficiency were extracted (online supplemental table 4) from a previous review.⁸ Genetic risk scores (GRSs) for total, LDL-cholesterol and HDL-cholesterol were computed using 223 SNPs overall as suggested previously.¹⁵ Briefly, the GRSs were calculated with each SNP being weighted by its relative effect size (β coefficient) obtained from the literature (online supplemental table 5).

Other covariates

Sociodemographic and lifestyle data were collected by questionnaire and included gender, age, educational level (low/middle/high), marital status (alone/couple), personal and family history of CVD, family history of dyslipidaemia, smoking (never/former/current) and alcohol consumption (yes/no). Number of other drugs (including or excluding non-prescribed, over-the-counter drugs) were considered as a proxy for the number of comorbidities.

Body weight and height were measured with participants barefoot and in light indoor clothes. Body weight was measured in kilograms to the nearest 100 g using a Seca scale (Hamburg, Germany). Height was measured to the nearest 5 mm using a Seca (Hamburg, Germany) height gauge. Body mass index (BMI) was calculated and categorised into normal ($< 25 \text{ kg/m}^2$), overweight ($25 \leq \text{BMI} < 30 \text{ kg/m}^2$) and obese ($\text{BMI} \geq 30 \text{ kg/m}^2$).

Blood pressure (BP) was measured using an Omron HEM-907 automated oscillometric sphygmomanometer after at least a 10-min rest in a seated position, and the average of the last two measurements was used. Hypertension was defined by a systolic blood pressure $\geq 140 \text{ mm Hg}$ or a diastolic blood pressure $\geq 90 \text{ mm Hg}$ or presence of antihypertensive drug treatment.

Eight-hour fasting blood samples were collected, and biological measurements were conducted in a Modular P apparatus (Roche Diagnostics, Basel, Switzerland) for the baseline and first follow-up, and in a Cobas 8000 (Roche Diagnostics, Basel, Switzerland) device for the second follow-up. The following analytical procedures [with maximum interbatch and intrabatch coefficients of variation (CVs)] were used: total cholesterol by CHOD-PAP (1.6%–1.7%) and high density lipoprotein (HDL)-cholesterol by CHOD-PAP+PEG+cyclodextrin (3.6%–0.9%). Glucose was assessed by glucose dehydrogenase (2.1%–1.0%) at baseline and by glucose hexokinase (1.6%–0.8%) at first and second follow-ups. Diabetes was defined as fasting plasma glucose $\geq 7.0 \text{ mmol/L}$ or presence of an antidiabetic drug treatment.

Statistical analysis

Statistical analyses were conducted using Stata v.16.1 (Stata Corp, College Station, TX, USA) separately for each survey. Results were expressed as number of participants (percentage) for categorical variables and as average \pm SD or median (IQR) for continuous variables. Bivariate comparisons between controlled and uncontrolled participants (using either ESC/EAS or GSLA criteria) were performed using chi-square for categorical variables and Student's t-test or Kruskal-Wallis nonparametric test for continuous variables. Multivariable analyses were conducted using logistic regression for categorical variables and results were expressed as multivariable-adjusted OR and 95% CI.

The associations between specific SNPs and management of dyslipidaemia were assessed by comparing the distribution of the genotypes according to controlled and uncontrolled participants (as defined by ESC/EAS or GSLA criteria) using Fisher's exact test.

Statistical significance was considered for a two-sided test with $p < 0.05$.

Patient and public involvement

None.

RESULTS

Prevalence of dyslipidaemia and changes in statin category

Overall, there were 709, 1056 and 1151 eligible participants at baseline, first and second follow-ups, respectively, of whom 92 (13.0%), 212 (20.1%) and 353 (30.7%) were excluded, leaving 617, 844 and 798 participants for analysis. The reasons for exclusion are indicated in online supplemental figure 1; the main reason was lack of genetic data. The number of participants treated for dyslipidaemia changed between surveys depending on the number of participants newly treated and the number of participants who dropped out. The characteristics of the included and the excluded participants are summarised in online supplemental table 6; excluded participants were less frequently born in Switzerland, while no other consistent difference was found.

The distribution of the different types of lipid-lowering treatments for the three surveys is provided in online supplemental figure 2, and of the statin generations and potency for the first and second follow-ups are provided in online supplemental figure 3. Statins represented the first type of hypolipidaemic drug, but their predominance decreased with time. Prevalence of first generation statins decreased and prevalence of third generation statins increased. Prevalence of low potency statins decreased and high potency statins increased. When posology was considered, statin potency was considerably reduced, but trends were similar (online supplemental figure 3). This decrease in potency was most marked for intermediate potency statins (online supplemental figure 4).

Prevalence and factors associated with control of dyslipidaemia, ESC/EAS criteria

Prevalence of adequately managed dyslipidaemia was 52%, 45% and 46% at baseline, first and second follow-ups, respectively. The results of the analysis using the ESC/EAS criteria stratified by survey are summarised in tables 1–3.

On bivariate analysis (table 1), controlled participants were younger, had lower levels of cardiovascular risk factors and CVD risk and a higher prevalence of parental history of CVD than inadequately controlled participants in all surveys. Controlled participants also had a lower BMI and were taking less drugs than inadequately controlled participants in the first and second follow-ups; prevalence of fibrates was higher among inadequately controlled participants at baseline and in the first follow-up. No differences were found regarding GRSs between controlled and inadequately controlled participants in all surveys. On multivariable analysis (table 2), increased age or CVD risk was negatively associated with control in all surveys; no association was found between type of hypolipidaemic drug or quartiles of the LDL GRS and dyslipidaemia control.

The distribution of the statin generation or potency according to dyslipidaemia control is provided in online supplemental table 7. Controlled participants had a higher prevalence of third generation (first follow-up) or high potency statins than inadequately controlled participants. When posology was used to estimate potency, no differences were found. The results of the multivariable analyses taking into account statin generation or statin potency irrespective of the posology are provided in table 3 and online supplemental table 8, respectively. In both analyses, increasing age or CVD risk led to a lower likelihood of being controlled, while increasing statin generation or potency led to a higher likelihood of being controlled. When posology was used to estimate potency, the association was no longer significant (online supplemental table 9).

Prevalence and factors associated with control of dyslipidaemia, GSLA criteria

Prevalence of adequately managed dyslipidaemia was 70%, 68% and 83% in the baseline, first and second follow-ups, respectively. The results of the analysis using the GSLA criteria stratified by survey are summarised in online supplemental table 10–15.

On bivariate analysis, controlled participants had lower CVD risk (all surveys), lower BMI (first and second follow-ups) and lower prevalence of smoking (first follow-up) than inadequately controlled participants; no differences were found regarding GRS (online supplemental table 10). On multivariable analysis, increased CVD risk was negatively associated with dyslipidaemia control in all surveys; men had a higher likelihood of being controlled (baseline and second follow-up) and alcohol consumption decreased likelihood of control (baseline); no association was found with LDL GRS or the

Table 1 Bivariate comparison of socioeconomic and clinical characteristics among participants treated for dyslipidaemia, according to controlled and uncontrolled status as per European Society of Cardiology/European Atherosclerosis Society criteria

	Baseline		First follow-up		Second follow-up		P value	Controlled	P value	Controlled	P value
	Uncontrolled	Controlled	Uncontrolled	Controlled	Uncontrolled	Controlled					
N	295	322	465	379	428	370					
Age (years)	64.0±8.4	59.5±8.0	67.4±8.6	61.0±7.7	71.4±8.2	64.4±8.9	<0.001		<0.001		<0.001
Women (%)	118 (40.1)	145 (44.9)	186 (40.0)	223 (58.8)	218 (50.9)	183 (49.5)	0.233		<0.001		0.678
Swiss national (%)	210 (71.4)	220 (68.1)	339 (72.9)	261 (68.9)	312 (72.9)	251 (67.8)	0.371		0.198		0.118
Education (%)							0.917		0.512		0.049
High	31 (10.5)	35 (10.8)	62 (13.3)	61 (16.1)	52 (12.2)	68 (18.4)					
Middle	61 (20.8)	71 (22.0)	102 (21.9)	83 (21.9)	99 (23.1)	81 (21.9)					
Low	202 (68.7)	217 (67.2)	301 (64.7)	235 (62.0)	277 (64.7)	221 (59.7)					
Married/couple (%)	199 (67.7)	229 (70.9)	271 (58.3)	234 (61.7)	233 (54.4)	215 (58.1)	0.388		0.308		0.298
BMI (kg/m ²)	28.2±4.4	27.8±4.6	28.1±4.6	27.1±4.9	28.1±4.6	27.0±5.1	0.217		0.002		0.002
BMI categories (%)							0.250		0.001		0.001
Normal	65 (22.1)	90 (27.9)	111 (23.9)	132 (34.8)	112 (26.2)	142 (38.4)					
Overweight	136 (46.3)	141 (43.7)	216 (46.5)	162 (42.7)	185 (43.2)	142 (38.4)					
Obese	93 (31.6)	92 (28.5)	138 (29.7)	85 (22.4)	131 (30.6)	86 (23.2)					
Smoking (%)							0.714		0.239		0.151
Never	101 (34.4)	109 (33.8)	153 (32.9)	142 (37.5)	168 (39.3)	122 (33.0)					
Former	117 (39.8)	138 (42.7)	224 (48.2)	161 (42.5)	190 (44.4)	175 (47.3)					
Current	76 (25.9)	76 (23.5)	88 (18.9)	76 (20.1)	70 (16.4)	73 (19.7)					
Alcohol drinker (%)	224 (76.2)	232 (71.8)	352 (75.7)	280 (73.9)	285 (73.3)	240 (71.2)	0.218		0.544		0.538
Treatment for (%)											
Hypertension	158 (53.7)	145 (44.9)	266 (57.2)	170 (44.9)	258 (60.3)	167 (45.1)	0.028		<0.001		<0.001
Diabetes	68 (23.1)	27 (8.4)	121 (26.0)	27 (7.1)	102 (23.8)	55 (14.9)	<0.001		<0.001		0.001
Parental history (%)	63 (21.4)	101 (31.3)	95 (20.4)	123 (32.5)	95 (22.2)	116 (31.4)	0.006		<0.001		0.003
CVD risk (%)							<0.001		<0.001		<0.001
Other	95 (32.3)	173 (53.6)	140 (30.1)	225 (59.4)	162 (37.9)	193 (52.2)					
High	66 (22.5)	118 (36.5)	98 (21.1)	107 (28.2)	73 (17.1)	94 (25.4)					
Very high	133 (45.2)	32 (9.9)	227 (48.8)	47 (12.4)	193 (45.1)	83 (22.4)					
Number of drugs											
Including OTC	-	-	4(3-6)	4(2-5)	5(3-7)	4(3-7)	<0.001*		<0.001*		<0.001*
Excluding OTC	-	-	4(2-6)	3(2-5)	4(3-7)	3(2-6)	<0.001*		<0.001*		<0.001*

Continued

Table 1 Continued

	Baseline		First follow-up		Second follow-up		P value
	Uncontrolled	Controlled	Uncontrolled	Controlled	Uncontrolled	Controlled	
		P value		P value		P value	
Genetic risk scores							
Total cholesterol	-2.8±9.4	0.149	-3.5±9.2	-3.6±8.9	-4.2±9.6	-3.2±8.1	0.147
LDL-cholesterol	-2.2±7.8	0.117	-2.5±7.5	-2.3±7.1	-3.2±7.6	-2.5±6.3	0.171
HDL-cholesterol	-6.5±3.5	0.215	-6.6±3.6	-6.9±3.6	-6.5±3.6	-6.9±3.6	0.124
Hypolipidaemic drug treatment (%)							
Statins	270 (91.5)	0.035	373 (80.4)	288 (75.8)	328 (76.6)	264 (71.4)	0.089
Fibrates	28 (9.5)	0.044	28 (6.0)	5 (1.3)	18 (4.2)	14 (3.8)	0.762
Other	11 (3.7)	0.834	72 (15.5)	79 (20.8)	84 (19.6)	85 (23.0)	0.249

Data from the baseline (2003–2006), first (2009–2012) and second (2014–2017) follow-ups of the ColausPsyCoLaus study, Lausanne, Switzerland. Results are expressed as number of participants (column %) for categorical variables and as average ± SD or as median (IQR) for continuous variables. Between-groups comparisons performed using χ^2 for categorical variables and Student's t-test or Kruskal-Wallis nonparametric test (*) for continuous variables. BMI, body mass index; CVD, cardiovascular disease; HDL^c, high density lipoproteins; LDL^c, low density lipoproteins; OTC, over the counter.

class of hypolipidaemic drug (online supplemental table 11).

The distribution of the statin generation or potency according to dyslipidaemia control is provided in online supplemental table 12. Controlled participants had a higher prevalence of third generation or high potency statins than inadequately controlled participants. When posology was used to estimate potency, no differences were found. The results of the multivariable analyses taking into account statin generation or statin potency irrespective of the posology are provided in online supplemental tables 13 and 14, respectively. In both analyses, increasing CVD risk led to a lower likelihood of being adequately controlled, while being male, increasing number of drugs or increasing statin generation or potency led a higher likelihood of being adequately controlled. When posology was used to estimate statin potency, the association was no longer significant (online supplemental table 15).

Specific SNPs

The p values for the associations between 51 specific SNPs and dyslipidaemia control are presented in online supplemental table 16. Most statistically significant associations were found for *SLCO1B1* (Solute Carrier Organic Anion Transporter Family Member 1B1), but no consistent association was found overall.

DISCUSSION

Our results show that individuals at high risk of CVD present an increased risk of mismanagement of dyslipidaemia, which was consistent among the three survey periods. The use of more potent statins increased the likelihood of dyslipidaemia control, while lipid GRSs were not associated with dyslipidaemia control.

Prevalence of controlled dyslipidaemia

Prevalence of controlled dyslipidaemia varied between 45% and 52% according to ESC/EAS criteria and between 68% and 83% according to GSLA criteria. Those values are higher than the values reported by the EUROASPIRE IV, where a third (32.7%) of participants achieved the target level of <2.5 mmol/L for LDL-cholesterol.¹⁶ Still, comparisons are difficult as EUROASPIRE IV focused on high-risk participants only. Importantly, prevalence rates of adequately managed dyslipidaemia were much higher using GSLA than ESC/EAS criteria. Hence, a clinician using GSLA criteria will lower LDL levels to a lesser level than using ESC/EAS criteria. Given that CVD risk decreases linearly with LDL-cholesterol levels (all other factors being equal),¹⁷ the decrease in CVD risk is expected to be lower using GSLA than using ESC/EAS criteria. It would be important to evaluate if people managed according to the GSLA criteria achieve the same level of protection against CVD as if they were managed according to the ESC/EAS criteria.

Factors associated with controlled dyslipidaemia

Participants at high risk of CVD had a higher likelihood of being inadequately managed in all surveys, irrespective

Table 2 Multivariable analysis of the factors associated with dyslipidaemia control as per European Society of Cardiology/European Atherosclerosis Society criteria

	Baseline		First follow-up		Second follow-up	
	OR (95% CI)	P value	OR (95% CI)	P value	OR (95% CI)	P value
Age (per 10 years increase)	0.44 (0.34 to 0.57)	<0.001	0.34 (0.27 to 0.43)	<0.001	0.35 (0.28 to 0.44)	<0.001
Man vs woman	0.89 (0.58 to 1.36)	0.599	0.48 (0.34 to 0.70)	<0.001	1.23 (0.85 to 1.79)	0.273
Swiss vs Non-Swiss	1.30 (0.85 to 1.97)	0.223	1.13 (0.79 to 1.64)	0.503	1.01 (0.69 to 1.48)	0.948
Education						
High	1 (ref.)		1 (ref.)		1 (ref.)	
Middle	1.11 (0.54 to 2.27)	0.782	0.83 (0.48 to 1.45)	0.521	0.61 (0.35 to 1.07)	0.087
Low	1.11 (0.59 to 2.11)	0.738	0.81 (0.49 to 1.33)	0.407	0.65 (0.40 to 1.07)	0.093
<i>P</i> value for trend	0.738		0.407		0.093	
Married vs not married	1.21 (0.80 to 1.82)	0.374	1.21 (0.86 to 1.71)	0.270	1.06 (0.74 to 1.5)	0.759
Body mass index categories						
Normal	1 (ref.)		1 (ref.)		1 (ref.)	
Overweight	0.59 (0.37 to 0.94)	0.028	0.85 (0.57 to 1.26)	0.413	0.61 (0.40 to 0.92)	0.019
Obese	0.92 (0.54 to 1.56)	0.753	0.96 (0.59 to 1.55)	0.852	0.64 (0.40 to 1.04)	0.074
<i>P</i> value for trend	0.753		0.852		0.074	
Smoking categories						
Never	1 (ref.)		1 (ref.)		1 (ref.)	
Former	1.54 (0.98 to 2.40)	0.059	1.10 (0.75 to 1.62)	0.618	1.38 (0.94 to 2.02)	0.099
Current	0.63 (0.36 to 1.08)	0.094	0.85 (0.52 to 1.39)	0.512	1.04 (0.61 to 1.75)	0.892
<i>P</i> value for trend	0.094		0.512		0.892	
Alcohol drinker (yes vs no)	0.66 (0.42 to 1.03)	0.068	0.91 (0.62 to 1.35)	0.645	0.64 (0.43 to 0.94)	0.024
Antihypertensive treatment (yes vs no)	1.05 (0.70 to 1.57)	0.820	1.18 (0.82 to 1.69)	0.368	0.77 (0.53 to 1.12)	0.176
Parental history (yes vs no)	1.00 (0.64 to 1.56)	0.998	1.08 (0.74 to 1.59)	0.690	0.79 (0.53 to 1.18)	0.256
CVD risk						
Other	1 (ref.)		1 (ref.)		1 (ref.)	
High	1.25 (0.76 to 2.04)	0.379	0.68 (0.45 to 1.04)	0.074	1.42 (0.88 to 2.28)	0.153
Very high	0.11 (0.06 to 0.18)	<0.001	0.12 (0.08 to 0.19)	<0.001	0.38 (0.25 to 0.59)	<0.001
<i>P</i> value for trend	<0.001		<0.001		<0.001	
LDL genetic risk score quartiles						
First	1 (ref.)		1 (ref.)		1 (ref.)	
Second	1.11 (0.65 to 1.89)	0.696	0.97 (0.61 to 1.54)	0.883	1.31 (0.81 to 2.12)	0.264
Third	0.70 (0.41 to 1.19)	0.185	0.96 (0.60 to 1.53)	0.866	1.57 (0.97 to 2.51)	0.064
Fourth	0.74 (0.43 to 1.25)	0.259	1.07 (0.67 to 1.72)	0.764	1.44 (0.89 to 2.32)	0.133
<i>P</i> value for trend	0.107		0.781		0.099	
Hypolipidaemic drug treatment						
Statins	1.56 (0.18 to 13.8)	0.690	1.00 (0.55 to 1.81)	0.998	1.42 (0.81 to 2.51)	0.223
Fibrates	0.67 (0.08 to 5.35)	0.707	0.13 (0.04 to 0.43)	0.001	1.20 (0.46 to 3.15)	0.714
Other	0.94 (0.34 to 2.61)	0.910	0.57 (0.31 to 1.04)	0.067	0.71 (0.40 to 1.26)	0.237

Data from the baseline (2003–2006), first (2009–2012) and second (2014–2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland. Results are expressed as odds ratio and (95% CI). Statistical analysis was done using logistic regression. CVD, cardiovascular disease.

of the criteria considered. Those findings are consistent with a recent review of European studies¹⁸ and a large cross-sectional European study,¹⁹ where control rates were lower than 20%. Possible explanations include the

fact that subjects at high CVD risk should have much lower lipid values, thus more difficult to achieve.

Similar to a Polish study¹¹ but contrary to a US study,²⁰ the prevalence of highly potent statins increased from

Table 3 Multivariable analysis of the factors associated with dyslipidaemia control as per European Society of Cardiology/European Atherosclerosis Society criteria

	First follow-up		Second follow-up	
	OR (95% CI)	P value	OR (95% CI)	P value
Age (per 10 years increase)	0.31 (0.23 to 0.41)	<0.001	0.35 (0.27 to 0.47)	<0.001
Man vs woman	0.60 (0.39 to 0.92)	0.018	1.40 (0.90 to 2.16)	0.134
Swiss vs Non-Swiss	1.17 (0.76 to 1.80)	0.474	1.16 (0.75 to 1.80)	0.502
Education				
High	1 (ref.)		1 (ref.)	
Middle	0.78 (0.40 to 1.53)	0.476	1.08 (0.54 to 2.15)	0.821
Low	0.88 (0.49 to 1.59)	0.681	1.04 (0.57 to 1.90)	0.895
<i>P value for trend</i>	0.681		0.895	
Married vs not married	1.23 (0.82 to 1.83)	0.317	1.19 (0.79 to 1.78)	0.407
Body mass index categories				
Normal	1 (ref.)		1 (ref.)	
Overweight	0.84 (0.52 to 1.35)	0.474	0.56 (0.35 to 0.92)	0.023
Obese	0.91 (0.53 to 1.58)	0.749	0.54 (0.31 to 0.95)	0.032
<i>P value for trend</i>	0.749		0.032	
Smoking categories				
Never	1 (ref.)		1 (ref.)	
Former	1.09 (0.70 to 1.71)	0.695	1.21 (0.78 to 1.88)	0.384
Current	0.84 (0.46 to 1.51)	0.560	0.93 (0.51 to 1.71)	0.820
<i>P value for trend</i>	0.560		0.820	
Alcohol drinker (yes vs no)	0.79 (0.50 to 1.25)	0.316	0.72 (0.46 to 1.12)	0.146
AntiHTA ttt (yes vs no)	0.97 (0.63 to 1.51)	0.903	0.80 (0.52 to 1.25)	0.337
Parental history (yes vs no)	1.27 (0.80 to 2.02)	0.310	0.76 (0.48 to 1.23)	0.267
CVD risk				
Other	1 (ref.)		1 (ref.)	
High	0.63 (0.39 to 1.02)	0.061	1.32 (0.76 to 2.31)	0.327
Very high	0.08 (0.05 to 0.14)	<0.001	0.35 (0.21 to 0.58)	<0.001
<i>P value for trend</i>	<0.001		<0.001	
LDL genetic risk score quartiles				
First	1 (ref.)		1 (ref.)	
Second	0.90 (0.53 to 1.54)	0.707	1.67 (0.95 to 2.93)	0.076
Third	0.89 (0.52 to 1.52)	0.665	1.79 (1.04 to 3.07)	0.036
Fourth	1.11 (0.65 to 1.92)	0.696	1.64 (0.93 to 2.86)	0.085
<i>P value for trend</i>	0.725		0.085	
Number of drugs (per one unit)	1.15 (1.05 to 1.25)	0.002	1.07 (0.99 to 1.15)	0.069
Statin generation				
First	1 (ref.)		1 (ref.)	
Second	1.90 (1.18 to 3.05)	0.008	1.90 (1.08 to 3.36)	0.026
Third	3.62 (1.65 to 7.92)	0.001	2.18 (1.05 to 4.51)	0.036
<i>P value for trend</i>	0.001		0.036	
Fibrates	NC		2.55 (0.19 to 34.1)	0.480
Other hypolipidaemic drugs	0.90 (0.38 to 2.11)	0.800	1.13 (0.51 to 2.51)	0.762

Data from the first (2009–2012) and second (2014–2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland. Analysis was done taking into account statin generation.

Results are expressed as OR and (95% CI). Statistical analysis was done using logistic regression.

NC, not computable; antiHTA ttt, antihypertensive drug treatment.

41% in 2009–2012 to 56% in 2014–2017. This value is higher than reported in the EUROASPIRE V⁴ study, where 49.9% of participants were on high-intensity therapy. Importantly, participants on high potency statins achieved better control, a finding also reported in the EUROASPIRE V⁴ and the DA VINCI¹⁹ studies. The 2019 ESC/EAS guidelines for management of dyslipidaemia recommend that high potency statins at highest recommended tolerable dose be initially applied to control lipid levels.³ Our results thus strengthen the importance of such recommendation, and general practitioners should be urged to shift to more potent statins to achieve better results. Still, our results also suggest that, despite a higher prescription rate of highly potent statins, those drugs are not prescribed at their full potency/posology, and that a sizeable fraction of treated subjects fails to reach lipid targets.

Other clinical and sociodemographic factors were associated with dyslipidaemia control, but associations were inconsistent between study periods or between ESC/EAS and GSLA criteria. Increasing age was negatively associated with dyslipidaemia control using ESC/EAS criteria but no association was found using GSLA criteria. Either no association,²¹ an inverse association²² or a positive association²³ between age and dyslipidaemia control have been reported. Similarly, men achieved better control than women using the GSLA criteria, while no consistent association was found using the ESC/EAS criteria. Better control rates have also been reported in the USA,²⁴ while the inverse association was reported in Germany.²² Such discrepancies might be related to the criteria applied, as age and gender might be stronger or weaker determinants of CVD risk in some risk equations compared with others. No association was found between nationality, education, marital status, job type or BMI categories and dyslipidaemia control. Our findings replicate those of other studies where no association between education,²¹ marital status^{21 22} and dyslipidaemia control was found. Overall, our results suggest that the sociodemographic factors associated with dyslipidaemia control differ according to country and to the criteria used to estimate CVD risk.

Genetic scores and individual SNPs

Several authors suggested that genetic profiling could be used to guide statin treatment and thus improve outcomes.^{7 8} A meta-analysis published in 2015 concluded that people with the highest burden of genetic risk derived the largest relative and absolute clinical benefit from statin therapy,²⁵ although such statement could also apply to people for whom cardiovascular risk was assessed using clinical data. Further, the initial promises regarding genetic testing of the kinesin-like protein 6 (KIF6) gene to guide statin prescription (the StatinCheck test) were not confirmed.²⁶ In this study, no association between genetic scores for lipid markers and statin efficiency was found. Possible reasons include the small effect of each individual SNP,²⁷ as a set of 95 SNPs explained <15% of

total lipid variance,²⁸ or the progressive blunting of the genetic effect by advanced ageing as found for BP.²⁹ Thus, our results suggest that genetic profiling of subjects prior to initiation of statin therapy might be clinically irrelevant, and such profiling is not stated in the current ESC/EAS guidelines for the management of dyslipidaemias.³ Nevertheless, several associations were found with the *SLCO1B1* gene. Some authors have suggested that genetic variations in this gene are associated with response to statins.⁸ Hence, this gene might be of interest to adapt statin treatment, and it would be important that other studies be conducted to confirm our findings.

Study limitations

This study has several limitations worth acknowledging. First, the sample size was relatively small and our study was likely underpowered to detect the minute associations between the genetic scores and dyslipidaemia control. Still, should those GRSs be applied in clinical practice, their effect should be large enough to allow choosing between several statins in a given individual. Second, the analysis was restricted to Switzerland, and findings might be generalisable to other countries or ethnicities. Still, most findings agree with larger studies such as EUROASPIRE V⁴ or DA VINCI.¹⁹ Third, there is no consensus regarding the management of dyslipidaemia, as thresholds for treatment vary according to country or scientific society.³⁰ Hence, our results cannot be extrapolated to other settings, and it would be important that similar studies be conducted in other countries.

CONCLUSION

Management of dyslipidaemia is suboptimal in Switzerland, especially for individuals at high cardiovascular risk. The effectiveness of high potency statins is hampered by low posology. GRSs are not associated with dyslipidaemia control, but the effect of *SLCO1B1* in statin therapy should be further investigated.

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Contributors VC: investigation, methodology, writing—original draft preparation, visualisation. PM-V: conceptualisation, methodology, data curation, formal analysis, writing—reviewing and editing, visualisation. NA: writing—reviewing and editing. JV: reviewing and editing. The authors had full access to the data and took responsibility for its integrity. All authors have read and agreed to the written manuscript. PM-V had full access to the data and is the guarantor of the study.

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Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval The institutional Ethics Committee of the University of Lausanne, which afterwards became the Ethics Commission of Canton Vaud (www.cer-vd.ch), approved the baseline (reference 16/03), the first (reference 33/09) and the second

(reference 26/14) follow-ups. The approval was confirmed in 2021 (reference PB_2018-00038, 239/09). The study was performed in agreement with the Helsinki declaration and its former amendments, and in accordance with the applicable Swiss legislation. All participants gave their signed informed consent before entering the study. Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request.

Non-identifiable individual-level data are available for researchers who seek to answer questions related to health and disease in the context of research projects who meet the criteria for data sharing by research committees. Please follow the instructions at <https://www.colaus-psycolaus.ch/> for information on how to submit an application for gaining access to CoLausPsyCoLaus data.

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SUPPLEMENTAL INFORMATION**Supplemental table 1:** statins classification regarding generation and potency.

Statin	ATC code	Generation	Potency, no posology	Potency, with posology
lovastatin	C10AA02	First	Low	20 low 40 intermediate
pravastatin	C10AA03	First	Low	<40 low 40-80 intermediate
fluvastatin	C10AA04	First	Low	10 low 40-80 intermediate
simvastatin	C10AA01	Second	Intermediate	20-40 intermediate 80 high
pitavastatin	C10AA08	Second	Intermediate	2 intermediate 4 high
atorvastatin	C10AA05	Second	High	10-20 intermediate 40-80 high
cerivastatin	C10AA06	Third	High	100 intermediate 300 high
rosuvastatin	C10AA07	Third	High	5-10 intermediate 20 high

Statins were ordered according to their potency. Adapted from ^{1,2}.

Supplemental table 2: LDL-cholesterol thresholds used to define control of dyslipidemia according to the European Society of Cardiology / European Atherosclerosis Society (ESC/EAS) criteria.

Risk categories according to SCORE	Control
<i>Very high risk</i>	
10–y risk >10%, diabetes or personal history of CVD	<1.8 mmol/L (70 mg/dL)
<i>High risk</i>	
10–y risk 5% – 10%	<2.6 mmol/L (100 mg/dL)
<i>Other categories</i>	
Less than 2 CVD risk factors and 10–y risk <5%	<4.0 mmol/L (155 mg/dL)

ESC/EAS criteria as stated in ³. Risk categories based on the SCORE equation recalibrated for the Swiss population ⁴.

Supplemental table 3: LDL-cholesterol thresholds used to define control of dyslipidemia according to the Swiss Group for Lipids and Atherosclerosis.

Risk categories *	Control
<i>High-risk</i>	
10-y risk >20%, diabetes or personal history of CVD	<2.6 mmol/L (100 mg/dL)
<i>Intermediate risk</i>	
At least two CVD risk factors and 10-y risk 10% – 20%	<3.4 mmol/L (130 mg/dL)
<i>Low risk</i>	
At least two CVD risk factors and 10-y risk <10%	<4.2 mmol/L (160 mg/dL)
<i>Very low risk</i>	
Less than 2 CVD risk factors and 10-y risk <10%	<4.2 mmol/L (160 mg/dL)

* Risk calculation as defined in <https://www.agla.ch/fr/calculateurs-outils/calculateur-de-risque-du-gsla>. According to ⁵.

Supplemental table 4: list of specific SNPs interacting with the efficacy of statin treatment.

Gene	SNP	Simvastatin	Pravastatin	Atorvastatin	Rosuvastatin	Lovastatin	Fluvastatin
<i>ABCB1</i>	rs1045642	X, ↑		O			
<i>ABCB1</i>	rs1128503	↑					
<i>ABCB1</i>	rs1922242	O MI	O MI low	O MI low			O
<i>ABCB1</i>	rs2032582	↑					
<i>ABCB1</i>	rs3789244	O MI	O MI	O MI			
<i>ABCG2/BCRP</i>	rs2199936				O		
<i>ABCG2/BCRP</i>	rs2231137				X		
<i>ABCG2/BCRP</i>	rs2231142			O	X		X
<i>ABCG8</i>	rs11887534			O			
<i>ACE</i>	rs1799752						↑
<i>APOA1</i>	rs670		O	O			
<i>APOA1</i>	rs5069			O			
<i>APOA5</i>	rs662799	O		O		O	
<i>APOC1</i>	rs4420638	O	O	O	O		
<i>APOE</i>	rs429358	O		↑		↓	
<i>APOE</i>	rs445925			X			
<i>APOE</i>	rs449647			↑			
<i>APOE</i>	rs71352238				O		
<i>APOE</i>	rs7412		↑	↑		↓	
<i>CETP</i>	rs5783961						O
<i>CETP</i>	rs5882	O					
<i>CETP</i>	rs708272	O	O	O			
<i>CLMN</i>	rs8014194	O	O	O			
<i>CYP2C9</i>	rs1057910						X
<i>CYP2D6</i>	rs3892097			X			
<i>CYP3A4</i>	rs2740574			↑			
<i>CYP3A4</i>	rs355599367	X, ↑					
<i>CYP3A4</i>	rs55951658	O					
<i>CYP3A5</i>	rs776746	↓		X, ↓		↓	
<i>CYP7A1</i>	rs3808607			↓			
<i>ESR1</i>	rs2234693			O			
<i>ESR1</i>	rs9340799			-			
<i>HMGCR</i>	rs12916			O			
<i>HMGCR</i>	rs17238540		-				
<i>HMGCR</i>	rs17244841		↓				
<i>HMGCR</i>	rs17671591			X			
<i>HMGCR</i>	rs5908			-			
<i>KIF6</i>	rs20455		O	O			
<i>LDLR</i>	rs5925						↑
<i>LPA</i>	rs10455872			-	O		
<i>LPL</i>	rs328				O		
<i>PCSK9</i>	rs17111584				O		
<i>PON1</i>	rs662		O				
<i>SCAP</i>	rs12487736	↑					
<i>SCCO1B1</i>	rs2306283		↓		X		↑
<i>SLCO1B1</i>	rs11045819						↑
<i>SLCO1B1</i>	rs4149015		X				
<i>SLCO1B1</i>	rs4149056	X, ↓	X, ↓	X, ↓	X		
<i>SOD2</i>	rs4880				↑		
<i>SREBF2</i>	rs4822063			↓			
<i>TLR4</i>	rs4986790		O				

↑, increases effect; ↓, decreases effect; X, safety issues; O, other; MI, myocardial infarction. Data adapted from ⁶.

Supplemental table 5: data used to compute the genetic risk scores for total, LDL and HDL-cholesterol levels.

Score / Locus	Chr	SNP	Alleles (+/-)	Minor allele	β for weighting
Total cholesterol					
<i>ASAP3</i>	1	rs1077514	T/C	C	-0.030
<i>LDLRAP1</i>	1	rs12027135	T/A	A	-1.22
<i>PCSK9</i>	1	rs2479409	G/A	G	1.96
<i>MOSC1</i>	1	rs2807834	G/T	T	-1.38
<i>ANGPTL3</i>	1	rs3850634	T/G	G	-2.60
<i>IRF2BP2</i>	1	rs514230	T/A	A	-1.36
<i>SORT1</i>	1	rs629301	T/G	G	-5.41
<i>EVI5</i>	1	rs7515577	A/C	C	-1.18
<i>INSIG2</i>	2	rs10490626	G/A	A	0.042
<i>UGT1A1</i>	2	rs11563251	T/C	T	0.037
<i>FAM117B</i>	2	rs11694172	G/A	G	0.028
<i>GCKR</i>	2	rs1260326	T/C	T	1.91
<i>APOB</i>	2	rs1367117	A/G	A	4.16
<i>LOC8431</i>	2	rs2030746	T/C	T	0.020
<i>ABCB11</i>	2	rs2287623	G/A	G	0.027
<i>ABCG5/8</i>	2	rs4299376	G/T	G	3.01
<i>RAB3GAP1</i>	2	rs6759321	T/G	T	1.18
<i>PXK</i>	3	rs13315871	G/A	A	-0.036
<i>RAF1</i>	3	rs2290159	G/C	C	-1.42
<i>CMTM6</i>	3	rs7640978	C/T	T	-0.038
<i>LRPAP1</i>	4	rs6831256	G/A	G	-0.022
<i>HMGCR</i>	5	rs12916	C/T	C	2.84
<i>CSNK1G3</i>	5	rs4530754	A/G	G	-0.023
<i>TIMD4</i>	5	rs6882076	C/T	T	-1.98
<i>LPA</i>	6	rs1564348	C/T	C	2.18
<i>HFE</i>	6	rs1800562	G/A	A	-2.16
<i>KCNK17</i>	6	rs2758886	A/G	A	0.023
<i>MYLIP</i>	6	rs3757354	C/T	T	-1.46
<i>HBS1L</i>	6	rs9376090	C/T	T	-0.025
<i>FRK</i>	6	rs9488822	A/T	T	-1.18
<i>GPR146</i>	7	rs1997243	G/A	G	0.033
<i>DNAH11</i>	7	rs2285942	T/C	T	1.70
<i>SOX17</i>	8	rs10102164	A/G	A	0.030
<i>CYP741</i>	8	rs1030431	A/G	A	1.26
<i>PLEC1</i>	8	rs11136341	G/A	G	1.34
<i>NAT2</i>	8	rs1961456	G/A	G	1.07
<i>PPP1R3B</i>	8	rs2126259	C/T	T	-3.14
<i>TRPS1</i>	8	rs2737229	A/C	C	-1.11
<i>TRIB1</i>	8	rs2954022	C/A	A	-2.30
<i>ABCA1</i>	9	rs1883025	C/T	T	-2.24
<i>VLDLR</i>	9	rs3780181	A/G	G	-0.044
<i>TTC39B</i>	9	rs581080	C/G	G	-1.57
<i>ABO</i>	9	rs651007	T/C	T	2.30
<i>VIM-CUBN</i>	10	rs10904908	G/A	G	0.025
<i>GPAM</i>	10	rs2255141	A/G	A	1.14
<i>MARCH8-ALOX5</i>	10	rs970548	C/A	C	-0.026

<i>SPTY2D1</i>	11	rs10832963	G/T	T	-1.06
<i>ST3GAL4</i>	11	rs11220463	T/A	T	2.01
<i>PHLDB1</i>	11	rs11603023	T/C	T	0.022
<i>FADS1-2-3</i>	11	rs174550	T/C	C	-1.78
<i>UBASH3B</i>	11	rs7941030	C/T	C	0.97
<i>APOA1</i>	11	rs964184	G/C	G	4.68
<i>PHC1-A2ML1</i>	12	rs4883201	A/G	G	-0.035
<i>LIPC</i>	15	rs1532085	A/G	A	1.54
<i>HPR</i>	16	rs2000999	A/G	A	2.34
<i>CETP</i>	16	rs3764261	A/C	A	1.67
<i>DLG4</i>	17	rs314253	T/C	C	-0.023
<i>OSBPL7</i>	17	rs7206971	A/G	A	1.01
<i>LIPG</i>	18	rs7239867	G/A	A	-1.94
<i>CILP2</i>	19	rs10401969	T/C	C	-4.74
<i>FLJ36070</i>	19	rs492602	G/A	G	1.27
<i>LDLR</i>	19	rs6511720	G/T	T	-7.09
<i>HNF4A</i>	20	rs1800961	C/T	T	-4.73
<i>ERGIC3</i>	20	rs2277862	C/T	T	-1.19
<i>MAFB</i>	20	rs2902940	A/G	G	-1.38
<i>TOP1</i>	20	rs4297946	C/G	C	1.52
<i>TOM1</i>	22	rs138777	A/G	A	0.021
<i>PPARA</i>	22	rs4253772	T/C	T	0.032
LDL cholesterol					
<i>LDLRAP1</i>	1	rs12027135	T/A	A	-1.10
<i>PIGV-NROB2</i>	1	rs12748152	T/C	T	0.05
<i>PCSK9</i>	1	rs2479409	G/A	G	2.01
<i>ANXA9-CERS2</i>	1	rs267733	A/G	G	-0.03
<i>MOSC1</i>	1	rs2807834	G/T	T	-1.09
<i>ANGPTL3</i>	1	rs3850634	T/G	G	-1.59
<i>IRF2BP2</i>	1	rs514230	T/A	A	-1.13
<i>SORT1</i>	1	rs629301	T/G	G	-5.65
<i>INSIG2</i>	2	rs10490626	G/A	A	-0.05
<i>UGT1A1</i>	2	rs11563251	T/C	T	0.03
<i>FN1</i>	2	rs1250229	C/T	T	-0.02
<i>APOB</i>	2	rs1367117	A/G	A	4.05
<i>LOC84931</i>	2	rs2030746	T/C	T	0.02
<i>EHBP1</i>	2	rs2710642	A/G	G	-0.02
<i>ABCG5/8</i>	2	rs4299376	G/T	G	2.75
<i>ACAD11</i>	3	rs17404153	G/T	T	-0.03
<i>CMTM6</i>	3	rs7640978	C/T	T	-0.04
<i>LRPAP1</i>	4	rs6831256	G/A	G	0.02
<i>HMGCR</i>	5	rs12916	C/T	C	2.45
<i>CSNK1G3</i>	5	rs4530754	A/G	G	-0.03
<i>TIMD4</i>	5	rs6882076	C/T	T	-1.67
<i>FRK</i>	6	rs11153594	C/T	T	-0.89
<i>LPA</i>	6	rs1564348	C/T	C	1.95
<i>HFE</i>	6	rs1800562	G/A	A	-2.22
<i>MYLIP</i>	6	rs3757354	C/T	T	-1.43
<i>DNAH11</i>	7	rs12670798	C/T	C	1.26
<i>NPC1L1</i>	7	rs217386	G/A	A	-1.17
<i>SOX17</i>	8	rs10102164	A/G	A	0.03
<i>CYP741</i>	8	rs1030431	A/G	A	0.95

<i>PLEC1</i>	8	rs11136341	G/A	G	1.40
<i>PPP1R3B</i>	8	rs2126259	C/T	T	-3.14
<i>TRIB1</i>	8	rs2954022	C/A	A	-1.84
<i>VLDLR</i>	9	rs3780181	A/G	G	-0.04
<i>ABO</i>	9	rs649129	T/C	T	2.05
<i>GPAM</i>	10	rs1129555	A/G	A	1.08
<i>ST3GAL4</i>	11	rs11220462	A/G	A	1.95
<i>FADS1-2-3</i>	11	rs174583	C/T	T	-1.71
<i>APOA1</i>	11	rs964184	G/C	G	2.85
<i>BRCA2</i>	13	rs4942486	T/C	T	0.02
<i>HPR</i>	16	rs2000999	A/G	A	2.00
<i>CETP</i>	16	rs247616	C/T	T	-1.45
<i>DLG4</i>	17	rs314253	T/C	C	-0.02
<i>OSBPL7</i>	17	rs7206971	A/G	A	1.01
<i>CILP2</i>	19	rs10401969	T/C	C	-3.11
<i>SNX5</i>	20	rs2328223	C/A	C	0.03
<i>MAFB</i>	20	rs2902941	A/G	G	-0.98
<i>SPTLC3</i>	20	rs364585	G/A	A	-0.03
<i>TOP1</i>	20	rs909802	T/C	T	1.41
<i>PPARA</i>	22	rs4253772	T/C	T	0.03
<i>MTMR3</i>	22	rs5763662	T/C	T	0.08
HDL cholesterol					
<i>HDGF-PMVK</i>	1	rs12145743	G/T	G	0.200
<i>PIGV-NROB2</i>	1	rs12748152	C/T	T	-0.051
<i>ZNF648</i>	1	rs1689800	G/A	G	-0.47
<i>ANGPTL1</i>	1	rs4650994	G/A	G	0.021
<i>PABPC4</i>	1	rs4660293	G/A	G	-0.48
<i>GALNT2</i>	1	rs4846914	G/A	G	-0.61
<i>APOB</i>	2	rs1042034	T/C	C	0.90
<i>COBLL1</i>	2	rs12328675	T/C	C	0.68
<i>IRS1</i>	2	rs1515100	A/C	C	0.46
<i>STAB1</i>	3	rs13326165	A/G	A	0.029
<i>ACAD11</i>	3	rs17404153	G/T	T	0.028
<i>RBM5</i>	3	rs2013208	T/C	T	0.025
<i>ATG7</i>	3	rs2606736	C/T	C	0.025
<i>GSK3B</i>	3	rs6805251	T/C	T	0.020
<i>C4orf52</i>	4	rs10019888	A/G	G	-0.027
<i>SLC39A8</i>	4	rs13107325	T/C	T	-0.84
<i>ADH5</i>	4	rs2602836	A/G	A	0.019
<i>FAM13A</i>	4	rs3822072	G/A	A	-0.025
<i>ARL15</i>	5	rs6450176	A/G	A	-0.49
<i>RSPO3</i>	6	rs1936800	C/T	C	0.020
<i>C6orf106</i>	6	rs2814944	A/G	A	-0.49
<i>CITED2</i>	6	rs605066	C/T	C	-0.39
<i>VEGFA</i>	6	rs998584	C/A	A	-0.026
<i>MLXIPL</i>	7	rs17145738	C/T	T	0.57
<i>TMEM176A</i>	7	rs17173637	T/C	C	-0.036
<i>SNX13</i>	7	rs4142995	G/T	T	-0.026
<i>KLF14</i>	7	rs4731702	C/T	T	0.59
<i>IKZF1</i>	7	rs4917014	G/T	G	0.022
<i>DAGLB</i>	7	rs702485	G/A	G	0.024
<i>TRIB1</i>	8	rs10808546	C/T	T	0.61

<i>LPL</i>	8	rs12678919	A/G	G	2.25
<i>TRPS1</i>	8	rs2293889	T/G	T	-0.44
<i>PPP1R3B</i>	8	rs9987289	A/G	A	-1.21
<i>ABCA1</i>	9	rs1883025	T/C	T	-0.94
<i>TTC39B</i>	9	rs643531	C/A	C	-0.72
<i>MARCH8-ALOX5</i>	10	rs970548	C/A	C	0.026
<i>OR4C46</i>	11	rs11246602	C/T	C	0.034
<i>KAT5</i>	11	rs12801636	A/G	A	0.024
<i>FADS1-2-3</i>	11	rs174601	T/C	T	-0.73
<i>AMPD3</i>	11	rs2923084	G/A	G	-0.41
<i>LRP4</i>	11	rs3136441	T/C	C	0.78
<i>MOGAT2-DGAT2</i>	11	rs499974	C/A	A	-0.026
<i>UBASH3B</i>	11	rs7115089	C/G	G	0.31
<i>APOA1</i>	11	rs964184	G/C	G	-1.50
<i>LRP1</i>	12	rs3741414	C/T	T	0.46
<i>SBNO1</i>	12	rs4759375	C/T	T	0.86
<i>ZNF664</i>	12	rs4765127	G/T	T	0.44
<i>PDE3A</i>	12	rs7134375	C/A	A	0.40
<i>MVK</i>	12	rs7134594	C/T	C	-0.44
<i>SCARB1</i>	12	rs838880	T/C	C	0.61
<i>LIPC</i>	15	rs1532085	G/A	A	1.45
<i>LACTB</i>	15	rs2652834	A/G	A	-0.39
<i>FTO</i>	16	rs1121980	G/A	A	-0.020
<i>LCAT</i>	16	rs16942887	G/A	A	1.27
<i>CMIP</i>	16	rs2925979	T/C	T	-0.45
<i>CETP</i>	16	rs3764261	C/A	A	3.39
<i>PGS1</i>	17	rs4082919	G/T	G	-0.40
<i>ABCA8</i>	17	rs4148008	G/C	G	-0.42
<i>STARD3</i>	17	rs881844	C/G	C	-0.51
<i>MC4R</i>	18	rs12967135	A/G	A	-0.42
<i>LIPG</i>	18	rs7241918	G/T	G	-1.31
<i>HAS1</i>	19	rs17695224	G/A	A	-0.029
<i>LILRA3</i>	19	rs386000	G/C	C	0.83
<i>ANGPTL4</i>	19	rs7255436	C/A	C	-0.45
<i>PEPD</i>	19	rs731839	A/G	G	-0.022
<i>LOC55908</i>	19	rs737337	C/T	C	-0.64
<i>HNF4A</i>	20	rs1800961	T/C	T	-1.88
<i>PLTP</i>	20	rs6065906	C/T	C	-0.93
<i>UBE2L3</i>	22	rs181362	T/C	T	-0.46

Chr, chromosome; SNP, single nucleotide polymorphism. Adapted from ⁷.

Supplemental table 6: bivariate comparisons of socio-economic and clinical characteristics between retained and excluded participants. Data from the baseline (2003-2006), first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland.

	Baseline			First follow-up			Second follow-up		
	Included	Excluded	p-value	Included	Excluded	p-value	Included	Excluded	p-value
N	617	92		844	212		798	353	
Age (years)	61.6 ± 8.5	58.1 ± 9.6	<0.001	64.5 ± 8.8	61.7 ± 10.4	<0.001	68.1 ± 9.2	67.5 ± 10.3	0.309
Women (%)	263 (42.6)	28 (30.4)	0.027	409 (48.5)	90 (42.5)	0.117	401 (50.3)	166 (47.0)	0.313
Swiss national (%)	430 (69.7)	59 (64.1)	0.282	600 (71.1)	116 (54.7)	<0.001	563 (70.6)	191 (54.1)	<0.001
Education (%)			0.301			0.027			0.714
High	66 (10.7)	5 (5.5)		123 (14.6)	47 (22.2)		120 (15.0)	54 (15.3)	
Middle	132 (21.4)	20 (22.0)		185 (21.9)	43 (20.3)		180 (22.6)	72 (20.4)	
Low	419 (67.9)	66 (72.5)		536 (63.5)	122 (57.6)		498 (62.4)	227 (64.3)	
Married/couple (%)	428 (69.4)	65 (71.4)	0.690	505 (59.8)	121 (57.1)	0.465	448 (56.1)	166 (53.9)	0.501
BMI (kg/m ²)	28.0 ± 4.5	28.1 ± 4.8	0.792	27.6 ± 4.8	28.0 ± 5.0	0.398			
BMI categories (%)			0.979			0.986			0.197
Normal	155 (25.1)	24 (26.1)		243 (28.8)	53 (28.2)		254 (31.8)	59 (26.6)	
Overweight	277 (44.9)	41 (44.6)		378 (44.8)	85 (45.2)		327 (41.0)	91 (41.0)	
Obese	185 (30.0)	27 (29.4)		223 (26.4)	50 (26.6)		217 (27.2)	72 (32.4)	
Smoking (%)			0.064			0.099			0.067
Never	210 (34.0)	39 (42.9)		295 (35.0)	73 (34.8)		290 (36.3)	103 (38.7)	
Former	255 (41.3)	26 (28.6)		385 (45.6)	83 (39.5)		365 (45.7)	102 (38.4)	
Current	152 (24.6)	26 (28.6)		164 (19.4)	54 (25.7)		143 (17.9)	61 (22.9)	
Alcohol drinker (%)	456 (73.9)	65 (70.7)	0.510	632 (74.9)	147 (69.3)	0.101	525 (72.3)	149 (62.3)	0.004
Treatment for									
Hypertension	303 (49.1)	49 (53.3)	0.457	436 (51.7)	101 (47.6)	0.296	425 (53.3)	190 (53.8)	0.859
Diabetes	95 (15.4)	24 (26.1)	0.010	148 (17.5)	40 (18.9)	0.650	157 (19.7)	69 (19.6)	0.960
Parental history (%)	164 (26.6)	24 (26.1)	0.920	218 (25.8)	52 (24.5)	0.698	211 (26.4)	64 (18.1)	0.002
CVD risk (%)			0.551			0.717			<0.001

Low	267 (43.3)	36 (39.1)	367 (43.5)	89 (42.0)	356 (44.6)	103 (29.2)
Intermediate	182 (29.5)	26 (28.3)	203 (24.1)	48 (22.6)	165 (20.7)	121 (34.3)
High	168 (27.2)	30 (32.6)	274 (32.5)	75 (35.4)	277 (34.7)	129 (36.5)

BMI, body mass index; CVD, cardio vascular disease. Results are expressed as number of participants (column %) for categorical variables and as average \pm standard deviation for continuous variables. Between-groups comparisons performed using chi-square for categorical variables and student's t-test for continuous variables.

Supplemental table 7: bivariate comparison of the statin generation and potency among participants treated for dyslipidemia, according to controlled and uncontrolled status as per European Society of Cardiology / European Atherosclerosis Society (ESC/EAS) criteria. Data from the first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland. Analysis restricted to participants taking statins.

	First follow-up			Second follow-up		
	Uncontrolled	Controlled	p-value	Uncontrolled	Controlled	p-value
N	373	288		428	370	
Generation			0.004			0.212
First	99 (26.5)	50 (17.4)		54 (16.5)	32 (12.2)	
Second	249 (66.8)	204 (70.8)		233 (71.0)	188 (71.8)	
Third	25 (6.7)	34 (11.8)		41 (12.5)	42 (16.0)	
Potency §			0.018			0.018
Low	99 (26.5)	50 (17.4)		54 (16.5)	32 (12.2)	
Intermediate	130 (34.9)	109 (37.9)		107 (32.6)	66 (25.2)	
High	144 (38.6)	129 (44.8)		167 (50.9)	164 (62.6)	
N	348	274		328	262	
Potency †			0.911			0.067
Low	166 (47.7)	126 (46.0)		117 (40.6)	72 (31.2)	
Intermediate	153 (44.0)	124 (45.3)		125 (43.4)	111 (48.0)	
High	29 (8.3)	24 (8.7)		46 (16.0)	48 (20.8)	

§, not considering posology; †, considering posology. Results are expressed as number of participants (column %). Between-groups comparisons performed using chi-square.

Supplemental table 8: multivariable analysis of the factors associated with dyslipidemia control as per European Society of Cardiology / European Atherosclerosis Society criteria. Data from the first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland. Analysis taking into account statin potency, but not considering posology.

	First follow-up		Second follow-up	
	OR (95% CI)	p-value	OR (95% CI)	p-value
Age (per 10 years increase)	0.31 (0.23 - 0.41)	<0.001	0.36 (0.27 - 0.47)	<0.001
Man vs. woman	0.59 (0.38 - 0.90)	0.014	1.36 (0.88 - 2.11)	0.171
Swiss vs. Non-Swiss	1.16 (0.76 - 1.78)	0.495	1.17 (0.75 - 1.81)	0.487
Education				
High	1 (ref.)		1 (ref.)	
Middle	0.76 (0.39 - 1.48)	0.415	1.04 (0.52 - 2.08)	0.908
Low	0.87 (0.48 - 1.57)	0.649	1.02 (0.56 - 1.87)	0.937
<i>p-value for trend</i>	0.649		0.937	
Married vs. not married	1.23 (0.83 - 1.84)	0.299	1.20 (0.80 - 1.79)	0.386
Body mass index categories				
Normal	1 (ref.)		1 (ref.)	
Overweight	0.86 (0.53 - 1.38)	0.523	0.57 (0.35 - 0.93)	0.025
Obese	0.93 (0.54 - 1.61)	0.807	0.53 (0.30 - 0.94)	0.031
<i>p-value for trend</i>	0.807		0.031	
Smoking categories				
Never	1 (ref.)		1 (ref.)	
Former	1.13 (0.72 - 1.77)	0.590	1.19 (0.77 - 1.85)	0.428
Current	0.86 (0.48 - 1.55)	0.614	0.90 (0.49 - 1.65)	0.729
<i>p-value for trend</i>	0.614		0.729	
Alcohol drinker (yes vs. no)	0.81 (0.51 - 1.27)	0.360	0.68 (0.43 - 1.08)	0.101
AntiHTA ttt (yes vs. no)	0.95 (0.62 - 1.47)	0.821	0.80 (0.51 - 1.25)	0.327
Parental history (yes vs. no)	1.25 (0.79 - 1.99)	0.338	0.75 (0.47 - 1.21)	0.240
CVD risk				
Other	1 (ref.)		1 (ref.)	
High	0.63 (0.39 - 1.02)	0.060	1.37 (0.78 - 2.40)	0.275
Very high	0.08 (0.05 - 0.14)	<0.001	0.35 (0.21 - 0.58)	<0.001
<i>p-value for trend</i>	<0.001		<0.001	
LDL genetic risk score quartiles				
First	1 (ref.)		1 (ref.)	
Second	0.89 (0.52 - 1.53)	0.680	1.63 (0.92 - 2.86)	0.092
Third	0.92 (0.54 - 1.57)	0.757	1.76 (1.02 - 3.03)	0.041
Fourth	1.11 (0.65 - 1.91)	0.702	1.58 (0.90 - 2.77)	0.112
<i>p-value for trend</i>	0.694		0.109	
Number of drugs (per 1 unit)	1.14 (1.05 - 1.25)	0.002	1.06 (0.99 - 1.15)	0.099
Statin potency, no posology				
Low	1 (ref.)		1 (ref.)	
Intermediate	1.98 (1.18 - 3.34)	0.010	1.50 (0.80 - 2.82)	0.209

High	2.08 (1.25 - 3.46)	0.005	2.25 (1.26 - 4.01)	0.006
<i>p-value for trend</i>	0.005		0.006	
Fibrates	NC		2.16 (0.16 - 29.1)	0.560
Other hypolipidemic drugs	0.95 (0.41 - 2.22)	0.905	1.07 (0.48 - 2.37)	0.867

OR, odds ratio; CI, confidence interval; antiHTA ttt, antihypertensive drug treatment; NC, not computable. Results are expressed as odds ratio and (95% confidence interval). Statistical analysis using logistic regression.

Supplemental table 9: multivariable analysis of the factors associated with dyslipidemia control as per European Society of Cardiology / European Atherosclerosis Society criteria. Data from the first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland. Analysis taking into account statin potency and posology.

	First follow-up		Second follow-up	
	OR (95% CI)	p-value	OR (95% CI)	p-value
Age (per 10 years increase)	0.31 (0.24 - 0.42)	<0.001	0.37 (0.27 - 0.50)	<0.001
Man vs. woman	0.56 (0.36 - 0.86)	0.008	1.45 (0.91 - 2.31)	0.120
Swiss vs. Non-Swiss	1.14 (0.74 - 1.78)	0.548	1.11 (0.69 - 1.76)	0.672
Education				
High	1 (ref.)		1 (ref.)	
Middle	0.74 (0.37 - 1.48)	0.396	0.95 (0.46 - 1.95)	0.882
Low	0.86 (0.47 - 1.56)	0.619	0.94 (0.50 - 1.76)	0.835
<i>p-value for trend</i>	0.619		0.835	
Married vs. not married	1.25 (0.83 - 1.88)	0.29	1.17 (0.76 - 1.80)	0.481
Body mass index categories				
Normal	1 (ref.)		1 (ref.)	
Overweight	0.87 (0.54 - 1.42)	0.586	0.62 (0.37 - 1.05)	0.073
Obese	0.89 (0.50 - 1.56)	0.677	0.56 (0.31 - 1.02)	0.058
<i>p-value for trend</i>	0.677		0.058	
Smoking categories				
Never	1 (ref.)		1 (ref.)	
Former	1.12 (0.71 - 1.77)	0.629	1.12 (0.7 - 1.78)	0.636
Current	0.91 (0.50 - 1.65)	0.747	0.82 (0.42 - 1.58)	0.545
<i>p-value for trend</i>	0.747		0.545	
Alcohol drinker (yes vs. no)	0.73 (0.45 - 1.16)	0.183	0.58 (0.35 - 0.95)	0.031
AntiHTA ttt (yes vs. no)	0.99 (0.64 - 1.55)	0.977	0.89 (0.55 - 1.42)	0.618
Parental history (yes vs. no)	1.24 (0.77 - 1.98)	0.382	0.85 (0.51 - 1.41)	0.529
CVD risk				
Other	1 (ref.)		1 (ref.)	
High	0.64 (0.39 - 1.04)	0.074	1.29 (0.71 - 2.33)	0.400
Very high	0.08 (0.05 - 0.15)	<0.001	0.35 (0.20 - 0.61)	<0.001
<i>p-value for trend</i>	<0.001		<0.001	
LDL genetic risk score quartiles				
First	1 (ref.)		1 (ref.)	
Second	1.01 (0.58 - 1.75)	0.979	1.63 (0.89 - 2.96)	0.111
Third	1.15 (0.66 - 1.99)	0.623	1.89 (1.06 - 3.36)	0.031
Fourth	1.24 (0.71 - 2.16)	0.452	1.68 (0.93 - 3.02)	0.085
<i>p-value for trend</i>	0.392		0.073	
Number of drugs (per 1 unit)	1.13 (1.03 - 1.23)	0.008	1.04 (0.96 - 1.13)	0.366
Statin potency, with posology				
Low	1 (ref.)		1 (ref.)	
Intermediate	1.10 (0.73 - 1.65)	0.656	1.37 (0.86 - 2.17)	0.183

High	1.35 (0.63 - 2.88)	0.437	1.69 (0.89 - 3.21)	0.110
<i>p-value for trend</i>	0.437		0.110	
Fibrates	NC		2.20 (0.16 - 31.2)	0.559
Other hypolipidemic drugs	0.91 (0.38 - 2.21)	0.839	1.06 (0.45 - 2.47)	0.898

OR, odds ratio; CI, confidence interval; antiHTA ttt, antihypertensive drug treatment; NC, not computable. Results are expressed as odds ratio and (95% confidence interval). Statistical analysis using logistic regression.

Supplemental table 10: bivariate comparisons of socio-economic and clinical characteristics among participants treated for dyslipidemia, according to controlled and uncontrolled status as per the Swiss Group for Lipids and Atherosclerosis criteria. Data from the baseline (2003-2006), first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland.

	Baseline			First follow-up			Second follow-up		
	Uncontrolled	Controlled	p-value	Uncontrolled	Controlled	p-value	Uncontrolled	Controlled	p-value
N	184	433		266	578		139	659	
Age (years)	61.5 ± 8.5	61.7 ± 8.5	0.728	64.5 ± 8.6	64.5 ± 8.9	0.947	67.1 ± 9.0	68.4 ± 9.3	0.139
Women (%)	81 (44.0)	182 (42.0)	0.648	119 (44.7)	290 (50.2)	0.142	72 (51.8)	329 (49.9)	0.688
Swiss national (%)	128 (70.0)	302 (69.6)	0.929	183 (69.1)	417 (72.0)	0.378	105 (74.5)	458 (69.7)	0.261
Education (%)			0.474			0.707			0.354
High	47 (10.9)	66 (10.7)		16 (6.0)	38 (6.6)		10 (7.2)	50 (7.6)	
Middle	98 (22.6)	132 (21.4)		39 (14.7)	89 (15.4)		17 (12.2)	53 (8.0)	
Low	288 (66.5)	419 (67.9)		50 (18.8)	125 (21.6)		23 (16.6)	95 (14.4)	
Married/couple (%)	127 (69.0)	301 (69.5)	0.903	353 (61.1)	505 (59.8)	0.279	74 (53.2)	374 (56.8)	0.448
BMI (kg/m ²)	28.4 ± 4.4	27.8 ± 4.5	0.150	28.2 ± 4.9	27.4 ± 4.7	0.038	28.6 ± 4.8	27.4 ± 4.9	0.008
BMI categories (%)			0.354			0.125			0.033
Normal	40 (21.7)	115 (26.6)		69 (25.9)	174 (30.1)		33 (23.7)	221 (33.5)	
Overweight	83 (45.1)	194 (44.8)		115 (43.2)	263 (45.5)		58 (41.7)	269 (40.8)	
Obese	61 (33.2)	124 (28.6)		82 (30.8)	141 (24.4)		48 (34.5)	169 (25.6)	
Smoking (%)			0.285			0.019			0.110
Never	152 (35.1)	210 (34.0)		218 (37.7)	295 (35.0)		235 (35.7)	290 (36.3)	
Former	182 (42.0)	255 (41.3)		259 (44.8)	385 (45.6)		312 (47.3)	365 (45.7)	
Current	99 (22.9)	152 (24.6)		101 (17.5)	164 (19.4)		112 (17.0)	143 (17.9)	
Alcohol drinker (%)	148 (80.4)	308 (71.1)	0.016	207 (77.8)	425 (73.5)	0.182	93 (74.4)	432 (71.9)	0.567
Treatment for									
Hypertension	98 (53.3)	205 (47.3)	0.179	41 (44.1)	395 (52.6)	0.121	79 (56.8)	346 (52.5)	0.352
Diabetes	36 (19.6)	59 (13.6)	0.061	13 (14.0)	135 (18.0)	0.339	34 (24.5)	123 (18.7)	0.118
Parental history (%)	134 (72.8)	319 (73.7)	0.828	69 (25.9)	149 (25.8)	0.960	94 (67.6)	493 (74.8)	0.081

CVD risk (%)			<0.001			<0.001			<0.001
Low	32 (17.4)	234 (54.0)		47 (17.7)	319 (55.2)		28 (20.1)	329 (49.9)	
Intermediate	70 (38.0)	114 (26.3)		74 (27.8)	130 (22.5)		36 (25.9)	131 (19.9)	
High	82 (44.6)	85 (19.6)		145 (54.5)	129 (22.3)		75 (54.0)	199 (30.2)	
Statins (%)	161 (87.5)	417 (96.3)	<0.001	200 (75.2)	461 (79.8)	0.134	88 (63.3)	504 (76.5)	0.001
Fibrates (%)	28 (15.2)	17 (3.9)	<0.001	17 (6.4)	16 (2.8)	0.012	7 (5.0)	25 (3.8)	0.497
Other (%)	7 (3.8)	15 (3.5)	0.835	55 (20.7)	96 (16.6)	0.152	46 (33.1)	123 (18.7)	<0.001
Number of drugs									
Including OTC	-	-		4 [3 - 6]	4 [2 - 6]	0.229 §	5 [3 - 7]	5 [3 - 7]	0.842 §
Excluding OTC	-	-		4 [2 - 6]	3 [2 - 5]	0.246 §	4 [2 - 6]	4 [2 - 6]	0.642 §
Genetic risk scores									
Total cholesterol	-2.7 ± 9.4	-3.7 ± 9.6	0.254	-3.7 ± 9.5	-3.5 ± 8.9	0.763	-3.3 ± 9.4	-3.8 ± 8.9	0.562
LDL-cholesterol	-2.4 ± 7.5	-2.9 ± 7.5	0.461	-2.5 ± 7.6	-2.4 ± 7.2	0.847	-2.5 ± 7.3	-2.9 ± 7.0	0.467
HDL-cholesterol	-6.5 ± 3.4	-6.7 ± 3.6	0.649	0.2 ± 7.4	-6.6 ± 3.5	0.312	-6.9 ± 3.8	-6.7 ± 3.6	0.539

BMI, body mass index; CVD, cardio vascular disease; OTC, over the counter. Results are expressed as number of participants (column %) for categorical variables and as average ± standard deviation or as median [interquartile range] for continuous variables. Between-groups comparisons performed using chi-square for categorical variables and student's t-test or Kruskal-Wallis nonparametric test (§) for continuous variables.

Supplemental table 11: multivariable analysis of the factors associated with dyslipidemia control as per the Swiss Group for Lipids and Atherosclerosis criteria. Data from the baseline (2003-2006), first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland.

	Baseline		First follow-up		Second follow-up	
	OR (95% CI)	p-value	OR (95% CI)	p-value	OR (95% CI)	p-value
Age (per 10 years increase)	1.17 (0.90 - 1.51)	0.231	1.05 (0.85 - 1.30)	0.627	1.18 (0.90 - 1.54)	0.221
Man vs. woman	1.70 (1.08 - 2.69)	0.022	1.15 (0.80 - 1.66)	0.458	1.59 (1.00 - 2.53)	0.050
Swiss vs. Non-Swiss	1.26 (0.81 - 1.96)	0.298	1.30 (0.90 - 1.86)	0.159	0.74 (0.45 - 1.21)	0.232
Education						
High	1 (ref.)		1 (ref.)		1 (ref.)	
Middle	1.23 (0.58 - 2.62)	0.586	1.12 (0.64 - 1.96)	0.690	0.44 (0.21 - 0.92)	0.030
Low	1.07 (0.55 - 2.05)	0.848	0.81 (0.49 - 1.32)	0.396	0.61 (0.30 - 1.22)	0.163
<i>p-value for trend</i>	0.848		0.396		0.163	
Married vs. not married	1.11 (0.71 - 1.72)	0.652	1.13 (0.81 - 1.59)	0.466	0.81 (0.52 - 1.26)	0.349
Body mass index categories						
Normal	1 (ref.)		1 (ref.)		1 (ref.)	
Overweight	0.75 (0.45 - 1.25)	0.268	0.92 (0.60 - 1.40)	0.691	0.57 (0.33 - 0.98)	0.043
Obese	1.05 (0.60 - 1.86)	0.854	0.96 (0.59 - 1.54)	0.854	0.58 (0.31 - 1.09)	0.091
<i>p-value for trend</i>	0.854		0.691		0.091	
Smoking categories						
Never	1 (ref.)		1 (ref.)		1 (ref.)	
Former	1.28 (0.78 - 2.08)	0.328	0.91 (0.62 - 1.35)	0.646	1.96 (1.19 - 3.23)	0.008
Current	1.29 (0.73 - 2.26)	0.380	0.91 (0.56 - 1.47)	0.692	1.76 (0.93 - 3.32)	0.081
<i>p-value for trend</i>	0.380		0.692		0.081	
Alcohol drinker (yes vs. no)	0.46 (0.28 - 0.77)	0.003	0.75 (0.50 - 1.12)	0.155	0.74 (0.45 - 1.23)	0.246
Antihypertensive treatment (yes vs. no)	1.50 (0.97 - 2.29)	0.065	1.48 (1.03 - 2.12)	0.036	1.04 (0.64 - 1.69)	0.862
Parental history (yes vs. no)	0.99 (0.61 - 1.59)	0.964	0.96 (0.65 - 1.42)	0.847	0.65 (0.40 - 1.06)	0.086
CVD risk (GSLA criterion)						
Low	1 (ref.)		1 (ref.)		1 (ref.)	

Intermediate	0.16 (0.09 - 0.28)	<0.001	0.19 (0.12 - 0.31)	<0.001	0.22 (0.11 - 0.42)	<0.001
High	0.08 (0.05 - 0.14)	<0.001	0.08 (0.05 - 0.13)	<0.001	0.100 (0.06 - 0.20)	<0.001
<i>p-value for trend</i>	<0.001		<0.001		<0.001	
LDL genetic risk score quartiles						
First	1 (ref.)		1 (ref.)		1 (ref.)	
Second	0.89 (0.50 - 1.58)	0.681	1.07 (0.67 - 1.69)	0.784	1.99 (1.06 - 3.74)	0.033
Third	0.54 (0.31 - 0.96)	0.034	1.11 (0.70 - 1.77)	0.645	0.93 (0.51 - 1.70)	0.815
Fourth	0.74 (0.42 - 1.30)	0.293	1.12 (0.70 - 1.77)	0.641	0.77 (0.43 - 1.38)	0.388
<i>p-value for trend</i>	0.125		0.617		0.108	
Hypolipidemic drug treatment						
Statins	0.35 (0.03 - 4.26)	0.411	1.19 (0.66 - 2.15)	0.570	2.21 (1.14 - 4.28)	0.019
Fibrates	0.06 (0.01 - 0.63)	0.019	0.29 (0.11 - 0.76)	0.012	0.61 (0.2 - 1.86)	0.389
Other	0.80 (0.28 - 2.27)	0.674	0.45 (0.25 - 0.81)	0.008	0.47 (0.25 - 0.89)	0.020

OR, odds ratio; CI, confidence interval. Results are expressed as odds ratio and (95% confidence interval). Statistical analysis using logistic regression.

Supplemental table 12: bivariate comparisons of the statin generation and potency among participants treated for dyslipidemia, according to controlled and uncontrolled status as per the Swiss Group for Lipids and Atherosclerosis criteria. Data from the first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland. Analysis restricted to participants taking statins.

	First follow-up			Second follow-up		
	Uncontrolled	Controlled	p-value	Uncontrolled	Controlled	p-value
N	200	461		88	502	
Generation			0.003			0.026
First	62 (31.0)	87 (18.9)		21 (23.9)	65 (13.0)	
Second	122 (61.0)	331 (71.8)		55 (62.5)	366 (72.9)	
Third	16 (8.0)	43 (9.3)		12 (13.6)	71 (14.1)	
Potency §			0.002			0.005
Low	62 (31.0)	87 (18.9)		21 (23.9)	65 (13.0)	
Intermediate	70 (35.0)	169 (36.7)		30 (34.1)	143 (28.5)	
High	68 (34.0)	205 (44.5)		37 (42.1)	294 (58.6)	
N	186	436		77	442	
Potency §			0.386			0.199
Low	94 (50.5)	198 (45.4)		34 (44.2)	155 (35.1)	
Intermediate	75 (40.3)	202 (46.3)		28 (36.4)	208 (47.1)	
High	17 (9.1)	36 (8.3)		15 (19.5)	79 (17.9)	

§, not considering posology; †, considering posology. Results are expressed as number of participants (column %). Between-groups comparisons performed using chi-square.

Supplemental table 13: multivariable analysis of the factors associated with dyslipidemia control as per the Swiss Group for Lipids and Atherosclerosis criteria. Data from the first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland. Analysis taking into account statin generation.

	First follow-up		Second follow-up	
	OR (95% CI)	p-value	OR (95% CI)	p-value
Age (per 10 years increase)	1.20 (0.93 - 1.56)	0.159	1.04 (0.73 - 1.48)	0.823
Man vs. woman	1.59 (1.03 - 2.45)	0.037	2.23 (1.23 - 4.02)	0.008
Swiss vs. Non-Swiss	1.25 (0.81 - 1.92)	0.315	0.80 (0.43 - 1.47)	0.468
Education				
High	1 (ref.)		1 (ref.)	
Middle	1.09 (0.54 - 2.19)	0.805	0.85 (0.32 - 2.22)	0.733
Low	0.83 (0.46 - 1.52)	0.551	1.13 (0.46 - 2.74)	0.791
<i>p-value for trend</i>	0.551		0.791	
Married vs. not married	1.24 (0.84 - 1.84)	0.287	0.97 (0.55 - 1.71)	0.917
Body mass index categories				
Normal	1 (ref.)		1 (ref.)	
Overweight	0.87 (0.53 - 1.45)	0.603	0.61 (0.29 - 1.28)	0.191
Obese	0.86 (0.49 - 1.50)	0.591	0.47 (0.21 - 1.06)	0.068
<i>p-value for trend</i>	0.591		0.068	
Smoking categories				
Never	1 (ref.)		1 (ref.)	
Former	0.91 (0.58 - 1.44)	0.688	1.79 (0.96 - 3.36)	0.068
Current	0.96 (0.54 - 1.72)	0.891	1.22 (0.56 - 2.66)	0.625
<i>p-value for trend</i>				
Alcohol drinker (yes vs. no)	0.67 (0.41 - 1.08)	0.099	0.95 (0.51 - 1.78)	0.882
AntiHTA ttt (yes vs. no)	1.19 (0.77 - 1.84)	0.438	1.11 (0.60 - 2.05)	0.733
Parental history (yes vs. no)	0.98 (0.61 - 1.58)	0.945	0.54 (0.28 - 1.03)	0.060
CVD risk (GSLA criterion)				
Low	1 (ref.)		1 (ref.)	
Intermediate	0.18 (0.10 - 0.32)	<0.001	0.19 (0.07 - 0.49)	0.001
High	0.05 (0.03 - 0.10)	<0.001	0.06 (0.02 - 0.14)	<0.001
<i>p-value for trend</i>	<0.001		<0.001	
LDL genetic risk score quartiles				
First	1 (ref.)		1 (ref.)	
Second	1.04 (0.61 - 1.76)	0.888	2.95 (1.29 - 6.72)	0.010
Third	1.21 (0.70 - 2.08)	0.492	1.06 (0.51 - 2.19)	0.881
Fourth	1.29 (0.75 - 2.20)	0.359	1.18 (0.57 - 2.47)	0.653
<i>p-value for trend</i>	0.298		0.666	
Number of drugs (per 1 unit)	1.14 (1.05 - 1.25)	0.003	1.16 (1.04 - 1.29)	0.008
Statins generation				
First	1 (ref.)		1 (ref.)	
Second	2.54 (1.61 - 4.01)	<0.001	4.17 (2.07 - 8.42)	<0.001
Third	2.59 (1.19 - 5.61)	0.016	3.44 (1.31 - 8.99)	0.012

<i>p-value for trend</i>	0.016		0.012	
Fibrates	NC		NC	
Other	0.62 (0.27 - 1.43)	0.263	1.09 (0.36 - 3.3)	0.880

OR, odds ratio; CI, confidence interval; antiHTA ttt, antihypertensive drug treatment; NC, not computable. Results are expressed as odds ratio and (95% confidence interval). Statistical analysis by logistic regression.

Supplemental table 14: multivariable analysis of the factors associated with dyslipidemia control as per the Swiss Group for Lipids and Atherosclerosis criteria. Data from the first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland. Analysis taking into account statin potency, but not considering posology.

	First follow-up		Second follow-up	
	OR (95% CI)	p-value	OR (95% CI)	p-value
Age (per 10 years increase)	1.22 (0.94 - 1.59)	0.127	1.06 (0.74 - 1.51)	0.748
Man vs. woman	1.57 (1.02 - 2.43)	0.040	2.13 (1.17 - 3.87)	0.013
Swiss vs. Non-Swiss	1.24 (0.81 - 1.92)	0.322	0.82 (0.45 - 1.52)	0.533
Education				
High	1 (ref.)		1 (ref.)	
Middle	1.07 (0.53 - 2.14)	0.856	0.77 (0.29 - 2.04)	0.601
Low	0.83 (0.45 - 1.50)	0.531	1.05 (0.43 - 2.58)	0.914
<i>p-value for trend</i>	0.531		0.914	
Married vs. not married	1.26 (0.85 - 1.86)	0.259	1.00 (0.57 - 1.76)	0.995
Body mass index categories				
Normal	1 (ref.)		1 (ref.)	
Overweight	0.89 (0.54 - 1.47)	0.637	0.64 (0.31 - 1.35)	0.245
Obese	0.85 (0.49 - 1.49)	0.570	0.49 (0.22 - 1.10)	0.085
<i>p-value for trend</i>	0.570		0.085	
Smoking categories				
Never	1 (ref.)		1 (ref.)	
Former	0.90 (0.57 - 1.42)	0.653	1.83 (0.97 - 3.46)	0.061
Current	0.96 (0.54 - 1.72)	0.895	1.18 (0.53 - 2.58)	0.688
<i>p-value for trend</i>	0.895		0.688	
Alcohol drinker (yes vs. no)	0.67 (0.42 - 1.09)	0.106	0.90 (0.48 - 1.70)	0.750
AntiHTA ttt (yes vs. no)	1.20 (0.77 - 1.85)	0.417	1.09 (0.59 - 2.02)	0.781
Parental history (yes vs. no)	0.98 (0.61 - 1.58)	0.942	0.52 (0.27 - 0.99)	0.049
CVD risk				
Low	1 (ref.)		1 (ref.)	
Intermediate	0.17 (0.10 - 0.31)	<0.001	0.20 (0.08 - 0.51)	0.001
High	0.05 (0.03 - 0.10)	<0.001	0.06 (0.02 - 0.15)	<0.001
<i>p-value for trend</i>	<0.001		<0.001	
LDL genetic risk score quartiles				
First	1 (ref.)		1 (ref.)	
Second	1.05 (0.62 - 1.79)	0.852	2.74 (1.20 - 6.24)	0.016
Third	1.19 (0.70 - 2.05)	0.519	1.01 (0.49 - 2.10)	0.982
Fourth	1.27 (0.74 - 2.18)	0.380	1.12 (0.53 - 2.35)	0.763
<i>p-value for trend</i>	0.329		0.586	
Number of drugs (per 1 unit)	1.14 (1.04 - 1.25)	0.003	1.15 (1.03 - 1.28)	0.012
Statins potency, no posology				
Low	1 (ref.)		1 (ref.)	
Intermediate	2.14 (1.30 - 3.54)	0.003	2.90 (1.35 - 6.24)	0.007
High	3.01 (1.82 - 4.98)	<0.001	5.13 (2.43 - 10.8)	<0.001

<i>p-value for trend</i>	<0.001		<0.001	
Fibrates	NC		NC	
Other	0.58 (0.25 - 1.34)	0.202	0.91 (0.30 - 2.76)	0.871

OR, odds ratio; CI, confidence interval; antiHTA ttt, antihypertensive drug treatment; NC, not computable. Results are expressed as odds ratio and (95% confidence interval). Statistical analysis

Supplemental table 15: multivariable analysis of the factors associated with dyslipidemia control as per the Swiss Group for Lipids and Atherosclerosis criteria. Data from the first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland. Analysis taking into account statin potency and posology

	First follow-up		Second follow-up	
	OR (95% CI)	p-value	OR (95% CI)	p-value
Age (per 10 years increase)	1.25 (0.96 - 1.63)	0.102	1.10 (0.76 - 1.60)	0.619
Man vs. woman	1.61 (1.04 - 2.51)	0.034	1.82 (0.97 - 3.42)	0.062
Swiss vs. Non-Swiss	1.25 (0.80 - 1.94)	0.321	0.81 (0.42 - 1.54)	0.515
Education				
High	1 (ref.)		1 (ref.)	
Middle	1.08 (0.53 - 2.19)	0.839	0.47 (0.16 - 1.40)	0.175
Low	0.80 (0.43 - 1.46)	0.465	0.65 (0.23 - 1.79)	0.402
p-value for trend	0.465		0.402	
Married vs. not married	1.33 (0.89 - 2.00)	0.163	1.06 (0.59 - 1.92)	0.842
Body mass index categories				
Normal	1 (ref.)		1 (ref.)	
Overweight	0.86 (0.51 - 1.43)	0.554	0.66 (0.31 - 1.40)	0.283
Obese	0.87 (0.49 - 1.55)	0.641	0.65 (0.28 - 1.48)	0.302
p-value for trend	0.641		0.302	
Smoking categories				
Never	1 (ref.)		1 (ref.)	
Former	0.95 (0.60 - 1.52)	0.836	1.72 (0.89 - 3.34)	0.109
Current	1.05 (0.58 - 1.89)	0.868	1.20 (0.52 - 2.78)	0.672
p-value for trend	0.868		0.672	
Alcohol drinker (yes vs. no)	0.67 (0.41 - 1.10)	0.113	0.93 (0.48 - 1.8)	0.831
AntiHTA ttt (yes vs. no)	1.35 (0.86 - 2.11)	0.187	1.20 (0.63 - 2.28)	0.575
Parental history (yes vs. no)	0.94 (0.58 - 1.52)	0.805	0.67 (0.34 - 1.33)	0.251
CVD risk				
Low	1 (ref.)		1 (ref.)	
Intermediate	0.19 (0.10 - 0.34)	<0.001	0.23 (0.09 - 0.59)	0.002
High	0.06 (0.03 - 0.11)	<0.001	0.07 (0.03 - 0.18)	<0.001
p-value for trend	<0.001		<0.001	
LDL genetic score quartiles				
First	1 (ref.)		1 (ref.)	
Second	1.07 (0.62 - 1.85)	0.800	1.89 (0.81 - 4.39)	0.141
Third	1.34 (0.77 - 2.34)	0.299	0.83 (0.38 - 1.79)	0.634
Fourth	1.25 (0.72 - 2.17)	0.422	1.00 (0.46 - 2.17)	0.997
p-value for trend	0.313		0.514	
Number of drugs (per 1 unit)	1.11 (1.01 - 1.21)	0.028	1.16 (1.02 - 1.30)	0.019
Statins potency, posology				
Low	1 (ref.)		1 (ref.)	
Intermediate	1.36 (0.91 - 2.06)	0.137	1.66 (0.88 - 3.11)	0.115
High	1.67 (0.81 - 3.45)	0.168	2.07 (0.89 - 4.82)	0.092

p-value for trend	0.168		0.092	
Fibrates	NC		NC	
Other	0.75 (0.31 - 1.84)	0.529	1.64 (0.43 - 6.28)	0.473

OR, odds ratio; CI, confidence interval; antiHTA ttt, antihypertensive drug treatment; NC, not computable. Results are expressed as odds ratio and (95% confidence interval). Statistical analysis by logistic regression.

Supplemental table 16: p-values for the different SNPs associated with statin effect regarding dyslipidemia control, according to the European Society of Cardiology / European Atherosclerosis Society criteria, and the Swiss Group for Lipids and Atherosclerosis strict and near control criteria. Data from the baseline (2003-2006), first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland.

Gene	SNP	ESC/EAS			GSLA strict control			GSLA near control		
		Baseline	1 st FU	2 nd FU	Baseline	First FU	2 nd FU	Baseline	First FU	2 nd FU
<i>ABCB1</i>	rs1128503	0.728	0.896	0.040	0.969	0.794	0.167	0.481	0.539	0.513
<i>ABCB1</i>	rs1922242	0.910	0.369	0.147	0.648	0.360	0.026	0.902	0.598	0.474
<i>ABCB1</i>	rs2032582	0.835	0.492	0.156	0.887	0.673	0.150	0.387	0.553	0.454
<i>ABCB1</i>	rs3789244	0.728	0.901	0.040	0.985	0.779	0.167	0.481	0.539	0.513
<i>ABCG2/BCRP</i>	rs2199936	0.485	0.861	0.444	0.649	0.381	0.141	0.452	0.295	0.507
<i>ABCG2/BCRP</i>	rs2231137	0.094	0.966	0.292	1.000	0.817	0.906	0.671	0.897	0.584
<i>ABCG2/BCRP</i>	rs2231142	0.411	0.894	0.444	0.592	0.310	0.141	0.452	0.329	0.507
<i>ABCG8</i>	rs11887534	0.541	0.671	0.002	0.945	0.210	0.196	1.000	0.031	0.603
<i>APOA1</i>	rs5069	0.232	0.858	1.000	0.072	0.245	1.000	0.501	0.219	0.680
<i>APOA1</i>	rs670	0.793	0.314	0.125	0.735	0.378	0.386	0.456	0.372	0.833
<i>APOA5</i>	rs662799	0.238	0.052	0.092	0.712	0.792	0.125	0.396	0.437	1.000
<i>APOC1</i>	rs4420638	0.847	0.089	0.567	0.514	0.697	0.085	0.092	0.208	0.503
<i>APOE</i>	rs429358	1.000	0.077	0.502	0.308	0.576	0.784	0.105	1.000	1.000
<i>APOE</i>	rs445925	0.718	0.623	0.788	1.000	0.141	0.856	0.414	0.606	1.000
<i>APOE</i>	rs449647	0.461	0.888	0.207	0.919	0.870	0.617	0.708	0.642	1.000
<i>APOE</i>	rs71352238	0.856	0.269	0.489	0.358	0.952	0.642	0.056	0.761	0.791
<i>APOE</i>	rs7412	0.246	0.154	0.145	0.792	0.694	0.803	0.702	0.347	1.000
<i>CETP</i>	rs5882	0.683	0.749	0.932	0.446	0.773	0.211	0.371	0.105	0.319
<i>CETP</i>	rs708272	0.088	0.841	0.459	0.593	0.580	0.247	0.785	0.182	0.453
<i>CLMN</i>	rs8014194	0.304	0.477	0.382	0.339	0.543	0.636	0.138	0.154	0.527
<i>CYP2C9</i>	rs1057910	0.798	0.064	0.060	0.392	0.060	0.163	0.393	0.064	0.302
<i>CYP2D6</i>	rs3892097	0.781	0.540	0.800	0.328	0.641	0.601	0.619	0.798	0.242
<i>CYP3A4</i>	rs2740574	0.394	0.305	0.430	0.805	0.729	0.220	0.165	0.473	0.703

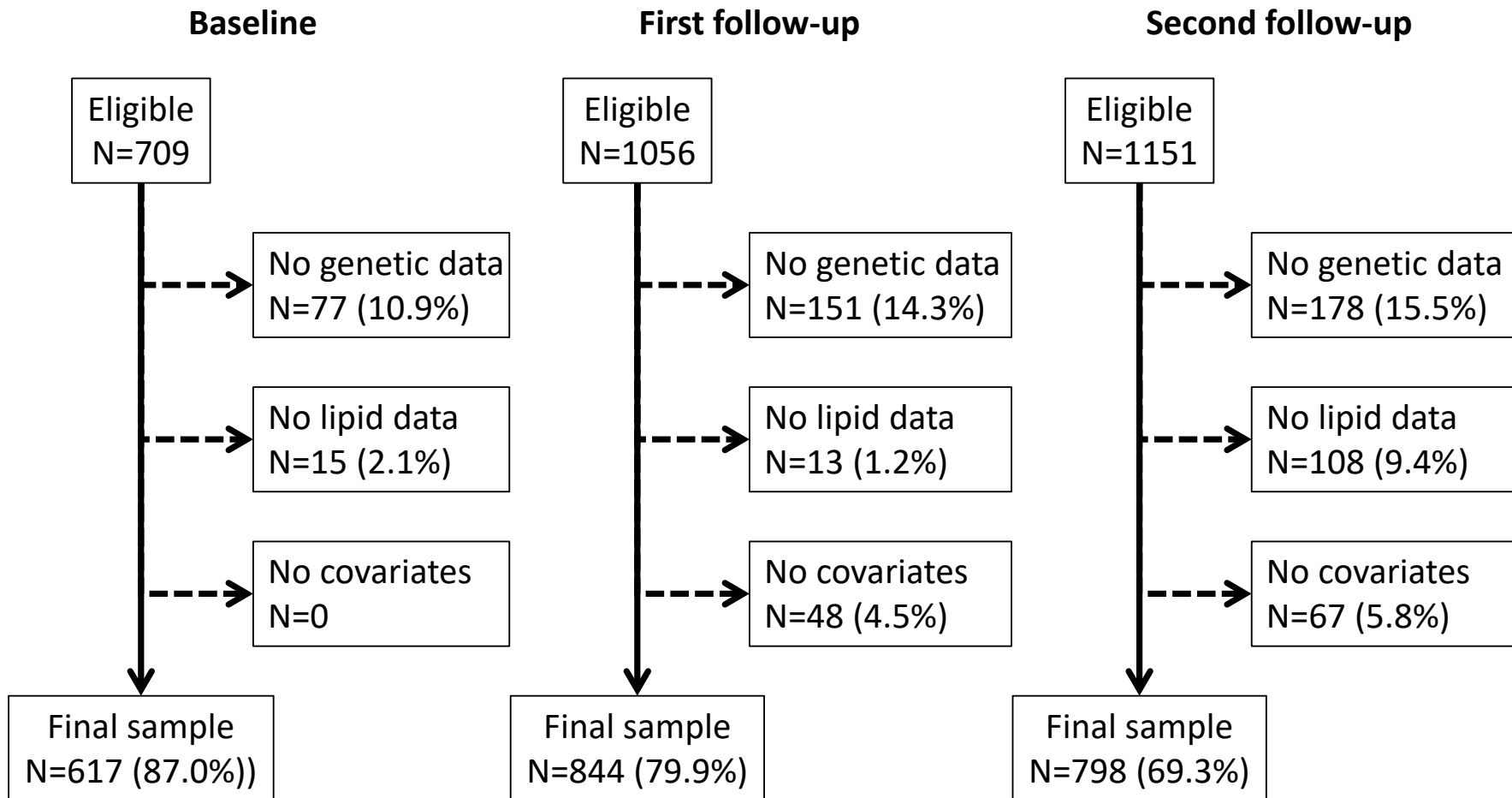
<i>CYP3A4</i>	rs55951658	NC	NC	NC	NC	NC	NC	NC	NC	NC
<i>CYP7A1</i>	rs3808607	0.043	0.573	0.267	0.017	0.153	0.601	0.647	0.149	0.766
<i>ESR1</i>	rs2234693	0.026	0.938	1.000	0.613	0.868	0.119	0.816	0.797	0.757
<i>ESR1</i>	rs9340799	0.139	0.899	0.569	0.420	0.778	0.156	0.036	0.390	0.619
<i>HMGCR</i>	rs12916	0.598	0.406	0.626	0.584	0.912	0.651	0.802	0.139	0.413
<i>HMGCR</i>	rs17238540	0.833	0.552	0.713	0.153	0.901	0.537	1.000	0.450	0.430
<i>HMGCR</i>	rs17244841	0.666	0.498	0.705	0.151	1.000	0.536	1.000	0.442	0.424
<i>HMGCR</i>	rs17671591	0.171	0.947	0.158	0.337	0.388	0.634	0.590	0.124	0.737
<i>HMGCR</i>	rs5908	0.102	0.507	0.632	1.000	1.000	0.466	0.271	1.000	1.000
<i>KIF6</i>	rs20455	0.307	0.029	0.209	0.602	0.323	0.609	0.094	0.124	0.058
<i>LDLR</i>	rs5925	0.408	0.311	0.015	0.206	0.281	0.348	0.553	0.889	0.935
<i>LPA</i>	rs10455872	0.266	0.659	0.332	0.021	0.340	1.000	1.000	0.461	0.452
<i>LPL</i>	rs328	0.372	0.397	0.674	0.186	0.654	0.835	0.087	1.000	0.878
<i>PCSK9</i>	rs17111584	0.462	0.359	0.054	1.000	0.197	1.000	0.380	0.027	1.000
<i>PON1</i>	rs662	0.069	0.637	0.911	0.130	0.407	0.684	0.730	1.000	0.749
<i>SCAP</i>	rs12487736	0.031	0.145	0.312	0.226	0.525	0.840	0.032	0.711	0.413
<i>SCCO1B1</i>	rs2306283	0.076	0.429	0.384	0.533	0.404	0.902	0.719	0.557	0.126
<i>SLCO1B1</i>	rs11045819	0.914	0.768	0.068	0.020	0.841	0.250	0.466	0.044	0.728
<i>SLCO1B1</i>	rs4149015	0.572	0.819	0.808	0.895	0.084	0.303	0.758	0.438	0.039
<i>SLCO1B1</i>	rs4149056	0.112	0.888	0.011	0.675	0.574	0.027	0.856	0.045	0.356
<i>SOD2</i>	rs4880	0.255	0.611	0.460	0.182	0.740	0.837	0.228	0.925	0.214
<i>TLR4</i>	rs4986790	0.654	0.873	0.013	0.727	0.124	0.662	1.000	0.166	1.000
<i>ABCB1</i>	rs1045642	0.789	0.500	0.619	0.877	0.160	0.350	0.461	0.578	0.269

NC, not computable as all participants were homozygous. FU, follow-up. Statistical analysis performed using Fisher's exact test. Values <0.05 are indicated in bold.

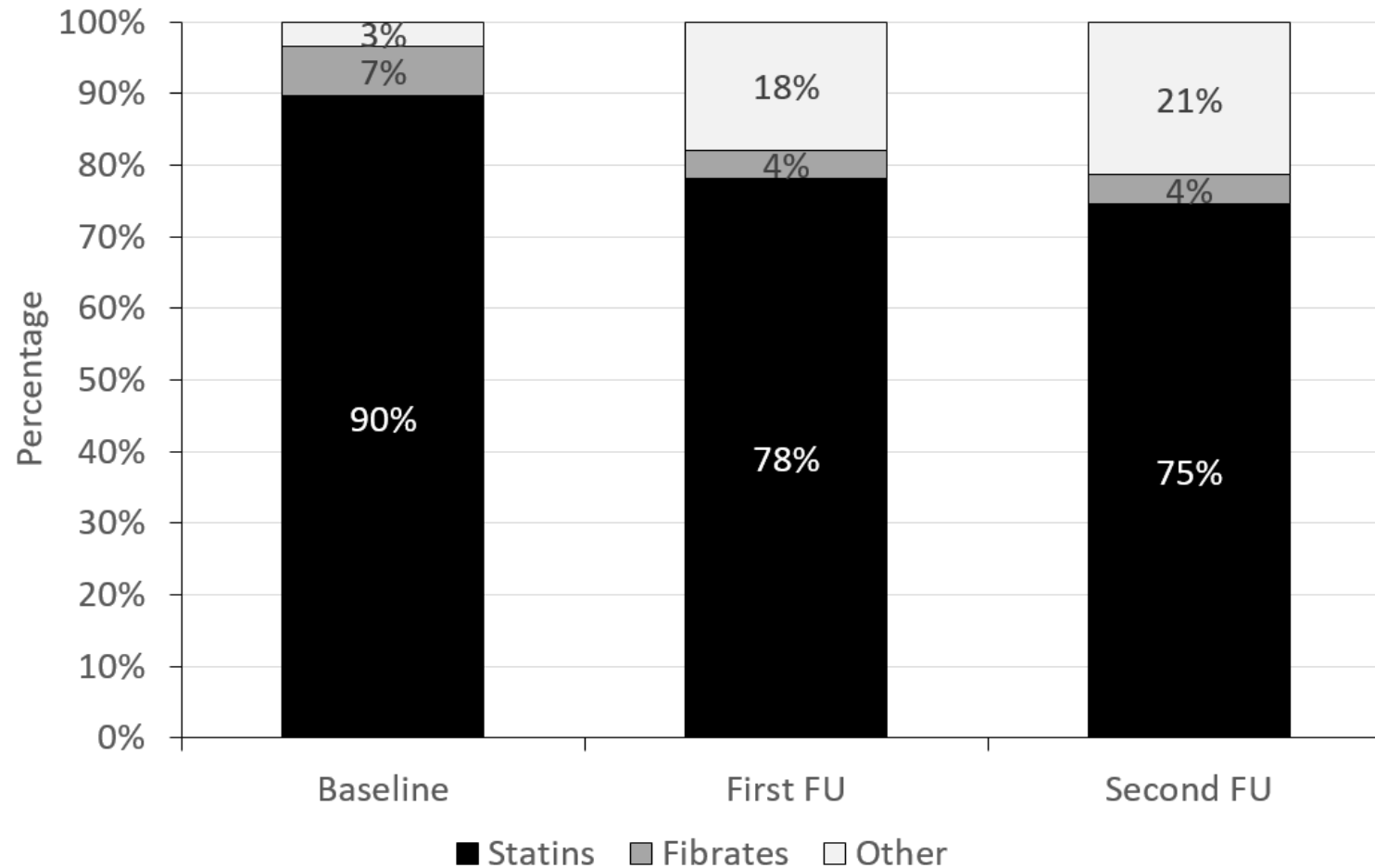
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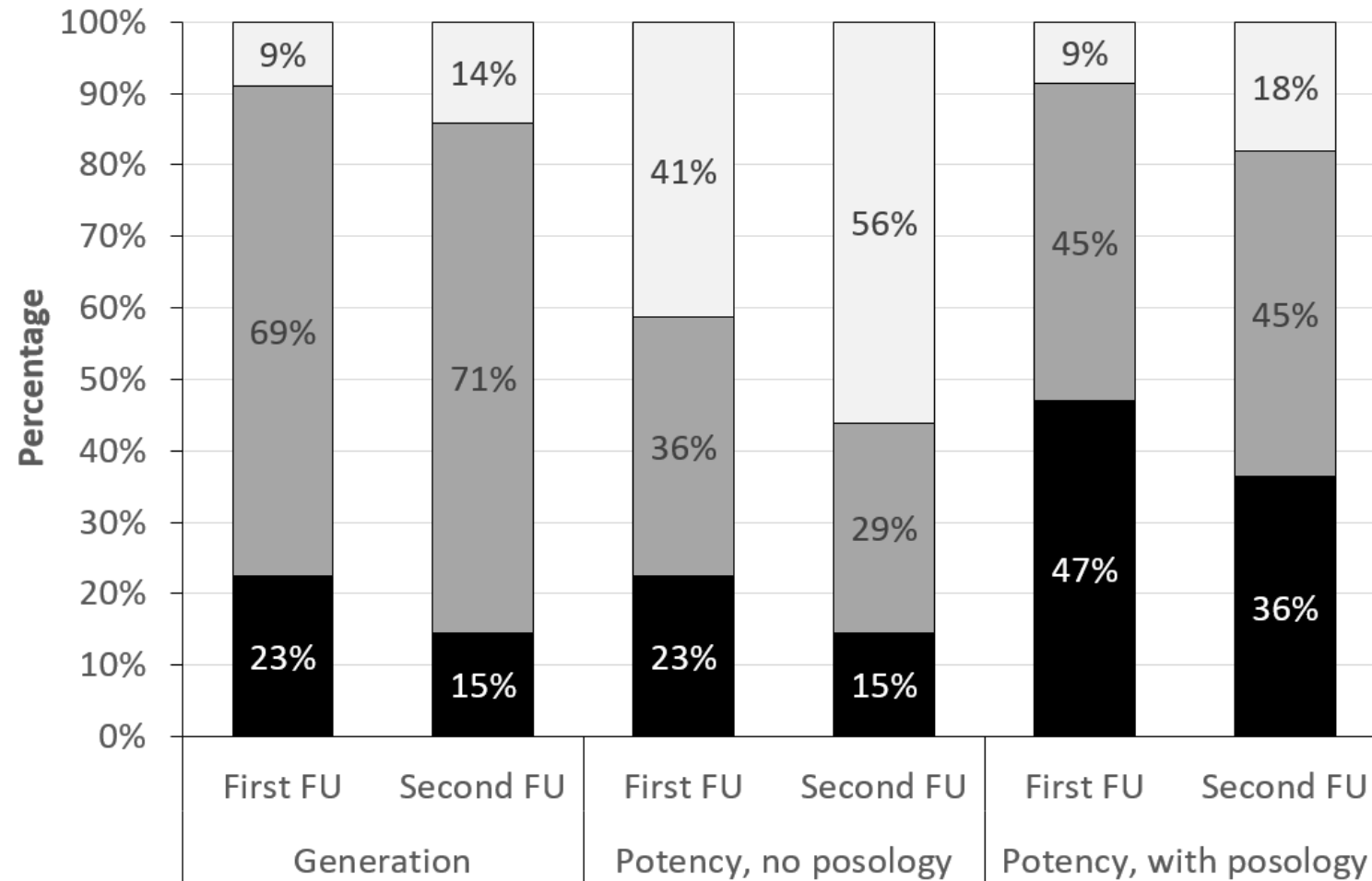
Supplemental figure 1: flowchart of the selection of participants, baseline (2003-2006), first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland



Supplemental figure 2: distribution of hypolipidemic drug classes, baseline (2003-2006), first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland



Supplemental figure 3: Generation and potency of statin drugs, first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland. Black: first generation or low potency; dark gray, second generation or intermediate potency; light gray, third generation or high potency.



Supplemental figure 4: comparison of statin potency considering or not posology, first (2009-2012) and second (2014-2017) follow-ups of the CoLaus|PsyCoLaus study, Lausanne, Switzerland.

First follow-up

		<i>With posology</i>		
		Low	Intermediate	High
<i>Without posology</i>	Low	85	87	0
	Intermediate	283	7	0
	High	0	256	70

Second follow-up

		<i>With posology</i>		
		Low	Intermediate	High
<i>Without posology</i>	Low	67	43	0
	Intermediate	197	8	0
	High	0	282	136