UNIL | Université de Lausanne
Faculté de biologie
et de médecine

IUMSP
Institut universitaire de médecine sociale et préventive

Mémoire de Maîtrise en médecine 1493

Association of blood pressure and dietary patterns with family history of hypertension in Switzerland

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Lausanne, 2013

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Abstract

Objective: Genetic factors are known to affect blood pressure. A positive family history of hypertension is associated with higher blood pressure levels than a negative history. To our knowledge, there are no population-based data across linguistic regions on this topic in Switzerland. Furthermore, little is known about the association of family history of hypertension with dietary patterns. We analysed the association of father's, mother's and siblings' history of hypertension with blood pressure and dietary habits in the Swiss Survey on Salt Intake.

Methods: We used data from 1448 participants to the Swiss Survey on Salt intake, a population-based study conducted in 2010-2012 in the general population aged 15 years and older. Participants were asked about their family history of hypertension, their behaviour and perception in relation to dietary salt consumption, their lifestyle. Office blood pressure was measured using a validated automatic device. We estimated salt, potassium and protein intakes using 24-hour urinary sodium, potassium and urea excretions. We used multiple logistic and linear regressions to explore factors associated with family history of hypertension and the association of blood pressure with a family history of hypertension.

Results: The 785 men and 863 women had mean[SD] age of 48.3[18.6] and 46.6[18.2] years and bmi of 26.1[4.3] and 24.4[4.8] , respectively. Systolic/diastolic blood pressure (mm Hg) was higher in people with a positive as compared to those with a negative family history of hypertension (133.4/79.0 vs 127.4/74.4 in men and 120.8/73.4 vs 117.2/71.2 in women, all P<0.0001). In adjusted analyses, a positive family history of hypertension was associated with 3.8/3.9 mm Hg higher SBP/DBP in men (P<0.001), whereas no association persisted in women. In adjusted analyses, we found no significant differences in urinary sodium, potassium and urea excretions by family history of hypertension. People with a positive family history of hypertension, as compared to those with a negative history, tended to report more frequently to pay attention to their salt consumption (Odds ratio[Standard error] (OR[SE])=1.49[0.29] in women, P=0.04 and 1.45[0.31] in men, P=0.09). We found no association between family history of hypertension and self-reported attempt to limit salt intake. People from the German-speaking region had higher odds of a positive family history of hypertension than people from the French-speaking region (OR[SE]=1.81[0.39] in men, P=0.01 and 1.56[0.30] in women, P=0.02), independently of hypertension status.

Conclusions: We found a positive family history of hypertension to be associated with higher blood pressure in this Swiss population-based sample. People with a positive family

history of hypertension reported to pay more attention to dietary salt intake, although they did not attempt to limit salt intake. This was confirmed by the fact that we found similar dietary salt intake, estimated from 24-hour urinary sodium excretion, in people with and without family history of hypertension. We found no difference in 24-hour urinary potassium and urea excretions in people with and without family history of hypertension. Finally, we found higher prevalence of positive family history of hypertension in the German speaking region of Switzerland as compared to the French-speaking region, independently of hypertension status. Family history of hypertension does not appear to greatly influence dietary patterns in Switzerland.

Introduction

Family history of cardiovascular disease and cardiovascular risk factors

Cardiovascular disease (CVD) is associated with a high burden in terms of morbidity and mortality worldwide. The risk factors for CVD are well known: age, sex, diabetes mellitus, dyslipidemia, family history of CVD, smoking and hypertension. CVD are known to aggregate in families. For instance, CVD in parents doubles the risk of CVD in offspring.(1) Furthermore, if siblings suffer from CVD, the risk for an incident is stronger, in particular if the onset of siblings' CVD occurred at a young age.(2) This is the reason of our interest in the relationship between a positive family history of hypertension and high blood pressure (BP) of the offspring, which is a cardiovascular risk factor.

Family history of hypertension and its association with offspring's blood pressure

Offspring with at least one or two hypertensive parents have higher BP levels that offspring of normotensive parents, with a dose-effect relationship.(3)(4)(5)(6)(7) This is already observed at young ages and continues throughout life.(3)(8)(9) Parental BP has a strong influence on the natural history of BP in their offspring from childhood into young adulthood.(9) This relationship depends on the age at which hypertension had been diagnosed in parents: the risk of hypertension in offspring increases if the parents were diagnosed before the age of 60 years. Hunt et al.(10) showed that this relationship also exists for siblings and twins, and is even stronger if a twin is hypertensive than if a parent or a sibling is hypertensive.

Familial aggregation and cardiovascular risk factors

People with hypertensive parents also have higher levels of other cardiovascular risk factors such as cholesterol and mean blood glucose concentrations, BMI, and serum uric acid concentrations than offspring of normotensive parents.(3)(10)(11)(12) Some studies also showed that there are changes in left ventricle size and function, and increased carotid stiffness associated with a positive family history of hypertension.(13)(14)(15) This indicates that cardiovascular risk factors tend to cluster into families (6)(16)(17), likely due to genetic factors and shared environmental conditions, among which diet is suspected to play an important role.

Familial aggregation exists for important cardiovascular risk factors such as total serum cholesterol and low-density lipoprotein cholesterol(18), diabetes mellitus(19) and smoking

habits(20) but also for obesity(21), nutrient intake (22) and physical activity (23). Moreover, parental habits are related to children's BP levels. Burke et al. (24) showed that father's alcohol intake and smoking had an influence on daughter's systolic blood pressure (SBP). SBP was also significantly related to paternal BMI in both sons and daughters, but the father's fat intake was only related to son's SBP.

As a child grows up, the number of shared behaviours with his family increases.(25) As smoking habits and high BMI tend to cluster into families, we can assume that offspring of smoking and/or overweighted parents may become current smokers and/or overweighted themselves. Many studies showed that these are risk factors for being prehypertensive or hypertensive.(26)(27)(28) These results highlight the importance of modifying family's and offspring's habits as soon as possible in the childhood. This also suggests that improved health behaviors in families at risk may show long-term benefits for offspring.

Sex of the hypertensive parent and higher blood pressure

There are contradictory findings on whether paternal or maternal hypertension is of greater importance as a risk factor for elevated BP in the offspring.(17)(29) Some studies (3)(30) found that only maternal BP, and not paternal BP, was significantly correlated with the BP of their offsprings, whereas others found paternal history to be a better predictor of high BP in male and female offspring than maternal history.(8)(24)(31)(32) Staessen et al.(33) showed that there were father-son and mother-daughter correlations for SBP, whereas the other parent-offspring correlations for SBP and DBP were not significant. However, Staessen et al. found that there was a father-daughter and a mother-son correlation for urinary sodium excretion. Goldstein et al.(5) found that men with one or two hypertensive parents had higher BP that women with both parents being hypertensive. These results suggest that there may be a relation between the sex of the parent and the sex of the offspring in predicting BP, which points toward sex-specific genetic factors

Reliability of reported family history of hypertension

Bochud et al.(34) showed that only 2/3 of patients were able to provide information about their siblings' history of hypertension, and that the history of hypertension in siblings reported by the participants was sensitive (89%) but not very specific (78%). A study has shown that women have better knowledge about their family history of CVD than men.(35) This shows us how difficult it is to have the right information about patient's family history and it may

explain why so many studies have different findings about the relation between mother/father history of hypertension and elevated BP in offspring.

Association of blood pressure and diet

We know that BP is influenced by many dietary factors. The best known of these factors is sodium(36)(37)(38), which has been shown to increase BP at least in selected subgroups of people, such as the elderly, obese people, people with kidney diseases and people of African descent (ESH, salt sensitivity). We find sodium in salt, but also in breads, cheeses, products derived from meat, industrial food, etc. The association of lower BP with a high intake of potassium is also known(38). Potassium consumption is reflected by fruits' and vegetables' consumption, among other dietary factors. Also, BP is higher in patients drinking a large amount of alcohol, and lower in those who drink caffeine in sufficient quantity.

Family history of hypertension and dietary patterns

Higher BP in association with a family history of hypertension is influenced by two major causes: genetic factors and shared environmental factors, among which diet likely plays an important role. So far, little is known about the dietary patterns of people with a positive family history of hypertension as compared to those with a negative history. In this work compared two groups: people with a negative family history of hypertension and those with a positive family history of hypertension. The aim of our study is to analyse whether people coming from a hypertensive family eat more salt and have different dietary habits than people coming from a normotensive family. We also explored whether we could confirm high BP levels in association with a positive family history of hypertension.

Subjects and methods

Sampling strategy

The data come from a cross-sectional population-based survey which took place in 9 Swiss cantons (Vaud, Geneva, Valais, Fribourg, Luzern, Basel, Zürich, St-Gallen and Ticino). This permitted to cover the French-, German- and Italian- speaking regions of Switzerland, reflecting the cultural and geographical diversity of Swiss population. The Swiss Federal Office of Public Health launched a nationwide program to reduce dietary intake in the Swiss population (Salt strategy 2008-2012). The Swiss Study on Salt intake (SSS) is part of this programme with the aim of estimating the salt intake in the Swiss population. The SSS's recruitment began in January 2010 and ended in March 2012. Participants had to live in

Switzerland and be aged ≥15 years to be admitted in this study. They were contacted first as a household, using the Swiss Federal Statistical Office phone directory, which is updated every three months, provided by a major phone provider in Switzerland. This directory is the most complete list of the Swiss households. Households were first contacted by sending an information letter inviting them to participate at this survey. Then, they were contacted by up to 3 phone call attempts, in different days and at different hours, including evening hours. Finally, one person per household was randomly selected to take part in the survey. Participation rate was 10%. Participants were separated in 8 sex- and age-stratified groups (men and women, aged 15-29, 30-44, 45-59 and ≥60 years). Young participants were difficult to recruit; this is why the study sample was completed with 277 participants recruited in schools and universities.

Data collection

The purpose of this study was to estimate the salt intake of the Swiss adult population by calculating the urinary sodium excretion using a single 24-hour urine collection. To avoid any change in their salt consumption, participants were told that the survey was about lifestyle and BP. Participants came twice to the study center, for measurements (made by trained health workers) and for urine collection. The urine samples' analyses were made by the central laboratory of the CHUV.

At the first visit, participants completed a questionnaire about their lifestyle: diet (fish, meat, fruits and vegetables, alcohol consumption), smoking status, physical activity. They also answered questions about medication, pre- or post-menopausal status and the use of oral contraceptive/ hormonal replacement therapy, medical history and the one of their family. Then weight and height were measured, and BMI was calculated and stratified (18-25: normal, 25-30: overweight, ≥30: obesity). They received two bottles (3 liters each) and instruction for the urine collection.

At the second visit, participants brought the bottles back. An optional blood collection was made by a nurse. Urine was weighted and frozen, then sent with the blood samples to the Central Chemical Laboratory of Lausanne University Hospital (CHUV, Lausanne, Vaud, Switzerland). This laboratory is certified ISO/CEI 17025 and ISO 15189.

Urinary sodium and potassium excretions were measured by indirect potentiometry. Urinary urea was measured by Urease-GLDH method. Urinary creatinine was measured by Jaffé

kinetic compensated method. These measurements were made in 24 hours urine by Roche Diagnostics, Switzerland.

Covariates

The linguistic region was defined according to the center participants attended (Vaud, Geneva, Valais and Fribourg for French part, Luzern, Basel, Zürich and St-Gallen for German part and Ticino for the Italian one).

The questionnaire contained participants' responses about age, sex, smoking status (coded as never smoker, current smoker, ex-smoker), alcohol consumption ("During the past 7 days, how many bottles of beer (3 dl), how many glasses of wine (2 dl) and how many glasses of spirit (0.25 dl) did you drink, ?"), diet (quantity of meat, fish, fruits and vegetables defined by the question "How many days per week do you eat …?") and beverages (defined by "What amount of liquid do you usually drink per day, without taking alcohol into account?"). They were also asked about any specific diet followed during the past 12 months, their interest in their salt consumption ("Are you interested in your salt consumption?") and their attempt to limit this consumption ("Do you try to limit your salt consumption?"). They also had to report their family history of hypertension ("Does (did) your biological mother/ biological father/ any of your full siblings suffer from hypertension?") and their antihypertensive treatment ("Have you ever been prescribed drugs to treat high BP by a doctor?").

BMI was calculated as weight[kg]/height[m]^2, their systolic and diastolic BPs were calculated using the average of 8 different measures (4 out of 5 measurements taken on two consecutive days, after having removed the first measurement) and hypertension was defined as ≥140/≥90 mmHg. The samples of urine were used to measure urinary sodium, potassium, urea and creatinine excretion as well as urine volume in 24 hours and. The glomerular filtration rate (eGFR) was estimated using the CKD-EPI equation and expressed in ml/min/1.73m^2.

Statistical analyses

Statistical analyses were made using Stata 12.0 (Stata Corp, College Station, USA). Results were expressed as mean ± standard deviation (continuous covariates) or in percentages (categorical covariates). Groups were compared using T-test and Chi-squared test for tables 1 and 2. Dietary habits (i.e. fish, meet, fruits, vegetables and liquid intake) were compared using a median test. Table 3 was made using simple and multiple linear regression models, going from minimal to full adjustment. We obtained values for systolic and diastolic BP

differences (expressed in mmHg) comparing participants from groups FH HT+ (positive family history of hypertension) and FH HT- (negative family history of hypertension). We adjusted the model using the following covariates: age, sex, BMI, treatment against hypertension, linguistic region, 24-hour urinary sodium excretion, 24-hour urinary potassium excretion, 24-hour urinary urea excretion and 24-hour urinary volume. Finally, we used multiple logistic regression for table 4 where the dependent variable was family history of hypertension (FH HT+ coded as 1 vs FH HT- coded as 0), including the following covariates: age, sex, BMI, hypertension, linguistic region, 24-hour urinary sodium excretion, 24-hour urinary potassium excretion, 24-hour urinary creatinine excretion, 24-hour urinary urea excretion and 24-hour urinary volume, self-reported intakes of meat, fish, fruits, and vegetables, attention payed to salt consumption and attempt to limit salt consumption.

Ethics

The Swiss Study on Salt intake was approved by the Institutional Ethics Committees of each center. The protocol was in accordance with the Helsinki Declaration. Participants had to give their written informed consent. If participants were under 18, parental consent was also provided.

Results

Participants' characteristics

A total of 1448 participants were included in this analysis. Table 1 shows participants' characteristics (separately for men and women) by family history of hypertension. A positive family history of hypertension (FH HT+) was found in 603 participants (265 men and 338 women) and 845 (434 men and 411 women) had a negative family history of hypertension (FH HT-). Some characteristics statistically differed between the group with positive (at least one hypertensive parent) and the group with negative family history of hypertension: age (men of group FH HT+ were older, mean[SD] 51.11 [15.5] vs 46.23 [19.7] years, p=0.01 and women of FH HT+ group were 50.29 [16.4] vs 43.8 [18.8] years, p<0.0001), BMI (23.98 [4.6] kg/m^2 in FH HT- women and 24.87 [5.1] kg/m^2 in FH HT+ women, p=0.01), smoking status in women (16.77% of FH HT+ women are current smokers vs 17.16% of FH HT- women, p=0.02), SBP (133.37 [13.6] mmHg in FH HT+ men vs 127.44 [13.2] mmHg in FH HT- men, p<0.0001, and 120.78 [16] mmHg in FH HT+ women vs 117.23 [14.5] mmHg in FH HT- women, p=0.01) and DBP (79.04 [10.3] mmHg in FH HT+ men vs 74.37 [10.3] mmHg in FH HT- men (p<0.0001) and 73.44 [9.3] mmHg in FH HT+ women vs 71.2 [9.2] mmHg in FH HT-

women, (p=0.01)), hypertension and antihypertensive treatment (the prevalences were lower in the FH HT- group for both men and women, p<0.0001), urinary potassium excretion (lower in the FH HT+ groups, 77.55 [25.1] vs 73.03 [25.9], p=0.02 for men, and 61.73 [21.8] vs 55.88 [21.1], p=0.01 for women), urinary volume in women (2084.8 [926.2] in the FH HT+ group vs 1943.51 [918.8], p=0.04) and finally eGFR (higher in both FH HT- groups).

We found no statistically significant differences for smoking status in men (17.87% of FH HT+ men are current smokers vs 19.25% of FH HT- men, p=0.2), alcohol consumption, urinary sodium excretion (184.45 [72.7] in FH HT+ men vs 179.71 [70.4] in FH HT- men, p=0.39, and 136.9 [58.8] in FH HT+ women vs 130.32 [56.6] in FH HT- women, p=0.12), urea excretion and urinary volume for men.

Dietary habits and their association with family history of hypertension

Participants were asked about their lifestyle and their dietary habits. Table 2 shows the differences between the FH HT+ and FH HT- groups, considering men and women separately. We found no statistically significant differences between these groups for the self-reported consumptions of fruits, vegetables, fish or meat, the diet participants followed or the amount of liquid they drank. Men showed no difference in trying to limit their salt consumption but we found this difference in women: 69.55% of FH HT+ vs 58.05% of FH HT-, p=0.001. By contrast, we found that both men and women with positive family history of hypertension were more interested in their salt consumption than those with a negative history (43.18% vs 35.02% for men, p=0.03 and 56.85% vs 42.65% for women, p<0.001).

Association of family history of hypertension with selected phenotypes

Table 3 shows the association of family history of hypertension with selected phenotypes, such as age, sex, linguistic region or urea excretion. Model 1 is unadjusted and shows that the SBP of participants FH HT+ was 5.93 [1] (p<0.001) mm Hg for men and 3.55 [1.1] (p=0.002) mm Hg for women higher than pressures of subjects of group FH HT-. Their DBP is higher with 4.67 [0.8] (p<0.001) mm Hg for men and 2.23 [0.7] (p<0.001) mm Hg for women. Age has a big influence on this numbers for men only when considering SBP and for both sexes when considering DBP: after adjustment, the difference was of 4.39 [0.9] (p<0.001) mm Hg for men and 0.52 [1] (p=0.58) mm Hg for women for SBP and 3.87 [0.8] (p<0.001) mm Hg for men and 1.47 [0.7] (p=0.03) mm Hg for women for DBP. Model 3 was adjusted for BMI and shows that there is a statistically difference in both BPs for men and for

women, even though men had a bigger difference than women. Model 4 shows differences when adjusted for linguistic region: 5.36 [1] (p<0.001) mm Hg for men and 3.23 [1.1] (p=0.004) mm Hg for women for SBP and 4.31 [0.8] (p<0.001) mm Hg for men and 2.1 [0.7] (p=0.002) mm Hg for women for DBP. Model 5 was adjusted for sodium excretion and model 6 for potassium excretion. The relationship between positive family history of hypertension and BP does not disappear when we adjust models for sodium or for potassium urinary excretion. From here on, none of following models are significant for women. In model 7 were added covariates for age, BMI and anti-hypertension treatment: 4.12 [1] (p<0.001) mm Hg more for SBP and 4.08 [0.8] (p<0.001) mm Hg more for DBP. In model 8 we added linguistic region to covariates of model 7 and found these numbers: 3.63 [1] (p<0.001) mm Hg more for SBP and 3.79 [0.8] (p<0.001) mm Hg more for DBP. Model 9 is only adjusted for age, anti-hypertension treatment and sodium excretion. The values for this model are: 4.43 [1] (p<0.001) mm Hg more for SBP and 4.44 [0.8] (p<0.001) mm Hg more for DBP. Model 10 is the same with potassium excretion instead of sodium excretion and shows that SBP was 4.43 [1] (p<0.001) mm Hg higher and DBP was 4.42 [0.8] (p<0.001) mm Hg higher. Model 11 is adjusted for age, anti-hypertension treatment and sodium- and potassium excretion. Numbers are significant for both BPs. In model 12 was added the BMI and we found following values: 4.29 [1] (p<0.001) mm Hg more for SBP and 4.22 [0.8] (p<0.001) mm Hg more for DBP. In model 13 we added the linguistic region as a covariate: 3.81 [1] (p<0.001) mm Hg more for SBP and 3.93 [0.8] (p<0.001) mm Hg more for DBP. Finally, model 14 is adjusted for age, BMI, anti-hypertension treatment, linguistic region, sodium and potassium excretion, urea excretion and urinary volume. The values we found are: 3.8 [1] (p<0.001) mm Hg for SBP and 3.94 [0.8] (p<0.001) mm Hg for DBP.

Association of family history of hypertension with dietary habits and selected phenotypes

Table 4 is the result of a multiple logistic regression where the dependent variable is a positive or a negative family history of hypertension, defined as positive for participants with at least one hypertensive parent. We checked the influence of different covariates on the fact of having a positive family history of hypertension. Covariates which were significantly associated with a positive family history of hypertension are: hypertension (OR[SE]=2.81[0.66], p<0.001 in men and OR 1.59[0.38], p=0.05 in women), being from the German-speaking part of Switzerland (OR 1.81[0.39], p=0.01 in men and OR 1.56[0.30], p=0.02 in women) and the attention payed to the salt consumption by women only (OR 1.49[0.29], p=0.04).

Family history of hypertension was not independently associated with age, BMI, being from the Italian-speaking part of Switzerland, urinary sodium or potassium excretions, urea or creatinin excretion, urinary volume, attempt to limit salt consumption, and portions of fruits, vegetables, meet or fish.

Discussion

Association of family history of hypertension with offspring's blood pressure

It is already known that offspring of hypertensive parents had a higher BP than offspring of normotensive parents. (3)(4)(5)(6)(7) It is also known that having a hypertensive sibling is a bigger risk to become hypertensive than having a hypertensive parent.(10) Furthermore, a study(9) shows that there is a dose-effect relationship which begins in childhood.

We confirmed these results by showing that participants with a positive family history of hypertension (including parents' and siblings' history) have a higher systolic and diastolic BP. This difference is stronger for men than for women. We also showed that being FH HT+ is a big risk for being hypertensive (defined as SBP >140 mmHg and DBP >90 mmHg) : 24% men of normotensive families were hypertensive but 47% men of hypertensive families were included in our definition of hypertension. This risk is almost doubled for women.

Besides, participants from FH HT+ group had more often an antihypertensive treatment. This is partly explained by their hypertension status, but the number of FH HT+ participants under antihypertensive treatment is more than twice the number of FH HT- participants taking the same treatment. We may assume than a part of participants of the FH HT+ group takes antihypertensive treatment without being included in our definition of hypertension.

Dietary habits in association with family history of hypertension

We found no difference in most of our covariates for dietary habits. Both groups eat the same quantity of meet and fish, and of vegetables and fruits. Despite of what we assumed at the beginning of this study, we found no difference in urinary sodium excretion in 24 hours urine. That means that people coming from hypertensive families do not eat more salt than people coming from normotensive families.

On the other hand, we found that people with a positive family history of hypertension tends have higher urinary potassium excretion, independently of hypertension stage, age and sex. We found indeed 77.55 mmol/24h vs 73.03 mmol/24h (p=0.02) for men and 61.73 mmol/24h vs 55.88 mmol/24h (p=0.0002) for women. We could not relate this to a high self-reported

consumption of fruits and vegetables. The source of this potassium excretion therefore remains unclear.

We found people (specially women) with a positive family history of hypertension to report to pay more attention to salt intake. 57% women from the FH HT+ group answered "yes" to the question ("Are you interested in your salt consumption?" when only 35% men from the FH HT- group answered "yes" to this question. However, this was not substantiate by lower urinary Na excretion. Our results suggest that even if people pay more attention they do not eat less salt. Furthermore, they do not seem to limit more than salt consumption.

Linguistic differences associated with reported family history of hypertension

We found regional differences with higher prevalence of positive family history of hypertension in the German speaking region of Switzerland. In table 4 we can see that men from the German speaking region have an Odd's Ratio of 1.8 (1.6 for women) compared with men from the French speaking region. It is known that prevalence of hypertension is higher in the German-speaking region (39), so we found more hypertensive families in this part of Switzerland. That explains partly our results. Another explanation may be that German-speaking people talk more frequently about health in family.

Strengths

The strengths of this study are its population-based nature and the use of 24-hour urine collection to estimate dietary salt, potassium and protein intake. Our description of hypertension is based on repeated measures. The laboratory measures were all done in Lausanne, CHUV. The prospective nature of this study allows us to have standardised measures. Finally, we studied three linguistic regions with three different cultures.

Limitations

The first limitation is the participation rate of 10%. As a consequence, this sample might not be representative of the general Swiss population aged 15 years and over, which limits the external validity of the findings. This is obvious when looking at the average age of our population. Young people were indeed difficult to recruit and it is well known that hypertension incidence increases with age. We can therefore assume that our sample has higher BP and has more often hypertensive parents because the latter are older than the underlying source population.

We found that women report more family history of hypertension than men, despite the fact that the number of men included in this study is similar to the number of women. This was already discussed in one study(35) about CVD in which authors explained it by a better knowledge by women of their family history of CVD. We may postulate that women are more interested in their medical family history, and may talk more with their family about it.

There is a risk that the 24-hour urine was under- or over-collected but our measures were adjusted for it, by adding both 24h urine volume and creatinine excretion per Kg body weight as covariates in the models.

Finally, we didn't use a validated food frequency questionnaire.

Conclusions

We found a positive family history of hypertension to be associated with higher blood pressure in this Swiss population-based sample. People with a positive family history of hypertension reported to pay more attention to dietary salt intake, although they did not attempt to limit salt intake. This was confirmed by the fact that we found similar dietary salt intake, estimated from 24-hour urinary sodium excretion, in people with and without family history of hypertension. We found no difference in 24-hour urinary potassium and urea excretions in people with and without family history of hypertension. Finally, we found higher prevalence of positive family history of hypertension in the German speaking region of Switzerland as compared to the French-speaking region, independently of hypertension status. Family history of hypertension does not appear to greatly influence dietary patterns in Switzerland.

Tables and figures

Table 1 : Participants' characteristics

	Men			Women		
	FH HT+	FH HT-	P value	FH HT+	FH HT-	P value
Number of participants	265	434		338	411	
Age	51.11 (15.50)	46.23 (19.65)	0.01	50.29 (16.37)	43.8 (18.82)	<0.0001
BMI	26.65 (4.27)	25.7 (4.18)	0.01	24.87 (5.12)	23.98 (4.55)	0.01
Current Smokers (%)	17.87	19.25	0.20	16.77	17.16	0.02
Alcohol units during the past 7 days. Median (IQR)	5 (2;9)	4 (1;8)	0.33	1 (0;4)	2 (0;4)	0.07
SBP (mm Hg)	133.37 (13.57)	127.44 (13.20)	<0.0001	120.78 (15.95)	117.23 (14.52)	0.01
DBP (mm Hg)	79.04 (10.30)	74.37 (10.33)	<0.0001	73.44 (9.26)	71.2 (9.20)	0.01
Hypertension (%)	46.97	24.19	<0.0001	26.63	13.87	<0.0001
Antihypertensive treatment (%)	32.58	11.52	<0.0001	20.41	8.76	<0.0001
Urinary Na excretion (mmol/24h)	184.45 (72.69)	179.71 (70.35)	0.39	136.9 (58.83)	130.32 (56.62)	0.12
Urinary K excretion (mmol/24h)	77.55 (25.08)	73.03 (25.89)	0.02	61.73 (21.81)	55.88 (21.10)	0.01
Urinary urea excretion (mmol/24h)	441.82 (148.25)	429.75 (141.39)	0.28	308.01 (100.67)	297.24 (103.42)	0.15
Urinary volume (ml/24h)	1952.43 (893.05)	1878.19 (873.11)	0.28	2084.8 (926.16)	1943.51 (918.81)	0.04
Urinary creatinine excretion (mmol/kg/24h)	21.15 (4.68)	22.00 (5.37)	0.03	16.51 (4.09)	17.09 (4.90)	0.08
eGFR (ckd-epi)(ml/min/1.73m^2)	86.68 (19.01)	91.68 (19.63)	0.01	87.94 (18.95)	91.43 (20.20)	0.02

Data are mean(SD), unless otherwise specified.

Table 2: Association of family history of hypertension with selected dietary patterns

	Men			Women		
	FH HT+	FH HT-	P value	FH HT+	FH HT-	P value
n	265	434		338	411	
Specific diet followed (%)	9.51	8.55	0.67	17.61	13.90	0.17
Amount of liquid (l/24h). Mean (SD)	1.15 (2.50)	1.30 (2.19)	0.40	1.41 (1.81)	1.11 (2.45)	0.06
Portions of fruits.	2 (1;2)	2 (1;2)	0.70	2 (2;3)	2 (2;3)	0.20
Portions of vegetables.	2 (1;2)	2 (1;2)	0.38	2 (2;3)	2 (2;3)	0.23
Quantity of meet (days/week).	4 (3;5)	4 (2;5)	0.41	3 (2;4)	3 (2;4)	0.42
Quantity of fish (days/week).	1 (0;1)	1 (0;1)	0.72	1 (0;1)	1 (0;1)	0.17
Attention payed to salt consumption (% yes)	43.18	35.02	0.03	56.85	42.65	<0.001
Attempt to limit salt consumption (% yes)	57.36	50.81	0.09	69.55	58.05	0.01

Data are medians (IQR), unless otherwise specified. IQR, interquartile range.

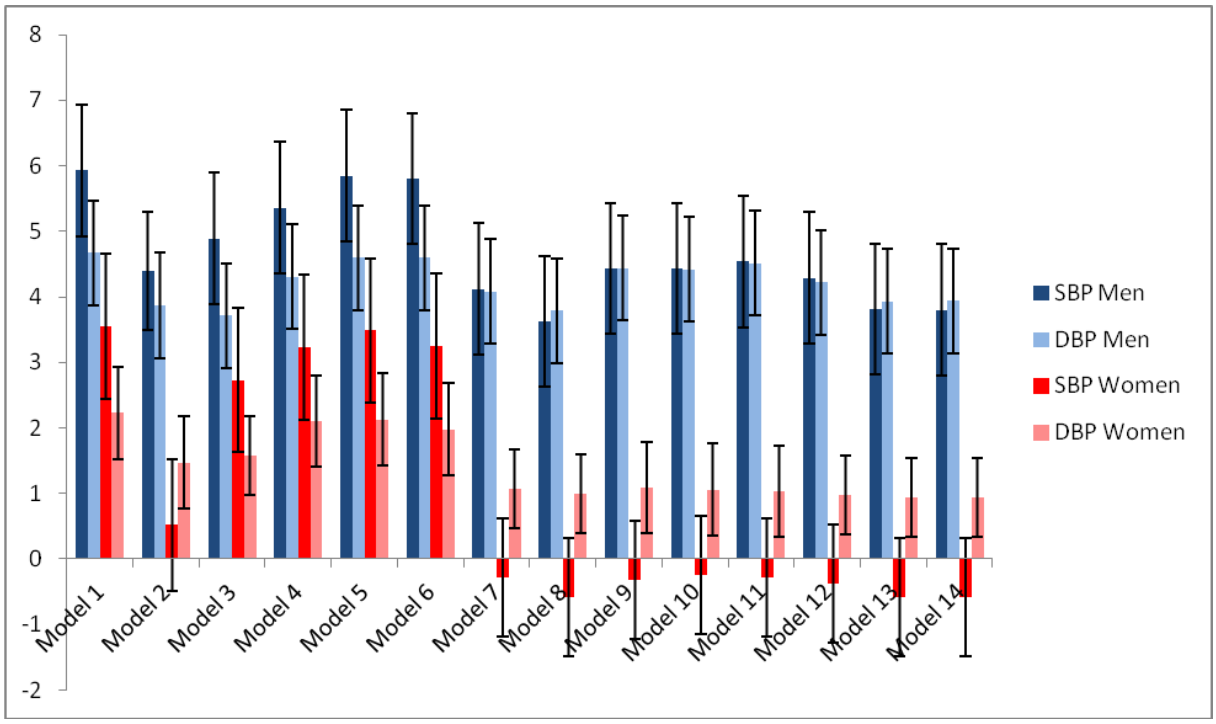
Table 3. Association blood pressure with family history of hypertension using selected adjustment models

Model	SBP				DBP			
	Men		Women		Men		Women	
	B(SE)	P value	B(SE)	P value	B(SE)	P value	B(SE)	P value
1	5.93 (1.04)	<0.001	3.55 (1.12)	0.01	4.67 (0.80)	<0.001	2.23 (0.68)	<0.001
2	4.39 (0.95)	<0.001	0.52 (0.96)	0.58	3.87 (0.78)	<0.001	1.47 (0.67)	0.03
3	4.89 (1.00)	<0.001	2.73 (1.08)	0.01	3.71 (0.76)	<0.001	1.58 (0.64)	0.01
4	5.36 (1.04)	<0.001	3.23 (1.11)	0.01	4.31 (0.80)	<0.001	2.10 (0.67)	0.01
5	5.85 (1.04)	<0.001	3.49 (1.12)	0.01	4.63 (0.80)	<0.001	2.13 (0.68)	0.01
6	5.80 (1.04)	<0.001	3.25 (1.12)	0.01	4.60 (0.80)	<0.001	1.98 (0.68)	0.01
7	4.12 (0.96)	<0.001	-0.29 (0.93)	0.76	4.08 (0.76)	<0.001	1.07 (0.64)	0.10
8	3.63 (0.97)	<0.001	-0.58 (0.92)	0.53	3.79 (0.76)	<0.001	0.99 (0.64)	0.12
9	4.43 (0.97)	<0.001	-0.32 (0.93)	0.73	4.44 (0.78)	<0.001	1.09 (0.67)	0.10
10	4.43 (0.98)	<0.001	-0.25 (0.94)	0.79	4.42 (0.79)	<0.001	1.06 (0.68)	0.12
11	4.54 (0.97)	<0.001	-0.29 (0.94)	0.76	4.51 (0.78)	<0.001	1.03 (0.67)	0.13
12	4.29 (0.96)	<0.001	-0.37 (0.93)	0.69	4.22 (0.76)	<0.001	0.98 (0.65)	0.13
13	3.81 (0.96)	<0.001	-0.59 (0.93)	0.53	3.93 (0.75)	<0.001	0.94 (0.64)	0.14
14	3.85 (0.96)	<0.001	-0.57 (0.93)	0.54	4.01 (0.75)	<0.001	0.95 (0.64)	0.14

Data are beta regression coefficients (SE) and P values.

Covariates included in the models
Model 1: none
Model 2: age
Model 3: BMI
Model 4: linguistic region
Model 5: sodium excretion
Model 6: potassium excretion
Model 7: age- and BMI- and anti-hypertension treatment
Model 8: age- , BMI-, anti-hypertension treatment- and linguistic region
Model 9: age-, anti-hypertension treatment- and urinary Na excretion
Model 10: age-, anti-hypertension treatment- and urinary K excretion
Model 11: age-, anti-hypertension treatment- and urinary K and Na excretion
Model 12: age-, BMI-, anti-hypertension treatment- and urinary K and Na excretion
Model 13: age-, BMI-, anti-hypertension treatment-, linguistic region- and urinary K and Na excretion
Model 14: age-, BMI-, anti-hypertension treatment-, linguistic region-, urinary K and Na excretion-, urea and creatinine excretion- and urinary volume

Figure 1. Association of family history of hypertension with blood pressure



Bars are mean values and whiskers are standard deviation. SBP, systolic blood pressure, DBP, diastolic blood pressure. Y axis, mmHg.

Model 1: unadjusted
Model 2: age-adjusted
Model 3: BMI-adjusted
Model 4: linguistic region-adjusted
Model 5: sodium excretion-adjusted
Model 6: potassium excretion-adjusted
Model 7: age- and BMI- and anti-hypertension treatment-adjusted
Model 8: age- , BMI-, anti-hypertension treatment- and linguistic region-adjusted
Model 9: age-, anti-hypertension treatment- and urinary sodium excretion-adjusted
Model 10: age-, anti-hypertension treatment- and urinary potassium excretion-adjusted
Model 11: age-, anti-hypertension treatment- and urinary potassium and sodium excretion-adjusted
Model 12: age-, BMI-, anti-hypertension treatment- and urinary potassium and sodium excretion-adjusted
Model 13: age-, BMI-, anti-hypertension treatment-, linguistic region- and urinary potassium and sodium excretion-adjusted
Model 14: age-, BMI-, anti-hypertension treatment-, linguistic region-, urinary potassium and sodium excretion-, urea excretion- and urinary volume-adjusted

Table 4. Logistic regression: Association of family history of hypertension with dietary habits and selected phenotypes

FH HT+	Odds Ratio (SE)		P value	
	Men	Women	Men	Women
Age (years)	1.00 (0.01)	1.01 (0.01)	0.80	0.26
BMI (kg/m^2)	0.97 (0.03)	0.99 (0.02)	0.27	0.81
Hypertension (mmHg)	2.81 (0.66)	1.59 (0.38)	<0.001	0.05
Linguistic region (French)	1	1		
Linguistic region (German)	1.81 (0.39)	1.56 (0.30)	0.01	0.02
Linguistic region (Italian)	1.09 (0.33)	1.27 (0.34)	0.78	0.36
Urinary Na excretion (mmol/24h)	1.00 (0.001)	1.00 (0.001)	0.53	0.80
Urinary K excretion (mmol/24h)	1.01 (0.001)	1.01 (0.01)	0.17	0.06
Urinary urea excretion (mmol/24h)	1.00 (0.001)	1.00 (0.001)	0.25	0.74
Urinary volume (ml/24h)	1.00 (0.001)	1.00 (0.001)	0.59	0.60
Urinary Creatinine excretion (mmol/kg/24h)	0.97 (0.03)	0.99 (0.3)	0.33	0.64
Attempt to limit salt consumption	0.98 (0.21)	1.16 (0.24)	0.94	0.48
Attention paid to salt consumption	1.45 (0.31)	1.49 (0.29)	0.09	0.04
Portions of fruits (portions/day)	0.98 (0.12)	1.01 (0.11)	0.85	0.93
Portions of vegetables (portions/day)	0.96 (0.14)	1.05 (0.12)	0.80	0.68
Quantity of meet (portions/day)	1.04 (0.06)	1.03 (0.05)	0.50	0.52
Quantity of fish (portions/day)	1.03 (0.10)	1.12 (0.10)	0.76	0.21

Data are odds ratio(SE) and P values.

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