Birth weight modifies the association between a healthy Nordic diet and office blood pressure in old age

Running head: Diet - blood pressure association by birth weight

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Abstract

A healthy diet reduces risk for high blood pressure. A small body size at birth increases risk for high blood pressure.

Our aim was to study whether birth weight modifies the association between a healthy Nordic diet, characterized by high intake of Nordic vegetables, fruits, and berries, whole-grain rye, oat, and barley, and rapeseed oil, and blood pressure.

Finnish men and women (n=960) born in 1934-1944 attended clinical visits including clinical measurements, and questionnaires in 2001-2004 and 2011-2013. Linear regression was applied to investigate the interactions between birth weight and Nordic diet (measured by the Baltic sea diet score, BSDS) on blood pressure change during the 10-year follow-up.

Baseline Nordic diet and birth weight showed a significant interaction on systolic blood pressure (SBP) (p =0.02), and pulse pressure (PP) (p <0.01) over a 10-year follow-up. In the lowest birth weight category (women <2951 g, men <3061 g), predicted SBP decreased across BSDS thirds (lowest (T1): 155 mmHg, highest (T3): 145 mmHg, p for linearity =0.01) as did predicted PP (T1: 71 mmHg, T3: 63 mmHg, p <0.01). In the middle birth weight category, predicted SBP increased across BSDS thirds (T1: 151 mmHg, ~~T2~~T3: 155 mmHg, p=0.02) as did predicted PP (T1: 67 mmHg, T3: 71 mmHg, p <0.01). In the highest birth weight category, no associations were found.

Higher adherence to a healthy Nordic diet was associated with lower SBP and PP in individuals with low birth weight but with higher SBP and PP in those with average birth weight.

Keywords: elderly, developmental origins of health and disease, fetal programming, dietary index, diet quality

Introduction

High blood pressure significantly reduces an individual's healthy life span and is a leading factor underlying the global disease burden [1]. The most important modifiable risk factors of high blood pressure are poor health behaviours including a high intake of sodium, overweight, physical inactivity, and smoking [2]. Individuals with low birth weight (birth weight) are susceptible to higher blood pressure from childhood throughout their life span [3,4]. Hypothesized mechanisms underlying the association between birth weight and blood pressure include a low nephron number [5-7], endothelial dysfunction, vascular remodelling [8], and nitric oxide (NO) – reactive oxygen species (ROS) imbalance.

Recent findings have suggested that the association between health behaviours and blood pressure is stronger among those born with a low birth weight. The association between adult BMI and blood pressure [9,10] as well as other cardiovascular outcomes [11,12] may be more pronounced in individuals born with low birth weight compared to those born with higher birth weight. Based on findings from the Nurses' Health Study II a combination of poor health behaviours (including diet, physical activity, alcohol consumption, non-narcotic analgesics, and BMI) together with low birth weight increased blood pressure more than the summarized individual increase in health behaviours and birth weight [13]. We have previously shown in Helsinki Birth Cohort Study that lower salt intake was associated with lower SBP only among those with low birth weight [14].

The healthy Nordic diet has been associated with blood pressure in clinical trials [15], and in epidemiological studies [16]. The healthy Nordic diet shares similar characteristics with the Dietary Approaches to Stop Hypertension (DASH) diet [17] but with emphasis on food culture and food availability in Nordic countries. This means special emphasis on Nordic foods such as Nordic vegetables, fruits and berries, whole-grain rye, oat, and barley, and rapeseed oil [18]. These factors form the basis of the Baltic sea diet score (BSDS).

This is the first study to prospectively assess whether the association between whole diet at baseline (years 2001-2004) and blood pressure at follow-up (years 2011-2013) is modified by body size at birth. Because of the study population, the most relevant diet to investigate was the healthy Nordic diet as measured by the BSDS.

Methods

Helsinki Birth Cohort Study includes men and women born as singletons in Helsinki in 1934–1944. Individuals who were alive in 1971 when a unique identification number when it was assigned to all Finns were included, in total 13 345 participants. From those who were born in the Helsinki University Central Hospital, a random sample to fulfill a target of 2000 were invited to a clinical examination in 2001–2004 (**Fig. 1**). The number of participants who attended the clinical visit in 2001-2004 was 2003 and in the follow-up visit (2011-2013) 1094. The number of participants with relevant data and whose gestational age was 259 days or more (i.e. who was not born pre-term), was 960. The authors assert that all procedures contributing to this work comply with the ethical standards of the Finnish National Board on Research Integrity and with the Helsinki Declaration of 1975, as revised in 2008, and has been approved by the Ethics Committee of the Helsinki and Uusimaa Hospital District. Informed consent was obtained from all participants.

Exposure variables were the healthy Nordic diet (i.e. BSDS), birth weight, and their interaction term. Outcome variables were SBP, diastolic blood pressure (DBP), pulse pressure (PP), and mean arterial pressure (MAP). Baseline covariates were age, sex, educational attainment (years), household income, gestational age, smoking (current smoker, former smoker, non-smoker), BMI, leisure-time physical activity (LTPA, times/week), energy intake, sodium intake, and mother’s pre-delivery BMI. Covariates including baseline and follow-up measures were use of blood pressure affecting medicine (yes/no), and weight change during follow-up. The effect modifier was birth weight.

Birth weight, gestational age, and mother’s pre-delivery BMI (measured when the mother entered the hospital to give birth) were collected from hospital birth records. Household income of the year 1995 was obtained from Statistics Finland. At baseline, participants filled in questionnaires, which provided data on educational attainment (years), LTPA (times per week), and smoking (current smoker, former smoker, non-smoker). LTPA was inquired as how many times per week on average the participant committed to moderate to vigorous physical activity during leisure time. Use of blood pressure affecting medication (yes / no), was asked at baseline and at follow-up.

At both clinical examinations, height and weight of the participants were measured to the nearest 0.1 cm, and nearest 0.1 kg, respectively, with the participant wearing light indoor clothing and without shoes. Before blood pressure measurement, the participants sat five minutes without talking. During the measurement the participants sat on a chair with their feet on the floor, legs uncrossed and had the arm at heart level. The recorded value was the mean of two readings of a standard sphygmomanometer (Omron Matsusaka Europe, Hoofdorp, the Netherlands) measured two minutes apart. Measurements were conducted by a trained study nurse. PP was calculated by equation $PP = SBP - DBP$, and MAP by equation $MAP = DBP + \frac{1}{3} \times (SBP - DBP)$.

<u>Dietary assessment</u>

At baseline (years 2001-2004), the participants completed a validated, semi-quantitative 128-item food-frequency questionnaire (FFQ) covering 12 food groups [19,20]. The reference period was the previous 12 months. In the questionnaire, number of frequency options of intake was nine, and they ranged from never or seldom to 6 or more times per day. After completing the questionnaire it was checked at the study center by a study nurse. The data were entered and processed at the National Institute for Health and Welfare with use of the National Food Consumption Database FINELI (National Institute for Health and Welfare, Helsinki, Finland) [21].

BSDS consists of eight components [22]. For Nordic fruits (apples, pears, and berries), Nordic vegetables (tomatoes, cucumbers, leafy vegetables, roots, cabbages, peas), Nordic cereals (rye, oat, and barley), Nordic fish (salmon and freshwater fishes), low-fat and fat-free milk, ratio of polyunsaturated fatty acids to saturated fatty acids (SFA) and trans-fatty acids, intake above the sex-specific quartiles served as cut-offs. Sex-specific quartiles assigned 0-3 points from lowest to the highest quartile. For red and processed meat, and total fat the quartiles were scored in reverse order. The final BSDS ranged from 0 to 24. Higher score indicates higher adherence to the Nordic diet. Before the scoring, the BSDS components were energy-adjusted using a density method [23].

Statistics

The descriptive results are presented as means/medians with standard deviations/interquartile ranges (IQR), or frequencies with proportions. Birth weight was divided in categories using two sex-specific cut-offs; mean + SD, and mean – SD, which generated three categories (birth weight I: <2951 g in women, <3061 g in men; birth weight II: 2951 – 3810 g in women, 3061-3959 g in men; and birth weight III: > 3810 in women, > 3959 g in men). BSDS was divided by sex-specific tertile cut-offs. For continuous variables, linearity of continuous variables across birth weight categories was tested by ANOVA, and linear contrasts were set to birth weight categories. For the ordinal variable BSDS, trend across birth weight categories was tested by Cuzick trend test. For categorical variables linearity across birth weight categories were tested by Cochran-Armitage trend test. Interaction of birth weight and BSDS on SBP, DBP, PP, and MAP were studied by linear regression analysis, where BSDS, birth weight, and their interaction term were included as independent variables, and SBP, DBP, PP, or MAP as outcome variables Because there was no additional interaction with sex on blood pressure parameters, men and women were analyzed together. The crude model included only energy intake as a covariate. The fully adjusted model additionally included baseline blood pressure parameters (SBP, DBP, PP, or MAP), age, sex, educational attainment, household income, gestational age, smoking, LTPA, BMI, weight change during follow-up, sodium intake, and use of blood-pressure affecting medication at baseline. Additional adjustment for alcohol intake did not change the results. Data on blood pressure affecting medication at follow-up (2011-2013) was missing for 137 participants, and maternal pre-delivery BMI was missing for 118 participants. These variables were therefore omitted from the main analysis. However, we performed sensitivity analyses with data on imputed blood pressure affecting medication and maternal pre-delivery BMI. Independent variables in the multiple imputation (10 imputations) model included age, sex, educational attainment, household income, gestational age, smoking, LTPA, BMI, weight change during follow-up, energy intake, BSDS, sodium intake, blood pressure parameters at baseline and at follow-up, and use of blood-pressure affecting medication at baseline. In addition to missing data for blood-pressure affecting medication and maternal pre-delivery BMI, four missing values for smoking and one for household income were imputed simultaneously. Description of examination of the missing data pattern is presented in Supplemental Tables S1 and S2.

Because the covariates associated with missingness of blood-pressure affecting medication at follow-up and maternal pre-delivery BMI could be included as independent variables in the imputation model, the assumption of missing at random was held. Diagnostics of observed, imputed, and completed data are presented in Supplemental Figures S3 and S4. Normality of the variables was approximated from histograms and homogeneity of variance was tested by Bartlett's test. All analyses were performed using STATA 14.1 (StataCorp, 4905 Lakeway Dr, College Station, TX, USA).

Results

Participants attending follow-up examinations had longer duration of education (13 vs. 12 years), were younger at baseline (61 vs. 62 years), had higher birth weight (3441 vs. 3381 g), were leaner (BMI 27 vs. 28 kg/m^2), were less likely to smoke at baseline (19 vs. 29%), had lower energy intake (2200 vs. 2280 kcal/d), and had higher BSDS (13 vs. 12 points) compared with those who did not participate (p for all<0.05).

Characteristics of participants are presented in **Table 1**. At baseline, linearity across birth weight categories was seen for SBP (p for linearity=0.03), PP (p for linearity <0.01), and use of blood pressure lowering medication (p for linearity p<0.01) (**Table 2**). At baseline in a cross-sectional analysis, no significant association of BSDS and blood pressure parameters by birth weight categories were observed (Supplemental Table S5).

Change of BSDS from baseline to follow-up in birth weight categories from the lowest to the highest were 1.2 points (SD 4.3), 1.0 points (SD 4.1), 0.8 points (SD 3.9), respectively (p for linearity=0.29). At follow-up, no significant difference was observed in blood pressure parameters or change in blood pressure affecting medication during follow-up across birth weight categories (Table 2). In a longitudinal analysis, the association of baseline BSDS and SBP by birth weight was statistically significant in both the crude model (adjusted for energy intake only) (p for interaction BSDS x birth weight = 0.01), and in the fully adjusted model (p (BSDS x birth weight) = 0.02) (Fig. 2). Similarly, the association between BSDS and PP by birth weight was significant in the crude model (p (BSDS x birth weight) = 0.03) and in the fully adjusted model (p (BSDS x birth weight) <0.01). A projection of linear predictions of SBP across birth weight deciles in thirds of the BSDS suggests that the turning point for the association was approximately 3200 g (Supplemental Figure S8). No association between BSDS and DBP by birth weight or BSDS and MAP by birth weight was found.

In birth weight i (lowest birth weight category) predicted SBP in the lowest BSDS third (T1) was 155 mmHg (95% CI: 150, 160) and in the highest BSDS third (T3) 145 mmHg (95% CI: 139, 151) (adjusted p for linearity across BSDS thirds =0.01). Predicted PP in T1 was 71 mmHg (95% CI: 67, 75) and in T3 63 mmHg (95% CI: 58, 68) (p for linearity <0.01) (Fig. 2). In the middle birth weight group (birth weight II), predicted SBP in T1 was 151 mmHg (95% CI: 148, 153) and in T3 155 mm Hg (95% CI: 153, 158) (p for linearity=0.02). Predicted PP in T1 was 67 mmHg (95% CI: 65, 68) and in T3 71 mmHg (95% CI: 69, 73) (adjusted p for linearity<0.01). In the highest birth weight group (birth weight III), no associations were found between BSDS and SBP or BSDS and PP.

Sensitivity analyses

The association between BSDS and blood pressure parameters by birth weight adjusted for change in blood-pressure affecting medication during follow-up and maternal pre-delivery BMI with missing data as well as with imputed data are presented in Supplemental Table S6. Inclusion of imputed covariates did not substantially change the effect estimates or p-values of interactions of birth weight and BSDS on blood pressure or effect estimates and confidence intervals of BSDS – blood pressure association in birth weight categories.

Components of BSDS

From individual components of BSDS, in birth weight I higher intake of fruits, fish, and cereals were associated with lower SBP (all p-values for linearity across quartiles of intake <0.05) (Supplemental Table S7). Higher intake of fruits and fish and lower intake of meat were associated with lower PP (p for linearity < 0.05). In birth weight II, higher intake of milk (p for linearity ≤ 0.05) was associated with higher SBP and PP, and higher intake of vegetables (p for linearity = 0.03) was associated with higher PP.

Discussion

In the present study, participants belonging to the lowest birth weight category (<2951 g in women, <3061 g in men), and with a higher adherence to a healthy Nordic diet had lower SBP and PP over a 10-year follow-up. In participants in the middle birth weight category (in women 2951–3810 g, in men 3061-3959 g), higher adherence to a healthy Nordic diet was associated with higher SBP and PP during follow-up. In participants with high birth weight (in women > 3810 g, in men > 3959 g) no associations ~~of~~ between the Nordic diet and SBP or PP were found. No association between the healthy Nordic diet and DBP or MAP by birth weight categories were found.

Thus far, to the best of our knowledge the only studies assessing an interaction between diet and birth weight on blood pressure have been in relation to salt intake [14,24,25]. A recent work based on findings in Helsinki Birth Cohort Study revealed that salt intake was associated with higher SBP only among those who were born with low birth weight, not among persons with higher birth weight [14]. This is in line with our finding of different response to dietary factors of those born with low birth weight. Due to lack of other diet-birth weight-interaction studies, studies focusing on adiposity and birth weight interaction on blood pressure are the closest relevant comparisons. Some, but not all, such studies suggest that the association between BMI and blood pressure is more pronounced in those born small [10,26,27]. In a recent study including a 20-year follow-up of the Nurses' Health Study II, Li et al. (2015) found that non-optimal lifestyle-related behaviours (combination of diet, physical activity, alcohol consumption, non-narcotic analgesics, and BMI) in combination with low birth weight increased hypertension above what each component (unhealthy behaviours or low birth weight) did independently [13]. Our results are in line with previous studies that suggest that the association between healthy lifestyles and blood pressure depend on birth weight.

In the present study among individuals with lower birth weight, an increase from the lowest to the highest third of BSDS equalled approximately -10 mmHg difference in SBP, and approximately -8 difference in PP. A meta-analysis of individual data from 61 prospective studies evidenced that a 2 mmHg decrease in SBP could cause a 10% reduction in stroke mortality and a 7% reduction in mortality due to vascular causes [28]. Another meta-analysis including three large cohorts showed that a 10 mmHg wider PP resulted in a 13% increase in risk of any coronary end point, and a 20% increase in cardiovascular mortality [29]. Thus, among individuals with low birth weight, a healthy diet, could have a lowering impact on cardiovascular mortality through lowering both SBP and PP.

Why would a Healthy Nordic diet act more effectively in persons with lower birth weight? One possible mechanism relates to nitric oxide – reactive oxygen species (NO-ROS) imbalance. Both compounds, NO and ROS, regulate endothelial function and vascular tone. Individuals with low birth may possess chronically low availability of endogenous NO [30]. This condition could create an increased affinity to dietary nitrate as well as for dietary compounds enhancing conversion of nitric compounds to NO. Such compounds are abundant in fruits and berries (vitamin C [31], folate [32], and polyphenols [33]), and in certain fish species (docosahexaenoic acid (DHA) [34]). Nutrients such as heme iron of red meat may accelerate ROS production [35], whereas antioxidants of fruits, berries, and vegetables may scavenge ROS, thereby alleviating its looping effect on NO-ROS imbalance. Several dietary components involved in the above mechanisms are also components of BSDS and were associated with SBP and PP in those born small. Other constituents of the Healthy Nordic diet through other mechanisms and possible mechanistic interactions may have also affected SBP and PP of individuals with born with a low birth weight. We have no explanation for the association between the Healthy Nordic diet and higher SBP and PP in those with average birth weight or for the lack of association in those with high birth weight.

The study has several strengths. The longitudinal setting and well-characterized study population are advantages. In addition, the validated FFQ used ~~in~~ at baseline and follow-up ensures good quality dietary data. Some limitations also need to be mentioned. Self-reported dietary intake and physical activity data may not be as accurate as those measured objectively. We cannot exclude the possibility of birth weight being a proxy of a maternal behaviour or condition during pregnancy. We have tried to address maternal health behaviours by adjustment for maternal pre-delivery BMI, which did not substantially change the results. Some residual confounding may be present because of possible unknown relevant confounders. Measurement of blood pressure only in a clinical setting is not as accurate as an ambulatory measurement over a longer time period. The differences in education, health behaviours, and blood pressure at baseline as well as birth weight between the participants and non-participants of the follow-up suggest that the study group does not optimally represent the overall HBCS cohort.

According to our results, beneficial effects on SBP and PP that can be obtained through a healthy diet and the effects are largest among those with a low birth weight. More studies focusing upon the association between dietary patterns and blood pressure by birth weight are needed. In addition, possible mechanisms behind associations and lack of association in different birth weight categories should be studied.

Availability of the data

The dataset and code supporting the conclusions of this article is available upon a reasonable request from the authors.

Conflicts of interest

The authors have no conflicts of interest.

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Table 1. Characeristics of the participants at baseline (2001-2004).

	Birth weight categories								
	I (n=142)		II (n=656)		III (n=162)		p for linearity *	All (n=960)	
	n	%	n	%	n	%		n	%
Women, n (%)	88	62	355	54	98	60	0.88	541	56
	Mean	SD	Mean	SD	Mean	SD		Mean	SD
Age, y	62	2	61	3	61	3	0.08	61	3
Education, y	13	4	13	4	12	3	0.28	13	4
Household income, Finnish marks/year‡	60900	53200	64100	67400	62600	48900	0.32	63400	62600
BMI, kg/m^2	27	4	27	4	28	5	<0.01	27	4
Birth weight, g	2776	184	3423	238	4096	230	<0.001	3441	437
Gestational age, d	275	11	281	10	285	9	<0.001	281	10
Fasting plasma glucose, mmol/l	5.7	1.0	5.7	1.1	5.6	0.9	0.20	5.7	1.1
Fasting plasma insulin, mmol/l	10.8	12.0	9.5	8.1	10.9	15.4	0.18	9.9	10.3
Plasma total cholesterol, mmol/l‡	5.9	1.1	5.9	1.0	5.8	1.0	0.64	5.9	1.0

Plasma triglycerides, mmol/l‡	1.5	0.7	1.4	0.8	1.3	0.6	0.09	1.4	0.7
Plasma HDL, mmol/l‡	1.6	0.4	1.6	0.4	1.6	0.5	0.91	1.6	0.4
Plasma LDL, mmol/l‡	3.6	0.9	3.6	0.9	3.6	0.8	0.70	3.6	0.9
Nutrition									
BSDS, points (range 0-24) (median, IQR)	11	6	12	6	12	5	0.10	12	6
Nordic fruits and berries g/d	132	172	136	149	141	132	0.66	136	150
Nordic vegetables g/d	265	187	268	163	285	179	0.91	271	169
PUFA/SFA ratio %	44	15	44	13	46	14	0.25	45	13
Cereals g/d	57	41	58	36	57	35	0.95	58	36
Meat g/d	107	87	110	92	104	78	0.41	108	89
Fish g/d	51	60	47	42	51	60	0.80	48	49
Fat E%	33	6	33	5	32	5	0.16	33	5
Milk g/d	174	221	198	236	201	247	0.40	195	236
Energy, kJ/d	9120	3420	9210	3100	9320	3350	0.58	9210	3190
Sodium, mg/d	3430	1510	3450	1280	3450	1290	0.37	3450	1320
Potassium, mg/d	4780	2000	4820	1590	4810	1750	0.53	4810	1680
	n	%	n	%	n	%		n	%

Leisure-time physical activity							0.76		
Less than once a week	15	11	79	12	13	8		107	11
1-3 times/week	93	65	421	64	111	69		625	65
4 times/week or more	34	24	156	24	38	23		228	24
Smokers‡									
Non-smoker	63	45	313	48	80	50	0.62	456	48
Former smoker	53	37	214	32	53	33		320	33
Smoker	26	18	128	20	26	17		180	19
Diabetes medication users	10	7	30	5	5	3	0.11	45	5
Participants with diabetes mellitus	8	6	39	6	6	4	0.44	53	6
Participants with experience of									
myocardial infarction	3	2	13	2	1	1	0.31	17	2
angina pectoris	11	8	30	5	6	4	0.11	47	5
stroke	2	1	7	1	0	0	0.19	9	1

BMI, body mass index, BSDS, Baltic sea diet score; E%, percentage of total energy intake. * ANOVA with linear contrasts (continuous variables), Cuzick trend test (ordinal variable BSDS), or Cochran-Armitage test for trend (categorical variables). †Energy intake adjusted for by including energy intake as an independent variable in a multivariate model (BSDS) and by dividing intake by total energy intake (foods) and residual method (sodium). ‡Missing values:

household income n=50, cholesterol n=1, triglycerides n=1, HDL n=1, LDL n=1, smoking n=4.

Birth weight categories: BW I: <2951 g in women, <3061 g in men; BW II: 2951 – 3810 g in women, 3061-3959 g in men; and BW III: > 3810 in women, > 3959 g in men.

Table 2. Blood pressure parameters and use of blood pressure affecting medication at baseline (2001-2004) and change in use of blood pressure affecting medication at follow-up (2011-2013).

	Birth weight categories								
	I (n=142)		II (n=656)		III (n=162)			All (n=960)	
	mean	sd	mean	sd	mean	sd	p for linearity	mean	sd
Baseline									
Systolic blood pressure, mm Hg	146	19	143	20	141	19	0.030	143	20
Diastolic blood pressure, mm Hg	89	9	88	10	89	11	0.84	88	10
Pulse pressure	57	16	55	15	52	14	<0.01	55	15
Mean arterial pressure	108	11	106	12	106	13	0.27	107	12
	n	%	n	%	n	%		n	%
Users of blood pressure affecting medication	54	38.0	213	32.0	38	23.0	<0.01	305	32.0
	mean	sd	mean	sd	mean	sd		mean	sd
Follow-up									
Systolic blood pressure, mm Hg	152	22	153	22	149	22	0.23	152	22
Diastolic blood pressure, mm Hg	84	10	84	11	84	11	0.90	84	10
Pulse pressure	69	18	69	18	66	17	0.11	68	17

Mean arterial pressure	107	12	107	13	106	13	0.54	107	13
	n	%	n	%	n	%		n	%
Change in use of blood pressure affecting medication during follow-up†									
total number of analyzed participants	125	100.0	558	100.0	140	100.0	0.27	823	100.0
stopped during follow-up	2	2	10	2	1	1		13	2
no change	78	62	399	72	99	71		576	70
started during follow-up	45	36	149	27	40	29		234	28

†Missing values 137. Birth weight categories: BW I: <2951 g in women, <3061 g in men; BW II: 2951 – 3810 g in women, 3061-3959 g in men; and BW III: > 3810 in women, > 3959 g in men. *ANOVA with linear contrasts (continuous variables) or Cochran-Armitage test for trend (categorical variables).

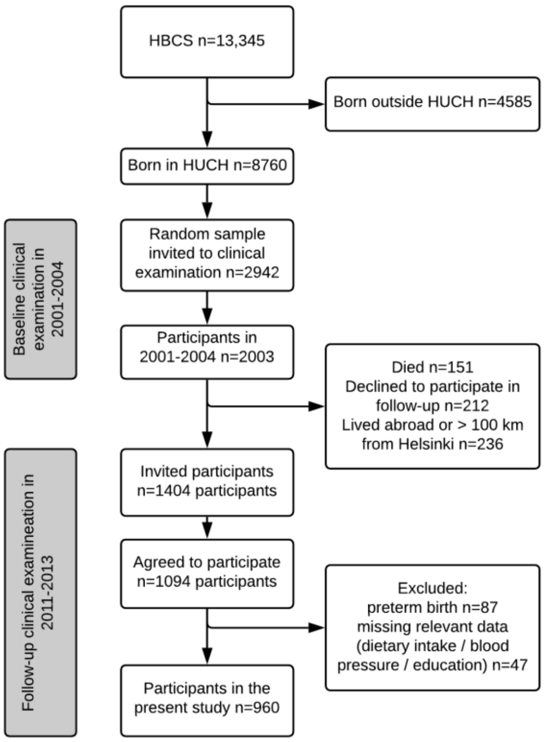

Figure 1. Selection of the participants of the present study from the original cohort of Helsinki Birth Cohort Study.

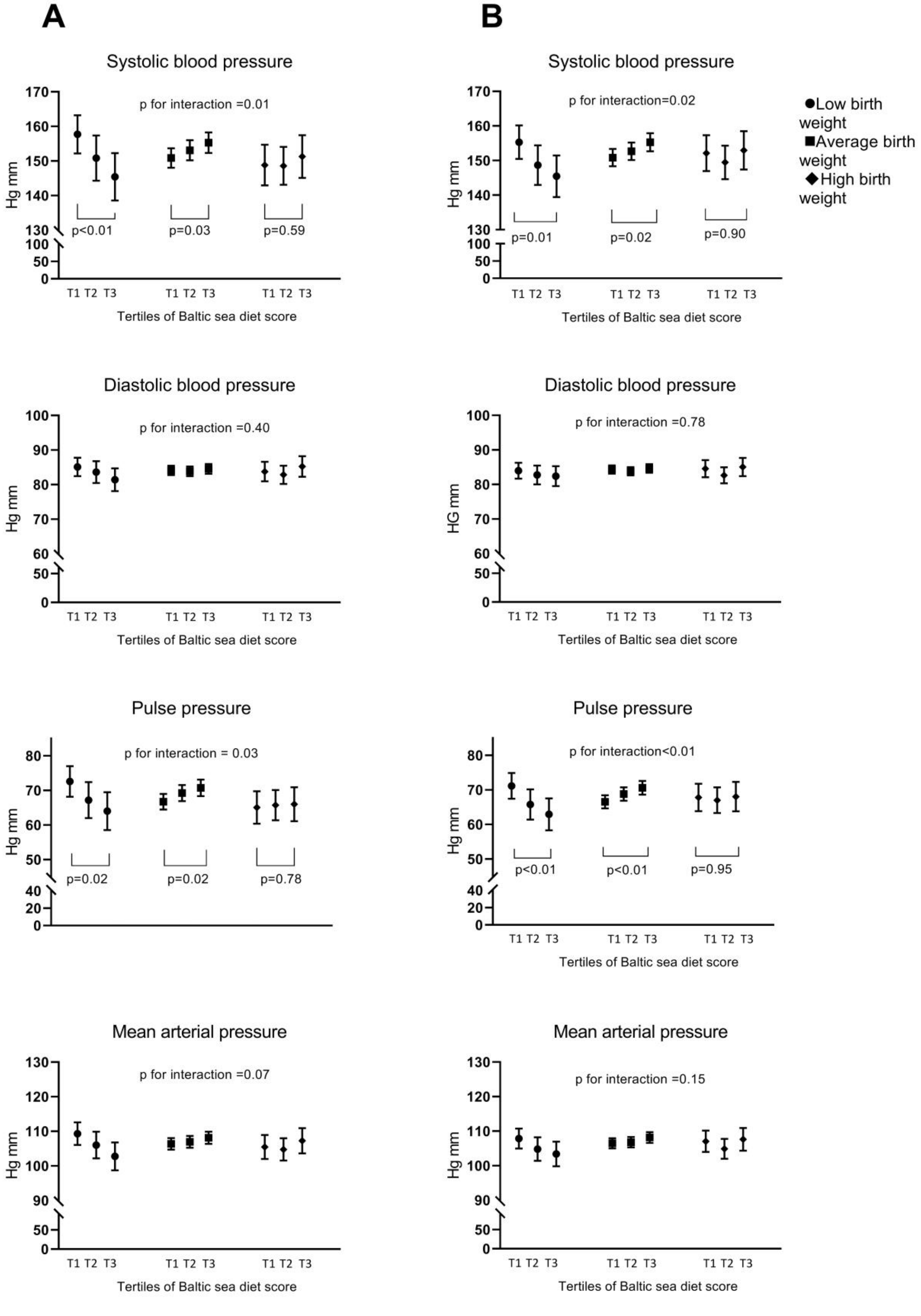

Figure 2. Systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse pressure (PP), and mean arterial pressure (MAP) at follow-up (2011-2013) in thirds of Baltic Sea Diet Score (2001-2004)

(T1, T2, and T3) by birth weight categories (birth weight I: <2951 g in women, <3061 g in men; birth weight II: 2951 – 3810 g in women, 3061-3959 g in men; and birth weight III: > 3810 in women, > 3959 g in men) as well as p-values for an interaction term BSDS x birth weight, and main effects of BSDS in each birth weight category. Analyses of column A was adjusted for bl (baseline, 2001-2004) energy intake and column B additionally for bl age, sex, educational attainment, household income, bl SBP / DBP / PP / MAP, gestational age, bl smoking, bl leisure-time physical activity, bl BMI, weight change during follow-up, bl sodium intake, and bl use of blood pressure affecting medication. For multivariate analyses (column B) the number of participants was 955 because data on smoking was missing from 4 participants, and data on household income for 1 participant.