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Cross-Sectional and Longitudinal Correlates of Home Blood Pressure

Sam Sivén



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CROSS-SECTIONAL AND LONGITUDINAL CORRELATES OF HOME BLOOD PRESSURE

Sam Sivén

University of Turku

Faculty of Medicine
Department of Clinical Medicine
Internal Medicine
Doctoral Programme in Clinical Research

Supervised by

Professor Teemu Niiranen, MD, PhD
Internal Medicine
Department of Clinical Medicine
Faculty of Medicine
University of Turku
Turku, Finland

Research Professor Antti Jula, MD, PhD
Department of Chronic Disease Prevention
Finnish Institute for Health and Welfare
Turku, Finland

Reviewed by

Professor Pekka Mäntyselkä, MD, PhD
General Practice
Faculty of Health Sciences
University of Eastern Finland
Kuopio, Finland

Adjunct Professor Jenni Koskela, MD, PhD
Faculty of Medicine and Health Technology
Tampere University
Tampere, Finland

Opponent

Associate Professor Maria Hagnäs, MD, PhD
Faculty of Medicine and Health Technology
Tampere University
Tampere, Finland

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To my family

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ABSTRACT

Hypertension remains a major public health concern worldwide, yet disparities in detection, management, and outcomes persist. In addition, the significance of home blood pressure (BP) as a predictor of future hypertension and hypertension-mediated organ damage (HMOD) in the general population remains unclear.

The objectives of this thesis were to examine the association of demographics and social inequalities with hypertension care assessed with office and home BP measurements, and the longitudinal associations of home versus office BP measurements with future hypertension and HMOD in the general population. Data from a Finnish population-based cohort, Health 2000, and its re-examination, Health 2011 was utilised. Home BP measurements were performed in a subsample of both cohorts (the Finn-Home Study).

Study I revealed that lower education, male sex, smoking, obesity, and physical inactivity were associated with poorer management and control of hypertension. The observed prevalence and control rates of office hypertension were verified with home BP. Study II demonstrated that both white-coat and masked hypertension significantly increased the risk of developing sustained hypertension, with masked hypertension showing the strongest association. Studies III and IV uncovered that home BP correlated more closely with the development of left ventricular hypertrophy and kidney disease than office measurements, indicating better predictive value for HMOD.

Together, these studies highlight two critical dimensions of hypertension research: first, the persistent social and demographic inequalities that hinder equitable hypertension care, and second, the superior prognostic accuracy of home BP monitoring for identifying individuals at risk of sustained hypertension, cardiac hypertrophy, and renal impairment. The findings advocate for broader implementation of home BP monitoring in clinical and public health practice, alongside targeted interventions to reduce disparities in hypertension management.

KEYWORDS: Home blood pressure, epidemiology, cardiovascular disease

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TIIVISTELMÄ

Kohonnut verenpaine on edelleen merkittävä kansanterveydellinen huolenaihe maailmanlaajuisesti, ja sen toteamisessa, hoidossa ja hoitotuloksissa esiintyy yhä eriarvoisuutta. Lisäksi kotona mitatun verenpaineen yhteys tulevaan verenpainetautiin ja verenpainetaudin aiheuttamiin elinvaurioihin väestötasolla on epäselvä.

Tämän väitöskirjan tavoitteena oli tutkia demografisten muuttujien ja sosiaalisen eriarvoisuuden yhteyttä verenpainetaudin hoitoon, kun verenpainetta arvioidaan koti- ja vastaanottomittauksin, sekä tarkastella kotona ja vastaanotolla mitatun verenpaineen yhteyttä tulevaan verenpainetautiin ja elinvaurioihin väestötasolla. Aineistona käytettiin suomalaista väestöpohjaista kohorttia Terveys 2000 -tutkimusta sekä sen uusintatutkimusta Terveys 2011:sta. Lisäksi osa osallistujista mittasi myös kotiverenpaineensa (Finn-Home-tutkimus).

Tutkimus I osoitti, että matalampi koulutustaso, miessukupuoli, tupakointi, lihavuus ja fyysinen passiivisuus olivat yhteydessä verenpainetaudin heikompaan hoitoon ja huonompaan hoitotasapainoon. Kotimittaukset tukivat näitä löydöksiä. Tutkimus II osoitti, että sekä valkotakkiverenpaine että piilevä verenpainetauti lisäsivät merkittävästi pysyvän verenpainetaudin riskiä. Tutkimuksissa III ja IV havaittiin, että kotiverenpaine korreloi vastaanottopainetta vahvemmin vasemman kammion liikakasvun ja munuaissairauden kehittymisen kanssa, mitä voisi hyödyntää verenpainetaudin aiheuttamien elinvaurioiden ennaltaehkäisyssä.

Yhteenvetona tutkimukset painottavat verenpainetutkimuksen kahta keskeistä näkökulmaa: verenpainetaudin huonompaan hoitoon yhdistettyä sosiaalista ja demografista eriarvoisuutta, sekä kotiverenpainemittausten vahvaa yhteyttä pysyvän verenpainetaudin, sydänlihaksen paksuuntumisen ja munuaisvaurion kehittymiseen. Tulokset tukevat kotiverenpainemittausten käytön laajentamista sekä kohdennettuja toimia erojen vähentämiseksi verenpainetaudin hoidossa.

AVAINSANAT: kotiverenpaine, epidemiologia, sydän- ja verisuonitaudit

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Abbreviations

ACC/AHA	American College of Cardiology/American Heart Association
BMI	Body mass index
BP	Blood Pressure
ECG	Electrocardiography
ECG-LVH	Electrocardiographically assessed left ventricular hypertrophy
eGFR	Estimated glomerular filtration rate
HMOD	Hypertension-mediated organ damage
LVH	Left ventricular hypertrophy

List of Original Publications

This dissertation is based on the following original publications, which are referred to in the text by their Roman numerals:

- I Sivén SS, Niiranen TJ, Aromaa A, Koskinen S, Jula AM. Social, lifestyle and demographic inequalities in hypertension care. *Scandinavian Journal of Public Health*, 2015 May;43(3):246–253.
<https://doi.org/10.1177/1403494815571031>
- II Sivén SSE, Niiranen TJ, Kantola IM, Jula AM. White-coat and masked hypertension as risk factors for progression to sustained hypertension: the Finn-home study. *Journal of Hypertension*, 2016 Jan;34(1):54–60.
<https://doi.org/10.1097/HJH.0000000000000750>
- III Sivén SSE, Niiranen TJ, Langén VLJ, Puukka PJ, Kantola IM, Jula AM. Home versus office blood pressure: longitudinal relations with left ventricular hypertrophy: the Finn-Home study. *Journal of Hypertension*, 2017 Feb;35(2):266–271.
<https://doi.org/10.1097/HJH.0000000000001152>
- IV Sivén SS, Langén VL, Puukka P, Sundvall J, Kantola IM, Jula AM, Niiranen TJ. Home and office blood pressure measurements as determinants of kidney disease in the general population: The Finn-Home Study. *European Journal of Preventive Cardiology*, 2019 Jan;26(2):208–210.
<https://doi.org/10.1177/2047487318784367>

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1 Introduction

During the early 20th century, elevated blood pressure (BP), later termed hypertension, was identified as a potential leading cause of cardiovascular disease. In the 1920s, the first large observational studies on the risks of hypertension were performed by the insurance industry, which collected information on factors associated with future cardiovascular disease events.¹ During the early 1960s, epidemiological cohort studies confirmed the role of hypertension as a leading risk factor for myocardial infarction.² Only a few years later, in 1967, pharmacological therapy of hypertension was proven effective in reducing mortality and morbidity.³ During the latter part of the 20th century, hypertension awareness and control increased, while numerous pharmaceutical trials examined the comparability of outcomes with the use of different classes and combinations of novel antihypertensive drugs.⁴

Hypertension is the leading global burden of disease risk factor, and it was the attributable cause of 10.8 million (19.2% of all) deaths in 2019.⁵ Despite the advances in the prevention, detection, and treatment of hypertension, its prevalence among adults worldwide remains high – approximately 32% in women and 34% in men.⁶ The overall prevalence of hypertension remains high also in Finland. In 2023, approximately half of the Finnish adult population has elevated BP or is on antihypertensive medication, with prevalence rates rising steadily with age, from one in five men and one in ten women aged 20–39 years to nearly 86% of women and 87% of men aged 75 years and older.⁷ In addition to its high prevalence, the awareness and control rates of hypertension are still suboptimal both globally and in Finland.^{6,8} To target healthcare resources most efficiently and cost-effectively, it would be of great importance to improve policymakers' and physicians' awareness of the socio-demographic and lifestyle factors associated with the prevalence, awareness, treatment and control of hypertension.⁹

In addition to recognizing hypertension as an important risk factor, BP measurement has developed significantly over the past century, since the first invasive BP measurements by Stephen Hales in 1733.⁴ These improvements include, but are not limited to, the first sphygmomanometer in 1855, the use of an inflatable cuff for arterial occlusion by Scipione Riva-Rocci, and the use of a stethoscope for arterial sound auscultation from the compressed artery by Nikolai Korotkoff in 1905.⁴

Auscultation of the Korotkoff sounds at the doctor's or nurse's office remained the primary method of measuring BP throughout the 20th century. However, these measurements could only be made at the nurse's or doctor's office, which made BP assessment difficult.¹

Office BP measurements remained the gold standard until the advent of automatic home BP monitors based on the oscillometric technique in the 1970s.¹⁰ However, the introduction of home measurements resulted in a new diagnostic problem, as the simultaneous use of office and home BP measurement often leads to conflicting BP classifications. For example, a patient's BP may be elevated at the office but normal at home. This phenomenon is known as white-coat hypertension. This white-coat phenotype has traditionally been considered to be low-risk, although limited recent data suggest that this may not be the case.^{11,12} The opposite phenomenon of elevated BP at home, but normal BP at the office is called masked hypertension.^{11,12}

Home BP has been shown to be superior to office BP in predicting cardiovascular disease events.¹³ Furthermore, home BP measurements have been proven to associate cross-sectionally more closely with hypertension-mediated end-organ damage (HMOD), such as left ventricular hypertrophy (LVH) or albuminuria, compared to office BP measurements.¹⁴ However, the longitudinal associations between home BP and HMOD remain scarcely studied.

Home BP measurement is currently widely recommended and used in clinical practice.¹⁵⁻¹⁷ Nevertheless, certain aspects of home BP measurement remain understudied. The overarching aim of this thesis was, therefore, to compare home and office BP measurements in assessing the prevalence, awareness, treatment, and control of hypertension and their correlates, in estimating the risk of progression to sustained hypertension, and in predicting longitudinal HMOD changes.

2 Review of the Literature

2.1 Global Burden of Hypertension

On a global scale, the prevalence of hypertension is high and shows an upward trend. A pooled analysis of 1,201 population-representative studies including 104 million participants estimated that, in 2019, 626 million women and 652 million men aged 30–79 had hypertension. Notably, approximately 82% of all individuals with hypertension – over one billion people – lived in low- and middle-income regions.⁶ As a leading risk factor for cardiovascular disease, hypertension is responsible for a large share of the global burden of ischaemic heart disease and stroke, which have a global prevalence of 315 million and 87 million, respectively.^{18,19} Stroke is the second-leading cause of death and the third-leading cause of death and disability combined,²⁰ while ischaemic heart disease had the highest global age-standardised, disability-adjusted life-years of all diseases.¹⁹ The percentage of disability-adjusted life-years attributable to high systolic BP in the general population of each country in 2019 is depicted in **Figure 1**. These figures highlight the pivotal role of hypertension in driving cardiovascular morbidity and mortality worldwide.

As the global population grows – projected to approach 10 billion by 2059 – and life expectancy increases, the burden of cardiovascular disease attributable to hypertension will remain high, particularly in low- and middle-income countries, where hypertension prevalence is rising and healthcare systems face increasing strain.^{19,21–23} Non-communicable diseases are estimated to result in global losses exceeding 600 billion USD in national income. Among these conditions, cardiovascular diseases account for the largest share of healthcare expenditure in most countries – for example, 12% of the healthcare budget in the European Union – representing a substantial financial burden both nationally and globally.²⁴

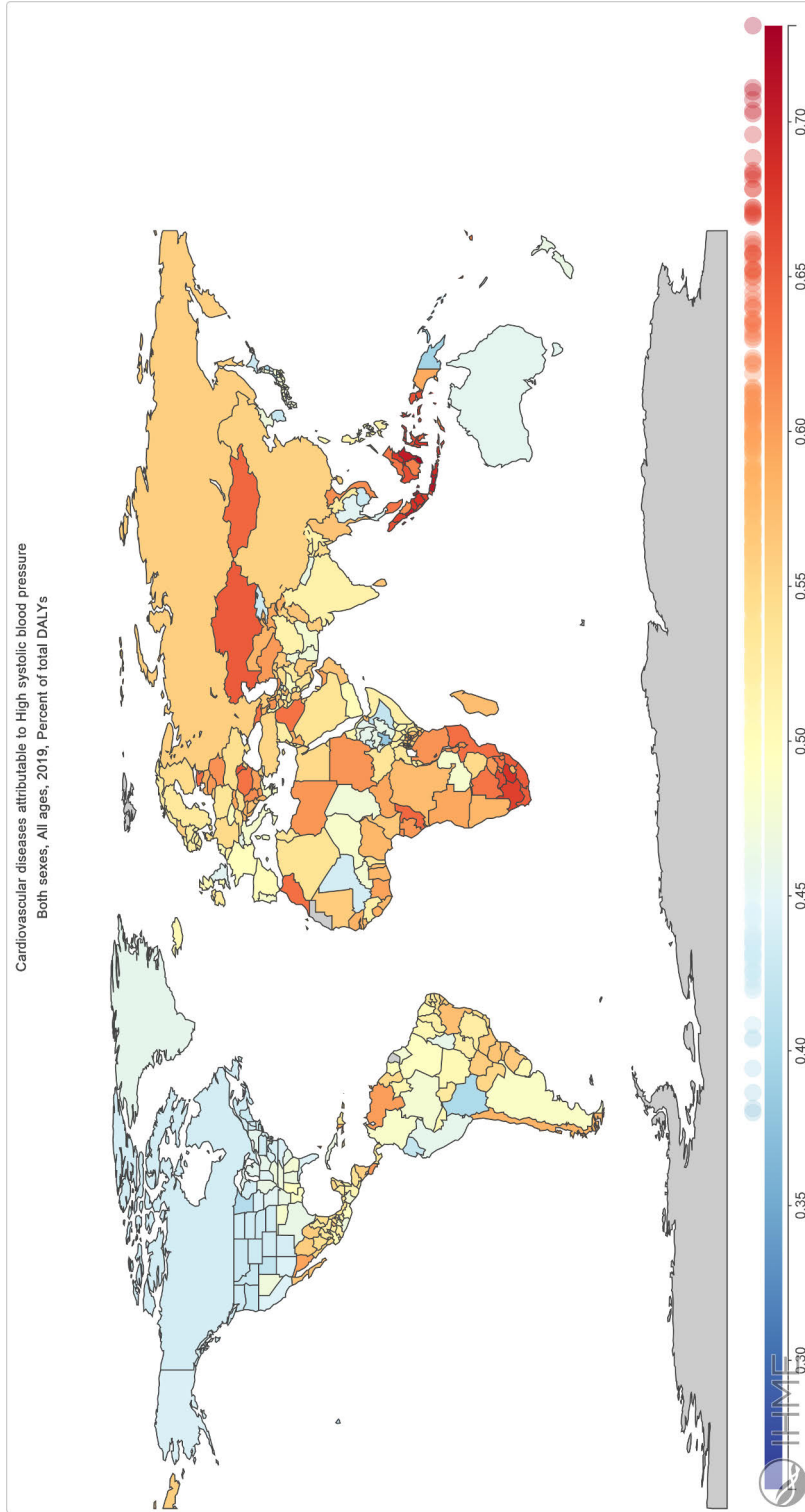


Figure 1. The percentage of total disability-adjusted-life-years (DALYs) attributable to high systolic BP in the general population of each country in 2019. Produced from data provided by the Institute for Health Metrics and Evaluation (IHME), Hans Rosling Center for Population Health, Seattle, WA, USA) in accordance with Creative Commons BY-NC-ND 4.0 license. Data available at <https://www.healthdata.org>.

Table 1. Prevalence, Awareness, Treatment, and Control of Hypertension Across Countries.

STUDY REGION	COUNTRY	AUTHOR, YEAR	SAMPLE SIZE	PREVAL. (%)	AWAREN. (%)	TREATM. (%)	CONTROL (%)
L	Finland	Kastarinen, 2009 †‡§	2,925	52.1 (m) 33.6 (w)	63.3 (m) 75.0 (w)	49.4 (m) 57.7 (w)	33.8 (m) 39.9 (w)
N	Finland	Koponen, 2018	5,534	57.6 (m) 48.3 (w)	NA	52.0 (m) 55.8 (w)	42.5 (m) 41.8 (w)
N	Portugal	Macedo, 2007 ¶	5,023	42.1	45.7	38.9	45.5
N	Czech	Cifková, 2010 ‡§	2,246	43.6	69.7	58.5	42.1
N	Turkey	Sengul, 2016	5,437	45.4	54.7	47.4	53.9
N	USA	Ong, 2007 ¶	4,872	45.5	66.5	53.7	63.9
N	Canada	Gee, 2012 ¶	3,473	45.6	82.5	78.9	78
N	USA	Guo, 2012 ¶	5,764	45.4	74	71.6	64.4
L	Israel	Abu-Saad, 2014	763	41.3	66	53.3	38.7
L	Iran	Malekzadeh, 2013	50,000	42.7	46.2	45.5	32.1
N	Nigeria	Ekunwufe, 2010	756	45.3	30	21	9
N	West Bank	Khdour, 2013	2,077	28	51	40	33
L	China	Ke, 2014 ¶	1,410	34	67	59	49
L	China	Wang H., 2013 ¶	17,400	45.5	54.3	46.3	39.4 †
N	China	Ahn, 2013 ‡§ #	18,700	NA	42.7	NA	NA
N	Singapore	Liew, 2019	10,000	45.3	51	38.1 †	37.6
N	Singapore	Wu, 2009	5,022	41.5	51.8	43.7	45.3
N	China	Wang J., 2014 ¶	50,000	45.5	42.6	34.1	45.4
N	Japan	Satoh, 2017	2,623	48.9	67.1	56.2	38.8
N	Japan	Asayama, 2017	99,000	NA	NA	NA	43.6
N	India	Mohanty, 2021 ¶*	64,000	41.9	54.4	50.8	56.7 †
M	Low and middle income	Geldsetzer, 2019 ¶	1,1 × 10 ⁶	45.4	39.2	45.6	34 †
M	Middle income	Basu, 2013 ¶	44,000	36.7	33.7 †	27.4 †	37.9 †
M	All incomes	Chow, 2013	142,000	40.8	46.5	40.6	32.5
GLOBAL		Zhou, 2021 ¶	104 × 10 ⁶	32 (m) 34 (w)	49 (m) 59 (w)	38 (m) 47 (w)	47 † (m) 49 † (w)

L, local; N, national; M, multinational; Preval., prevalence; Awaren., awareness; Treatm., treatment. (m), men; (w) women.

|| Age 95% CI: 42.2–42.7

‡ Longitudinal study

§ Results for latest timepoint shown.

* Represents adults ≥45y & spouses

† Values calculated from presented results.

¶ Sampling weights and/or adjustments used to compute representative statistics.

Hypertensives only, mean (SD) age 70.3 (7.3)

2.2 Prevalence, Awareness, Treatment, and Control of Hypertension

The prevalence, awareness, treatment, and control of hypertension have been extensively studied, as demonstrated by numerous local and national studies^{8,25–45}, multinational investigations^{46–49}, and global analyses^{6,18,23,50–54} (Table 1).

Estimates of hypertension prevalence, awareness, treatment, and control vary widely across studies.⁵³ In Finland, recent data show that among individuals with elevated BP or on antihypertensive medication, 18% of women and 24% of men were unaware of their condition. Among those identified with elevated BP or who reported taking antihypertensive medication in the past week, 64% of women and 59% of men were on medication. However, less than half of those receiving antihypertensive treatment achieved target BP, with 46% of women and 48% of men reaching treatment goals.⁷

2.3 Inequalities in Hypertension Care

Hypertension is the leading risk factor for global disease burden and accounts for 7% of global disability-adjusted life years.⁵⁵ While advances in medical science have improved efforts to manage hypertension, concerns about disparities in care provision persist.⁵⁶ These disparities encompass a range of factors, including access to healthcare services⁵⁷, socioeconomic status⁵⁸, racial and ethnic disparities⁵⁹, and gender inequities⁶⁰.

2.3.1 Social Factors

Several studies have detected a link between hypertension and social factors, such as income level, education, and occupation.^{35,49,61} Leng et al. conducted a large meta-analysis on these associations, incorporating 51 original studies in their analysis.⁶¹ They found that the risk of hypertension was increased in the lowest categories of all studied socioeconomic indicators, namely income level, education, and occupation. The lowest income group had a 19% increased prevalence of hypertension compared with the highest income group. Similar and statistically significant results were uncovered for occupational categories. The association between education and hypertension was even stronger, with the risk of hypertension being two-fold higher in the lowest educational group compared to the highest educational group. These findings were consistent in the high-income countries. In contrast, the research group found inverse associations of both income and occupational indices with hypertension in Africa. It should be noted that the authors of this meta-analysis did not discuss these contrasting findings between high-income countries and Africa in their research article.⁶¹ An epidemiologic shift could explain these results.⁶² It is a theory that aims

to elucidate the transition in the prevalence and impact of diseases, moving from primarily infectious diseases to non-communicable diseases, which occurs alongside phenomena such as urbanisation and technological modernisation. As a result of this transition, diets in some parts of Africa are increasingly relying on highly processed foods. Accelerated marketing and the increased availability of such foods over a relatively short time have contributed to the creation of ‘obesogenic’ environments, which are closely linked to the pathophysiology of hypertension.⁶³

When the outcome is defined as an improvement in the awareness, treatment, and control of hypertension, rather than its prevalence, the effect of the socioeconomic indicators seems less consistent. One reason for this impression might be the lack of larger meta-analyses on this research question. Several smaller studies on this topic have been published, but they often differ substantially in their methodologies, which could contribute to their contrasting results.

To illustrate, income has not been associated with hypertension awareness^{35,64}, treatment^{35,64,65} and control^{35,64} in several studies. In contrast, some authors have reported an association between income level and hypertension treatment^{35,66} or control^{67,68}.

Furthermore, the association between education and control of hypertension is inconsistent in the literature. Liew et al. found that a lower educational level was associated with untreated hypertension. In contrast, income was not associated with it or the awareness or control of hypertension in a multi-ethnic Asian population in Singapore.³⁵ Another study conducted in South Korea corroborated the association between lower educational level and untreated hypertension.⁶⁹ However, several other studies have not detected an association between education and hypertension treatment.^{64,65,67} In addition, education has been associated with neither hypertension awareness nor control in some studies.^{64,67} Guessous et al.⁷⁰ and Appleton et al.⁷¹ have even reported an unexpected positive association between educational level and untreated hypertension in their studies. Guessous et al. described that the reasons for this finding are not clear. They pointed out that individuals possessing a substantial educational background may perceive themselves as capable of controlling their BP and could, therefore, opt not to adhere to their physician’s counsel.

Of note, Liew et al. did not provide information about exclusion of potential multicollinearity among the socioeconomic indicators, such as variance inflation factor values for the regressors,³⁵ and neither have the other often cited articles in this domain.^{64,65} While socioeconomic indicators measure different aspects, they are often related.⁷² Some authors have encouraged researchers to consider the diagnostic for multicollinearity as a crucial, major step in the regression analysis process, when the purpose of a multivariable regression analysis is to explain the individual effects of the predictors on an outcome variable.⁷³ Potential multicollinearity between the predictors, i.e. socioeconomic indicators in this case, could pose

a risk of misleading conclusions regarding the role of each collinear predictor in the regression model.⁷³

2.3.2 Lifestyle Factors

In the preceding section, social factors were discussed as potential contributors to disparities in hypertension on a general level. These factors may influence lifestyle behaviours – such as dietary patterns, alcohol consumption, tobacco use, and obesity-related behaviours – which are examined in greater detail in the present section.

The landmark Dietary Approaches to Stop Hypertension (DASH) trial (459 participants) demonstrated that a combination diet rich in fruits, vegetables, and low-fat dairy foods, and reduced in saturated fat, total fat, and cholesterol reduced systolic BP significantly more in African Americans (6.8 mmHg) than in whites (3.0 mmHg).⁷⁴ While the greater BP reduction observed among African American participants in the DASH trial may reflect higher baseline BP and greater salt sensitivity, it is likely that differences in baseline dietary quality and nutrient intake – notably lower intakes of fruits, vegetables, and low-fat dairy before the intervention – contributed to the more pronounced response. In a systematic review and meta-analysis covering 19 high-income countries, de Mestral et al. found that individuals of lower socioeconomic status consumed, on average, 14% more sodium – equivalent to about 500 mg per day – than their higher socioeconomic status counterparts.⁷⁵ Such disparities in sodium intake may partly account for the higher prevalence and poorer control of hypertension among disadvantaged groups. In addition to differences in sodium consumption, economic constraints may directly limit access to healthier dietary patterns. Darmon and Drewnowski demonstrated that foods of lower nutritional value generally cost less per calorie and were therefore more frequently consumed by individuals of lower socioeconomic status.⁷⁶ These findings suggest that financial barriers contribute to the persistence of dietary inequalities that underpin disparities in hypertension risk.

Another contributing factor to hypertension disparities is alcohol. Dose-response evidence shows nearly linear increases in hypertension risk above 12 g/day, with risk rising more sharply in White women and lower susceptibility observed in Black populations, potentially due to genetic or environmental factors. Alcohol may elevate BP through several mechanisms, including activation of the renin-angiotensin-aldosterone system, increased sympathetic nervous system activity, decreased baroreceptor sensitivity, and enhanced intracellular calcium in vascular smooth muscle, with hormonal differences contributing to sex-specific effects.⁷⁷ Evidence uncovered through systematic review indicates that low socioeconomic status increases vulnerability to alcohol-related hypertension, even at similar or lower levels of consumption, likely due to differences in drinking behaviours, clustering with other risky

lifestyle factors, differential healthcare access, and social or environmental contexts.⁷⁸ Research specifically examining socioeconomic status, alcohol use, and hypertension remains scarce, highlighting a need for more studies.

Furthermore, the relationship between smoking and hypertension is complex. While smoking is not traditionally seen as a direct cause of hypertension, it increases arterial stiffness through sympathetic activation, impaired baroreflex, and reduced arterial compliance.⁷⁹ When combined with high BP, smoking exacerbates vascular damage beyond the effect of either factor alone.⁸⁰ Social context further shapes these disparities: the Chicago Community Adult Health Study showed that disadvantaged neighbourhoods, with higher tobacco exposure, had greater hypertension prevalence, whereas affluent neighbourhoods had lower prevalence. Incorporating neighbourhood-specific variables in the statistical models reduced racial, ethnic and educational disparities in prevalence, though treatment and control gaps persisted.⁸¹ Tobacco use, vascular physiology, and social environment together drive inequalities in hypertension.

Obesity is a major risk factor for hypertension, with large-scale epidemiological and longitudinal studies showing that excessive weight gain, particularly visceral adiposity, may account for 65–75% of incident hypertension risk. Mechanistically, obesity-related hypertension arises from the overactivation of the renin-angiotensin-aldosterone system and the sympathetic nervous system, as well as overstimulation of adipokines, insulin resistance, immune dysfunction, and structural and functional changes in renal, cardiac, and adipose tissues.⁸² Weight loss interventions, such as exercise or bariatric/metabolic surgery, lower BP, but persistent socioeconomic and environmental barriers indicate that obesity-related hypertension disproportionately affects marginalised populations.^{82–84}

Collectively, these lifestyle behaviours – shaped by socioeconomic and environmental contexts – contribute to the unequal burden of hypertension, underscoring the need for interventions that target both individual habits and the social determinants of health.

2.3.3 Demographic Factors

Demographic characteristics contribute significantly to disparities in hypertension, influencing risk, prevalence, and management across factors such as age, gender, race and ethnicity, and geographic location.

Age and gender further shape hypertension risk. In the United States, data from the Centers for Disease Control and Prevention for 2015–2018 indicate that BP – and consequently hypertension prevalence – rises steadily with age. Younger men are more often hypertensive than women, with 34.5% of men aged 20–44 affected compared with 17.6% of women in the same age group. In older age groups, women

exhibit a higher prevalence, reaching 74.3% among those aged 65–74 compared with 66.7% among men, and 86% among women over 75 compared with 81.5% among men.⁸⁵ These trends reflect the cumulative effects of ageing on vascular stiffness, renal sodium handling, sympathetic nervous system activity, and sex-specific hormonal influences.⁸⁵

Rural–urban differences are a key demographic determinant of hypertension. In lower-income countries, awareness, treatment, and control of hypertension are typically higher in urban communities than in rural ones, as shown by the Prospective Urban Rural Epidemiology study.⁴⁹ This gap reflects differences in healthcare access, socioeconomic status, and exposure to modifiable risk factors such as diet and physical activity. Rural populations often face barriers to screening and medication availability, leading to lower rates of diagnosis and control. However, rural–urban disparities are not limited to developing settings. In the United States, adults living in rural areas have significantly higher odds of hypertension and elevated systolic BP compared with those in urban regions, likely reflecting similar challenges in access to preventive and clinical care.⁸⁶

Geographic variation can also influence hypertension patterns. In Finland, an east–west gradient in BP has been observed. Historically, BP was higher in eastern Finland, but large-scale prevention efforts, notably the North Karelia Project, have substantially reduced cardiovascular risk factors in the region. Over the decades, surveys have indicated that BP levels in eastern Finland have declined markedly, and in more recent assessments, they have been comparable to levels in southwestern Finland.^{87–89} These improvements reflect the sustained impact of interventions targeting diet, lifestyle, and hypertension management.

Racial and ethnic disparities are also evident. Black populations, particularly African Americans, often experience higher hypertension prevalence and poorer control compared with White populations, reflecting a combination of social, environmental, and biological factors.⁹⁰ Data from the Multi-Ethnic Study of Atherosclerosis show that hypertension prevalence was significantly higher in African Americans (60%) than in Whites (38%), whereas Hispanic (42%) and Chinese participants (39%) did not differ significantly from Whites. Among hypertensive participants, the proportion with treated but uncontrolled hypertension was higher in African Americans (35%), Chinese (33%), and Hispanics (32%) compared with Whites (24%). However, only African American ethnicity remained significantly associated with treated but uncontrolled hypertension after controlling for socioeconomic factors.⁹¹

These findings underscore persistent disparities in hypertension management that extend beyond prevalence differences and highlight the importance of targeted interventions for high-risk populations.

2.3.4 Blood Pressure Monitoring in Relation to Prevalence, Awareness, Treatment, and Equity Considerations

The method of BP assessment – whether performed at home or in a clinical setting – can substantially influence estimates of hypertension outcomes, including prevalence, awareness, treatment, and control.^{92–95} Home BP monitoring offers advantages in detecting masked hypertension and may reduce misclassification caused by white-coat effects, thereby improving diagnostic accuracy.^{92–94} Population-based studies such as the Ohasama in Japan, the Pressioni Arteriose Monitorate E Loro Associazioni (PAMELA) in Italy, and the Finn-Home in Finland suggest that reliance solely on office measurements can misclassify a substantial proportion of individuals, either overlooking masked hypertension or labelling white-coat hypertension as uncontrolled.^{92–94} Consequently, when relying exclusively on office readings, true hypertension prevalence may be underestimated.^{93,94}

Awareness of hypertension is shaped by measurement settings. Home BP monitoring promotes self-awareness and engagement, as individuals regularly observe their own BP values and better understand the chronic nature of hypertension.^{96,97} Additionally, use of home BP monitoring has been associated with improved adherence to follow-up and treatment recommendations.⁹⁷

However, access to validated home BP monitoring devices, training, and correct adherence to measurement protocols can vary across populations.^{95,98,99} Substantial disparities exist across racial, socioeconomic, and demographic lines, which may affect the access to home BP monitors, although direct evidence on their impact on hypertension outcomes remains limited. For example, Black adults and unemployed individuals had lower odds of owning home BP monitors compared to White and employed adults. Even though higher-income adults were more likely to monitor their BP, they were still less likely to share readings with providers.^{98,100} Few studies explicitly evaluate how home BP monitoring impacts hypertension disparities, particularly among rural, low-income, and certain racial/ethnic minority populations, highlighting critical gaps in real-world implementation evidence.⁹⁹

Treatment patterns and hypertension control are also influenced by the measurement context. Home BP monitoring, particularly when combined with structured feedback, telemonitoring, or clinician-guided titration, has been associated with earlier initiation of antihypertensive therapy, better adherence to medications, and higher rates of achieving target BP when compared with office-based care alone.^{95–97} These benefits are most apparent in patients actively engaged with healthcare services, but may not be equitably distributed given disparities in device access and health system support.^{98,99}

In summary, home BP monitoring modifies not only the accuracy of hypertension diagnosis, but also the social patterning of awareness, treatment, and control. While home BP monitoring has the potential to narrow gaps through more precise and patient-centred management, unequal access to technology, training, and health

literacy may conversely widen disparities unless programmes explicitly addressing these barriers are introduced.^{95,98,99} Overall, the evidence suggests that disparities in home BP monitoring remain a critical area for future research, particularly to evaluate their impact on hypertension outcomes and to identify implementation strategies that can advance equity.

2.4 Risk of Progression to Sustained Hypertension by Office and Out-of-Office Blood Pressure Phenotypes

The current hypertension guidelines from the Finnish Society of Hypertension, the European Society of Hypertension, the European Society of Cardiology, and the American College of Cardiology/American Heart Association (ACC/AHA) recommend a thorough examination of the patient's BP status for the physician to be able to determine the correct course of action with accuracy.^{15,16,101,102} Because BP is a continuous variable and cardiovascular disease risk starts to increase exponentially from an office BP of 110/70 mmHg upwards, the thresholds on which the diagnosis of hypertension is based are largely arbitrary.¹⁰³ The challenge of defining hypertension is further highlighted by the fact that the thresholds have considerably decreased over the past 60 years.¹⁰⁴ In the 1960s, it was still the belief of many experts that hypertension was a natural process of ageing (and that a systolic BP of "age + 100" was a normal level).¹⁰⁴ In contrast, the American hypertension guidelines lowered the definition of hypertension to 130/80 mmHg in 2017, increasing the prevalence of hypertension in the United States from 32% to 46% overnight.¹⁰⁵ Current Finnish and international hypertension management guidelines differ in the definition of hypertension and its division into different stages and phenotypes.^{15,16,101,102} These differences in classifications based on office BP are presented in **Table 2**. Apart from the American ACC/AHA guidelines, which recommend a lower threshold of 130/80 mmHg to diagnose hypertension, a mean office BP of 140 mmHg systolic and 90 mmHg diastolic is considered hypertension by the other guidelines.

2.4.1 Definition of Blood Pressure Phenotypes

Out-of-office BP measurement, which includes home and ambulatory BP monitoring, has several important benefits compared to relying solely on office readings. Compared to office BP, the use of out-of-office BP measurements allows for a more accurate prediction of cardiovascular disease risk through an increased number of BP measurements that are free from the white-coat effect.^{106,107} This is why all aforementioned hypertension guidelines, apart from the European Society of Hypertension guidelines, recommend using office BP measurements only for hypertension

Table 2. Office Blood Pressure Categories in Various Hypertension Guidelines.

	OFFICE BP	FINLAND 2020	ESH 2023	ESC 2024	ACC/AHA 2025
SYSTOLIC	<120	Optimal BP	Optimal BP	Non-elevated BP	Normal BP
	120–129	Normal BP	Normal BP	Elevated BP	Elevated BP
	130–139	High-normal BP	High-normal BP	Elevated BP	Stage 1 HT
	140–159	Grade 1 HT	Grade 1 HT	HT	Stage 2 HT
	160–179	Grade 2 HT	Grade 2 HT	HT	Stage 2 HT
	≥180	Grade 3 HT	Grade 3 HT	HT	Stage 2 HT
DIASTOLIC	<70	Optimal BP	Optimal BP	Non-elevated BP	Normal BP
	70–80	Optimal BP	Optimal BP	Elevated BP	Normal BP
	80–84	Normal BP	Normal BP	Elevated BP	Stage 1 HT
	85–89	High-normal BP	High-normal BP	Elevated BP	Stage 1 HT
	90–99	Grade 1 HT	Grade 1 HT	HT	Stage 2 HT
	100–109	Grade 2 HT	Grade 2 HT	HT	Stage 2 HT
	≥110	Grade 3 HT	Grade 3 HT	HT	Stage 2 HT

BP, Blood Pressure; HT, Hypertension.

screening, whereas out-of-office measurements are necessary for confirming the diagnosis of hypertension.^{15,16,101,102}

As the cardiovascular disease risk increases more rapidly per 1 mmHg increase in out-of-office BP than in office BP, the diagnostic thresholds for hypertension vary between various BP measurement methods.^{106,107} Currently, all major guidelines agree on comparable thresholds for different BP measurement techniques that are equivalent to an office BP level of 140/90 mmHg, the most commonly used threshold for diagnosing hypertension (**Table 3**).^{15,16,101,102}

Table 3. Out-of-office Thresholds Equivalent to Office Thresholds of 140/90 mmHg.

MEASUREMENT METHOD	SYSTOLIC (MMHG)	DIASTOLIC (MMHG)
OFFICE BP	140	90
HOME BP	135	85
AMBULATORY BP		
DAYTIME	135	85
NIGHTTIME	120	70
24H	130	80

BP, Blood Pressure.

The diagnosis of hypertension is often based on both office and out-of-office BP measurements, which may differ markedly due to the white-coat effect or other reasons. Therefore, BP may be elevated with one BP measurement method but normal with another method. Cross-classification of patients according to office and out-of-the-office BP measurement delineates four groups: normotension, where patients have normal office and out-of-office BP; white-coat hypertension, where patients have hypertension in office BP measurements but normal out-of-office BP; masked hypertension, where patients have normal office BP, but hypertension according to out-of-office BP measurements; and sustained hypertension, where patients have hypertension according to both office and out-of-the-office BP measurements. These BP phenotypes, that are also mentioned in all major hypertension guidelines, are visually presented in **Table 4**.^{15–17,101,102,105,108,109}

Table 4. Categorisation of Blood Pressure Phenotypes.

OFFICE BP	OUT-OF-OFFICE BP	
	24-H AMBULATORY BP <130/80 HOME BP <135/85	24-H AMBULATORY BP ≥130/80 HOME BP ≥135/85
≥140/90	White-coat hypertension	Sustained hypertension
<140/90	Normotension	Masked hypertension

BP, blood pressure.

White-coat hypertension has been conventionally considered a benign phenotype that does not warrant pharmaceutical therapy. Masked hypertension, in contrast, has been thought of as a rare phenotype with an unknown prognosis. However, results from the population-based International Databases of Home and Ambulatory BP in Relation to Cardiovascular Outcome consortia have demonstrated that these phenotypes are neither rare nor benign.^{107,110} The prevalence of white-coat hypertension and masked hypertension in the general population is 10–15%, depending on whether the diagnosis is based on home or ambulatory BP measurements (**Table 5**).^{107,110} In addition, masked hypertension, in particular, is linked with a significantly higher cardiovascular disease risk than normotension, suggesting that it is not a benign phenomenon. In fact, white-coat and masked hypertension may represent intermediate phenotypes between normotension and sustained hypertension.

Table 5. Prevalence and Prognosis of Blood Pressure Phenotypes in the International Databases on Ambulatory and Home Blood Pressure in Relation to Cardiovascular Outcome.

PHENOTYPE		PREVALENCE, %	HR FOR CVD COMPARED WITH NORMOTENSION
AMBULATORY BP	WCHT	10.6	1.22; $P>0.05$
	MHT	14.6	1.62; $P<0.001$
	SHT	25.5	1.82; $P<0.001$
HOME BP	WCHT	14.3	1.42; 95% CI [1.06–1.91]; $P=0.02$
	MHT	9.8	1.55; 95% CI [1.12–2.14]; $P<0.01$
	SHT	24.5	2.13; 95% CI [1.66–2.73]; $P<0.0001$

CVD, cardiovascular disease; WCHT, white-coat hypertension; MHT, masked hypertension; SHT, sustained hypertension; HR, hazard ratio; CI, confidence interval.

2.4.2 Blood Pressure Phenotype Changes During Follow-Up

Only a few studies have examined the risk of developing sustained hypertension in individuals with white-coat and masked hypertension.

In the first publication in this domain, Bidlingmeyer et al. assessed the risk of sustained hypertension in 81 untreated patients with white-coat hypertension.¹¹¹ The authors reported that 72% of these patients developed ambulatory hypertension over a 3-year period. In contrast, normotensive controls did not progress to sustained hypertension in that period.

In another study with a relatively small sample size and short follow-up, Colombo et al. investigated whether white-coat hypertension might be an early transitional phase leading to sustained hypertension.¹¹² They enrolled 173 patients who had elevated office BP and normal ambulatory BP. Of the 173 patients, 102 ($\approx 58.9\%$) progressed to sustained hypertension over a follow-up of five years. In addition, the authors observed that stress reactivity (i.e. a positive BP and heart rate response in mental arithmetic) might help distinguish patients who were more likely to convert to sustained hypertension.

In a third article by Polónia et al., the authors investigated whether untreated white-coat hypertensive individuals tended to develop ambulatory hypertension over time and compared their progression with that of normotensive individuals.¹¹³ Over a mean follow-up of 3.5 years, four out of 36 (11%) untreated white-coat hypertensive subjects developed sustained hypertension compared to three out of 52 (6%) normotensive control subjects.

Two larger population-based studies, both published in 2009, have also assessed whether home and office BP-based white-coat hypertension is a risk factor for sustained hypertension.^{12,114} In a Japanese population-based sample of 777 participants aged ≥ 40 , participants with white-coat hypertension had a highly significant 2.86-fold odds for progression to sustained hypertension compared to normotensive individuals over a mean follow-up of 8.2 years.¹¹⁴ In addition, the probability of developing home hypertension increased with higher baseline home BP. In another population-based study of 1412 individuals living in Monza, Italy, Mancina et al. assessed the risk of sustained hypertension in white-coat and masked hypertensives who underwent both ambulatory and home BP monitoring.¹² As compared with normotensives, the adjusted and significant odds of developing sustained hypertension were higher for white-coat and masked hypertensive subjects over a follow-up of 10 years (odds ratios 2.51 and 1.78, respectively). Similar results were obtained when the definition of these conditions was based on home BP.

2.5 Longitudinal Association between Home Blood Pressure and Hypertension-Mediated Organ Damage

Hypertensive target organ damage, also known as hypertension-mediated organ damage (HMOD), refers to structural and/or functional injury to specific organs as a result of chronically elevated BP.¹⁵ These changes are key indicators of disease progression and risk of cardiovascular events. The most commonly used markers of subclinical HMOD are LVH, proteinuria, retinopathy, atherosclerosis, and arterial stiffness.¹⁵

In cross-sectional studies, home BP has been observed to be superior to office measurements regarding their association with preclinical organ damage, such as echocardiographic LVH and albuminuria.^{14,115} Correspondingly, the prospective long-term associations between home BP and cardiovascular outcomes are, in general, stronger than those observed for office BP.¹⁰⁶

However, only a few longitudinal studies regarding the predictive value of home BP on the changes in HMOD in the general population and treated hypertensive patients have been conducted (**Table 6**). In two of these studies home BP was more strongly associated with changes in organ damage than office BP^{116,117}, whereas one study contains contradicting evidence¹¹⁸. One study focused on night-time home BP, in which BP was measured three times during the night with a timer-equipped home monitor.¹¹⁹ This study uncovered that night-time home BP was a particularly good predictor of longitudinal changes in HMOD. Of note, left ventricular mass index has

Table 6. Longitudinal Associations Between Home Blood Pressure and Hypertension-Mediated Organ Damage.

STUDY / YEAR	COHORT	HOME BP MEASURE	HMOD	MAIN FINDING	KEY INTERPRETATION
TSUNODA ET AL., 2002	207 treated hypertensive patients, 5 yrs	Morning & evening HBP	LVMI	LVMI ↑ only in those with uncontrolled or rising home SBP; Δhome SBP correlated with LVMI change; office BP ns	Long-term control of home SBP <135 mmHg prevents LVMI progression
ISHIKAWA ET AL., 2012	854 Japanese adults, cross-sectional + 1 yr follow-up	Nighttime automated HBP	Albuminuria, LVMI	Nighttime home SBP was better predictor of renal/cardiac damage than office or daytime BP	Nocturnal home SBP most strongly linked to renal & cardiac damage
KARPETTAS ET AL., 2014	153 treated patients, 13 mo	Morning & evening HBP (7 days)	LVMI & PWV	Δhome SBP was best predictor of LVMI regression; office BP ns; ambulatory BP was strongest predictor of PWV	HBP changes track structural regression better than office BP
VELOUDI ET AL., 2016	286 patients, 12 mo	Morning & evening HBP (7 days)	LVMI & PWV	HBP changes were not significantly different between the groups with and without organ damage regression	HBP and office BP did not differ in predicting organ damage regression.

BP, blood pressure; HMOD, hypertension-mediated organ damage; LVMI, left ventricular mass index; SBP, systolic blood pressure; UACR, urinary albumin-creatinine ratio; PWV, pulse wave velocity; HBP, home blood pressure.

been assessed in all these studies, whereas only single studies have assessed the relation between home BP and changes in albuminuria and arterial stiffness (**Table 6**).

2.6 Benefits and Limitations of Blood Pressure Measurement Methods for Assessing Longitudinal Risk

Different BP measurement methods vary in their ability to predict long-term cardiovascular risk. Office BP has been the most common method of BP measurement and forms the basis of many epidemiological studies and of nearly all randomised clinical trials.^{120,121} It is easy to obtain and standardised, but it is influenced by white-coat and masked hypertension, providing only a snapshot of BP.^{121,122} Consequently, it has moderate predictive value for long-term risk.¹²³

Home measurements better reflect usual BP and can identify white-coat and masked hypertension. Repeated readings also improve BP estimation. However, accuracy depends on the patient's technique and the device's reliability.^{124–127} Overall, home BP measurement offers better longitudinal risk prediction than office BP.^{14,123,128} Home BP is particularly suitable for repeated BP measurements, as the patient can easily perform it in the comfort of their own home.¹²⁹

Ambulatory BP monitoring records BP over 24 hours, capturing circadian patterns such as nighttime BP and morning surge, which are strong predictors of cardiovascular outcomes.¹¹⁹ It minimises white-coat effects and provides highly reliable data.¹³⁰ Despite being the gold standard, its use is limited by cost, patient discomfort, and impracticality for frequent monitoring.^{131–133}

For longitudinal risk prediction, the relative accuracies and predictive strengths are as follows: ambulatory BP > home BP > office BP.^{123,132,134} Combining methods, for example, baseline ambulatory BP measurements with regular home BP follow-up, offers the most reliable approach. Incorporating measures such as nighttime BP further enhances long-term cardiovascular risk prediction.¹³⁰ A comparison of BP measurement methods is presented in **Table 7**.

2.7 Summary

Hypertension is the leading global burden of disease risk factor, both worldwide and in Finland.^{7,19} Numerous studies have examined the rates of prevalence, awareness, treatment, and control of hypertension in varying cohorts. However, many of these studies have several limitations. These include being conducted with small patient or community cohorts or having data available on only the most basic background variables, such as age and sex. Additionally, most have not included the awareness, treatment, and control rates simultaneously in the analyses. Earlier large-scale

population studies have provided information on prevalence and treatment rates of hypertension in Finland, but have not examined the associated socioeconomic and lifestyle factors.^{7,8} In addition, and what is particularly relevant for this thesis, no previous studies have verified the observed prevalence and control rates of hypertension using home BP, a more reliable method for BP measurement than office BP.

White-coat hypertension has been traditionally considered a relatively benign phenomenon. However, recent studies have shown that white-coat hypertension may not be harmless, as cardiovascular disease risk may already increase from normotension to white-coat hypertension.^{107,110} In contrast, masked hypertension has been more convincingly shown to carry an increased cardiovascular disease risk.^{107,110,113} It was also still unclear whether white-coat hypertension and masked hypertension are predictive of future sustained hypertension, however. Smaller studies with a limited follow-up duration have provided inconsistent results.^{111,112} In two larger population-based studies, white-coat hypertension was found to be an independent risk factor for sustained hypertension.^{12,114} However, only one of these studies assessed the risk of transition from masked hypertension to sustained hypertension.¹¹⁴ In addition, the populations of these studies were recruited from a single rural or urban community instead of larger geographical areas.

Several cross-sectional studies have demonstrated that home BP has a stronger relation to HMOD than office BP.^{14,115} However, temporal relationships between exposure and outcome are difficult to determine from cross-sectional studies, and only a few relatively small patient cohorts with mixed results have previously suggested that change in home BP could be associated with change in HMOD.¹¹⁶⁻¹¹⁹

Table 7. Comparison of Blood Pressure Measurement Methods.

METHOD	PREDICTIVE VALUE FOR LONGITUDINAL RISK	MAIN STRENGTH	MAIN LIMITATION
OFFICE BP	Moderate	Widely available, evidence-based	Unable to identify White-coat/masked effects
HOME BP	High	Reflects true BP, captures variability	User-dependent accuracy
ABPM	Very high	24h profile, best prognostic data	Cost, limited repeatability

BP, blood pressure; Office BP, BP measurements performed by doctor or nurse; Home BP, BP measurements performed at home by oneself; ABPM, ambulatory BP measurement.

3 Aims

The main aim of this thesis was to compare home and office BP measurements in assessing the prevalence, awareness, treatment, and control of hypertension and their correlates, in estimating the risk of progression to sustained hypertension, and in predicting longitudinal HMOD changes.

The specific objectives were:

1. To assess hypertension prevalence, awareness, treatment, and control rates of hypertension in Finland using both home and office BP measurements, and the associated socioeconomic and lifestyle factors. (Study I)
2. To determine the risk of progression from white-coat and masked hypertension to sustained hypertension in the Finnish population over an 11-year follow-up. (Study II)
3. To compare the correlation of longitudinal home versus office BP change in relation to longitudinal change in electrocardiographically assessed left ventricular hypertrophy (ECG-LVH). (Study III)
4. To examine the association of home and office BP with prevalent and incident kidney disease. (Study IV)

4 Materials and Methods

4.1 Study Samples

Studies I–IV were performed using data from the Health 2000 study, a nationwide representative multidisciplinary epidemiological survey, and its follow-up study, the Health 2011 study. These studies were conducted by the Finnish National Institute for Health and Welfare between 2000–2001 and 2011. The study involved participants from 80 municipalities (**Figure 2**).

For the Health 2000 study, a nationwide sample of 9,922 persons aged 18 years or older was drawn from the population register to represent the Finnish adult population. The final sample consisted of 8,028 participants aged 30 years or older in the main study and of 1,894 participants in the study of young adults aged 18–29. Of the 8,028 participants, 6,771 (84.3%) participated in the health examination, of which 3,687 (84%) participants were 45 to 74 years of age. Of these 3,687 participants, 2,120 (57.4%) were selected to participate in the home BP sub-study, the Finn-Home study, based on the participants' willingness and availability of home monitors.

Of the 9,922 participants of the Health 2000 study, 8,135 participants were still alive and willing to participate in the Health 2011 follow-up study in July of 2011. In total, 4,729 (58%) participated in the health examination. Of the 2,120 participants of the Finn-Home survey, 1,898 were still alive, and 1,401 participated in the health examination. A total of 1,194 individuals participated in the Finn-Home study, both at baseline and follow-up. The Finn-Home follow-up was carried out from August 2011 to June 2012.^{135–137}

4.1.1 Study I

The Health 2011 study data were used for study I. Of the 4,729 participants who participated in the health examination, participants with missing health interview (n=452) or physical examination (n=47) were excluded, after which a total of 4,230 participants was included in the analyses. A subsample of 1,107 subjects aged 45–74 who participated in the home BP measurement substudy (Finn-Home) was included in the analyses concerning these measurements.

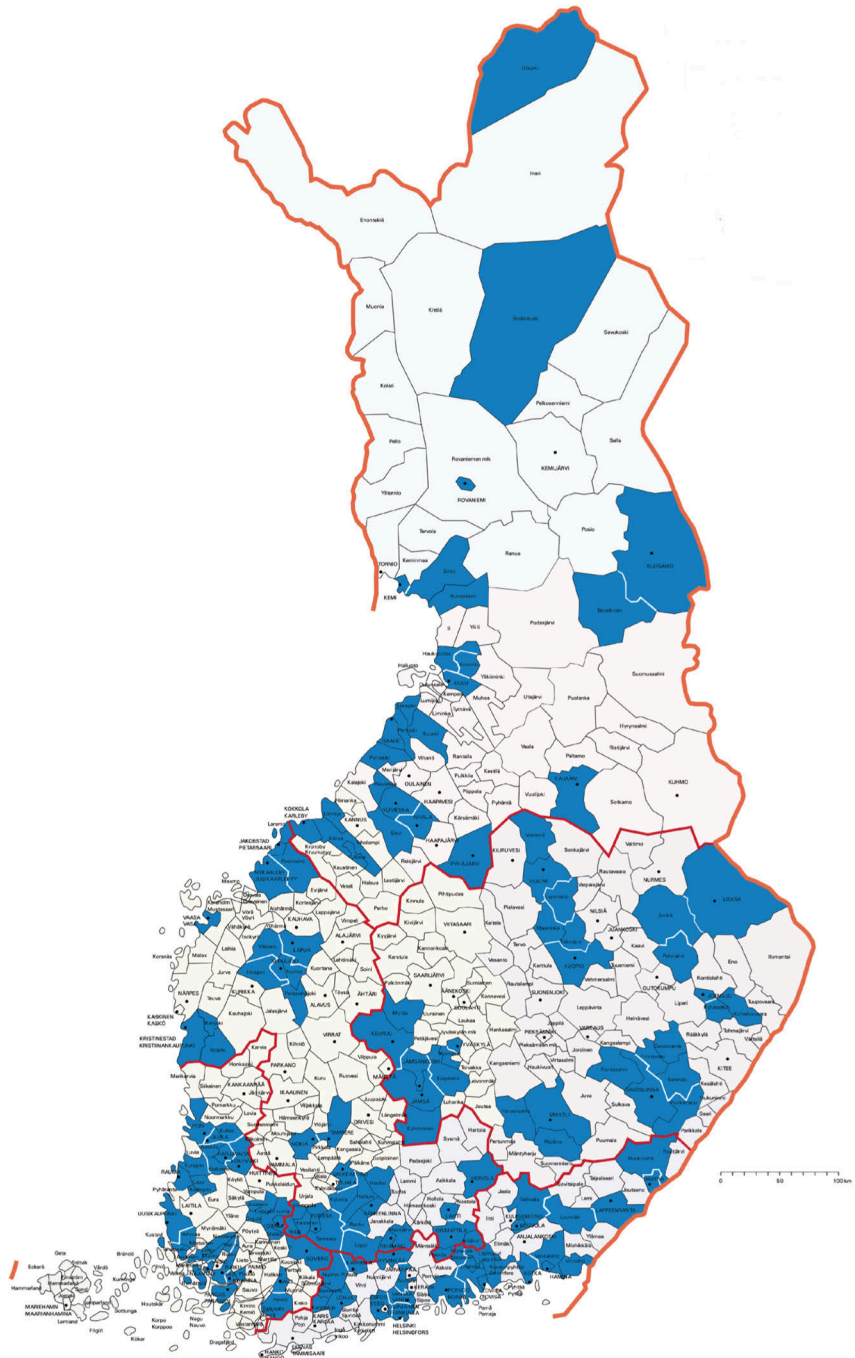


Figure 2. Survey areas of the Health 2000 study. Heistaro S, Kansanterveyslaitos. Methodology Report: Health 2000 survey. Helsinki: National Public Health Institute; 2008. 248 p. <<https://urn.fi/URN:NBN:fi-fe201204193320>>. Reprinted with permission of the National Institute for Health and Welfare.

4.1.2 Study II

Data from both the Health 2000 (baseline) and Health 2011 (follow-up) studies were used for study II. In total 1,194 individuals participated in the home BP measurement sub-study at baseline and at follow-up. Those with missing data (n=11) and antihypertensive medication at baseline (n=239) were excluded, resulting in a study sample of 944 participants.

4.1.3 Study III

Study III consisted of 1,194 participants with home BP measurements both at baseline (Health 2000) and follow-up (Health 2011). After excluding participants with less than 14 home BP measurements or missing office BP measurements (n=30), missing ECG data (n=72), prolonged QRS duration (≥ 120 ms, n=68)¹³⁸, and antihypertensive medication at baseline or follow-up (n=409, combined), the final study sample consisted of 615 participants. Participants on antihypertensive medication were excluded to minimise confounding effects.

4.1.4 Study IV

Study IV utilised data from both the Health 2000 and Health 2011 studies. Of the 2,120 participants with home and office BP measurements from the Health 2000 study, 111 participants were excluded due to missing data, resulting in a sample of 2,009 participants who were included in the baseline cross-sectional analyses.

At follow-up, 1,805 of those included in the cross-sectional analyses were alive, and 1,350 (74.8%) agreed to participate in the home measurement substudy. A total of 82 participants were excluded due to missing data, resulting in a sample of 1,268 individuals included in the longitudinal analyses.

4.2 Flow of Study

The methods used in both Health 2000 and Health 2011 studies were identical and have been described in detail by the study organisation.^{135,136}

In brief, both studies consisted of questionnaires and an interview performed by a centrally trained interviewer. A health examination was carried out either at an office or at home for those unable to travel, by centrally trained healthcare professionals.

The interviewers gathered information about the participant's health and illnesses, including, but not limited to, age, sex, marital and employment status, social factors, level of education, use of medication, use of health services, cigarette smoking, alcohol use, and physical activity. Those selected for the Finn-Home sub-study

received the home BP monitors after the health interview. For the Health 2000 study, the interview was performed at the participant's home, 1–6 weeks prior to the health examination. In the Health 2011 study, the interview was performed simultaneously with the physical health examination.

The physical examination consisted of multiple measurements, including the participant's height, weight, office BP, resting ECG, samples of blood, spot urine, and a clinical examination of oral health by a dentist. During the Health 2000 study, the average duration of the physical health examination was 4.5 hours.

The study protocol was approved by the Ethics Committee of the Helsinki and Uusimaa Hospital Region, and all participants signed written informed consent.^{135,136}

4.3 BP Measurements

The methodology for BP measurements was identical in both the Health 2000 and Health 2011 studies.^{135,136} In brief, office BP was measured during the physical health examination. The two measurements were taken at 1-minute intervals after a 5-minute rest with a calibrated mercury sphygmomanometer (Health 2000 study: Mercurio 300; Speidel & Keller, Jungingen, Germany; Health 2011 study: Riester Diplomat Presameter Desk Set; Rudolf Riester GmbH, Jungingen, Germany). The cuff size was chosen according to the proximal circumference of the upper arm. Systolic BP was defined as the appearance of Korotkoff sound I with a two-mmHg accuracy, while diastolic BP was defined as the disappearance of the latter sound of Korotkoff sound V, again with a two-mmHg accuracy.

Home BP measurements were performed on seven consecutive days, every morning and evening. Two measurements were taken at 1-minute intervals for a total of 28 measurements. The cuff size was selected according to the manufacturer's instructions. The used BP monitor was a validated, automatic oscillometric device (Health 2000 study: Omron HEM-722C; Health 2011 study: Omron HEM-7211-E; both Omron Matsusaka Co., Kyoto, Japan).^{139,140}

4.4 ECG Recordings

During the health examination, a standard resting 12-lead ECG was digitally recorded by a centrally trained nurse, using General Electric MAC 5000 or MAC 5500 systems (GE Healthcare, Milwaukee, WI, USA). The quality of the recording was verified during the examination, and the recording was repeated when deemed necessary.

The ECG recordings were analysed with specialised software (Magellan software, Marquette Electronics Inc., Milwaukee, WI, USA), overread and corrected to ensure correct placement of measurement points on the ECG, including durations and amplitudes of P waves, QRS complexes and T waves, as well as the durations

of conduction times. Measurement points were measured to the nearest 4 ms and 0.5 mm, respectively.

For Study III, four common ECG-LVH indices were determined for each participant, for which the specific measurements are depicted in **Figure 3**:

- | | |
|--|--|
| 1. The Sokolow-Lyon index ¹⁴¹ | $S_{V1} + R_{V5/6}$ |
| 2. The Cornell voltage ¹⁴² | men: $R_{aVL} + S_{V3}$; women: $R_{aVL} + S_{V3} + 6\text{mm}$ |
| 3. The Cornell product ¹⁴³ | Cornell voltage x QRS duration |
| 4. R_{aVL} voltage (hereafter R_{aVL}) ¹⁴⁴ | R wave amplitude in aVL lead |

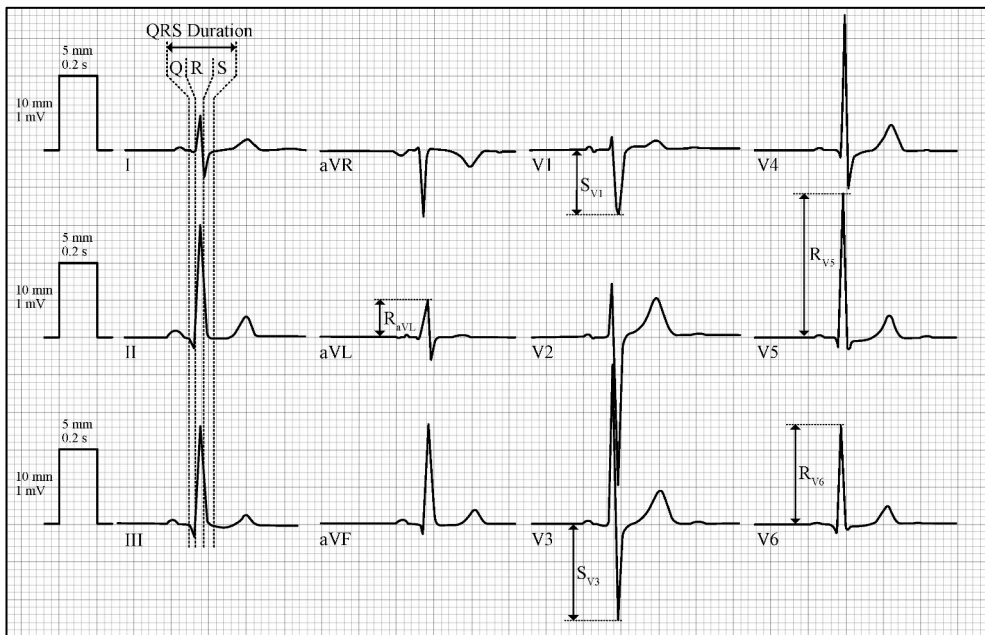


Figure 3. Normal ECG with criteria used to diagnose ECG-LVH.
Author's own work.

4.5 Follow-up of Non-fatal Events

For Study II, data on non-fatal cardiovascular events were collected from the National Hospital Discharge and Mortality Registers using the International Classification of Diseases, 10th revision codes. Data from these events, both before and during the follow-up period, were used. The data collection process has been detailed previously.¹⁴⁵

The mean (SD) follow-up time was 11.0 (0.1) years. The primary endpoint used in study II was the first event of non-fatal myocardial infarction, non-fatal stroke, new onset heart failure, percutaneous coronary intervention, or coronary bypass surgery.

4.6 Definitions

For Studies I–IV, office BP was defined as the mean of both measurements, and home BP was defined as the mean of all available measurements.

In Study I, office hypertension was defined as office BP $\geq 140/90$ mmHg or the use of antihypertensive medication, whereas home hypertension was defined as home systolic/diastolic BP $\geq 135/85$ mmHg or the use of antihypertensive medication. Awareness of hypertension was determined as a self-reported diagnosis of hypertension among participants with hypertension. Treatment of hypertension was defined as the use of antihypertensive medication among participants with hypertension. Furthermore, control of hypertension was specified as an office systolic/diastolic BP $< 140/90$ mmHg or home systolic/diastolic BP $< 135/85$ mmHg among participants with antihypertensive medication. Body mass index (BMI) was calculated as body mass (kg) divided by height (m) squared. Overweight and obesity were defined as BMI ≥ 25 kg/m² and ≥ 30 kg/m², respectively. Alcohol consumption per week was estimated using a questionnaire¹⁴⁶ and participants were divided into three groups according to approximate alcohol use: non-drinkers, modest users, and heavy users (> 288 g/week for men and > 192 g/week for women). Leisure-time physical activity was assessed using the four-point Gothenburg scale with grades from none to strenuous, with the two highest categories combined into one.¹³⁵ Finally, smoking was determined using a self-report questionnaire, and subjects were categorised into non-smokers, former smokers, and current smokers.

In Study II, sustained hypertension was defined as home BP $\geq 135/85$ mmHg and office BP $\geq 140/90$ mmHg or the use of antihypertensive medication, while white-coat hypertension was determined as office BP $\geq 140/90$ mmHg and home BP $< 135/85$ mmHg. Masked hypertension was defined as home BP $\geq 135/85$ mmHg and office BP $< 140/90$ mmHg. Smoking was categorised as daily use of tobacco products. Diabetes was defined as fasting glucose ≥ 7.0 mmol/l or the use of antidiabetic medication. Hypercholesterolemia was defined as a total plasma cholesterol level of > 7.0 mmol/l or the use of cholesterol-lowering medication.

In Study IV, microalbuminuria was defined as urine albumin-creatinine-ratio equal or higher than 2.5 mg/mmol for men and 3.5 mg/mmol for women.¹⁴⁷ The estimated glomerular filtration rate (eGFR) was calculated through serum creatinine concentration. More precisely, the eGFR (ml/min/1.73 m²) was calculated using the Chronic Kidney Disease Epidemiology Collaboration formula modified for SI units, where serum creatinine (Scr) is in $\mu\text{mol/L}$ and age is in years.¹⁴⁸ Decreased eGFR was defined as eGFR < 60 ml/min/1.73 m² and incident decreased eGFR was defined as the decline of kidney function from normal eGFR (≥ 90 ml/min/1.73 m²) to decreased eGFR during the follow-up period.¹⁴⁹

$$eGFR = 141 \times \left(\frac{Scr}{\kappa}\right)^\alpha \times 0.993^{Age} \times (1.018 \text{ if female})$$

$$\kappa = \begin{cases} \text{male: } 79.6 \\ \text{female: } 61.9 \end{cases}$$

$$\text{if } \left(\frac{Scr}{\kappa}\right) \leq 1, \text{ then } \alpha = \begin{cases} \text{male: } -0.411 \\ \text{female: } -0.329 \end{cases}$$

$$\text{if } \left(\frac{Scr}{\kappa}\right) > 1, \text{ then } \alpha = -1.209$$

4.7 Statistical Analyses

All statistical analyses were performed on SAS, version 9.3 for studies I and II, and version 9.4 for studies III and IV (SAS Institute Inc, Cary, North Carolina, USA), with the exception of analyses involving Steiger's test in study III, which were conducted using the COCOR package in R software, version 3.2.2.¹⁵⁰ *P*-values of <0.05 were considered statistically significant.

In Study I, sampling weights were used to adjust for non-response and over-sampling in the Health 2011 study, similarly to the original study population drawn from the population register for the Health 2000 study. The PROC SURVEYFREQ command was used to calculate the prevalence, awareness, treatment and control rates of hypertension for the entire population and various subgroups. Chi-square test was used to compare the between-group differences in frequencies, while mean values for population characteristics were calculated using the PROC SURVEYMEANS command. Furthermore, the PROC SURVEYLOGISTICS command was used for logistic regression models to assess the associations between various factors and prevalence, awareness, treatment and control of hypertension. The kappa coefficient was utilised to study the agreement between office and home BP measurements for the prevalence and control rates of hypertension.¹⁵¹

Similarly to Study I, the Chi-square test was used to compare differences in categorical variables between categories in Study II. For comparison of differences in continuous variables between BP categories, analysis of variance was used. The pairwise analyses were adjusted with the Tukey–Kramer method.¹⁵² The Mantel–Haenszel chi-square calculations were performed to test trends in incidence of sustained hypertension by BP category and baseline level.¹⁵³ To study the relative risks for progression from white-coat and masked hypertension to sustained hypertension, log-binomial regression modelling was used. Furthermore, regression models were adjusted for other hypertension risk factors (sex, age, smoking, BMI, diabetes, and hypercholesterolemia).

In study III, the associations between changes in home and office BP, and changes in the indices of ECG-LVH were studied using Pearson's correlation coefficients.¹⁵⁴ The comparison of these correlations was performed with Steiger's modification of Dunn and Clark's *Z* test to assess which of the BP measurement methods associated more strongly with changes in ECG-LVH indices.¹⁵⁵ The independent associations between changes in home or office BP and changes in ECG-LVH indices were examined using multivariable linear regression models. The differences between associations of BP changes and ECG-LVH index between the two BP measurement methods were examined through mixed regression model analyses. The reported interaction *P*-values obtained from the mixed regression model analyses for the interaction term for change in BP and BP measurement method describe the significances of differences between the associations of change in home and office BP with changes in ECG-LVH indices. All regression models were adjusted for age, sex, BMI, corresponding baseline BP, and corresponding ECG-LVH at baseline. To investigate the impact of increasing number of home BP measurements on the association between changes in home BP and ECG-LVH indices, the correlations between ECG-LVH indices and home BPs based on the mean of an increasing number of measurement days were examined.

In study IV, the associations of baseline office and home BP with baseline albuminuria and incident decreased eGFR were analysed using logistic regression. Linear regression was used to assess the association between baseline office and home BP with a change in eGFR from baseline to follow-up. Both home and office BP were included as covariates in all models. Other covariates for the adjustment of the multivariable models were baseline age, gender, diabetes, and smoking, with additional inclusion of baseline eGFR as a covariate in longitudinal models.

5 Results

5.1 Study Sample Characteristics

The characteristics available across all the study samples for studies I–IV are presented in **Table 8** while study-specific characteristics are presented in separate tables.

All study samples were from the Health 2000 study, and its follow-up, the Health 2011 study. However, the sample characteristics differed between studies to some extent due to varying inclusion and exclusion criteria. Additionally, in study I, the characteristics were calculated as weighted means to take into account the oversampling of elderly subjects and to maintain the representativeness with respect to the Finnish adult population. Despite these differences, the mean baseline age for each study was very similar, around 55 years. Studies II and III, which excluded participants with

Table 8. Sample Characteristics for Studies I–IV.

CHARACTERISTIC		STUDY I	STUDY II	STUDY III		STUDY IV	
		Health 2011	ALL	Health 2000	Health 2011	Cross-sectional analyses	Longitudinal analyses
STUDY POPULATION	n	4230	944	615	615	2009	1268
AGE, Y	mean (SD)	55.2 (13.8)	54.1 (7.4)	53.7 (7.2)	64.7 (7.2)	56.4 (8.5)	55.2 (7.7)
MEN	n (%)	1883 (44.5)	432 (45.8)	260 (42.2)		929 (46.2)	567 (44.7)
HYPERCHOLESTEROLEMIA	n (%)		225 (23.8)	137 (22.3)			
DIABETES	n (%)		23 (2.4)	15 (2.4)			
BMI, KG/M²	mean (SD)	27.1 (4.8)	26.7 (3.9)	26.0 (3.7)	26.3 (4.0)	27.4 (4.5)	27.3 (4.4)
CIGARETTE SMOKING	n (%)	814 (19.2)	210 (22.3)	140 (22.8)			

SD, standard deviation.

antihypertensive medication, comprised slightly younger and leaner individuals with mean (SD) BMI of 26.7 (3.9) kg/m² and 26.0 (3.7) kg/m², respectively, compared to the mean BMI of 27.1 (4.8) kg/m² in study I. Study IV included those with antihypertensive medication, individuals with higher probability for other comorbidities including obesity, resulting in a mean BMI of 27.4 kg/m². During follow-up in Study III, BMI increased only slightly from 26.0 kg/m² to 26.3 kg/m². From study to study, the variation in sex distribution was within a few percent, with a minimum of 42.2% men in Study II and maximum of 46.2% men in Study IV.

5.1.1 Study I

The basic characteristics for the study sample in Study I are presented in **Table 8**, and the more detailed socioeconomic and demographic characteristics are presented in **Table 9**.

The Study I sample maintained the representativeness of the sociodemographic composition of the Finnish adult population in 2011 due to use of sample weights in the statistical analyses.

Roughly half of the study population were either current or past smokers, whereas only 15.6% did not use alcohol. Most of the study population, 80.0%, used alcohol moderately, while 4.4% were classified as heavy users. The mean weekly alcohol consumption was 65 g/week with a standard deviation of 204 g/week indicating a very high variability. For physical activity, for which the four-point Gothenburg scale was used, 27.2% of the population were characterised as having minimal physical activity, while 52.3% had average and 20.5% had above average physical activity.

Regarding the socioeconomic characteristics, 73.9% were currently married and 16.4% had been married. Most of the study population had either vocational training (45.0%) or an education corresponding to a bachelor's degree (30.3%), while 13.6% had continued their education to a master's degree or higher. Elementary school was the highest education for 11.1% of the study population. Almost all of the study participants were currently employed (60.5%) or retired (35.3%), while only 4.2% were unemployed.

The hypertension characteristics for this study sample were calculated using office BP, as home BP was not available for all participants. Approximately half of the sample (47.1%) had hypertension, based on elevated BP or current use of antihypertensive medication. Of those with hypertension, 56.5% were aware of their condition, and 50.9% were receiving treatment. Among treated individuals, 48.3% achieved BP control, defined as office BP lower than 140/90 mmHg.

Although many diseases include a regular contact to a health care provider, only 642 individuals of the study sample (15.2%) had visited a health care provider during the preceding year.

5.1.1.1 Agreement between Office and Home Measurements

For the 1,107 participants who had home BP measurements available, the agreement in prevalence and control rates for hypertension, based on either office or home measurements, were assessed. This subsample consisted of 44.5% men (n=493), with a mean (SD) BMI of 27.5 (4.0) kg/m². Their mean (SD) age was 65.8 (6.8) years. This difference in mean age is due to the inclusion criteria of the Finn-Home subsample of the original baseline Health 2000 study, in which only individuals aged 45–74 years were invited to participate.

Table 9. Detailed Study Sample Characteristics for Study I.

CHARACTERISTIC		HEALTH 2011
CIGARETTE SMOKING, N (%)	Never	2,263 (53.5)
	Former	1,153 (27.3)
	Current	814 (19.2)
ALCOHOL USE, N (%)	No	660 (15.6)
	Moderate	3,384 (80.0)
	Heavy	186 (4.4)
ALCOHOL CONSUMPTION, MEAN (SD), G/WK		65.6 (204.2)
PHYSICAL ACTIVITY, N (%)	Minimal	1,149 (27.2)
	Average	2,213 (52.3)
	Above average	868 (20.5)
MARITAL STATUS, N (%)	Never	411 (9.7)
	Former	693 (16.4)
	Current	3,126 (73.9)
EDUCATION, N (%)	Elementary School	470 (11.1)
	Vocational Training	1,902 (45.0)
	Bachelor's Degree	1,282 (30.3)
	Master's or Higher	576 (13.6)
OCCUPATIONAL STATUS, N (%)	Employed	2,560 (60.5)
	Unemployed	177 (4.2)
	Retired	1,493 (35.3)
HYPERTENSION, N (%)	Prevalence	1,993 (47.1)
	Aware of hypertension	1,126 (56.5)
	Antihypertensive medication	1,015 (50.9)
	Under control with treatment	490 (48.3)
≤12 MONTHS FROM VISIT TO HEALTH CARE PROVIDER, N (%)		642 (15.2)

BMI, body mass index; SD, Standard Deviation.

The prevalence rates of hypertension defined using office and home BP measurements were 62.6% and 56.4%, respectively. This resulted in a kappa coefficient of 0.71 (95% CI: 0.66–0.75), indicating substantial agreement. For hypertension control, the corresponding rates were 48.6% and 48.1%, respectively. This resulted in a kappa coefficient of 0.42 (95% CI: 0.33–0.51), indicating moderate agreement.

5.1.2 Study II

In Study II, the 944 participants had no antihypertensive medication at baseline. The mean (SD) age of the sample was 54.1 (7.4) years. The mean (SD) follow-up time was 11.0 (0.1) years. At baseline, 55.9% (n=528) of the participants were normotensive, 6.7% (n=63) had masked hypertension, 15.0% (n=142) were classified as having white-coat hypertension, and 22.4% (n=211) had sustained hypertension. The characteristics for the sample of Study II are detailed in **Table 10**.

Table 10. Detailed Study Sample Characteristics for Study II.

CHARACTERISTIC	ALL	NT (1)	WCHT (2)	MHT (3)	SHT (4)	P<0.05 FOR PAIR-WISE DIFFERENCE	P-VALUE FOR OVERALL DIFFE- RENCE
STUDY POPULATION, N (%)	944 (100)	528 (55.9)	142 (15.0)	63 (6.7)	211 (22.4)		
BASELINE							
AGE, YEARS, MEAN (SD)	54.1 (7.4)	52.8 (6.8)	55.3 (7.8)	54.9 (7.8)	56.2 (7.7)	1–2, 1–4	<0.0001
SEX, MALE, N (%)	432 (45.8)	206 (39.0)	73 (51.4)	37 (58.7)	116 (55.0)		<0.0001
HC, N (%)	225 (23.8)	112 (21.2)	32 (22.5)	17 (27.0)	64 (30.3)		0.06
DIABETES, N (%)	23 (2.4)	3 (0.6)	2 (1.4)	5 (7.9)	13 (6.2)		NA
BMI, KG/M², MEAN (SD)	26.6 (3.9)	25.5 (3.5)	26.6 (3.3)	28.5 (4.2)	29.9 (4.0)	1–3, 1–4, 2–3, 2–4	<0.0001
CURRENT SMOKING, N (%)	210 (22.3)	126 (23.9)	27 (19.0)	19 (30.2)	38 (18.0)		0.11

HC, hypercholesterolemia; BMI, body mass index; SBP, systolic blood pressure. DBP, diastolic blood pressure. NT, normotensives. WCHT, group of white-coat hypertensives. MHT, group of masked hypertensives. SHT, group of sustained hypertensives.

While age and BMI at baseline were significantly different between hypertension categories (normotension, white-coat hypertension, masked hypertension, and sustained hypertension) and increased correspondingly, no statistically significant differences were observed for smoking, diabetes and hypercholesterolemia even though they showed a trend similar to age and BMI. The baseline mean age (SD) for the BP categories were 52.8 (6.8) years for the normotensives, 55.3 (7.8) years for white-coat hypertensives, 54.9 (7.8) years for masked hypertensives, and 56.2 (7.7) years for those with sustained hypertension. Regarding sex distribution, 39.0% of normotensives were male, while the categories of white-coat hypertensives, masked hypertensives, and sustained hypertensives had 51.4%, 58.7%, and 55.0% of male participants, respectively.

To examine if the representativeness was maintained during the follow-up period, baseline age and sex were compared to those who were still alive in 2011 but did not participate in the follow-up examinations (n=704). Nonparticipants were found to be slightly older with mean age (SD) of 56.7 (9.1) vs. 54.1 (7.4) years ($P<0.001$), while no statistically significant difference was found for sex distribution (45.8% men in participants vs. 44.5% in nonparticipants; $P=0.60$).

The baseline BP values are represented in detail in **Table 11**. At baseline, the mean (SD) home systolic BP was 123.8 (17.0) mmHg and home diastolic BP was 78.0 (9.0) mmHg, and for office BP the means (SD) were 132.3 (18.8) and 82.3 (10.4), respectively. Regarding the group of subjects classified in the normotensives, all the mean BPs were lower in comparison to the overall mean values. For the white-

Table 11. Blood Pressure Related Characteristics of Study II Sample at Baseline.

CHARACTERISTIC	ALL	NT (1)	WCHT (2)	MHT (3)	SHT (4)	P<0.05 FOR PAIR- WISE DIFFEREN CE	P-VALUE FOR OVERALL DIFFEREN CE
HOME SBP, MMHG, MEAN (SD)	123.8 (17.0)	113.2 (9.9)	123.7 (7.3)	139.5 (8.5)	145.9 (12.2)	1-2-3-4	<0.0001
HOME DBP, MMHG, MEAN (SD)	78.0 (9.0)	72.8 (5.8)	77.6 (4.9)	85.5 (5.4)	89.1 (6.7)	1-2-3-4	<0.0001
OFFICE SBP, MMHG, MEAN (SD)	132.3 (18.8)	120.3 (10.6)	146.1 (10.1)	130.2 (7.1)	153.9 (16.2)	1-2-3-4	<0.0001
OFFICE DBP, MMHG, MEAN (SD)	82.3 (10.4)	76.7 (7.2)	88.0 (7.9)	82.1 (5.5)	92.8 (9.4)	1-2-3-4	<0.0001

SBP, systolic blood pressure. DBP, diastolic blood pressure. NT, normotensives. WCHT, group of white-coat hypertensives. MHT, group of masked hypertensives. SHT, group of sustained hypertensives.

coat hypertensives, the home BP values were higher than normotensives, but lower than overall mean, whereas office BP was higher both for systolic and diastolic BP. Masked hypertensives had both home systolic and diastolic BP higher than the overall means, while office BP measurement provided a lower than overall average. The group of sustained hypertensives had a higher than overall mean result in home and office, and systolic and diastolic BP.

The follow-up BP characteristics are represented in detail in **Table 12**. During follow-up the systolic BP means for normotensives, white-coat hypertensives and the masked hypertensives increased in both home and office BP measurements. Diastolic values remained the same or decreased slightly for normotensives, white-coat hypertensives and overall, but increased in the masked hypertensives and sustained hypertensives groups. While sustained hypertensives had higher diastolic BP at follow-up, their systolic BP was lower at follow-up compared to baseline. This is likely due to the increase in antihypertensive medication from baseline to follow-up. The proportion of individuals with antihypertensive treatment initiated during follow-up increased from normotensives (9.5%) to white-coat hypertensives (21.8%), to masked hypertensives (49.2%) and to sustained hypertensives (57.8%).

Table 12. Blood Pressure Related Characteristics of Study II Sample at Follow-up.

CHARACTERISTIC	ALL	NT (1)	WCHT (2)	MHT (3)	SHT (4)	P<0.05 FOR PAIR-WISE DIFFERENCE	P-VALUE FOR OVERALL DIFFERENCE
HOME SBP, MMHG, MEAN (SD)	129.3 (14.2)	123.3 (12.0)	133.1 (11.2)	137.3 (13.7)	139.5 (13.5)	1-2, 1-3, 1-4, 2-4	<0.0001
HOME DBP, MMHG, MEAN (SD)	74.8 (7.8)	72.6 (6.9)	76.2 (7.4)	78.0 (8.0)	78.5 (8.2)	1-2, 1-3, 1-4, 2-4	<0.0001
OFFICE SBP, MMHG, MEAN (SD)	136.3 (18.1)	130.2 (14.8)	144.8 (16.6)	140.1 (19.1)	144.9 (20.1)	1-2, 1-3, 1-4	<0.0001
OFFICE DBP, MMHG, MEAN (SD)	79.6 (9.7)	77.8 (8.2)	82.3 (10.3)	80.7 (10.8)	81.7 (11.3)	1-2, 1-4	<0.0001
ANTIHYPERTENSIVE TREATMENT, N (%)	234 (24.8)	50 (9.5)	31 (21.8)	31 (49.2)	122 (57.8)		NA

SBP, systolic blood pressure. DBP, diastolic blood pressure. NT, normotensives. WCHT, group of white-coat hypertensives. MHT, group of masked hypertensives. SHT, group of sustained hypertensives.

5.1.3 Study III

After exclusions due to missing data or antihypertensive medication either at baseline or follow-up, 615 participants had home BP measurements and ECG data available for analyses. The basic study sample characteristics are presented in **Table 8**, while the detailed BP and ECG characteristics are presented in **Table 13** and **Table 14**, respectively.

During the 11-year follow-up period in Study III, the mean (SD) BMI increased from 26.0 (3.7) kg/m² to 26.3 (4.0) kg/m², indicating only a slight increase in body weight.

At baseline, men had a higher systolic and diastolic BP in both home and office measurements compared to women. This difference remained during the follow-up. However, BP increases during follow-up were greater in men than in women. The mean (SD) home systolic BP increased 6.5 (11.7) mmHg in men and 10.1 (10.2) mmHg in women. For office systolic BP, the increases were 5.5 (16.0) mmHg for men and 9.5 (16.4) mmHg for women. Home diastolic BP remained the same or decreased slightly during follow-up, with a change of -2.0 (6.9) mmHg for men and

Table 13. ECG-Related Characteristics of the Study III Population at Baseline and Follow-up.

CHARACTERISTIC, MEAN (SD)	ALL, N=615		MEN, N=260		WOMEN, N=355	
	2000	2011	2000	2011	2000	2011
SOKOLOW-LYON VOLTAGE INDEX, MM	24.2 (7.2)	23.0 (7.3)	26.3 (7.7)	24.1 (8.0)	22.7 (6.3)	22.3 (6.7)
CHANGE, MM		-1.2 (4.8)		-2.3 (5.2)		-0.4 (4.3)
CORNELL VOLTAGE INDEX, MM	17.6 (5.3)	18.5 (5.7)	16.1 (5.8)	16.5 (5.9)	18.6 (4.6)	20.0 (5.0)
CHANGE, MM		0.9 (3.3)		0.4 (3.3)		1.3 (3.1)
CORNELL PRODUCT INDEX, MM X MS	1580.1 (547.3)	1724.9 (604.1)	1541.6 (634.6)	1636.7 (665.3)	1608.4 (472.2)	1789.5 (547.1)
CHANGE, MM X MS		144.8 (360.7)		95.1 (392.1)		181.2 (331.8)
RAVL VOLTAGE INDEX, MV	0.43 (0.29)	0.56 (0.33)	0.49 (0.31)	0.59 (0.33)	0.38 (0.27)	0.54 (0.32)
CHANGE, MV		0.13 (0.18)		0.10 (0.19)		0.15 (0.17)

The change in each ECG-LVH criteria were calculated subtracting the baseline value from the follow-up value.

-0.6 (6.2) mmHg for women. For diastolic BP, the corresponding changes were -2.1 (10.2) mmHg for men and 0.5 (9.8) mmHg for women.

At baseline, the mean ECG-LVH indices were higher in men than women for Sokolow-Lyon and RaVL, whereas women had a higher mean Cornell voltage and Cornell product. These differences remained at the end of follow-up.

During follow-up, the change in mean (SD) Sokolow-Lyon in the whole study sample was -1.2 (4.8) mm. This change was more substantial in men with a mean change (SD) of -2.3 (5.2) mm, whereas in women the observed change was -0.4 (4.3) mm. For Cornell voltage, the mean (SD) change was 0.9 (3.3) mm for all. This change was less pronounced in men than in women, with mean (SD) changes of 0.4 (3.3) mm in men, compared to 1.3 (3.1) mm in women. Similar trends were observed for Cornell product and RaVL voltage. For the Cornell product, the change in mean (SD) was 144.8 (360.7) mm×ms for all, 95.1 (392.1) mm×ms for men, and 181.2 (331.8) mm×ms for women. For RaVL voltage, the mean (SD) change for the whole study sample was 0.13 (0.18) mV, 0.10 (0.19) mV for men and 0.15 (0.17) mV for women.

Table 14. Blood Pressure-Related Characteristics of the Study III Sample at Baseline and Follow-up.

CHARACTERISTIC, MMHG, MEAN (SD)	ALL, N=615		MEN, N=260		WOMEN, N=355	
	2000	2011	2000	2011	2000	2011
HOME SYSTOLIC BP	118.5 (14.0)	127.1 (14.0)	123.6 (12.7)	130.1 (13.7)	114.8 (13.7)	124.9 (13.7)
Δ HOME SYSTOLIC BP		8.6 (11.0)		6.5 (11.7)		10.1 (10.2)
HOME DIASTOLIC BP	75.3 (7.4)	74.1 (7.7)	78.1 (7.0)	76.1 (7.8)	73.3 (7.0)	72.7 (7.3)
Δ HOME DIASTOLIC BP		-1.2 (6.5)		-2.0 (6.9)		-0.6 (6.2)
OFFICE SYSTOLIC BP	127.6 (16.4)	135.3 (17.7)	130.1 (15.4)	135.6 (16.9)	125.7 (17.0)	135.1 (18.2)
Δ OFFICE SYSTOLIC BP		7.8 (16.4)		5.5 (16.0)		9.5 (16.4)
OFFICE DIASTOLIC BP	79.9 (9.3)	79.3 (9.3)	82.6 (8.9)	80.5 (9.9)	77.9 (9.1)	78.4 (8.8)
Δ OFFICE DIASTOLIC BP		-0.6 (10.0)		-2.1 (10.2)		0.5 (9.8)

BP, Blood Pressure. The change in each BP variable was calculated subtracting the baseline value from the follow-up value.

5.1.4 Study IV

For Study IV, 2,009 participants were included in the cross-sectional analyses, and 1,268 (63.1%) participants were included in the longitudinal analyses after exclusions. The lower number of participants included in the longitudinal analyses was mainly due to non-participation in the follow-up study, which was most likely due to death, higher age and higher morbidity. This resulted in a selection of slightly younger, and most likely healthier sample. The basic characteristics for these study samples are presented in **Table 8**, while BP- and kidney function-related characteristics are presented in **Table 15**.

The study sample included in the cross-sectional analyses had a mean (SD) age of 56.4 (13.8) years, whereas the sample included in the longitudinal analyses was slightly younger with a mean (SD) age of 55.2 (7.7) years. The study sample included in the longitudinal analyses, as compared with the cross-sectional sample, had fewer men (44.7% vs. 46.2%) and was slightly leaner (mean (SD) BMI of 27.3 (4.4) vs. 27.4 (4.5) kg/m²).

Table 15. Blood Pressure and Kidney Function Related Characteristics of the Study IV Sample at Baseline.

CHARACTERISTIC, MEAN (SD)	CROSS-SECTIONAL ANALYSES (N=2,009)	LONGITUDINAL ANALYSES (N=1,268)
HOME SBP, MMHG	129.9 (18.6)	127.2 (17.7)
HOME DBP, MMHG	80.3 (9.2)	79.5 (9.1)
OFFICE SBP, MMHG	137.5 (20.0)	135.5 (19.4)
OFFICE DBP, MMHG	83.8 (10.6)	83.6 (10.3)
SERUM CREATININE, μ MOL/L	72.2 (14.1)	71.5 (12.4)
URINE CREATININE, MMOL/L	9.2 (5.2)	9.1 (4.9)
URINE ALBUMIN EXCRETION, MG/L	17.9 (135.1)	7.6 (37.5)
URINE ALBUMIN-CREATININE RATIO, MG/MMOL	2.0 (13.1)	0.9 (3.5)
MEAN EGFR, ML/MIN/1.73 M ²	90.8 (13.5)	Baseline: 91.9 (12.1) Follow-up: 82.5 (14.1)

BP, blood pressure; SBP, systolic BP; DBP, diastolic BP; eGFR, estimated glomerular filtration rate.

The study sample included in the longitudinal analyses had lower mean office and home BP compared to the cross-sectional sample. The same trend was visible also for eGFR and urine albumin-creatinine ratio.

5.2 Social, Lifestyle, and Demographic Inequalities in Hypertension Care (Study I)

The prevalence, awareness, treatment and control rates of office hypertension in subgroups, along with age- and sex-adjusted, and multivariable-adjusted odds ratios and 95% confidence intervals are presented in **Table 16**, **Table 17**, **Table 18**, and **Table 19**, respectively. Only the variables with significant differences in the univariable analyses were included in the multivariable models for prevalence, awareness, treatment, and control of hypertension, respectively.

5.2.1 Factors Associated with Prevalence of Office Hypertension

The detailed results regarding factors associated with prevalence of office hypertension are presented in **Table 16**.

In the univariable analyses, participants who were older, male, divorced or widowed, less educated, unemployed or retired, obese, non-smokers, or physically inactive had more hypertension than their counterparts. Additionally, moderate alcohol consumption was associated with a lower hypertension prevalence.

In the age- and sex-adjusted analyses, those with an education of a master's degree or higher and those with an above average physical activity were less likely to have hypertension. In contrast, unemployment and retirement, being overweight or obese, and heavy alcohol consumption were associated with greater odds of hypertension.

In the multivariable analyses, higher age and male gender were strongly associated with greater odds of hypertension. The same observation was made for being retired, overweight, obese, and with heavy alcohol consumption. An education level of a master's degree or higher was associated with lower odds of hypertension. Surprisingly, former cigarette smoking was associated with lower odds of hypertension. Physical activity was not significantly associated with odds of hypertension in the multivariable analysis.

Table 16. Socioeconomic Status and Lifestyle Factors Association with Hypertension Prevalence; Univariate, Age- and Sex-adjusted and Multivariate Analyses.

	UNIVARIATE %	AGE&SEX ADJUSTED OR (95% CI)	MULTIVARIATE OR (95% CI)
AGE, YEARS	***		
<55	29.7		1.00
55 -- 75	61.9		2.51 (2.07–3.05) ***
>75	66.7		2.41 (1.65–3.52) ***
GENDER	***		
MALE	49.4		1.39 (1.19–1.61) ***
FEMALE	42.8		1.00
MARRIED	***		
NEVER	39.0	1.00	1.00
FORMER	55.1	0.84 (0.63–1.12)	1.16 (0.86–1.57)
CURRENT	44.8	0.96 (0.77–1.19)	1.19 (0.94–1.51)
EDUCATION	***		
ELEMENTARY SCHOOL	57.9	1.00	1.00
VOCATIONAL TRAINING	50.6	1.22 (0.95–1.56)	1.12 (0.87–1.45)
BACHELOR'S DEGREE	39.6	1.07 (0.82–1.39)	1.07 (0.80–1.42)
MASTER'S OR HIGHER	31.1	0.68 (0.51–0.89) **	0.74 (0.55–1.00) *
EMPLOYMENT	***		
EMPLOYED	34.1	1.00	1.00
UNEMPLOYED	45.5	1.41 (1.04–1.92) *	1.21 (0.87–1.69)
RETIRED	67.1	1.29 (1.06–1.58) *	1.99 (1.64–2.41) ***
BODY MASS INDEX	***		
NORMAL	28.6	1.00	1.00
OVERWEIGHT	48.5	2.03 (1.75–2.35) ***	2.05 (1.78–2.37) ***
OBESE	68.6	5.15 (4.26–6.23) ***	5.04 (4.12–6.15) ***
CIGARETTE SMOKING	**		
NEVER	46.7	1.00	1.00
FORMER	48.3	1.00 (0.84–1.18)	0.79 (0.66–0.95) *
CURRENT	40.5	1.04 (0.86–1.25)	0.85 (0.70–1.03)
ALCOHOL USE	***		
NO	56.3	1.00	1.00
MODERATE	43.0	0.96 (0.80–1.17)	0.93 (0.76–1.15)
HEAVY	59.4	2.00 (1.39–2.87) ***	1.85 (1.23–2.80) **
PHYSICAL ACTIVITY	***		
MINIMAL	50.3	1.00	1.00
AVERAGE	47.9	0.88 (0.74–1.04)	1.07 (0.90–1.27)
ABOVE AVERAGE	34.7	0.64 (0.52–0.80) ***	0.80 (0.64–1.01)
≤12 MO FROM VISIT TO HCP	NA	NA	NA

NA, not available; *, $P<0.05$; **, $P<0.01$; ***, $P<0.001$ for statistical significance between groups in univariate analysis; OR (95% CI), odds ratio (95% confidence interval); HCP, health care provider.

5.2.2 Factors Associated with Awareness of Office Hypertension

Of participants with hypertension, 56.5% were aware of their condition (**Table 9**). The detailed results regarding factors associated with awareness of office hypertension are presented in **Table 17**.

No statistically significant difference in hypertension awareness was observed between groups by different educational levels. Additionally, information on recent visits to a health care provider was not available for analysis. All other analysed variables were significantly associated with hypertension awareness. In univariate analyses, younger participants, and especially those under 55 years, were less aware of their hypertension compared to participants in the 55–75 and over 75 age ranges. Men and those who were never married, of normal weight, current smokers, heavy alcohol users, or had an above average physical activity were less often aware of their hypertension as compared with their counterparts. Divorce, retirement, obesity, alcohol abstinence, and low physical activity was associated with increased hypertension awareness.

In the age- and sex-adjusted analyses, those who were retired, overweight, or obese had greater odds of hypertension. In contrast, current smoking, average physical activity, and above average physical activity were associated with lower odds of hypertension awareness.

In the multivariable analyses, older age was strongly linked to higher odds of hypertension awareness, while male sex was associated with lower odds of awareness. Marital status did not reach statistical significance. For employment status, only retirement correlated with higher odds of awareness. Being overweight and obese were both strong correlates of higher odds of hypertension awareness. Current smoking was also significantly associated with lower odds of hypertension awareness. Alcohol use was not associated with hypertension awareness, whereas those with an average or an above average physical activity had lower odds of hypertension awareness.

Table 17. Socioeconomic Status and Lifestyle Factors Association with Hypertension Awareness; Univariate, Age- and Sex-adjusted and Multivariate Analyses.

	UNIVARIATE %	AGE&SEX ADJUSTED OR (95% CI)	MULTIVARIATE OR (95% CI)
AGE, YEARS	***		
<55	39.2		1.00
55 -- 75	62.4		1.84 (1.36–2.38) ***
>75	71.9		2.17 (1.38–3.43) **
GENDER	***		
MALE	50.9		0.78 (0.65–0.94) **
FEMALE	61.1		1.00
MARRIED	***		
NEVER	51.2	1.00	1.00
FORMER	66.5	0.94 (0.62–1.43)	1.17 (0.77–1.75)
CURRENT	53.6	0.85 (0.61–1.18)	0.94 (0.68–1.31)
EDUCATION			
ELEMENTARY SCHOOL	62.3	1.00	1.00
VOCATIONAL TRAINING	55.6	1.10 (0.82–1.49)	
BACHELOR'S DEGREE	53.7	1.17 (0.87–1.57)	NA
MASTER'S OR HIGHER	53.2	1.08 (0.71–1.65)	
EMPLOYMENT	***		
EMPLOYED	43.9	1.00	1.00
UNEMPLOYED	48.7	1.20 (0.77–1.87)	1.19 (0.75–1.9)
RETIRED	67.7	1.38 (1.01–1.87) *	1.67 (1.27–2.2) ***
BODY MASS INDEX	***		
NORMAL	45.4	1.00	1.00
OVERWEIGHT	56.6	1.70 (1.29–2.23) ***	1.60 (1.22–2.11) ***
OBESE	62.0	2.34 (1.76–3.10) ***	1.99 (1.5–2.62) ***
CIGARETTE SMOKING	***		
NEVER	58.9	1.00	1.00
FORMER	58.5	1.08 (0.88–1.33)	1.03 (0.84–1.26)
CURRENT	43.1	0.77 (0.59–0.99) *	0.70 (0.53–0.92) *
ALCOHOL USE	***		
NO	64.9	1.00	1.00
MODERATE	54.2	0.90 (0.69–1.17)	0.94 (0.72–1.23)
HEAVY	48.2	0.85 (0.54–1.35)	0.85 (0.52–1.39)
PHYSICAL ACTIVITY	***		
MINIMAL	62.5	1.00	1.00
AVERAGE	54.6	0.65 (0.53–0.80) ***	0.66 (0.53–0.83) ***
ABOVE AVERAGE	47.7	0.58 (0.44–0.77) ***	0.62 (0.46–0.85) **
≤12 MO FROM VISIT TO HCP	NA	NA	NA

NA, not available; *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$ for statistical significance between groups in univariate analysis; OR (95% CI), odds ratio (95% confidence interval); HCP, health care provider.

5.2.3 Factors Associated with Treatment of Office Hypertension

Altogether 50.9% of participants with hypertension were on antihypertensive medication. Data on recent visits to a health care provider were not available, but all other examined variables were significantly associated with hypertension treatment rates. The detailed results regarding factors associated with treatment of office hypertension are presented in **Table 18**.

In the univariate analyses, men and those under 55 years were less often treated for their hypertension than their counterparts. Those who were either never or currently married were less often treated for their hypertension compared with divorced individuals. Regarding education, a higher education level was related to a lower rate of hypertension treatment. Regarding employment status, those who were employed had the lowest rate of hypertension treatment (36.5%), while 63.9% of those who were retired were treated for hypertension. Participants with a normal BMI had a lower-than-average treatment rate (40.1%), while overweight individuals had an average treatment rate, and obese participants had an above average treatment rate (56.8%). Current smokers had a lower-than-average treatment rate of 37.4%, while alcohol abstainers were more often treated for hypertension than moderate or heavy users. A similar trend was observed for physical activity as 57.1% of participants with minimal activity were treated for hypertension, whereas 48.7% and 42.7% of those with average and above average physical activity were treated for their hypertension.

In the age- and sex-adjusted analyses, being retired had an odds ratio (95% confidence interval, CI) of 1.45 (1.07-1.95) for being treated for hypertension compared to those who were currently employed. Those who were overweight or obese were also more likely to be treated for their hypertension with odds ratios (95% CI) of 1.66 (1.27-2.17) and 2.41 (1.83-3.18), respectively. In contrast, an increasing physical activity was linked with lower odds of being treated, with odds ratios of 0.64 (0.51-0.79) for average physical activity and an odds ratio of 0.60 (0.45-0.79) for above average physical activity.

In the multivariable analyses, increasing age and BMI were associated with increased odds of hypertension treatment. Retirement was also observed to be linked with increased odds of treatment. Current smoking was associated with lower odds of treatment, as was an average or above average level of physical activity.

Table 18. Socioeconomic Status and Lifestyle Factors Association with Hypertension Treatment; Univariate, Age- and Sex-adjusted and Multivariate Analyses.

	UNIVARIATE %	AGE&SEX ADJUSTED OR (95% CI)	MULTIVARIATE OR (95% CI)
AGE, YEARS	***		
<55	31.1		1.00
55 -- 75	57.7		2.17 (1.64–2.88) ***
>75	69.6		2.72 (1.74–4.25) ***
GENDER	***		
MALE	46.2		0.88 (0.73–1.06)
FEMALE	54.6		1.00
MARRIED	***		
NEVER	45.9	1.00	1.00
FORMER	61.6	0.87 (0.58–1.31)	1.10 (0.73–1.65)
CURRENT	47.8	0.80 (0.57–1.11)	0.89 (0.64–1.25)
EDUCATION	***		
ELEMENTARY SCHOOL	59.0	1.00	1.00
VOCATIONAL TRAINING	50.2	1.06 (0.79–1.44)	1.09 (0.81–1.47)
BACHELOR'S DEGREE	47.0	1.11 (0.81–1.51)	1.20 (0.87–1.64)
MASTER'S OR HIGHER	45.5	0.96 (0.63–1.44)	1.13 (0.73–1.74)
EMPLOYMENT	***		
EMPLOYED	36.5	1.00	1.00
UNEMPLOYED	41.5	1.22 (0.78–1.92)	1.18 (0.75–1.87)
RETIRED	63.9	1.45 (1.07–1.95) *	1.82 (1.39–2.38) ***
BODY MASS INDEX	***		
NORMAL	40.1	1.00	1.00
OVERWEIGHT	50.7	1.66 (1.27–2.17) ***	1.56 (1.19–2.05) **
OBESE	56.8	2.41 (1.83–3.18) ***	2.05 (1.56–2.69) ***
CIGARETTE SMOKING	***		
NEVER	53.3	1.00	1.00
FORMER	53.1	1.06 (0.86–1.31)	1.01 (0.81–1.26)
CURRENT	37.4	0.78 (0.59–1.03)	0.71 (0.52–0.97) *
ALCOHOL USE	***		
NO	60.6	1.00	1.00
MODERATE	48.2	0.87 (0.68–1.13)	0.91 (0.71–1.16)
HEAVY	43.8	0.90 (0.56–1.44)	0.87 (0.52–1.45)
PHYSICAL ACTIVITY	***		
MINIMAL	57.1	1.00	1.00
AVERAGE	48.7	0.64 (0.51–0.79) ***	0.65 (0.52–0.82) ***
ABOVE AVERAGE	42.7	0.60 (0.45–0.79) ***	0.64 (0.47–0.85) **
≤12 MO FROM VISIT TO HCP	NA	NA	NA

NA, not available; *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$ for statistical significance between groups in univariate analysis; OR (95% CI), odds ratio (95% confidence interval); HCP, health care provider.

5.2.4 Factors Associated with Control of Office Hypertension

Of the 1,015 participants on antihypertensive medication, a total of 490 (48.3%) participants had their hypertension controlled. The detailed results regarding factors associated with control of office hypertension are presented in **Table 19**.

In the univariable analyses, only age and employment were associated with hypertension control rates. The control rates decreased with an increasing age category, dropping from 56.1% to 48.6% to 37.6%. Regarding employment, a similar decreasing trend of hypertension control was observed from employment (54.7%) to unemployment (49.6%) to retirement (44.7%). Those who had visited a health care provider recently had similar control rates compared to those who had not (47.9% vs. 48.5%).

In the age- and sex-adjusted analyses, none of the variables were significantly linked with hypertension control.

In the multivariable analysis, only age above 75 years was associated with lower odds of hypertension control compared to those under 55 years old. Interestingly, and contrary to other analyses, male sex was associated with greater, but non-significant, odds of hypertension control.

Table 19. Socioeconomic Status and Lifestyle Factors Association with Hypertension Control; Univariate, Age- and Sex-adjusted and Multivariate Analyses.

	UNIVARIATE %	AGE&SEX ADJUSTED OR (95% CI)	MULTIVARIATE OR (95% CI)
AGE, YEARS	**		
<55	56.1		1.00
55 -- 75	48.6		0.84 (0.57–1.25)
>75	37.6		0.57 (0.34–0.97) *
GENDER			
MALE	51.0		1.20 (0.92–1.55)
FEMALE	45.8		1.00
MARRIED			
NEVER	51.5	1.00	
FORMER	41.6	0.93 (0.53–1.61)	NA
CURRENT	50.1	1.05 (0.65–1.69)	
EDUCATION			
ELEMENTARY SCHOOL	42.4	1.00	
VOCATIONAL TRAINING	47.6	1.05 (0.72–1.55)	NA
BACHELOR'S DEGREE	52.5	1.19 (0.77–1.82)	
MASTER'S OR HIGHER	52.6	1.18 (0.66–2.13)	
EMPLOYMENT	*		
EMPLOYED	54.7	1.00	1.00
UNEMPLOYED	49.6	0.85 (0.41–1.77)	0.84 (0.41–1.73)
RETIRED	44.7	1.01 (0.68–1.49)	0.82 (0.57–1.16)
BODY MASS INDEX			
NORMAL	52.9	1.00	
OVERWEIGHT	46.2	0.75 (0.52–1.08)	NA
OBESE	48.1	0.78 (0.55–1.10)	
CIGARETTE SMOKING			
NEVER	45.9	1.00	
FORMER	48.8	1.05 (0.78–1.41)	NA
CURRENT	56.7	1.28 (0.87–1.88)	
ALCOHOL USE			
NO	48.4	1.00	
MODERATE	48.2	0.82 (0.61–1.10)	NA
HEAVY	46.9	1.00 (0.54–1.85)	
PHYSICAL ACTIVITY			
MINIMAL	49.6	1.00	
AVERAGE	47.9	0.96 (0.72–1.26)	NA
ABOVE AVERAGE	45.7	0.75 (0.51–1.11)	
≤12 MO FROM VISIT TO HCP			
YES / NO	47.9 / 48.5	1.00 / 1.08 (0.85–1.37)	NA

NA, not available; *, $P<0.05$; **, $P<0.01$; ***, $P<0.001$ for statistical significance between groups in univariate analysis; OR (95% CI), odds ratio (95% confidence interval); HCP, health care provider.

5.2.5 Summary

In the univariable analyses, older, male, divorced or widowed, less educated, unemployed or retired, obese, non-smoking and physically inactive participants were more likely to have hypertension, while moderate alcohol consumption was associated with a lower prevalence of hypertension. Participants who were younger, men, not married, lean, heavy users of alcohol, current smokers, or had higher physical activity, were less aware of their hypertension and had lower treatment rates. Higher education associated with lower treatment rates, but for those with treatment, hypertension was more often in control. To note, young and employed participants had better control rates.

In age- and sex-adjusted analyses, a master's degree and above-average physical activity were associated with lower probability of hypertension. Those who were retired, unemployed, obese or heavy alcohol consumers had increased probability of having hypertension. Awareness of hypertension was more prevalent among those who were retired and overweight, while physically active participants and smokers were less likely to be aware of their hypertension. No statistically significant results were observed in variables regarding education, marital or smoking status, or alcohol use, as the variation in treatment rates was small. Similarly, variation in hypertension control rates was marginal, and no statistically significant odds ratios were observed.

In the multivariable analyses participants who were older, male, less educated, retired, obese, non-smokers, or heavy alcohol consumers were more likely to have hypertension. Those who were younger, smokers, not retired, had normal body weight or higher physical activity, were less aware and had lower treatment rates. Male sex was associated with a lower treatment rate, while participants older than 75 years of age had lower control rates.

To summarise, the results from the age- and sex-adjusted analyses were mainly identical to the multivariable analyses where higher age, higher BMI, and retirement were strongly linked with increased hypertension prevalence, awareness and treatment rates. Male sex was similarly associated with increased hypertension prevalence and awareness, whereas former smoking and heavy alcohol use correlated with higher hypertension prevalence. Current smoking and increasing physical activity correlated negatively with hypertension awareness and treatment rates.

5.3 White-coat and Masked Hypertension as Risk Factors for Progression to Sustained Hypertension (Study II)

5.3.1 Changes in Blood Pressure Phenotype Categories During Follow-Up

At baseline, the pairwise and overall differences in home and office BPs were statistically significant (**Table 11**). The baseline mean home BP increased from normotension to white-coat hypertension to masked hypertension to sustained hypertension, while the baseline mean office BP increased from normotension to masked hypertension to white-coat hypertension to sustained hypertension.

The distribution of participants at follow-up by baseline BP phenotype categories is illustrated in **Figure 4**. During the follow-up period, 393 participants (41.6%) moved to another BP category, while 551 participants (58.4%) maintained their baseline BP category. The incidence of sustained hypertension increased from 18.2% in baseline normotensives to 52.1% in baseline white-coat hypertensives, and further to 73.0% in baseline masked hypertensives. Notably, 83.4% of those with sustained hypertension at baseline remained as sustained hypertensives at follow-up. The trend for moving to and remaining in sustained hypertension was statistically significant (P for trend <0.0001).

At follow-up (**Figure 4**), the home BP levels differed significantly between the BP phenotypes ($P < 0.05$), except for the differences between masked and white-coat hypertension groups (systolic: $P = 0.10$, diastolic: $P = 0.33$), and between masked and sustained hypertension groups (systolic: $P = 0.63$, diastolic: $P = 0.97$). The differences in mean office BP between follow-up BP categories were statistically significant for

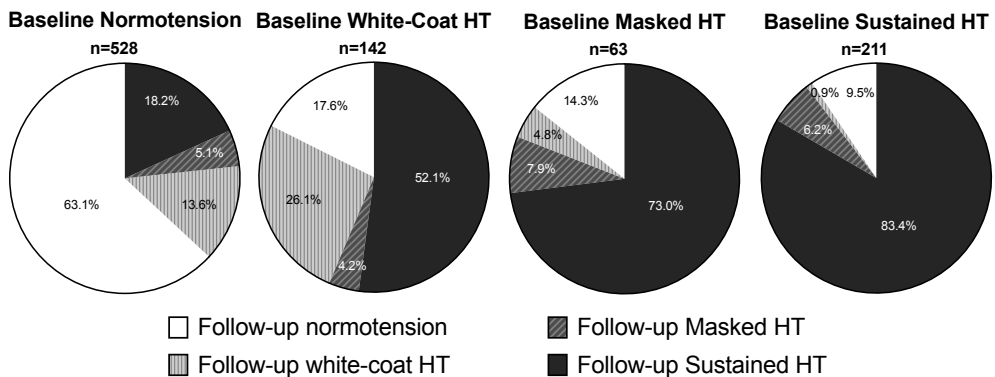


Figure 4. Distribution of Participants in Blood Pressure Categories at Follow-up According to Baseline Category. HT, hypertension. Author's own work.

the following pairs: normotension – white-coat hypertension (systolic and diastolic BP), normotension – masked hypertension (systolic BP), and normotension – sustained hypertension (systolic and diastolic BP). Of those in the sustained hypertension category at follow-up, 234 (59.7%) were on antihypertensive medication.

The progression rates towards sustained hypertension, relative to the baseline home and office BP levels are presented in **Original Publication II, Figure 2 and Supplemental Figure**, respectively. In this analysis, excluding those with sustained hypertension at baseline (n=211), the study population was categorised only by either home or office BP measurements at 10 mmHg increments for systolic BP and at 5 mmHg increments for diastolic BP. With these BP thresholds, altogether 216 participants transitioned to sustained hypertension during follow-up. The proportion of participants progressing to sustained hypertension in each BP level category during follow-up became higher in accordance with increase in the baseline BP level. This trend was also evaluated as statistically significant (P for trend <0.0001).

5.3.2 Risk of Progression to Sustained Hypertension

To examine the risk of progression to sustained hypertension in individuals with normotension, white-coat hypertension, and masked hypertension, a multivariable-adjusted log-binomial model was used. Age, sex, BMI, diabetes, hypercholesterolemia, and smoking were used as baseline covariates. Compared to baseline normotensives, white-coat hypertensives had a relative risk of 2.8 (95% CI 2.2-3.6; $P<0.0001$) and masked hypertensives a relative risk of 3.8 (95% CI 2.9-5.0; $P<0.0001$) to progress to sustained hypertension. The other covariates did not exhibit statistically significant associations. These results are depicted in **Original Publication II, Figure 3**.

5.3.3 Non-fatal Cardiovascular Events during Follow-up

The distribution of nonfatal cardiovascular events during follow-up for each BP category was also investigated. While excluding 36 participants that had suffered a cardiovascular event before baseline, altogether 62 participants suffered a non-fatal major adverse cardiovascular event. Determined by baseline BP category, 12 (2.4%) events occurred for normotensive, 14 (10.4%) for white-coat hypertensive, 10 (16.4%) for masked hypertensive, and 26 (12.7%) for sustained hypertensive participants ($P<0.0001$).

5.4 Home Versus Office Blood Pressure – Longitudinal Relations with Hypertension-Mediated Organ Damage (Studies III and IV)

5.4.1 Home and Office Blood Pressure Changes and Their Relation to Changes in ECG-LVH

The correlations of changes in home and office BP with changes in ECG-LVH indices from baseline to follow-up examination are presented in detail in **Original Publication III, Table 2**. All correlation coefficients between home or office BP and ECG-LVH indices were individually statistically significant ($P < 0.001$ for all systolic and $P \leq 0.004$ for all diastolic correlation coefficients).

For systolic BP, changes in Cornell voltage (Home: 0.28, Office: 0.17, $P_{diff} = 0.007$), Cornell product (Home: 0.25, Office: 0.16, $P_{diff} = 0.03$), and RaVL (Home: 0.32, Office: 0.20, $P_{diff} = 0.003$) correlated more strongly with home BP changes than with office BP changes. No statistically significant differences were observed for Sokolow-Lyon voltage (Home: 0.21, Office: 0.18, $P_{diff} = 0.42$). For changes in diastolic BP, only change in RaVL was more strongly associated with home BP change than with office BP change (Home: 0.28, Office: 0.19, $P_{diff} = 0.02$), whereas the correlations were not different for other ECG-LVH indices ($P_{diff} \geq 0.15$ for all).

Multivariable-adjusted linear regression models were used to analyse the associations between changes in home or office BP and changes in ECG-LVH indices. All models included age, sex, BMI, corresponding baseline BP and ECG-LVH index as covariates. The results are depicted in **Figure 5** and described in detail in **Original Publication III, Table 3**. Changes in both home and office BP were significantly associated with change in ECG-LVH indices across all models ($P \leq 0.009$), except the association between change in diastolic BP and change in Sokolow-Lyon index (home BP: $P = 0.08$, office BP: $P = 0.14$).

In multivariable-adjusted mixed models, change in home systolic BP was more strongly associated with changes in Cornell voltage ($P < 0.001$), Cornell product ($P = 0.004$) and RaVL ($P < 0.001$) than office systolic BP. For diastolic BP, a significant difference in the strength of the associations was only observed for RaVL, in favour of home BP ($P = 0.007$).

As a sensitivity analysis, the same mixed model analyses were repeated using only two home BP measurements from 1) the first morning; or 2) the third morning to define home BP. These analyses indicated that change in home systolic BP of the first measurement day morning associated more strongly with changes in Cornell voltage ($P = 0.005$) and Cornell product ($P = 0.02$) compared with office systolic BP. Furthermore, change in the home systolic BP on the morning of the third

measurement day associated more strongly with changes in Cornell product ($P=0.02$) than office systolic BP. No significant differences were observed for diastolic BP.

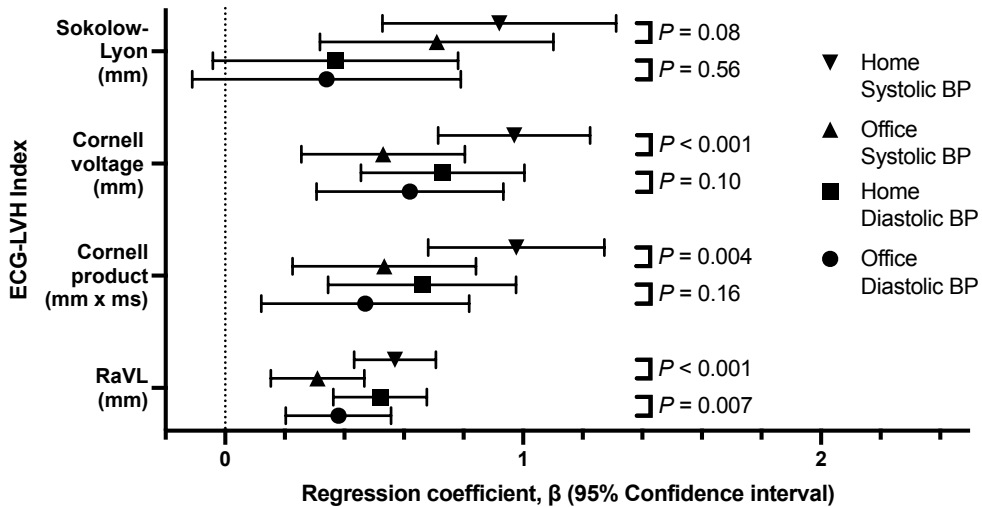


Figure 5. Association between change in blood pressure (BP) and change in electrocardiographically assessed left ventricular hypertrophy (ECG-LVH) index by linear regression analysis. The reported interaction P -values obtained from the mixed regression model analyses for the interaction term for change in BP and BP measurement method describe the significances of differences between the associations of change in home and office BPs with changes in ECG-LVH indices. Cornell product coefficient values are divided by 100 to fit in same scale. Author's own work.

5.4.2 Association between Home BP Change and ECG-LVH Change with an Increasing Number of BP Measurement Days

The correlations between office or home BP change with ECG-LVH index changes were assessed. The results are presented in **Table 20** and visually in **Original Publication III, Figure 1**. For both systolic and diastolic BP, the correlations between change in home BP and changes in ECG-LVH indices became arithmetically greater already after the first or second home BP measurement day in general, stabilising by the fourth day. The correlations were greater for systolic BP compared to diastolic BP.

Table 20. Pearson Correlation Coefficients for Change in Office BP and Home BP, and Change in ECG-LVH Indices.

	ECG-LVH INDEX	OFFICE BP	NUMBER OF HOME BP MEASUREMENT DAYS						
			1	2	3	4	5	6	7
SYSTOLIC	Δ Sokolow-Lyon	0.29	0.25	0.28	0.29	0.29	0.29	0.29	0.29
	Δ Cornell	0.20	0.24	0.25	0.26	0.26	0.27	0.27	0.27
	Δ Cornell product	0.15	0.19	0.19	0.19	0.19	0.19	0.19	0.19
	Δ RaVL	0.22	0.24	0.28	0.30	0.30	0.30	0.30	0.29
DIASTOLIC	Δ Sokolow-Lyon	0.22	0.20	0.21	0.23	0.23	0.23	0.23	0.22
	Δ Cornell	0.17	0.24	0.25	0.26	0.25	0.26	0.25	0.25
	Δ Cornell product	0.13	0.17	0.18	0.18	0.17	0.17	0.17	0.17
	Δ RaVL	0.19	0.24	0.25	0.26	0.25	0.25	0.25	0.25

BP, blood pressure; ECG-LVH, electrocardiographically assessed left ventricular hypertrophy; Δ , change in specific ECG-LVH index.

5.4.3 Association of Home and Office Blood Pressure with Baseline Albuminuria

This cross-sectional analysis of prevalent albuminuria was performed in a sample of 2,009 participants, of whom 143 had albuminuria at baseline. In the unadjusted models, only home systolic BP (OR 1.74; 95% CI 1.40–2.17; $P < 0.001$) and home diastolic BP (OR 1.59; 95% CI 1.28–1.97; $P < 0.001$) were significantly associated with prevalent albuminuria, whereas these associations for office systolic BP (OR 1.23; 95% CI 0.99–1.53; $P = 0.06$) and office diastolic BP (OR 1.01; 95% CI 0.81–1.27; $P = 0.91$) were not. In the multivariable-adjusted models, home systolic BP (OR 1.41; 95% CI 1.11–1.80; $P = 0.006$), home diastolic BP (OR 1.40; 95% CI 1.11–1.76; $P = 0.004$), and office systolic BP (OR 1.40; 95% CI 1.11–1.76; $P = 0.004$) were significantly associated with albuminuria, whereas office diastolic BP was not (OR 1.21; 95% CI 0.96–1.52; $P = 0.10$). The odds ratios and 95% confidence intervals are presented in Figure 6 and additional information is presented in **Original Publication IV, Table 1**.

5.4.4 The Association Between Home and Office Blood Pressure with eGFR change

The incident decrease of eGFR and change in eGFR from baseline to follow-up was analysed for 1,268 participants with normal eGFR at baseline, of which, 100 (7.9%) developed a decline in eGFR to <60 ml/min/1.73 m² during follow-up. Baseline home systolic BP was linked to incident eGFR decrease in the unadjusted models (OR 1.62; 95% CI 1.21–2.16; $P=0.001$), whereas office systolic BP was not (OR 1.00; 95% CI 0.75–1.33; $P=0.99$). Home and office diastolic BP were not significantly associated with incident decline in eGFR in the unadjusted models. Only higher baseline home systolic BP was associated with decline in eGFR in the unadjusted model (β -1.23; SE 0.39; $P=0.002$). In the multivariable-adjusted models, neither home systolic nor diastolic BP, nor office systolic nor diastolic BP were associated with a decline in eGFR. These are visualised in **Figure 6** and shown in greater detail in **Original Publication IV, Table 1**.

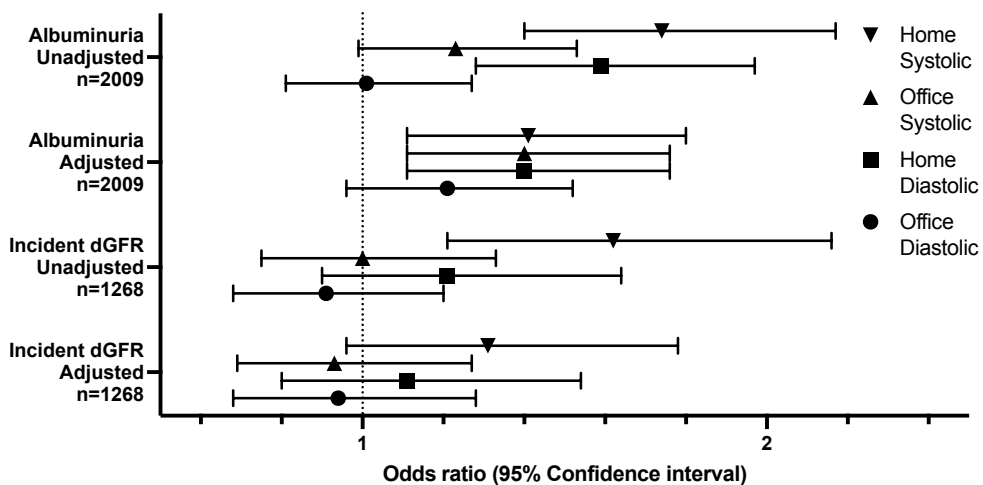


Figure 6. Associations of a one standard deviation increase in baseline home and office blood pressure with prevalent albuminuria at baseline (n=2,009) and incident decrease of estimated glomerular filtration rate to <60 ml/min/1.73 m². (dGFR, n=1,268). Author's own work.

6 Discussion

Hypertension and its complications remain a major public health challenge worldwide, with prevalence, awareness, treatment, and control rates often suboptimal even in high-income countries. This thesis aimed to assess these aspects in the Finnish population using home and office BP measurements, to examine the prognosis of white-coat and masked hypertension, and to investigate the relationship between home BP and HMOD, including LVH and chronic kidney disease. The findings provide insights into risk stratification, clinical management, and the utility of home BP monitoring.

6.1 Office and Home Hypertension Prevalence, Awareness, Treatment and Control, and Their Associations with Sociodemographic Factors

The analyses presented in this thesis demonstrate that hypertension is highly prevalent in Finland. The following studies are presented and referenced in more detail in **Table 1**. The observed prevalence rate of 47.1% from Study I is consistent with other Finnish studies and in most cases above the prevalence rates reported in similar nationwide studies from other countries, and especially those from high income countries. Hypertension awareness rates vary greatly from as low as 30% in Nigeria to as high as 82.5% in Canada, while in large meta-analyses awareness rates average between 40 to 55 percent (calculated average from Zhou et al. 2021: men 49%, women 59%)⁶. The hypertension awareness rate of 56.5% published in Study I is also in line with other studies from high income countries that have well-organised healthcare systems. The previously reported global treatment rates vary greatly, from 21 to 78.9 percent. The treatment rate of 50.9% observed in Study I is on par with rates reported from other western industrialised countries, while also higher rates have been reported from both Europe and North America. According to the available literature, Finland has hypertension treatment rates that are slightly above the global average.^{6-8,45} Similarly, the rate of those with treatment having their hypertension under control varies globally. The control rate of 48.3% observed in Study I is slightly higher than that reported in other Finnish studies, and slightly higher than what has been

observed in other middle- to high-income countries.^{8,45,48,49} However, some North American studies have reported control rates of up to 64 to 78 percent.^{31,32}

When considering the progression of Finnish hypertension prevalence, awareness, treatment, and control rates from past to present, it is clear that a lot of effort has been made to increase hypertension awareness and to educate the Finnish people of the importance of hypertension detection and treatment to minimise hypertension-related disease burden. For example, the North Karelia Project was the first community-based cardiovascular disease prevention project in the world.^{8,156} The main objectives of the North Karelia Project Hypertension Programme were: (1) to detect as many hypertensive people as possible; (2) to control BP in as many of them as possible; (3) to establish up-to-date and uniform diagnostic and therapeutic methods among physicians; and (4) to collect data on the epidemiology of hypertension and on hypertension-related work of the health services.^{157,158} In the early stages of these efforts, several risk factors correlated with elevated BP and adverse cardiovascular events were identified, such as age, gender, obesity, physical inactivity, adverse dietary habits including high intake of saturated fat and salt, and smoking.^{8,156} In addition, the interventional aspects of the North Karelia project were extremely successful, as mean BP levels in the Finnish population decreased over 10 mmHg from 1972 to 2012.¹⁵⁹ However, in recent years the trend of decreasing BP seems to have plateaued indicating the acute need for new more vigorous efforts to improve hypertension prevention and care.^{7,45}

With this historical context, Study I examined whether sociodemographic and lifestyle inequalities persisted in Finland in the year 2010. The findings of Study I suggest that after decades of public health efforts, significant disparities remain in hypertension care.

When interpreting the associations of these sociodemographic and lifestyle factors with hypertension, the potential multicollinearities across these factors must also be considered. Age is commonly recognised as the most important risk factor for hypertension and cardiovascular disease. This is mostly caused by the progression of atherosclerosis and arterial stiffening, resulting in increased pulse pressure (difference between systolic and diastolic BP).¹⁶⁰ Older age is also related to higher prevalence of other diseases, such as lung conditions, obesity, chronic kidney disease, and musculoskeletal problems, which may lead to physical inactivity and, in turn, higher systolic BP.

Hypertension is one of the most important, if not the most important, risk factor for cardiovascular disease. Thus, while it is important to consider the underlying causes of hypertension, its pharmaceutical and lifestyle therapy are just as important when trying to reduce the disease burden caused by hypertension.¹⁶¹ Poor hypertension control was predominantly observed among elderly participants. Among elderly and retired participants, awareness and treatment were higher, but hypertension

control was poorer, a pattern previously linked to arterial stiffening and cautious antihypertensive use in older populations.¹⁶²

Male sex was associated with higher prevalence and lower awareness of hypertension in Study I, a trend that has also been observed in other Finnish publications.^{8,45} Potential explanations underlying these findings are that women may be more often aware of healthy lifestyles and may have more frequent healthcare contacts related to reproductive health and preventive care. Elevated awareness and treatment rates were also observed in individuals that have more frequent contacts with a health care provider, such as those with obesity, diabetes, or other comorbidities.^{163,164}

Lifestyle factors, and especially obesity, were strongly associated with hypertension prevalence in Study I multivariable analyses, consistent with earlier reports that have demonstrated that hypertension in industrialised societies is largely driven by adverse lifestyles.¹⁶⁵ Former smoking was associated with a lower prevalence of hypertension, possibly reflecting a higher frequency of healthcare visits or greater health awareness among those who have quit smoking. In contrast, presumed perception of healthy lifestyles through average or above average physical activity was linked to lower awareness and treatment rates. Overall, individuals with a lower hypertension risk – lean, young, physically active, employed – had lower awareness and treatment rates, a pattern previously attributed to perceived good health reducing engagement with cardiovascular risk screening and treatment.⁴⁴ Lower awareness and treatment rates were also found to be associated with current smoking, possibly through neglect of healthy lifestyles.

Socioeconomic factors, such as education, occupation and retirement status, were associated directly and indirectly with hypertension prevalence, awareness, treatment, and control, which is consistent with previous literature.^{58,166} In Study I, in both age- and sex-adjusted and the multivariable analyses, retirement remained a statistically significant factor for hypertension prevalence, awareness, and treatment. This would suggest that retirement may result in more healthcare contacts and provide more time to focus on personal health through visits to a health care provider.

In summary, vigorous screening should be performed in individuals with traditional cardiovascular disease risk factors to better identify those with hypertension. Hypertension treatment should be initiated without delay and achievement of hypertension control should be monitored actively. However, awareness rates are lower in individuals without traditional risk markers and with healthy lifestyles. Therefore, screening should also be performed in these individuals to recognise hypertension for reducing inequalities in hypertension care.

6.2 White-Coat and Masked Hypertension as Predictors of Sustained Hypertension

In the longitudinal analysis of Study II, half of white-coat hypertensives and three-quarters of masked hypertensives progressed to sustained hypertension during the 11-year follow-up period. Multivariable analyses indicated that white-coat hypertension and masked hypertension were associated with a 3-fold and 4-fold higher risk of developing sustained hypertension compared to normotension, respectively. These findings clearly indicate that white-coat and masked hypertension are not benign but represent high-risk prehypertensive states.

In comparison to previous studies, Bidlingmeyer et al. reported that 72% of white-coat hypertensives developed ambulatory hypertension (12-hour daytime ambulatory BP mean over 140/90 mmHg) over three years of follow-up, while Colombo et al. observed a progression rate of 59% over a five-year period. However, these studies had relatively small sample sizes (81 and 173 subjects, respectively) and had short follow-up periods.^{111,112} However, these studies reported results that were inconsistent with those of Polónia et al., who reported a progression to hypertension in only 11% of participants over 3.5 years of follow-up (52 normotensive subjects, 36 untreated white-coat hypertensive subjects; 26 to 59 months follow-up).¹¹³ These studies were published in 1996, 2000, and 1997, respectively. A larger study, the Ohasama study, which consisted of altogether 777 subjects and was published in 2005, followed 128 white-coat hypertensives over an 8-year period and reported an odds ratio of 2.86 for the white-coat hypertensives to develop home hypertension compared to normotensives.¹¹⁴ Results from another larger study, the Pressioni Arteriose Monitorate e Loro Associazioni (PAMELA) study, by Mancia et al. (2009) indicated odds ratios of 2.51 and 1.78 for progression from white-coat hypertension, and masked hypertension to sustained hypertension, compared with normotensives, in a population of 1412 subjects during a ten-year follow-up.¹² In Study II, the relative risk ratios for progression from white-coat hypertension and masked hypertension to sustained hypertension were 2.8 and 3.8, respectively, when compared to progression from normotension to sustained hypertension. The findings from Study II were therefore relatively consistent with the two latter studies, which were larger and population-based, as compared with the studies by Bidlingmeyer, Colombo and Polónia.

Of note, the risks (nonfatal major adverse cardiovascular events) related to each hypertension phenotype increased from normotensives (2.4%, n=12) to white-coat hypertensives (10.4%, n=14), masked hypertensives (16.4%, n=10) and sustained hypertensives (12.7%, n= 26). The risk for adverse cardiovascular events is separate, but in line with the risk to develop sustained hypertension. The International Databases on Ambulatory (IDACO) and Home (IDHOCO) BP in Relation to Cardiovascular Outcome studies have similarly reported that white-coat hypertension and

masked hypertension carry a risk of cardiovascular outcomes that is intermediate to normotension and sustained hypertension.^{106,107,110} In fact, the most recent evidence demonstrates that masked hypertension is linked with a cardiovascular risk that is similar to, or only slightly lower, than sustained hypertension.¹⁶⁷

Regarding the differences in Study II sample characteristics between baseline hypertension phenotypes (**Table 10**), the risk factor profiles for white-coat and masked hypertensives were intermediate between normotensive and sustained hypertensive participants, including higher systolic BP and prevalence of hypercholesterolemia. Men and participants with higher BMI were more likely to exhibit white-coat, masked, or sustained hypertension. Importantly, white-coat and masked hypertension have been associated with increased risk of cardiovascular events, underscoring the need for close follow-up and lifestyle evaluation in these individuals, as demonstrated in earlier studies.^{110,168}

The exact pathophysiological mechanisms underlying white-coat hypertension and masked hypertension remain incompletely understood. The intermediate risk factor profiles observed in Study II, including elevated systolic BP, higher BMI, and increased prevalence of hypercholesterolemia, are consistent with proposed mechanisms involving early vascular changes and metabolic dysfunction.^{169–171} Sympathetic nervous system imbalance has also been implicated, potentially causing nocturnal non-dipping or other changes in the 24-hour BP profiles each carrying its own hypertension risk profile.^{172–175} These different 24-hour BP profiles can only be exactly determined with ambulatory BP measurements and could present themselves as white-coat or masked hypertension when ambulatory BP is not assessed. Other lines of research have examined the associations of subclinical cardiac damage and arterial damage as early markers in the progression from white-coat and masked hypertension towards sustained hypertension.^{176–178} Also, genetic determinants for overall risk of hypertension have been researched to benefit risk stratification and to find potential biological pathways for antihypertensive treatment in the future.^{179,180} These pathophysiological questions therefore warrant further investigation to fully characterise the risk profiles associated with each hypertension phenotype.

To summarise, the presented results from this thesis align with recent large population-based studies demonstrating that white-coat and masked hypertension predict future sustained hypertension and cardiovascular events.^{12,110,114} Earlier studies with smaller sample sizes and shorter follow-ups yielded inconsistent results, emphasising the added value of long-term, well-powered cohorts.^{111–113} Notably, progression to sustained hypertension increased across baseline BP categories from normotension to white-coat to masked hypertension, highlighting a continuum of risk.

6.3 Home versus Office BP in Predicting Target Organ Damage

The 11-year longitudinal studies presented in this thesis demonstrated that home BP is more strongly associated with both LVH progression and chronic kidney disease outcomes than office BP. Changes in home BP demonstrated stronger correlations and regression coefficients with changes in ECG-LVH compared with office BP, suggesting superior predictive value for hypertension-mediated organ damage, as reported in previous studies.^{14,92} Furthermore, home systolic BP was the only BP parameter consistently associated with incident albuminuria and GFR decline, while office BP associations were weaker and inconsistent, as observed in prior research.^{181,182}

Prior to the original publications of this thesis, longitudinal evidence on the association between home BP and target organ damage was sparse. The findings from Studies III and IV addressed an important gap identified in the literature review, in which longitudinal evidence on the links between home BP and HMOD was limited, as prior studies were predominantly cross-sectional or were conducted in selected patient populations, as summarised in **Table 6**. Study III addressed these limitations by utilising a population-based sample, an 11-year follow-up period, and mixed regression model analyses that directly assessed the longitudinal associations of home versus office BP with ECG-LVH changes. These results establish that home BP is superior to office BP in tracking ECG-LVH progression.

The choice of using ECG to assess LVH, such as in Study III, rather than echocardiography deserves some discussion. For study III, the decision was simple because no echocardiography data were available. However, even though ECG-LVH has some well-recognised limitations, including the varying sensitivity according to the used ECG-LVH index, it is widely available even in primary healthcare.^{183–186} Although echocardiography is often used for assessing LVH as it provides true anatomical measurements of the heart, it is usually only available in secondary or tertiary care. While magnetic resonance imaging is considered the most accurate method to assess left ventricular mass due to its repeatability and lack of imaging bias, as compared to echocardiography, it is expensive, time-consuming, and has poor overall availability. Nevertheless, in a validation study that used cardiac MRI as the reference standard, ECG-based RaVL was still shown to correlate well with MRI-based measurements of left ventricular mass.¹⁸⁵ In addition, ECG-LVH has been shown to predict adverse outcomes independently of changes in echocardiographic left ventricular mass.^{187,188} Therefore, while echocardiography and cardiac MRI provide superior anatomical assessment, ECG-LVH remains a prognostically meaningful and the most commonly used biomarker, which justifies its use in Study III and this thesis.

Study IV focused on changes in renal function and its association with office or home BP. The results from Study IV demonstrate that home BP is more consistently associated with albuminuria than office BP, although the longitudinal associations with eGFR decline reached significance only in unadjusted analyses. This was most likely due to lack of statistical power as there were only a small number of incident decreased eGFR cases (n=100, 7.9%). The difference between the results for albuminuria versus eGFR may also reflect pathophysiological differences. Albuminuria is considered an early, pressure-sensitive marker of glomerular damage, whereas eGFR decline is considered to represent later-stage nephron loss.¹⁴⁹ Still, these findings are aligned with those from previous studies, demonstrating the superiority of home BP for predicting renal outcomes.^{181,182}

The superiority of home BP is likely driven by multiple factors: measurement in a familiar environment, self-measurement by the participant leading to absence of the white-coat effect, and a greater number of readings, which reduce variability and capture day-to-day fluctuations.^{92,189} The Finn-Home study utilised a seven-day protocol, which likely contributed to the strong associations observed between home BP and target organ damage outcomes. Both reported cardiac and renal findings reinforce the importance of using home BP as a primary tool for risk stratification and follow-up of HMOD, and particularly for assessing risk of LVH and chronic kidney disease.

6.4 Strengths and Limitations

This thesis consists of four independent studies, which, when considered together, augment each other and provide a broad understanding of how home BP measurements should be used to screen for hypertension, assess the risk of progression of hypertension and the longitudinal risk of HMOD. However, before deciding on any clinical actions, one must understand the strengths and limitations of each study individually and combined.

6.4.1 Strengths

An important strength of all these studies is their population-based design, which was meticulously planned and performed in the Health 2000 and Health 2011 studies. These studies were designed as nationwide surveys to represent the entire Finnish adult population. As a part of the main study, the Finn-Home substudy, while performed in a specific age range, was able to maintain the representativeness of the entire adult population of that age range. In addition, the Finn-Home substudy utilised standardised and recommended measurement techniques and devices for home BP measurements. This greatly reduces selection bias in the study population and random error in the collected data.

Another important strength is the substantial 11-year follow-up period between the baseline Health 2000 and the follow-up Health 2011 studies. This study design enabled the longitudinal analyses reported in this thesis. This longitudinal study setting allowed for assessing emerging and evolving health conditions, namely hypertension and HMOD, over time. Only a few population-based studies used this approach previously over a similar, long follow-up period.^{116–119}

Additionally, a clear strength of the studies included in this thesis is the use of objective outcome measures that are clinically relevant. ECG-LVH was assessed with standard ECG criteria well known to clinicians. In addition, the laboratory parameters used to evaluate kidney function, albumin excretion, and urinary albumin-creatinine ratio are used by clinicians in daily clinical practice. Furthermore, the risk of information bias was minimised through the use of objective measurement-based endpoints and re-examinations, compared to self-reported outcomes.

The statistical analyses of this thesis should also be considered a strength. Each covariate was first individually analysed, then where applicable, analysed in an age- and sex-adjusted model, and then finally included in multivariable models to assess the independent relations of the covariates in comparison to other possible confounding factors. Also, sensitivity analyses were performed to test the robustness of the findings.

Finally, these studies analysed both home and office BP measurement data, where available, which enabled comparing the predictive accuracy of these two measurement methods in a large nationwide population.

6.4.2 Limitations

Several limitations are shared across the studies included in this thesis, whereas some may concern a specific study.

First, the studies were observational, which precludes causal inferences. Although prospective follow-up establishes temporal relationships, several factors causing possible interference may remain. As an example, the baseline interview and examination may cause changes in the behaviour of the study participant due to awareness of being studied, often by performing better or conforming to perceived expectations (Hawthorne effect).¹⁹⁰

Second, office BP measurements were limited to a single measurement occasion with two individual measurements, whereas home BP measurements included two measurements twice a day for seven consecutive days. The number of home measurements therefore often exceeded the number of office measurements. Although this reflects real-life practice, it also limits the direct comparability of the two methods. Additionally, different devices were utilised for home and office BP measurements, potentially introducing measurement bias.

Third, the exclusion of participants receiving antihypertensive therapy reduced the study sample sizes in some analyses. Furthermore, the participation rate of the Finn-Home substudy was 65.0%, which in combination with the exclusion criteria further reduced statistical power. Although sampling weights were applied in Study I to reduce bias, the possibility of selection bias due to healthier individuals being more likely to participate cannot be excluded.

Fourth, for the assessment of LVH, ECG indices were used rather than echocardiography. While ECG-LVH indices have modest sensitivity compared with anatomical LVH assessment, according to some studies, it is still recognised as an important predictor for cardiovascular events and is more feasible in population-based study settings.^{183,185–188}

Fifth, for chronic kidney disease, some associations were statistically significant only in unadjusted models, most likely due to limited statistical power. The incidence of chronic kidney disease endpoints in this sample of the general population was relatively low and constrained the precision of effect estimates.

Sixth, lifestyle and dietary factors, including sodium intake, may have been incompletely captured. Therefore, residual confounding from unmeasured or unknown confounders may have influenced the observed associations.

Finally, a substantial limitation for the generalisability of these findings on a global scale is the fact that the study population consisted predominantly of ethnic Finnish adults. The applicability of these findings in other ethnic settings with different healthcare systems, guidelines and socioeconomic situations therefore requires further research.

7 Summary/Conclusions

The main findings and conclusions of this thesis were:

1. This thesis shows that in addition to the high prevalence and moderate treatment rates of hypertension, significant health inequalities in hypertension and its management exist within the Finnish general population. These rates are not affected largely by the method of BP measurement when the currently recommended method-specific diagnostic thresholds are used. Although hypertension is primarily associated with aging and unhealthy lifestyles, socioeconomic factors appear to contribute, both directly and indirectly through interactions, to the development and treatment of hypertension. In general, individuals at greater risk of developing hypertension are more likely to be aware of their condition and to receive treatment. Conversely, hypertension tends to be underdiagnosed and undertreated among those with seemingly healthy lifestyles. Therefore, health education and awareness campaigns should also target individuals considered at low risk for hypertension. Specific measures could be to include more comprehensive health examinations, such as home BP monitoring, within occupational health care, particularly for young adults and for those at the early stages of hypertension development (Study I).
2. The findings in this thesis indicate that masked and white-coat hypertension cannot be regarded as harmless or benign phenomena. Both conditions are associated with a three- to four-fold higher risk of progression to sustained hypertension compared with normotension. Home BP monitoring should be encouraged to identify prehypertensive individuals at an increased cardiovascular risk. Individuals with white-coat hypertension and masked hypertension require close follow-up and careful lifestyle assessment (Study II).
3. This thesis demonstrates, for the first time, the superiority of home BP measurements over office measurements in the follow-up of ECG-LVH. These results also suggest that home BP could be more strongly associated with prevalent and incident chronic kidney disease than office BP in the general population. The findings from this and previous studies highlight the importance of using out-of-

office BP monitoring as the primary method for assessing BP levels. Although the optimal home and office BP targets are still being debated, these findings align with previous work and provide additional evidence of the superiority of home BP over office BP (Studies III and IV).

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