
ESTIMATED SALT INTAKE AMONG SOMALIS IN NORWAY

A cross-sectional study employing 24-hour urinary sodium excretion to estimate salt intake among Somali adults in the Sagene district of Oslo, Norway

SAIRAH LAI FA CHEN

Main supervisor: Ahmed A. Madar

Co-supervisor: Cecilie Dahl



Department of Community Medicine
Institute of Health and Society
Faculty of Medicine
University of Oslo

May 2018

Thesis submitted as part of the Master of Philosophy Degree in International Community
Health

ACKNOWLEDGEMENTS

I first wish to extend my gratitude to my thesis supervisors, Ahmed A. Madar and Cecilie Dahl.

Ahmed, for your guidance, cheer, and trusting me with a project dear to you.

Cecilie, for your patience and moral support. A rock.

To all participants, the Somali community, and Sagene community – thank you for welcoming us in.

To the Department of Community Medicine for offering this programme. You have allowed us invaluable flexibility and diversity.

To my fellow classmates – this process could not have been as enjoyable or inspiring without you.

Lastly, to those dearest:

Rafeena

Steve

Azha

Eivind

Thank you for your love and unwavering words of encouragement.

FOREWORD

This thesis is being submitted in partial fulfilment of the Master of Philosophy degree in International Community Health. It follows guidelines according to “Option 2 – One article submitted to an international peer reviewed journal and a summary” outlined in *Requirements of the master thesis*, Spring 2016, Department of Community Medicine.

The thesis includes the introduction, literature review, methods, and methodological considerations in full detail. The results and discussion are not included, as per Option 2. They may be found as part of the submitted article. A manuscript of this article has been included at the end of this document in *Manuscript of Submission* on page 93. The manuscript was submitted to the international peer reviewed journal, *European Journal of Nutrition*, on the 23rd of March 2018, entitled: “Estimation of salt intake assessed by 24-hour urinary sodium excretion among Somali adults in Oslo, Norway”.

This study was funded by the Norwegian Institute of Public Health and the Norwegian Health Directorate. Funding parties did not take part in this study’s design, sample, data analysis, or the writing of the associated article.

ABSTRACT

Purpose: High dietary salt intake is associated with increased blood pressure (BP) and cardiovascular disease (CVD) risk. The migration of Somalis from East-Africa to Norway may have altered their dietary habits, making them vulnerable to adverse health outcomes. Since little is known about the lifestyle and health status of this population, the purpose of our study was to estimate salt intake in Somali adults in Oslo, Norway.

Methods: A cross-sectional study involving 162 Somali adults (76 men, 86 women) from the Sagene borough in Oslo, Norway. Sodium and potassium excretion was assessed through the collection of 24-hour urine. Creatinine-based exclusions were made to ensure completeness of urine collections.

Results: Sodium excretion corresponded to estimated dietary salt intake of 8.66 ± 3.33 g/24 h in men and 7.28 ± 3.59 g/24 h in women ($p = 0.013$). An estimated 72% of participants consumed > 5 g salt/day. The Na/K ratio was 2.5 ± 1.2 in men and 2.4 ± 1.1 in women ($p = 0.67$).

Conclusions: Estimated salt intake was, while above the WHO recommendation, within the lower range of estimated salt intakes globally and in Western Europe. Further research is required to assess the health benefits of sodium reduction in this Somali immigrant population

LIST OF ABBREVIATIONS

4DFR	4-Day Food Record
AHA	American Heart Association
Ang II	Angiotensin II
ASA24	Automated Self-Administered 24-hour recalls
BMI	Body Mass Index
BP	Blood Pressure
CDC	Centre for Disease Control
CE	European Conformity
CI	Confidence Interval
CNS	Central Nervous System
CVD	Cardiovascular Disease
DAG	Directed Acyclic Graph
DASH	Dietary Approaches to Stop Hypertension
DBP	Diastolic Blood Pressure
ECF	Extra-Cellular Fluid
EO	Endogenous Ouabain
FFQ	Food Frequency Questionnaire
GBD	Global Burden of Disease
HIC	High-Income Country
HUBRO	Oslo Health Study
ID	Identification
ISE	Ion-Selective Electrode
IVDD	In Vitro Diagnostic Directive
LMIC	Low- and Middle-Income Countries
NCD	Non-Communicable Disease

NIPH	Norwegian Institute of Public Health
NNR	Nordic Nutrition Recommendations
PABA	Para-Aminobenzoic Acid
PURE	Prospective Urban and Rural Epidemiological study
RAAS	Renin Angiotensin Aldosterone System
RCT	Randomised Control Trial
REK	Regional Committees for Medical and Health Research Ethics
SBP	Systolic Blood Pressure
SD	Standard Deviation
STEPS	WHO STEPwise approach to non-communicable disease and risk factor Surveillance
TSD	Services for Sensitive Data
UNDP	United Nations Populations Division
UNHCR	United Nations Refugee Agency
UNN	University Hospital of North Norway
WHO	World Health Organisation

TABLE OF CONTENTS

Acknowledgements.....	II
Foreword.....	III
Abstract.....	IV
List of Abbreviations.....	V
Table of contents.....	VII
1. Introduction.....	1
2. Literature review.....	3
2.1 Salt in health and disease.....	3
2.1.1 Salt in human physiology.....	3
2.1.2 Recommended salt intake.....	4
2.1.3 The blood pressure response to salt.....	5
2.1.3.1 Defining blood pressure.....	5
2.1.3.2 Defining hypertension and its global significance.....	6
2.1.3.3 Pathogenesis for sodium-induced hypertension.....	6
2.1.3.1 Epidemiology of sodium-induced hypertension.....	7
2.1.4 Health outcomes from high sodium intake.....	9
2.1.5 Salt sensitivity.....	10
2.1.6 Potassium.....	11
2.2 Salt intake.....	12
2.2.1 Global status.....	12
2.2.2 Salt status in sub-Saharan Africa.....	13
2.2.3 Somali population salt status.....	13
2.2.4 Norwegian population salt status.....	14
2.2.5 Summary.....	14
2.3 Estimating dietary salt intake.....	15
2.3.1 Dietary assessment.....	15
2.3.2 Urine assessment.....	16
2.3.3 Summary.....	19
2.4 Somalis in Norway.....	20
2.4.1 General geography and anthropology.....	20
2.4.2 Settlement in Norway – demographics.....	21
2.4.3 Health and dietary habits.....	21
3. Rationale for this study.....	24
4. Research Aims and objectives.....	25
5. Methodology.....	26
5.1 Overview.....	26
5.2 Design.....	27
5.3 Participants.....	27

5.3.1	Defining the study area and population	27
5.3.2	Sampling strategy.....	28
5.3.3	Inclusion criteria	28
5.3.4	Exclusion criteria	29
5.4	Recruitment	29
5.4.1	Community-based efforts and cultural sensitivity	29
5.5	Measurement tools	30
5.5.1	Urine collection.....	30
5.5.2	Laboratory analysis.....	31
5.5.3	Questionnaire	31
5.5.4	Blood pressure testing.....	31
5.6	Urine-based exclusion criteria.....	32
5.7	Data collection procedure.....	32
5.8	Sample flow.....	34
5.9	Study variables	35
5.10	Ethical considerations	36
5.11	Incentives	37
5.12	Data handling	37
5.13	Statistical Analysis	38
6.	Methodological considerations.....	41
6.1	Study power and reliability	41
6.2	Internal validity	42
6.2.1	Study design.....	42
6.2.2	Selection bias	43
6.2.3	Information bias.....	44
6.2.4	Confounder bias	46
6.3	External validity.....	46
7.	Implications & Conclusions	49
	References.....	51
	Appendix A.....	60
	Appendix B.....	61
	Appendix C.....	62
	Appendix D.....	64
	Appendix E1	76
	Appendix E2	79
	Appendix F1.....	81
	Appendix F2.....	83
	Appendix G.....	84
	Manuscript of the Submission	85

1. INTRODUCTION

Sodium chloride (NaCl), more commonly known as *salt*, is an essential compound for human life. Salt is critical for nerve and muscle function as well as for regulating body fluids. It plays a role in the mechanisms maintaining blood volume, blood pressure (BP), and pH balance. Salt uptake occurs through dietary consumption, where approximately every 2.54g of salt consumed yields 1g of sodium. Once salt is dissolved in the body, sodium acts as a major serum cation in the body's extra-cellular fluid (ECF), establishing electrochemical gradients across membranes to move water and other ions around, out, and into the body. We cannot live without salt – nor, it seems, can we live with too much of it.

Salt is thought to have long-term health consequences when consumed in large amounts, over a long period of time. It is considered a major risk factor for hypertension (1), placing elevated strain on the heart and blood vessels. Hypertension has, consequently, been the leading risk factor for cardiovascular disease (CVD), death, and disability over the past quarter century worldwide (2). The burden of hypertension and CVD has further stimulated research on understanding salt and its health outcomes as well as public health campaigns aimed at monitoring, evaluating, and reducing salt intake.

This thesis reports on a cross-sectional study that was conducted between December 2015 and October 2016. It aims to estimate salt intake among Somali adults in the Sagene district of Oslo, Norway using one-time 24-hour urine collection. The assessment of 24-hour urinary excretion is the gold standard method for estimating sodium intake. In addition to fulfilling the main aim, this thesis secondarily estimates potassium intake and the sodium-to-potassium (Na/K) intake ratio. It also models the association between socio-demographic background variables and 24-hour sodium excretion. As is standard practice among studies such as this (3-6), the association between BP and 24-hour urinary sodium and potassium excretion will additionally be assessed.

It is important to note that the focus of this thesis is not on the relations between salt intake, BP, and/or CVD. Adequate assessment requires longitudinal design, very large sample size, and/or multiple 24-hour urine collections, which are outside the scope of a Master's project.

Providing high quality data to estimate salt intake in an individual population is what can feasibly be achieved – still beneficial to the scientific community and public health as we work towards improving health and achieving health equity for minority populations.

This thesis is will first provide a background context through a literature review, followed by a rationale and the aims and objectives of the study. Then, the methods used to carry out the study will be presented along with methodological considerations. The associated article manuscript lies in *Appendix*, where the results, discussion, and conclusion of the study can be found.

2. LITERATURE REVIEW

This chapter aims to provide a comprehensive report of the already-established sphere of knowledge relevant to the study. This literature review is organized into themed subsections which highlight the strengths, weaknesses, and gaps of current knowledge, theories, and hypotheses surrounding salt consumption as it relates to: adult salt intake, health outcomes, immigrant health, health among Somalis in Norway, dietary acculturation, and methods for its estimation.

The literature search itself was conducted primarily through PubMed Central[®], Ovid EMBASE[®], and Cochrane Library (Wiley Online Library[®]). The United Nations Refugee Agency (UNHCR), the Oslo Municipality Statistics Bank and Statistics Norway (SSB) also provided informative statistical data, beneficial for understanding the Somali context inside and outside of Norway. The literature search was last updated on 28 April 2018. A literature search flow chart can be found in *Appendix A*.

The terms *salt* and *sodium* are used interchangeably (although, specifically when referring to measurements), according to that used in the literature being addressed. For all intents and purposes, they are equivalent – *salt* being the form sodium is consumed in, holding dietary relevance, and *sodium* being the physiologically significant component of salt, thought to induce health effects. It is the element excreted in the urine which can be used to estimate sodium (and thus, salt) intake.

2.1 *Salt in health and disease*

2.1.1 Salt in human physiology

As one of the most abundant cations in the human body, sodium plays a central role in a number of vital functions. Nervous system signalling is allowed by the presence of sodium in the ECF to carry out action potentials, facilitating brain function and muscle function. The role of salt in the regulation of ECF osmolality, blood volume, and thus BP is primarily

achieved through the renal system. Renal regulation of sodium concentration in the ECF is critical for the maintenance of arterial BP. An individual's uptake of sodium into the bloodstream will almost always differ strikingly from hour to hour based on diet. However, the ECF concentration of sodium remains within a remarkably narrow range and must do so to maintain vital homeostasis (7). This places a great deal of responsibility on the kidneys to appropriately filter or retain sodium and/or water from the blood to maintain body fluid volume, and thus BP at every moment, irrespective of sodium or water consumption. The filtrate of this process is urine. (8)

The kidneys operate under the Renin-Angiotensin-Aldosterone System (RAAS) – a hormonal network, which forms a negative feedback loop to decrease BP when certain thresholds of sodium concentration and fluid volume are reached. At the lower levels of salt intake or detected low BP, healthy kidneys will reabsorb sodium and water to increase blood plasma osmolality, which result in water movement into the blood vessels to increase blood volume and BP. However, at the extreme highs of salt intake, normal kidneys excrete large amounts of sodium in the urine to prevent water retention in the blood vessels (8).

2.1.2 Recommended salt intake

Although sodium is an essential dietary mineral, there is no defined minimum requirement – a unique quality among all other essential minerals. The minimum amount of salt intake required per day is likely to differ between populations due to population/individual characteristics (background diet, level of physical activity, genetics) and geographical characteristics (climate and altitude) (9). While it is still possible for humans to consume dangerously low amounts of salt, the practical significance of such a guideline has generally been deemed unnecessary. The accessibility to salt used in home-cooking, in processed foods, and in restaurant foods is universal, with the exception of few 'unglobalized' populations, constituting less than 0.1% of the human population (10). As such, most agree there is virtually no risk of consuming too little salt (11).

The limit of interest, for effectively every existing public health institution, is the maximum salt recommendation. While the WHO carries the most well-known guideline, several other

more regional recommendations exist and provide a foundation for more local salt reduction campaigns. *Table 1* displays some of the current salt recommendations. There is wide variation, indicative of a level of non-consensus. However, all recommendations are far lower than current salt intake in most populations (12).

Table 1. International and national salt intake recommendations for adults

Institution	Maximum recommended (g/day)
World Health Organization (WHO) (11)	5
American Heart Association (AHA) (13)	3.8
Centre for Disease Control (CDC) (14)	5.8
Nordic Nutritional Recommendations (NNR) (15)	6*
Norwegian Health Directorate (16)	5

*Proposed target for Nordic population sodium reduction

2.1.3 The blood pressure response to salt

2.1.3.1 *Defining blood pressure*

The pressure exerted by circulating blood on the internal walls of the blood vessels is known as BP. Pressure changes drastically throughout the cardiac cycle. Therefore, BP is given the operational definition of systolic BP (SBP) over diastolic BP (DBP) – SBP being the maximum pressure in the blood stream occurring immediately after contraction of the heart ventricles (systole) and DBP being the lowest pressure in the blood stream occurring during heart filling and ventricular relaxation (diastole). Normal, healthy blood pressure is considered to be 120/80 mm Hg (8).

In a healthy person, BP is under tight physiological control. As described above, renal function plays a major role in short-term regulation by maintaining the delicate salt and water balance. Baroreceptor reflexes also detect changes in BP which can adjust the force and speed of heart contractions, signal to alter vascular tone, and signal to up- or down-

regulate the RAAS involved in renal function. Regulation of these processes can keep BP stable from a second to second basis (8).

2.1.3.2 Defining hypertension and its global significance

Hypertension is clinically defined as BP exceeding 140 mm Hg in SBP and/or 90 mm Hg in DBP (8). Hypertension increases the risk of vascular complications, thus leading to CVD such as stroke, ischemic heart disease, myocardial infarction, and heart failure as well as kidney disease (2, 8). According to the Global Burden of Disease Study 2015, hypertension has been the leading cause of premature death and disability over the past quarter century (1) and is the leading risk factor for CVD, which is responsible for one third of all global deaths (2). In a comprehensive review, the global age-standardized prevalence of hypertension was estimated to be 31.1% in 2010 (17).

Hypertension has, historically, been considered to only affect populations in High-Income Countries (HICs) due to sedentary lifestyles and affordable access to foods high in salt and empty calories. Today however, the burden of hypertension is greatest in low- and middle-income countries (LMICs) (17). In Africa, the prevalence of hypertension varies across studied populations. It is thought to be related to a rapid increase in development, differences in urban and rural lifestyles, and different diets, among others (18, 19). There is currently no reliable estimate for the prevalence of hypertension among the Somali population.

The level of salt intake is considered by many to be the leading modifiable risk factor for hypertension (10, 20, 21). Other prominent modifiable risk factors for hypertension include tobacco use, low physical activity, heavy alcohol consumption, and low potassium intake (WHO, 2013).

2.1.3.3 Pathogenesis for sodium-induced hypertension

The scientific community is in general agreement that the body's tightly regulated BP system and strong appetite for salt is indicative of a long-conserved adaptation to retain sodium (7, 22). Despite wide acceptance of the salt-BP relationship, the precise mechanisms underlying the relationship between sodium and BP remain

somewhat unclear. However, over the past 25 years many ground-breaking studies have begun to elucidate a newly discovered endocrine system involving the central nervous system (CNS) and its effect on cardiovascular, renal, and brain function with implications for the development of hypertension. The focus and scope of this Master's thesis does not allow for us to delve into its details. In short, several studies have found that the CNS houses a hormonal network responsible for the chronic increase in BP (7, 23, 24). Blaustein et al. (7) describe this as a “slow neuro-modulatory pathway”, termed the ‘Aldosterone-Endogenous Ouabain-Angiotensin II’ (Aldo-EO-Ang II) pathway, and it is critical for the development of salt-induced hypertension (7). The BP set-point, known as the *central barostat*, is regulated by the CNS and modulated by the Aldo-EO-Ang II pathway, leading to a realignment of the BP set point at high sodium intake (24).

Ang II and aldosterone also seem to upregulate the local (hypothalamic) production of the steroid, endogenous ouabain (EO) (25). This hormone has been shown to have high affinity for an inhibitory receptor on Na/K-ATPase transport pumps, preventing the transport of sodium out of the CSF, thus maintaining high sodium concentration there. It also acts on the calcium signalling, leading to further elevation of BP (26).

Pathophysiological mechanisms for the development of salt-induced hypertension remain in its early stages. Overall, modulatory peripheral and CNS control appear capable of altering the body's carefully regulated BP set-point, which in normotensive (i.e. normal BP) humans is kept in check by the acute systems (7).

2.1.3.1 Epidemiology of sodium-induced hypertension

Epidemiological evidence for the relationship between BP and estimated salt intake can be divided into two main types: 1) Evidence for an association in observational cross-sectional or prospective studies; 2) Evidence of BP changes when salt intake is actively reduced (i.e. effects measured from a sodium reduction intervention). In general, studies demonstrate a weak, but significantly positive association between BP and salt intake in the general population (10, 20, 27). However, the associations are non-homogeneous across subgroups, including ethnicity, age, grade of estimated sodium intake, and health status (20, 27, 28).

The INTERSALT study was the first, large-scale epidemiological study to measure the association between sodium and BP in several populations worldwide (10). Published in 1988, they included 52 populations from 32 countries (n=10 079). INTERSALT found a significant and positive adjusted association between estimated sodium intake and SBP from pooled results between all 52 populations, revealing increased SBP of 1.22 mmHg for 1 gram/day increase in salt intake. Within-population adjusted analyses revealed significant positive associations between sodium intake and BP in 8 of the 52 centres, 25 centres with positive associations not reaching significance, and 2 centres reporting a statistically significant negative association. The associations for pooled and within-population analyses were adjusted for age, sex, body mass index (BMI), and alcohol consumption. They also reported that age-related increases of BP became significantly steeper with increased sodium excretion.

Results from INTERSALT have been heavily criticized for ecological fallacy, prone to confounding bias, and improper statistical methods (29, 30). When four out of the 52 centres were removed, which many considered to be outlier populations, the result was weakened to 0.48 mmHg increase in SBP for every 1 g/day increase in salt intake (31). These four centres represented four 'un-globalized' populations that had extremely low salt and alcohol intake, low body weight and BP.

The Prospective Urban and Rural Epidemiological (PURE) Study (n=102 216) is the second and most recent large-scale epidemiology study to estimate the association between salt intake and BP (27). The study was conducted between 2002 and 2009 in 18 different countries. The results from PURE supported findings from INTERSALT, revealing a significant positive association between estimated sodium intake and BP, with steeper association for increased age. The overall association in PURE was much steeper compared to INTERSALT. Additionally, PURE found hypertensive individuals had greater increases in BP for each 1 gram increment of estimated sodium intake compared to normotensive individuals.

Although on a smaller scale, Stolarz-Strzypek et al. (32) produced the first longitudinal study examining the association between BP and estimated salt intake, based on two prospective studies on white-Europeans (total n=4 547). After 6.1 years follow-up, changes

in SBP were positively and significantly related to changes in estimated sodium intake(1.7 mmHg for every 100 mmol increase in sodium (CI: 0.7, 2.6))

In a recent 2017-updated Cochrane Review, Graudal et al. (28) estimated the BP effect of high vs. low sodium intake in randomized control trials on hypertensive and normotensive samples (RCTs). They reported a significant, positive relationship between BP and salt intake and a steeper slope of association in hypertensives, supporting PURE and INTERSALT results. The authors also found the effect was greater in black and Asian populations. However, there few studies on racial minorities available for inclusion (28).

Several publications have criticized the used of short-term RCTs (32, 33), such as in Graudal et al. (34), to extrapolate lasting BP changes. In a second Cochrane Review, He et al. (20) found longer term modest salt reduction resulted in significantly decreased BP. Again, hypertensives and ‘non-whites’ experienced a stronger association than normotensives (20).

2.1.4 Health outcomes from high sodium intake

Although hypertension is the leading risk factor for CVD, recent and ongoing scientific controversy has not been able to robustly conclude that increased salt intake is associated with CVD events. Several recent meta-analyses have reported no evidence or inconclusiveness of decreased risk of CVD events and/or mortality due to salt reduction from cohort studies and RCTs (35-37). Interestingly, this includes the WHO report in which they conducted their own meta-analyses, which was used as justification for their salt recommendation (11). Only one large-scale study by Strazzullo et al. (36) – a meta-analysis – found a significant association between sodium intake and stroke (36).

The PURE study identified evidence that the risk for CVD and mortality (just as demonstrated with BP) differs between normotensives and hypertensives, with hypertensives demonstrating a stronger association with health outcomes (33, 38). They also found evidence that low (< 7.62 g/day) and high (> 15.23 g/day) salt intake was associated with increased CVD events and mortality (33, 38). Due to the implications of these recent studies challenging this relationship so deeply rooted in public health, further research is required to establish consensus across various stakeholder parties.

Although less studied, there is some evidence that salt intake is related to other the onset of other non-communicable diseases (NCDs), such as kidney disease (39), gastric cancer (40), and osteoporosis (41). Additionally, in a meta-analysis conducted in 2017, Moosavian et al. (42) reported that high salt intake was associated with increased BMI and waist circumference (both indicators for obesity). However these studies were not especially conclusive.

2.1.5 Salt sensitivity

Part of the difficulty in establishing a safe range of salt intake for the general public is the marked heterogeneity of the BP response to salt intake. Some individuals manifest large changes in BP in response to altered dietary salt – termed *salt sensitive* – while others show no variation in BP, termed *salt resistant* (43). Salt sensitivity is estimated to be present in 51% of hypertensives and 26% of normotensives, explaining the greater response to higher salt intake in hypertensives (44). It appears to represent a complex interaction of genetic and environmental factors which, independently of altered salt intake, can control susceptibility for BP changes and cardiovascular health (45).

There is growing evidence that genetic mechanisms underlying the BP response to salt are critical for salt sensitivity (45). To be brief, the presence of alleles that code for protein factors directly and indirectly involving salt transport across cell membranes – such as sodium and potassium channels and enzymes – appear to alter the extent of salt and water retention as well as arterial compliance (43, 45, 46). It thus lends the argument that certain ethnic groups that share a similar gene pool may present greater salt sensitivity than others (47). However importantly, phenotypic expression of the allele is often altered by environmental factors.

Little is known about salt sensitivity in the Somali population, residing in the Horn of Africa or elsewhere. Only one study was found in the literature search, which reported that Somalis exhibited a less efficient baroreceptor reflex compared to “whites” (unknown ethnicity) when on a high compared to a low salt diet (48). The authors state this result indicates “blacks” are slower to decrease BP to normal levels when subjected to high blood volume. However, there were several problematic aspects of this study. Firstly, it was based on a

very small sample (20 individuals; 10 Somalis and 10 “whites”). Secondly, long-term effects on BP are unknown since the participants were only followed for seven days on the high and low salt diet. Thirdly, the term “black” was used to generalize results. However, this is misleading, as “black” is not an ethnic group and race has little genetic foundation – especially among people of African origin.

The majority of the research on African populations has been conducted on African Americans. While there is evidence they are more salt sensitive than other groups (49) the African American gene pool is a unique subset of a native Western African gene pool. Therefore, results should not be generalized to other African populations.

Diet quality is a key modifiable risk factor for salt sensitivity. The Dietary Approaches to Stop Hypertension (DASH) diet was introduced in 1995 and involves the consumption several portions of fruits, vegetables, and low-fat dairy products daily (50). The DASH diet was used in a large RCT (n=456) and found that those who adhered to the DASH diet were less salt-sensitive compared to those on a control diet. The DASH diet is considered to be a high-quality diet in general due to the concentration of appropriate nutrients, such as potassium. Potassium, has over the past two decades, been shown to have an especially strong relationship to salt-sensitivity

2.1.6 Potassium

Potassium, also an essential dietary mineral, is the most abundant cation in the ECF (8). Along with sodium, it is involved in maintaining osmolality of blood plasma, cells, and the ECF and in the excitability of axon potentials. Significantly, dietary potassium can exert a dose-dependent effect on sodium sensitivity (46, 49, 51). It has shown to have moderate BP lowering effects independently and by decreasing sensitivity to salt, as reported by INTERSALT (10) and the PURE study (27). Authors involved in the PURE study also reported estimated potassium intake of over 1.50 g/day was associated with a significant reduction in the risk of mortality and CVD events (38). In a systematic review conducted in 2011 on the effect of increased potassium intake on cardiovascular risk factors, the authors reported that, in addition to reducing BP, higher dietary potassium had a significant inverse relationship with incidence of stroke (52). On the basis of the evidence provided by this

analysis, the WHO established its first guideline for potassium intake in 2012, recommending adults consume over 90 mmol/day (equivalent to 3.51 g/day) (53).

Estimating the sodium-to-potassium (Na/K) ratio in studies has become routine due to their opposing relationship to BP changes and the ability to easily estimate the intake of both electrolytes when employing a urinary excretion method. Diets based on processed foods tend to be sodium-rich and potassium poor (high Na/K ratio), whereas diets based on high fruit and vegetable intake tend to be potassium-rich and sodium-poor (low Na/K ratio) (46).

2.2 *Salt intake*

2.2.1 Global status

Quantification of global, regional, and national levels of dietary salt intake has been a cornerstone for the monitoring and implementation of salt reduction interventions – a priority for major global and national organizations to reduce the burden of non-communicable disease (54, 55). In the majority of literature estimating salt intake in various populations, salt intake status is compared to the guideline administered by the WHO in 2012 (11), recommending an upper limit of five grams of salt intake per day. However, other more regional guidelines do exist (see *Table 1*).

The most recent systematic analysis of global salt intake was conducted as part of the Global Burden of Disease (GBD), Injuries, and Risk Factors Study 2010 collection. According to the GBD 2010, global mean sodium intake was 3.95 g/day. However the range of sodium intakes varied substantially across populations globally – from 1.48 g/day in Kenya to 5.63 g/day in Uzbekistan, corresponding to 3.63 and 14.3 grams of salt/day, respectively (12). The lowest average salt intake was found in populations in Sub-Saharan Africa, Latin America, and the Caribbean (12). Wide variation in sodium intake, with relatively low uncertainty was identified in Western Europe – ranging from 3.28 (2.99, 3.59) g/day in Denmark to 4.43 (4.23, 4.62) in Italy, whereas the highest intake was found in Asian populations (5.11 g/day) (12). In all regions, sodium intake was modestly higher for men compared to women, in the range of 8.9% higher in South Asia to 10.7% higher in Western

Europe. Age differences in sodium intake were small – around a 6% increase from age 25-29 to 40-44, and plateauing subsequently (12).

2.2.2 Salt status in sub-Saharan Africa

In the literature search for estimates of salt intake among populations in sub-Saharan Africa, two systematic reviews were identified as relevant, including the GBD 2010 (12). The GBD 2010 estimated sodium intake of 2.47 g/day, corresponding to 6.27 g/day of salt, in sub-Saharan Africa, based on 20 studies. Compared to other regions, the estimate from sub-Saharan Africa had the fewest data points, resulting in high uncertainty. Findings ranged from 2.18 (2.05-2.32) in Eastern sub-Saharan Africa to 2.76 (2.58-2.95) in Western sub-Saharan Africa (12).

The second relevant source found was a systematic review and meta-regression of salt intake estimates in sub-Saharan Africa was published in 2016 by Oyeboode et al. (56). They found there have been no estimates of salt intake below five grams (the WHO recommended limit) reported from Sub-Saharan Africa since the 1990s. Contrary to the two global systematic reviews (12, 57), Oyeboode et al. found no gender difference in estimated salt intake. From this, the authors propose that population-specific research is required since demographic and lifestyle risk factors likely differ across populations. Oyeboode et al. (56) did not provide an average estimated salt intake among regions or across all populations in Sub-Saharan Africa.

The implementation of salt reduction strategies in sub-Saharan Africa is hindered by the lack of national strategies, reflected in the relative scarcity of salt intake estimates in this region of the world (58).

2.2.3 Somali population salt status

There were no studies identified from the literature search estimating salt intake in Somalia or Somaliland. There was, however, one study found which estimated salt intake among Somali immigrants to Florence, Italy (59), and it is also the only study we were able to find on salt intake among adult immigrant groups. See *Appendix A* for search flow chart.

This single study, conducted in 1992 by Modesti et al. (59), reported salt intakes at baseline

(within two days of arrival to Florence) and at six months of residence in Florence. Salt intake increased significantly from 2.80 g/day at baseline to 5.67 g/day at six months (59). The baseline measurement may be a crude reflection of salt intake among Somali adults residing in Somalia or Somaliland since data was collected almost immediately upon their arrival to Italy. However, the study is relatively old (over 20 years), sample size was very small (n=25) and the method to estimate salt intake (24-hour urinary excretion) is almost completely influenced by day-to-day variability of salt in the diet (discussed more in *Chapter 2.3.2*). Their diets may have changed substantially within the one to two days of residence in Florence merely due to food availabilities, meaning the baseline estimate would reflect the amount of salt eaten over the previous day in Italy as opposed to what was recently or habitually eaten in their home country. However, there is no doubt an enormous increase in salt intake after six months, which may reflect their habitual salt intake as settled Somali residents in Florence, Italy.

2.2.4 Norwegian population salt status

The only current estimate on salt intake in the Norwegian population comes from a population in Tromsø, in the northern region of Norway. Presented at the EuroPrevent 2017 conference hosted by the European Society of Cardiology, H. Meyer et al. (60) found men consumed 10.2 grams/day and women consumed 7.4 g/day of salt estimated from the collection of 24-hour urine.

Only one other study was found estimating salt intake a Norwegian population, but is not recent. Published in 1983, Omvik et al. (61) reported salt intake of 192.5 mmol/day. It corresponds to slightly higher salt intake than that found in the Tromsø population.

2.2.5 Summary

It appears that nearly every adult population studied within the past 25 years is estimated to consume over five grams of salt per day – the upper limit recommended by the WHO. Mozzafarian et al. (62) estimated that 99.2% of the global adult population exceed this cut-off. Nevertheless, there are large regions of the world that have not been studied. Results

from the literature search point specifically to knowledge gaps for populations residing in the global south as well as immigrant populations.

2.3 *Estimating dietary salt intake*

Regular population estimation of salt intake is critical for monitoring and reporting on salt intake targets and recommendations. Currently, a variety of methods are available and used for measuring dietary salt intake. However, all methods are met with particular challenges, which make precise and accurate estimation of intake in individuals and/or the population demanding and often problematic (6, 63). Recognition of these challenges, and thus interest in assessing the validity of various methods for measuring salt intake, has increased dramatically within the past two decades. They are important discussions to be aware of prior to conducting a study of salt intake estimation to appropriately align aims with methods and implications with limitations.

2.3.1 Dietary assessment

Common dietary assessment methods to estimate salt intake include 24-hour dietary recall, dietary record, and the food frequency questionnaire (FFQ). These methods are advantageous over urine methods for their potential to collect detailed information on the dietary sources of sodium intake as opposed to only a quantification of estimated salt intake as a whole (64). Knowledge of the foods most associated with high salt intake within a population can inform public health on how to most effectively reduce salt intake based on interventions targeting processed food marketing and consumer education (63, 65).

Food frequency questionnaires (FFQs), in particular, are advantageous for their self-administration and machine-readability, making them practical and cost-effective (65). They also allow relatively long-term, habitual diet to be assessed. On the other hand, dietary recall and prospective dietary records can only feasibly track the diet for a few days, are labour intensive for participant and researcher, and can be expensive. However, they are more precise than FFQs (66).

Although dietary assessment has high practical potential, the validity of the method as a tool for measuring salt intake in a population is questionable (63). Particularly concerning is that significant bias can easily be introduced by both participant response and interpretation by the researcher since the methods are highly dependent on accurate and precise quantification of foods. Underreporting of general intake and of specific foods/nutrients by participants has consistently been found in previous studies, across all studied types of dietary assessment, which generally perform poorly in sensitivity analyses (65, 67-69). In particular, the tendency for under-reporting of energy intake introduces bias for sodium intake estimation since sodium intake is highly correlated with total energy intake (64, 70). In a recent study published in 2018, Y. Park et al. (71) found Automated Self-Administered 24-hour recalls (ASA24s) 4-Day Food Records (4DFRs), and Food Frequency Questionnaires (FFQs) underestimated sodium intake by 15-17%, 18-21%, and 29-34% respectively, when compared to the 24-hour urinary sodium biomarker. Other studies generally found similar or greater under-reporting of sodium intake.

2.3.2 Urine assessment

In contrast to dietary assessment, urine assessment involves the measurement of the concentration of sodium ions in the urine, often through an ion electrode. It is estimated approximately 98% of sodium in food is absorbed into the blood stream and approximately 90% is detectable in the urine over a given 24-hour period (72). The 10% loss has been attributed to sweat and faeces, and can be variable across seasons (63). Despite these losses, the collection of 24-hour urine is considered to be the most valid biochemical indicator of daily dietary sodium and thus, widely regarded as the 'gold standard' method for sodium intake. As per its name, it involves the collection of all urine throughout a 24-hour period, thereby enabling the estimation of daily sodium intake.

However, 24-hour urine has its challenges. Practically, 24-hour urine collection is burdensome for participants undertake, which can deter many from recruitment and from successful completion of participation, thus lowering the response rate and introducing selection bias. This was a particular concern raised at the WHO NCD surveillance monitoring and evaluating consultation, held in February 2012 in Oslo, Norway (73). A

wide range of response rates have been reported in recent studies – the most successful of which hover around the 50% margin. Some studies have reported extremely low response rates – around 20% in a study from Australia in 2014 (6) and 10% in a study from Switzerland in 2012 (74), when using random sampling methods. Response rates as low as these bring representativeness of study samples into question. As M. A. Land and colleagues argue, in these situations “the random sample effectively becomes a volunteer sample” (6) when they observed their volunteer sample produced similar findings to a random sample in Lithgow, Australia. As such, difficulties in obtaining a representative sample population providing 24-hour urine has the potential to introduce bias, compromising internal validity and limit the generalizability of findings.

The burdensome nature of the 24-hour urine procedure has also resulted in under-collection and over-collection. However, there are various methods used to detect incomplete samples. Oral administration and urinary recovery of para-aminobenzoic acid (PABA) is a common method and considered the gold standard for completeness detection (75). It is excreted in the urine at the same rate as sodium, thereby allowing detection of under-collection. However, there are reported issues including decreased excretion with increased age and non-adherence to oral administration instructions, greatly reducing its potential as the most objective indicator available (76, 77).

Assessment of 24-hour creatinine excretion, 24-hour urine volume, self-report, or a combination of these methods have also been widely used in population studies (3, 4, 6, 78, 79), including in the INTERSALT study (10). However, as A. Wielgosz and colleagues found, there is significant variation in estimates of sodium excretion depending on which method is used and which thresholds are chosen when compared against the use of PABA as the gold standard (75). They suggest administration of PABA should be used in all 24-hour urine studies to estimate population intake. However, as discussed above, PABA has its own complications that can undermine standardized detection of completeness. Balancing effectivity and feasibility for detecting completeness of 24-hour urine collection is still under investigation and currently a topic receiving attention in the field of dietary biomarkers.

When complete, 24-hour urine is an excellent measure of the sodium ingested by an individual over the given 24-hour period. However, it is a less accurate measure of habitual

sodium intake. Day-to-day variability in the diet is common. Therefore, 24-hour sodium excretion will be as variable as intake (72). As such, the collection of repeated 24-hour urine samples is recommended to gain an understanding of habitual (or average) individual sodium intake, particularly when studying its relationship to health outcomes (80). However, 24-hour urine collection can provide a valid estimation of population average and range of sodium intake (63, 72, 80).

Due to concerns of low response rate and the burdensome impracticalities associated with the collection of 24-hour urine, the collection of spot (or ‘casual’) urine to estimate 24-hour sodium excretion has become increasingly popular. Spot urine collection has many advantages over 24-hour urine collection. In many cases, it requires only one encounter (providing one single urine void) and can be collected on-site, as participants visit a survey centre for example. It also requires less training for staff (81). This enables spot urine to be easily included in large population health and nutrition studies (63).

Several equations exist, which aim to convert spot urinary sodium concentration to estimated 24-hour sodium concentration. Three methods are most commonly used – the Kawasaki method (82), the INTERSALT method (83), and the Tanaka method (84). Validation studies comparing various spot urine methods to the 24-hour urine gold standard have, over the past 10 years, been published extensively.

The efficacy of spot urine to estimate 24-hour sodium excretion still remains under fierce investigation (81, 85-87). Spot urinary sodium is likely a reflection of sodium intake consumed in the very short term – a few hours prior to urination perhaps (63). This suggests that variation of urinary sodium concentration throughout the day is likely highly variable depending on the timing of eating and the contents of the diet. Evidence of this diurnal, variation at the individual level has been reported, showing that night-time samples tend to have lower sodium concentrations than those collected during the day (88).

In a recent study published by Allen et al. (89), they assessed the validity of four predictive equations – Kawasaki, Tanaka, INTERSALT, and Mage – for estimation 24-hour sodium excretion from four timed urine samples (overnight, morning, afternoon, and evening) in a multi-ethnic sample population from the United States. They found, while the Tanaka

equation produced the least bias, none of the equations performed uniformly well across varying demographic groups (gender, race/ethnicity). In a similar, recently published study, Zhou et al. (90) found the Kawasaki, INTERSALT, and Tanaka methods resulted in very high bias for a Chinese, adult population. However, several studies demonstrate that spot urine collection using various predictive equations to estimate 24-hour urine is an adequate estimation of mean urinary sodium excretion, despite significant individual bias (85, 91).

2.3.3 Summary

Two main methods for estimating salt intake were reviewed in this sub-chapter.

1. Dietary assessment
2. Urine assessment

Prospective dietary record, 24-hour dietary recall, and FFQs are considered dietary assessment and do not employ a biomarker as an indicator for sodium intake. They rely on the participant to report consumption of foods and knowledge of salt content of foods in order to estimate sodium intake. While they provide valuable insight to particular food groups or foods that may be contributing to high salt intake in a population, dietary assessment methods are not the most reliable for estimating salt intake.

Urine collection, instead employs sodium concentration in the urine as a biomarker for sodium intake. The collection of 24-hour urine is considered the gold standard method for estimating sodium intake. However, its burdensome nature can compromise response rates and thus increase bias. Spot urine collection could provide a solution, as an estimate of 24-hour urinary sodium. However, predictive equations for estimation have been shown to be biased at the individual level. At the population level, the use of spot urine is considered to be adequate, but still uncertain due to insufficient research.

Despite the challenges and potential shortcomings of the 24-hour urine method, the overwhelming majority of literature supports 24-hour urine collection as the best indicator, and therefore ‘gold standard’ of salt intake. A major conclusion drawn from this section of the literature review is that estimating salt intake is challenging, even in HICs.

2.4 Somalis in Norway

2.4.1 General geography and anthropology

Ethnic Somalis originate from a region on the north-eastern African continent known as the *Horn of Africa*, which today includes the countries of Somalia, Somaliland, Djibouti, Ethiopia, and Kenya. For the most part, Somalis share a uniform language, *Somali*, are primarily Sunni Muslim, and belong to a patrilineal society forming *clans* and *sub-clans* based on Islamic and traditional practice. Such groups are linked to specific geographic origin, identify all Somalis and their affiliations, forming the basis for their social structure – hierarchy and roles.

A significant portion of Somalis in the Horn of Africa engage in a nomadic or semi-nomadic way of life, with around 25% percent of the population in Somalia defined as nomadic by the United Nations Populations Fund (UNFPA) (92). Their non-sedentary, pastoral lifestyle has meant that national borders were often crossed, in following natural resources, resulting in a people more defined by ethnicity and clan than geographic location or political border. While *Somalian* refers to those of residing in the nation of Somalia, *Somali* refers to their shared ethnic identity, transcending borders, and is the relevant term for this thesis.

The Somali civil war officially began in 1988 as an armed resistance to the state. Since then, state opposition transitioned into inter-clan fighting and further into intra-clan fighting, fuelling armed conflict that continues to this day. The widespread poverty and instability pushed many Somalis to refugee camps in neighbouring countries and to migrate to Western countries, creating a complex diaspora (93).

According to the United Nations Refugee Agency (UNHCR), there were 870 000 people from Somalia registered as refugees in the Horn of Africa and Yemen at the beginning of 2018, the majority of which reside in refugee camps in Kenya and Ethiopia (94). However, these numbers are likely greater if taking into account all ethnic Somalis in the Horn of Africa who may be affected by the unrest. Many have also migrated en-masse to Europe, North America, and the Middle East where numbers are steadily increasing as displaced Somalis seek asylum, often via dangerous routes. The number of ethnic Somalis worldwide

remains somewhat uncertain, but estimated to be around two million by the UN Populations Division (UNDP) in 2015 (95).

Somalis therefore constitute a substantial diaspora, plagued by recent and current hardships. As a group, they can be challenging to define as an outsider, unlinked to nationality. Long-term mass migration due to war and famine can negate their ties to the Somali state (as many are born in refugee camps or abroad), further complicated by their persisting quasi-borderless nomadic lifestyle. Ethnic Somalis are a rapidly changing global distribution and truly a people spread across borders.

2.4.2 Settlement in Norway – demographics

Norway is an HIC characterized by stable socialist politics, low unemployment, offering a high quality of life, and consistently ranked first on the Human Development Index (95). Immigration of people from Sub-Saharan Africa is a relatively new occurrence in Norway, with cultural enclaves only established over the past 30 years. According to Statistics Norway, there were approximately 30 000 Somalis living in Norway in 2017, with around 40% who reside in the Oslo Municipality (96). Consequently, Somalis constitute the largest non-European immigrant group in Norway and the second largest in Oslo (96).

The vast majority of Somalis have come to Norway seeking asylum and family reunion (96). According to Statistics Norway, Somalis in Norway are among those with lower education and less employed compared to Norwegians (97). Out of all the major immigrant groups in Norway, Somalis have the highest unemployment rates (98). Moreover, over 10% of Somalis over the age of 16 in Norway have not been formally educated (99). The most recent arrivals tend to be the least educated as they have spent the longest residing in Somalia and the neighbouring regions where infrastructure has been in worsening states of devastation over the past 25 years. Nevertheless, Somalis are the most satisfied with their living conditions out of the major immigrant groups, according to Statistics Norway (100).

2.4.3 Health and dietary habits

There is little known about the health and nutrition status of Somalis in Norway. Somalis in Norway tend to be a relatively young group. Morbidity and mortality patterns are still

emerging, however Norwegian national data suggests the risk of CVD is slightly higher among Somali immigrants than that in the Norwegian population (101).

Other studies found that duration of residence in Norway was associated with higher prevalence of diabetes and obesity most attributable to a decrease in physical activity and drastic change in diet as they adjust to a comparatively sedentary lifestyle and novel food availabilities in Norway (102, 103). However, the studies did not delve into the details of diet.

There is strong evidence from studies on other immigrant populations that the burden of chronic conditions related to dietary change increases with length of residence in the host country (104). The change in nutritional availabilities from their home-country to host-country is likely immense, as they are faced with affordable high caloric and low nutritional foods (105, 106). In Canada, for example, longer length of residence is associated with increased sodium and fat intake (104). A study from the United States found African immigrant men to New York City had low levels of fruit and vegetable consumption (thus low potassium) and little understanding of the benefits of these foods (107). Albeit, the interaction between migration, nutrition, and health is likely not all negative. For example, immigrants to HICs have been shown to consume more calcium, vitamin D, and iron – thus avoiding deficiencies that are prevalent in LMICs (108).

Moreover, cultural differences in health perception can alter the way an ethnic group assesses a health situation. In a study by Renzaho (109), he states that obesity is not defined nor seen as a disease with health consequences in many Sub-Saharan African countries. There is evidence Somalis, like other ethnicities in the region, prefer larger body sizes (106) – a product of their recent history and sustained battle with infectious disease and poverty (109). Interestingly, a survey conducted by Statistics Norway in 2016 reported that immigrants thought their health was ‘good’ or ‘very good’ (73%), which was lower, but close to the Norwegian population (83%).

After a detailed search of literature, the specifics of dietary and lifestyle risk factors for disease are unknown. Assumptions about susceptibilities to disease among Somalis in

Norway should be made cautiously due to the specificity and variation within each population.

3. RATIONALE FOR THIS STUDY

Robust evidence from epidemiological studies and meta-analyses demonstrates that salt is a risk factor for increased BP. Due to the high and increasing prevalence of hypertension globally, it is critical to monitor salt intake. The recent questioning of the safe range of salt intake and the relationship between CVD/mortality and salt intake furthers this importance. There is mounting evidence to suggest that a population-specific understanding of salt intake is necessary. This is for two main reasons. Firstly, salt intake differs remarkably across populations, thus demanding different approaches and targets for interventions intending to bring salt intake to healthy levels. Secondly, sensitivity to salt is heterogeneous across populations. Its specificity is bound to both genetic and environment context.

Somalis and their associated diaspora constitute an understudied population worldwide, on which there is no recent or accurate information on their salt intake status. There is indirect evidence to suggest Somalis in the diaspora may be at high risk of developing hypertension and CVD due to rapid lifestyle changes. As one of the largest immigrant groups in Norway, these knowledge gaps pose a barrier to achieving health equity between Somali immigrants and their host population.

This study aims to provide a baseline salt intake estimate of a Somali population in Norway, which is a priority for Norwegian health authorities. It employs the gold standard biomarker for salt intake – 24-hour urinary sodium excretion – measured through the collection of 24-hour urine. Obtainment of high quality data will serve as a baseline for building a well-informed profile of Somali immigrant health and associated risk factors. This has valuable application as it can inform effective health promotion to meet targets in the global effort to prevent hypertension and cardiovascular events as well as bring us closer to achieving consensus on safe ranges of salt intake.

4. RESEARCH AIMS AND OBJECTIVES

MAIN AIM

The aim of this thesis is to estimate the daily salt intake among adult (age 20-67) Somalis in the Sagene district of Oslo, Norway.

OBJECTIVES

- 1) Estimate the dietary salt intake through the analysis of 24-hour urine
- 2) Measure the association between estimated sodium intake and socio-demographic factors
- 3) Estimate the dietary potassium intake through the analysis of 24-hour urine
- 4) Measure the association between blood pressure and estimated sodium intake, estimated potassium intake, and estimated Na/K intake ratio

5. METHODOLOGY

This chapter provides a description and justification for the methods used to answer the research questions and fulfil the research aims. It opens with a brief overview of methods, followed by study design, participant sampling/recruitment, data collection tools, the procedure, ethics, methodological considerations, and lastly, a description of the statistical analysis carried out.

The methods employed throughout the study were guided by the *WHO STEPwise approach to non-communicable disease risk factor surveillance* (STEPS) – a tool developed by the WHO in 2003 to assist countries in undertaking NCD risk factor surveys (110). It aims to provide feasible, standardized protocol and tools for surveyors such that data areas consistent as possible and can enable comparisons across populations and time. In line with STEPS, this study emphasizes the collection of small amounts of high quality data over large amounts of low quality data through adherence to standardized methods as well as the assembling of risk factor information from a standardized questionnaire before moving to more complex biochemical analyses of urine (110).

5.1 Overview

This study is part of a larger project to survey risk factors for NCDs among Somalis in Oslo, Norway. It employed a community-based, descriptive, cross-sectional design. The study was conducted in the *Sagene* district of Oslo, Norway where the purpose of sampling was to recruit *all* eligible members living in that district. The sample population consisted of adults (age 20-67) of Somali background. Data collection occurred between December 2015 and October 2016. To estimate sodium and potassium intake, 24-hour urinary electrolyte excretion was measured. Samples were collected in Oslo and sent for analysis at the Medical Laboratory at the University Hospital of North Norway. Demographic variables were collected through a structured questionnaire. A validated oscillometric monitor and standardized protocol were used to measure BP. The final sample consisted of 159 participants. All data were assembled in the Services for Sensitive Data (TSD) IT-platform,

which is a remote, secure server where data analysis took place. Statistical analysis was performed using SPSS, Version 24.

5.2 Design

A cross-sectional, descriptive design was deemed appropriate for answering the principal research objective of estimating salt intake among Somali immigrants in Oslo, Norway. Additionally, the objective of measuring the association between salt intake and potential risk factors also supports the use of a cross-sectional design. As discussed in *Chapter 1*, this study's primary aim is not to assess the causal relationship between salt intake and BP or health outcomes. Therefore, a longitudinal design was not deemed necessary. However, this study design does provide a baseline survey for a follow-up study to be conducted to better assess the association and directionality of the relationship between salt intake and health outcomes in a longitudinal design. Adherence to standardized protocol guided by WHO STEPS for this baseline study will prove beneficial for future follow-up and cross-population comparison. However, for the purposes of the principal study objective, a cross-sectional design was sufficient and feasibly ideal.

5.3 Participants

5.3.1 Defining the study area and population

Oslo is Norway's largest and most culturally diverse city, divided into 15 districts which operate their own community organizations and activities. The district of Sagene in Oslo, Norway is just 3.1 km², with a total population of 39,918 in 2015 (111), and lies near to the metropolitan centre of the city. It was estimated there were 1,370 Somalis living in Sagene in 2015, making Sagene the third-most Somali-populous district in Oslo (and therefore in Norway) (112). The Somali community, in this district, is dense, and has a reputation for being a tightly-knit community. Numerous Somali organisations exist, allowing excellent access to participants. For these reasons, Sagene was chosen as an ideal study area to recruit the sample population.

The target population is adults (20-67 years old) with a Somali background living in the Norway. This includes Somali immigrants of any immigration status and all Norwegians born to Somali immigrants. Somali was defined as any person who self-identifies as ethnic Somali and/or identifies as having a Somali background.

5.3.2 Sampling strategy

A non-probability sampling strategy was employed. Conventional probability sampling methods – namely random sampling – often used in quantitative studies were not applied in this study since previous attempts to do so in immigrant populations in Norway resulted in low response rates (113). For example, Somalis were not included in the Oslo Health Study (HUBRO) (113) due to low response rate. HUBRO recruited participants through letters sent via post to a randomized sample of Somalis using demographic data from Statistics Norway. Reflection upon HUBRO revealed that Somalis were less trusting of impersonal, written information and thus may be more receptive to oral communication among people of the same background and shared language. In order to improve the response rate for this study, a community-based, voluntary sampling strategy in one district was deemed more appropriate. The goal of sampling was therefore to recruit *all* eligible Somali adults in the Sagene district. For this reason, a sample size calculation was not conducted prior to recruitment.

5.3.3 Inclusion criteria

Potential participants were required to fulfil the following criteria to be included in the study:

- Must be between the ages of 20 and 67 years old
- Must have Somali background (self-defined)
- Must have their home address registered in the Sagene district of Oslo, Norway
- Any genders were permitted

There were 272 potential participants who were identified as eligible during the recruitment process. The term “Somali background” was used during recruitment so as to not exclude potential participants who were not direct immigrants from Somali regions. Inclusion based on ethnicity was self-defined by the participants. Citizenship was not a factor for inclusion.

5.3.4 Exclusion criteria

Participants were excluded from the study if:

- Pregnant
- Begun diuretic medication less than 2 weeks prior to recruitment
- Diagnosed with kidney failure
- Diagnosed with haemorrhage
- Diagnosed with liver disease

These conditions disturb, what would otherwise be, physiologically regular urinary electrolyte and/or volume excretion in response to dietary intake (114, 115). Since 24-hour urinary electrolyte excretion was used as the biomarker to estimate electrolyte intake in this study, known conditions that prevent urinary excretion from reflecting intake were excluding factors. There were 20 potential participants excluded from the study based on these grounds, leaving 252 people who were eligible to give consent.

5.4 *Recruitment*

5.4.1 Community-based efforts and cultural sensitivity

In order to reach the greatest number and widest range of people of Somali background in the district, a community-based effort was mobilized. This included the help of Somali organizations, the district healthy life centre, volunteer centre, district medical officer, and community centre. Also, ten youth volunteers and three project assistants were identified by partner organizations. They aided in the recruitment of participants and in data collection. Contact with these parties was established and they worked to disseminate information about the study through Somali radio, community centre activities, and other informal methods, such as oral communication. The Somali organizations were particularly instrumental in establishing contact with potential participants since the majority of Somalis residing in Sagene are members. The district medical officer's office, healthy life centre and the community development centre also assisted in the dissemination of information in the

attempt to reach the remaining eligible Somalis who were not members of the Somali organizations.

An official information meeting was held at the Sagene community centre on the 29th of November 2015. Partner organizations posted information sheets to notify and encourage eligible members to come (See *Appendix B* for information sheet). At the meeting, members were informed about the purpose of the study, the various processes they would be involved in as participants, and the potential risks and benefits of participation. A question period was also held. They were given contact information and were encouraged to arrange an appointment with the researchers for signing of consent and data collection.

Cultural sensitivity was integral throughout the recruitment process for adequate recruitment. As such, oral communication between partner organizations and potential participants was encouraged to establish trust. In addition, potential participants were notified that Somali female project assistants were available during the data collection process – important for a balanced recruitment of men and women.

5.5 *Measurement tools*

5.5.1 Urine collection

All components of the urine collection kit were purchased from the medical and laboratory equipment company, Sarstedt AG & Co.

- 1) A 3.0 litre urine container for 24-hour storage and a 500 mL receptacle (UriSet24®)
- 2) An integrated unit for closed urine transfer using a vacuum system (V-Monovette®)
- 3) A 2 mL polypropylene micro-tube

Both UriSet24® and the V-Monovette® system are European Conformity (CE) and In-Vitro Diagnostic Directive (IVDD) certified (116). The receptacle and urine container system was ideal since it allowed women to easily collect urine – a barrier that can be a deterrent for women to participate. Also, the 3.0L container was equipped with a transparent inspection strip to easily measure total urine volume, such that feedback could be given to participants immediately. The closed-urine transfer system to the micro-tube was an advantage as well,

preventing contamination of the urine in the sample sent to the laboratory for analysis. See *Appendix C* for full urine collection kit specifications

5.5.2 Laboratory analysis

Urine samples were analysed in one-batch at Medical Laboratory, University Hospital of North Norway (UNN) for 24-hour urine creatinine, sodium, and potassium concentration. The Medical Laboratory, UNN has been certified by Norwegian Accreditation according to the standard, *NS-EN ISO 15189 TEST 209*. The laboratory at UNN employed the IVDD approved Roche Hitachi system – an indirect ion-selective electrode (ISE) to determine ion concentration (117).

5.5.3 Questionnaire

To collect socio-demographic variables, an interviewer-administered questionnaire in Norwegian was employed (see *Appendix D* for questionnaire). The project leader or project assistants were responsible for asking the questions and filling the questionnaire. All assisting members were proficient in Somali and Norwegian to assist with proper translation and clarification. The project leader was always present to ensure standard comprehension and recording.

The questionnaire was an updated version of that used in HUBRO (113). A pilot study was conducted, primarily to test areas which were not relevant for this particular sub-study. Since the questionnaire was used for the entirety of the broader project on risk factors for chronic disease among Somali immigrants in Oslo, the majority of the assessments did not pertain to this particular study. See *Table 2* for description of variables that were collected from the questionnaire. Variables chosen were identified as possible associated socio-demographic factors for salt intake by construction of Directed Acyclic Graphs (DAGs) as informed by the literature review:

5.5.4 Blood pressure testing

Blood pressure was measured using the automated device, *Omron HBP 1300*. The device has been validated in accordance with international protocol from the European Society of

Hypertension(118). Hypertension was defined as >140 mmHg systolic and/or >90 mmHg diastolic. Those who were on antihypertensive medication were also considered hypertensive.

5.6 *Urine-based exclusion criteria*

Participants with incomplete urine collections were excluded from the analysis. This was assessed through a combination of factors, where participants were excluded if they met any of the following criteria:

1. Creatinine < 4 mmol/24h for women and < 6 mmol/24h for men
2. Urine volume > 500 mL/24-hour for either gender
3. Reported missed collection

A total of two participants were excluded from the sample due to creatinine levels below the threshold.

5.7 *Data collection procedure*

The following is a step-by-step recount of the data collection phase of the study:

Recruitment and data collection began quickly after the information meeting, commencing in December 2015 and was completed by October 2016. A private office was provided by the district. It served a meeting point for the project leader and participants for the signing of informed consent and the first phase of data collection. Having been given telephone and address contact information for the private offices during the information meeting, willing participants were able to either schedule an appointment at a designated time or drop-in.

One participant was permitted in the office at a time with the project leader. It began with orally ensuring the participant understood the terms of consent in Norwegian or Somali and signing of the consent form, also in Norwegian (*Appendix E1*) or Somali (*Appendix E2*). A key list was updated including the participant's full name, telephone number, and an identification (ID) code (to be used as their anonymous identity on all future data).

The questionnaire was filled in the same appointment and was reviewed by the project leader. Blood pressure was also taken in the office, after questionnaire filling. If abnormalities in BP were identified, the project leader informed the participant and encouraged them to contact their general practitioner.

The participants were then asked if they wished to participate in the 24-hour urine collection phase of the study. For those who agreed, this was noted in the key list and they were provided with the urine collection kit as well as oral and written instructions for collection. Written instructions were given in Norwegian (see *Appendix F1*) and in Somali (see *Appendix F2*). The project leader and participant organized a date for when the urine collection kit and instruction sheet should be completed and returned to the office. The participant's ID code was noted on the instruction sheet. If the participant wished, it was organized for the project leader or project assistants to collect the urine kit from their home. An SMS reminder to complete the urine collection was sent and a reminder telephone call was made if the participant did not deliver on time.

Participants were instructed to empty the bladder into the toilet when waking up on their day of collection and to note its time. They were instructed to collect urine throughout the rest of the day by using the receptacle to catch urine and to then transfer this to the larger container. The last urination collection was instructed as the first urination of the following day. The urine container was to be kept cool throughout collection and missed collections to be noted.

After each participant returned the urine collection kit, the researcher recorded the total volume, mixed the urine container and took a 2 mL sample. Participants received feedback on their urine volume immediately. The researcher then moved samples of the 24-hour urine to the Norwegian Institute of Public Health (NIPH) biobank to be stored in a -20 °C freezer. This continued as participants returned their urine kits until the end of recruitment in October 2016. All samples (n=169) were taken by a researcher to UNN for analysis. All samples were analysed in one batch using one kit.

5.8 Sample flow

There were 272 individuals identified as eligible for participation during the recruitment process. However, 20 individuals were excluded due to their health condition and 30 individuals were absent or refused participation. Therefore, 222 individuals were consenting and completed the first stage of data collection (questionnaire and BP test). Of these, 169 participants returned their urine kits and samples were sent to the laboratory, while 53 remained unreturned. For 8 samples, data for urine volume, creatinine, potassium, or sodium excretion was missing and 2 samples did not meet the urine-based exclusion criteria. Therefore, the final sample population consisted of 159 individuals. See *Figure 1* for flow chart.

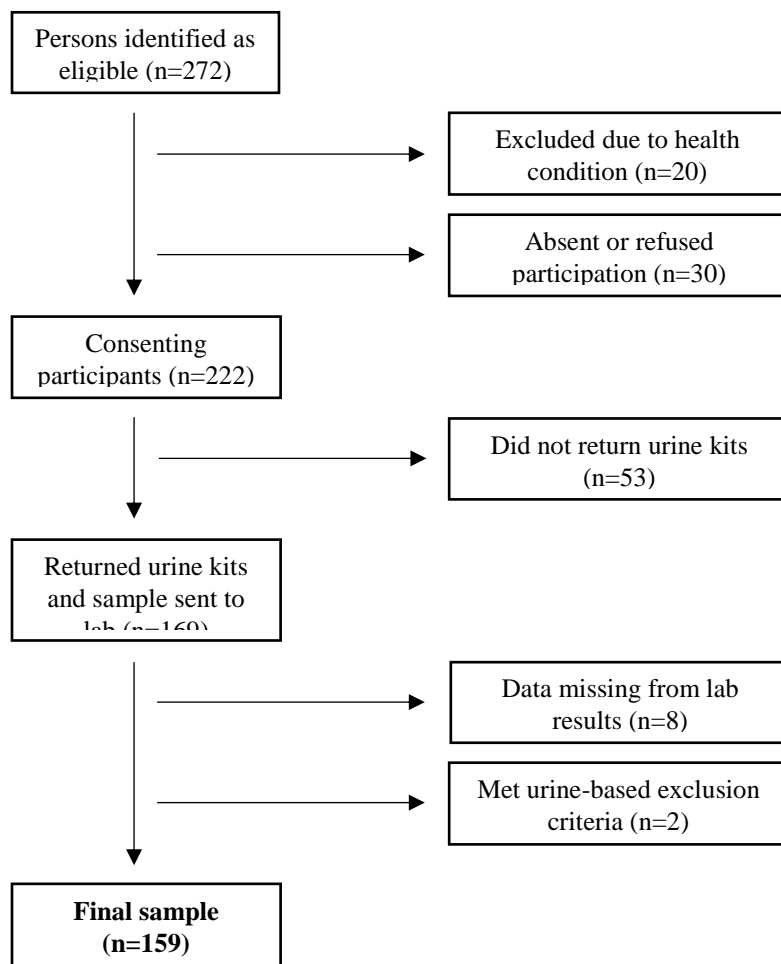


Figure 1. Flow chart exemplifying steps towards achieving a final sample

5.9 Study variables

The following table is a summary of all relevant variables collected in this study (crude and in their final form, ready for analysis)

Table 2. Description of variables used in the study and their derivation

Variable name	Description
<i>Variables obtained from 24-hour urine analysis</i>	
24-hour urine volume	The volume of fluid noted from the indicator on the V-Monovette 3 Litre container. This measurement intends to denote the amount of urine excreted over exactly 24-hours by one individual from the point after the first morning void to (and including) the next morning void. Measured in millilitres (<i>mL/24h</i>).
Urinary sodium concentration	The concentration of sodium in the 2 mL sample of the 24-hour urine. It is indicative of the concentration of sodium in the entire 24-hour urine sample. Measured in millimoles per liter (<i>mmol/L</i>)
24-hour urinary sodium excretion	‘Urinary sodium concentration’ multiplied by ‘24-hour urine volume’. Units are millimoles per 24-hour (<i>mmol/24h</i>).
Estimated 24-hour sodium intake	‘24-hour urinary sodium excretion’ is the gold standard biomarker for sodium intake, keeping in mind that approximately 10% of the sodium has been lost to other excretion (eg. feces, sweat, etc). This study maintains the unadjusted estimate of sodium intake due to the variability of the 10% loss. Units are millimoles per day (<i>mmol/24h</i>)
Estimated daily salt intake	A derivation of ‘estimated sodium intake’. Units are grams per day (<i>g/24h</i>). See statistical analysis for calculation.
Urinary potassium concentration	Same as ‘urinary sodium concentration’ but potassium instead of sodium
24-hour urinary potassium excretion	‘Urinary potassium concentration multiplied by ’24-hour urine volume’
Estimated 24-hour potassium intake	Same as ‘estimated 24-hour sodium intake’ but with potassium instead of sodium
Urinary creatinine concentration	Same as ‘urinary sodium concentration’ but creatinine instead of sodium

24-hour creatinine excretion ‘Urinary creatinine concentration’ multiplied by ‘24-hour urine volume’

Variables obtained from questionnaire

Gender Self-identified dichotomous variable – either ‘man’ or ‘woman’.

Age Number of years determined from date of birth

Length of residence Number of years having lived in Norway

Education Number of years of completed education

Variables obtained from BP monitor

SBP Systolic BP measured in milligrams of mercury (*mm Hg*). Taken as the average of the second and third SBP readings.

DBP Diastolic BP measured in milligrams of mercury (*mm Hg*). Taken as the average of the second and third DBP readings.

5.10 Ethical considerations

The study was approved by the Regional Committee for Medical and Health Research Ethics (study code: 2015/1552 REK South-East). Refer to *Appendix G* for approval document.

Standard requirements for free and informed consent were followed, adhering to the guided template provided by the Regional Committees for Medical and Health Research Ethics (REK) (119). The consent form, which can be found in Norwegian (*Appendix E1*) and Somali (*Appendix E2*), was a self-contained unit which informed participants about the study’s aims, potential risks and benefits to the individual, confidentiality, the nature of consent, and the consent signing. This information was also disclosed orally during the information meeting held on the 29th of November 2015 and during the signing of consent.

This consent form was used for the entirety of the larger Somali project “Surveying of risk factors for chronic disease among Somali immigrants in Oslo, Norway”. They were informed that the main benefit of the study was to gain knowledge about the health situation

of Somalis in Norway and through this engage in health promotion targeting this immigrant population. Participants could decline to take part in any portion of the project, such as the collection of 24-hour urine, which was one of the more burdensome aspects of the project. Other than the burdensome nature, there was no risk of harming the participants. Consent could be withdrawn at any point during the study.

5.11 *Incentives*

Participants were given an incentives gift card in appreciation for their participation in the study. A gift card of 100 Norwegian Kroner (NOK) was given to those who participated in the sociodemographic and clinical data collection phase (questionnaire and BP testing). An additional gift card of 100 NOK was given to those who participated in the urine collection phase for their effort.

Participants had full rights to their results from the study, as disclosed on the consent form. Because BP results were available immediately to the researcher, this was disclosed to the participant during the appointment along with the clinical diagnosis of normotensive or hypertensive. This was an important benefit for participants since there is evidence Somali immigrants partake less in preventative health exams (such as regular physician exams) than their native counterparts in HICs (120, 121). If laboratory data returned abnormal, participants were contacted and informed and were recommended on how to proceed.

5.12 *Data handling*

Questionnaires and BP measurements were taken individually in a private office with the researcher. Participants were assigned ID codes to establish anonymity such that data could not be used to track back to participants. As such, the questionnaire and urine sample were demarcated from the individual through use of this ID code. There was only one key list made during the recruitment process which linked participants to their ID code for the purpose of informing participants of their health status after laboratory results were returned. Otherwise only the ID code was used.

The key list, all questionnaires and BP recordings were stored in separate locked cabinets first at the office in Sagene and later at the Institute of Health and Society, University of Oslo. Only the project leader had access to these documents. Data was transferred to the electronic platform, TSD, for use in analysis and secure storage. The identities of the participants are not possible to discern in the results of the associated article manuscript. Lastly, prior to December 2020, the code list will be destroyed. Participants were informed of data handling procedures prior to giving consent.

5.13 Statistical Analysis

Some preliminary computations were necessary before statistical analysis. Since 24-hour urinary sodium excretion was used as the biomarker for estimated 24-hour sodium intake, estimated daily salt intake (NaCl g/24h) was then derived using the formula:

$$NaCl (g/24h) = Na (mmol/24h) \cdot \frac{58.4(g)}{1000 (mmol)}$$

The 58.4 grams represent the molar mass of sodium chloride, thus using a standard conversion from moles of an element to grams of a compound. The conversion is also supported by Huang et al. (85) who published a systematic review on salt intake from 24-hour urinary excretion and spot urine.

The molar ratio of 24-hour urinary sodium to potassium was calculated as such:

$$\frac{Na (mmol/24h)}{K (mmol/24h)}$$

Mean, medians, and standard deviation (SD) were described for all continuous variables. Number and percentage were reported for categorical variables. Sodium intake is likely to differ by gender. Therefore, results are shown separately for men and women in addition to the combined total. Differences between gender were tested with the independent samples T-test. The Mann-Whitney U-test was used to test the difference in data sets of non-parametric distribution.

For normally distributed variables, Pearson's r was computed to assess correlation between demographic variables and 24-hour sodium excretion. Spearman's Rho correlation

coefficient was used to assess correlations for non-parametric distributions. This was similarly conducted to assess the correlation between 24-hour sodium excretion and both systolic BP (SBP) and diastolic BP (DBP), as well as between the sodium-potassium molar ratio and both SBP and DBP.

Measurements of associations in this study were achieved through the analysis of two separate models. The main model aimed to provide insight on the association between demographic variables and estimated salt intake (using Na-excretion as the indicator), involving four 'sub-models' – one for each demographic exposure variable. Multiple linear regression (β , 95% Confidence Interval) was used to examine the association – after a check of assumptions – between demographic variables (including gender, age, number of years in Norway, and number of years of education) and 24-hour sodium excretion. Two DAGs can be seen below (*Figure 2* and *Figure 3*) for number of years lived in Norway and number of years of education, since they require adjustment. Gender and age exposures did not require adjustment as they are unmodifiable risk factors.

Figure 2. DAG:

Exposure is number of years lived in Norway

Outcome is 24-hour sodium excretion

Potential confounding variables are 'gender', 'age', and 'years of education', which should be adjusted for in the analysis.

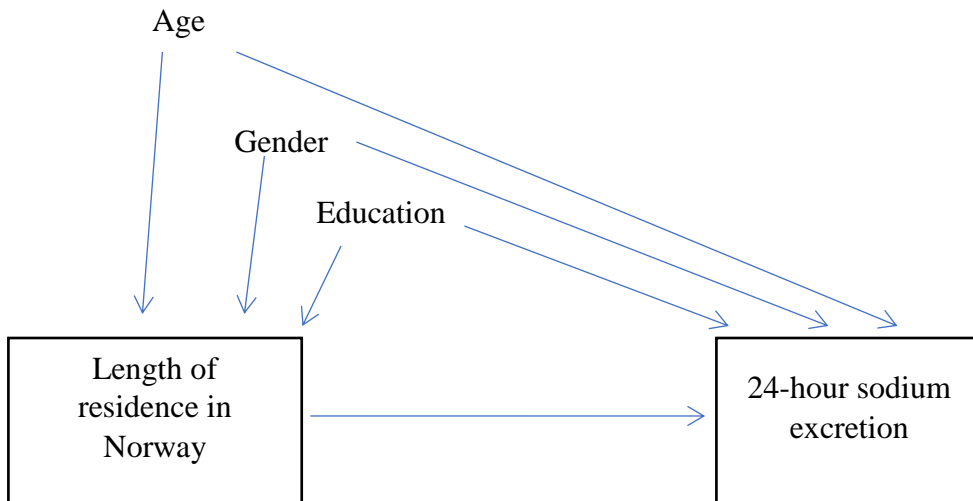
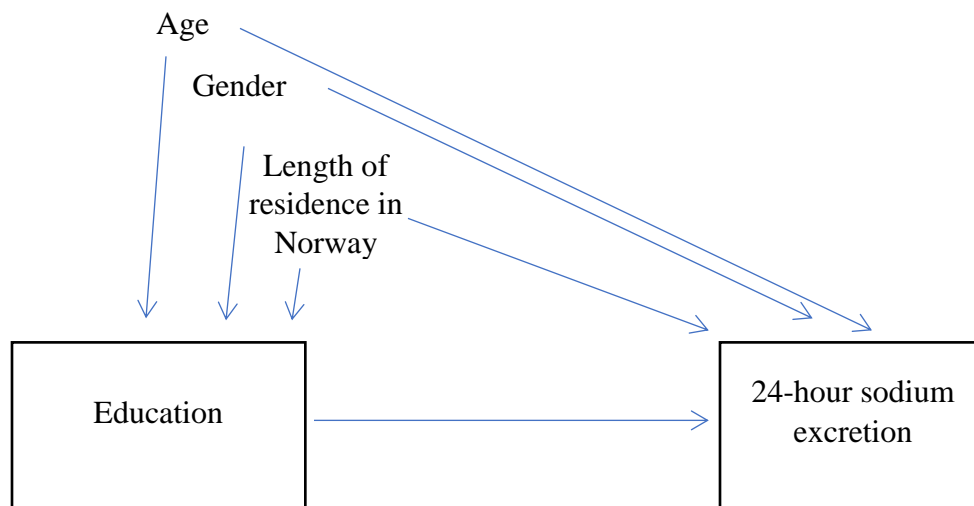


Figure 3. DAG:

Exposure is number of years of education

Outcome is 24-hour sodium excretion

Potential confounding variables are 'gender', 'age', and 'years lived in Norway', which should be adjusted for in the analysis.



The secondary model aimed to assess the relations between 24-hour electrolyte excretion (Na, K, and Na:K ratio) and BP (SBP and DBP). These associations were also modelled using multiple linear regression, controlling for relevant confounding variables based on the literature search and DAGs.

Variables included were checked for co-linearity with the exposure and only included in the model if $VIF < 10$. Collected data was described and analysed using SPSS Statistics Software, Version 24. Tests for differences were two-sided. The significance level was set to $p < 0.05$.

6. METHODOLOGICAL CONSIDERATIONS

This section reflects on how reliability, internal and external validity may have been impacted by the methods employed in this study to understand the quality of data generated and its generalizability for practical application.

6.1 *Study power and reliability*

When assessing reliability, we must consider the stability or consistency of the measurements. The quality of the data collection tools and their associated procedures can constitute an important source of error. Efforts were made to reduce this error by following standard and consistent procedures in areas where the researcher was involved. In particular, measurement of BP was done by the project leader, allocating five minutes of seated rest before taking three BP measurements in one-minute intervals. Further, the questionnaire was interviewer administered, with oral clarification in Somali and/or Norwegian, while the project leader was present. The consistency of these procedures following high quality standardized protocol likely increased the level of reliability and precision of the results. In areas where the researcher was not involved, as in the collection of 24-hour urine, emphasis was placed on giving clear instructions and providing contact information if the participants were ever unsure of how to proceed. However, we cannot know for certain if instructions were followed.

As discussed in *Chapter 2.3.2*, one-time collection of 24-hour urine does not provide a reliable estimate for habitual sodium intake for an individual. Because of high day-to-day variability in the diet, and thus of sodium and potassium intake, very large sample size and/or multiple collections of 24-hour urine from each participant would be necessary to robustly detect a relationship (or lack thereof) (80). Most likely this study was underpowered to reveal a sodium- or potassium-BP relationship, which is why this study focused on estimating population salt intake and potentially associated background factors.

Calculation of sample size to achieve the standard 80% power was not performed prior to data collection since the aim of sampling was to enrol as many eligible participants as

possible. However, a post-hoc analysis of effect size was performed using the Biomath Calculator (122). Power ($1 - \beta$) was set to 80% and the significance (α) set to 0.05. It demonstrated an approximate difference of 26.0 mmol/24h of sodium excretion between men and women was able to be detected when applying our sample size of 159 (75 men and 84 women) and a standard deviation of 57.0 mmol/24h (men). Our results were just under, with a 24.0 mmol/24h difference in sodium excretion between men and women. This demonstrates precision was likely adequate enough with the sample size to reliably detect this difference.

6.2 *Internal validity*

Internal validity is the level at which the results from a study are findings of unbiased occurrences within the source population.

6.2.1 Study design

The appropriateness of the study design for fulfilling the aims and objectives is critical to internal validity. The main aim of this study was to estimate salt intake among Somali adults in Oslo, Norway. The descriptive, population-based nature of this aim lent itself well to the cross-sectional design – a powerful tool in public health for surveying risk factors for disease. The purpose of the study was not to assess changes in population salt intake. However, this study will ideally serve as a baseline measurement for follow-up in a longitudinal design.

The cross-sectional design is also well-suited to measuring the association between salt intake (outcome) and background socio-demographic variables (exposures). Still, it cannot provide explanation for whether a given estimated salt intake was *caused* by a background variable. The same applies to measuring the association between BP and estimated sodium and potassium intake – also part of the specific study objectives. Causation cannot be inferred from the regression analysis performed on the cross-sectional data due to the simultaneous measurement of exposures and outcomes. Even in the case here, where the null hypothesis was not rejected (no association), we cannot conclude that changes in estimated sodium intake have no effect on BP in our sample population. As such, the cross-sectional

design is well-suited to fulfilling the aims and objectives of this study as long as care is taken to not conflate causality with correlation or association.

6.2.2 Selection bias

Selection bias are a results of the procedure used to select participants into the study, which can distort the true value or association. It can occur when characteristics of the sample population associated with the outcome (salt intake and/or BP) do not accurately reflect that of the population the study was sampled from. With respect to this study's main outcome – salt intake – selection bias could have skewed the detected mean intake from the true salt intake in the population of eligible members, thus compromising internal validity. Two aspects may be responsible for introducing selection bias in this study 1) The non-probability sampling method employed and 2) loss of participants throughout stages of the study (see sample flow chart in *Figure 1* to see where eligible members were lost).

The burdensome nature of collecting 24-hour urine may have excluded subsets of the target population who have different salt intake and/or BP profiles compared to the sample population. For example, those who work long hours, on weekends, and shifts may have declined participation or failed to return their urine collection kit (*non-response bias*) due to the inconvenience of collecting 24-hour urine with their schedules. It could be a reasonable assumption that these individuals' diets differ from the sample population, perhaps having less time to make home-cooked meals. Relying more on prepared foods could mean their salt intake was higher than that found in the sample population. As such, this study may have underestimated salt intake in the target population.

Steps were taken, however, to assess the representativeness of the sample population to first- and second-generation immigrants defined as having a Somalian nationality in all of Norway. Using data gathered from Statistics Norway, socio-demographic information was compared. Age distribution and education level were comparable (99), indicating a level of representativeness. However, these are crude comparisons of means since it was not possible to access the entire dataset from Statistics Norway, thus losing information on the distribution around the mean. To investigate *non-response bias*, socio-demographic factors

were compared between respondents (n=169) and non-respondents (n=53), finding no significant difference. Still, random sampling may have minimized selection bias A

6.2.3 Information bias

Information bias is the deviation of data from the true value caused by differential measurements of variables. Although cautions were taken to minimize this bias, we cannot exclude the possibility that the data collection tools did not lend themselves to information bias. Firstly, the questionnaire was interviewer-administered, predominantly by the researcher but sometimes by any one of the three project assistants. Bias can be introduced if the different administrators of the tool (in this case, the questionnaire) employed them in systematically different ways. However, the variables measured from the questionnaire demanded straightforward categorical or numbered responses that were not highly sensitive in nature. Somalis who participated in the study can all be assumed to be legal immigrants since only documented migrants would have had an address registered to the Sagene district – a criterion for inclusion in the study. Therefore, there is no reason to assume that participants would have given untrue information on the number of years they had lived in Norway or the number of years of education they had completed.

Several measures were taken to minimize observer and systematic error from BP assessment. A single, validated oscillometric BP monitor was administered by the same researcher throughout the study. Additionally, as in standard practice, the participant was given a 5-minute rest period and BP was measured three times in one-minute intervals. Despite this, normal circadian fluctuations of blood pressure throughout the day could have resulted in biased measures of blood pressure. Even measuring BP at the same time of day for every participant would not correct for this, since circadian cycles are dependent on sleep/wake times and other highly variable environmental and genetic factors. To our knowledge, epidemiological studies do not attempt to correct for the circadian cycle due to the complexity of circadian variation.

The assessment of 24-hour urinary sodium excretion to estimate sodium intake is possibly the greatest potential source of information bias in this study, despite it being the gold standard method. (The same applies to potassium intake, which was assessed the same way.

However, to simplify, only sodium will be referred to in the following). As stated *Chapter 2.3.2*, roughly 90% of ingested sodium over a 24-hour period is excreted in the urine. The remaining 10% is variable across individuals and populations, excreted in the feces or sweat. This study was not able to account for these losses. Therefore, our findings are likely a slight underestimate of true salt intake. Additionally, it is possible that seasonal variability affected the proportion of sodium collected in the urine where less sodium finds its way to the urine compared to in the winter. Since the study was conducted over an approximate one-year period, seasonal variability may have introduced bias. To gain a crude idea of if this took place, excretion results from participants who collected urine between October and March were compared to those who collected between April and September. No significant difference was found. However, this does not exclude the possibility of bias due to variable sodium loss.

We aimed to reduce information bias in the urine collection by applying urine-based exclusion criteria to assess completeness of the 24-hour urine collection. Exclusion criteria is important for reducing the possibility of under collections, over collections, or tampered collections, which can be common in studies employing 24-hour urine collection methods due to the burdensome nature for participants. The use of PABA recovery, for assessing completeness was not feasible in this study, because of time and resource limits. Additionally, the added complexity and burden of PABA administration had the potential to decrease the response rate. Instead, a combination of urine volume, creatinine excretion, and self-report exclusions were used, which has been deemed adequate (63). Because men and women tend to demonstrate significant differences in creatinine excretion, we chose to adhere to a gender-based exclusion, as used in Land et al. (6). Moreover, instructions for the urine collection were given clearly, orally and in writing, with contact information included if they had questions in the effort to minimize incomplete collections. Despite for clear instruction and use of a combination of urine volume and creatinine exclusion criteria, it is possible incomplete collections entered the final sample analysis. If this occurred, findings from this study may have underestimated complete 24-h salt intake.

6.2.4 Confounder bias

This study attempted to minimize confounder bias by collecting data on variables suspected to be confounders as informed by the literature review, construction and analysis of DAGs which provided a minimum sufficient adjustment set. However, there is a possibility that confounders were missed, thus distorting the association between the exposure and outcome. Body Mass Index (BMI) is a variable that may have been an important confounder of the association between sodium intake and BP. Similar epidemiological studies have adjusted for BMI, with result being that the strength of association between BP and salt intake is attenuated (10, 123). However, we did not find an association between sodium intake and BP.

6.3 *External validity*

External validity refers to the degree of generalizability that can be achieved when making inferences about wider and different populations based on the findings of the study. Generalizability is a complex extrapolation and carries heavy responsibility, as it can have implications for populations the study claims generalizability to. Careful consideration of external validity allows us to better translate research into public health practice – a cornerstone aim of epidemiology. The ability to generalize results is firstly bound to the purpose of the study and the study design. For example RCTs aim to understand causal relationships – using a carefully controlled set of participants to ensure high internal validity. For instance, the ability to trust findings from an RCT that included only people of a certain ethnicity and gender to assess the salt effect on BP would not be compromised even though the sample is not reflective of the general population.

As Rothman et al. (124) explain, descriptive or survey analyses require a different treatment of generalizability. An important distinction is that statistical representativeness plays a central role in the ability to generalize descriptive findings to other populations. Findings from descriptive analyses are highly dependent on the complex characteristics/demographics of the population, time, and geography. This study falls into this category, as it is of descriptive nature. It sampled 159 Somali adults living in Sagene – one of 15 districts in Oslo, Norway – to estimate their salt intake and associated socio-demographic factors. This

was commissioned by the NIPH to gain an understanding of salt intake among Somalis in Norway with the intention of promoting healthy living.

Although generalizability cannot be claimed with certainty, there were several measures taken throughout the study and results from post-hoc comparisons which increase the likelihood that our findings are generalizable to the adult Somali population in Norway. Firstly, participants were sampled from the geographic confines of the Sagene district – a modest neighbourhood in Oslo with average personal intake close under the national median intake, according to Statistics Norway. Secondly, as described in *Chapter 6.2.2*, socio-demographic data between our sample and data from Statistics Norway were comparable. Thirdly, it is possible we made contact with the vast majority of Somali adults in Sagene due to the community-based sampling method, of which 58% provided 24-hour urine samples that were included in the analysis.

However, lack of updates in the Norwegian address registry meant that potentially eligible participants residing in the Sagene district could not participate or could not be reached due to an outdated address. While there is an estimate of the total number of immigrants from Somalia living in Sagene from Statistics Norway, the number of adults is unknown. Moreover, these data do not categorize groups by self-defined ethnicity or origin, but rather the nationality of first generation immigrants and the nationality of the parents of Norwegian citizens born to first-generation immigrants. As discussed in *Chapter 2.4.1*, national borders in the Horn of Africa do not align with ethnicity. As such, we cannot know exactly what percentage of the ethnic Somali population residing in Sagene this study achieved in sampling.

It is also possible the results are more generalizable to Somali adults living in urban regions of Norway. Urban-rural differences in salt intake have been reported in the literature in other countries (18). For example, people living in urban areas may eat restaurant food more frequently than rural counterparts – a major source of salt intake. However, these are speculations, which may not apply to Norway. Further research on food availabilities across Norway and dietary behaviour among Somalis would offer complementary knowledge to our findings.

The results of this study should not be generalized to Somali populations in other countries or other age groups. Salt intake is highly connected to the specific country's food availabilities, which are, for the most part, dependent on national or regional food industries. Findings from this study are also therefore temporally bound. Some country populations are particularly reliant on prepared-food consumption, which has been shown to be a major source of salt (125). The amount of processed foods consumed by Somali adults in Sagene and in Norway is unknown. With respect to low generalizability to other age groups, children and the very elderly consume less food and therefore most likely less salt than adults. It is also likely adolescents and teenagers have a different dietary profile compared to adults. Additionally, the findings from this study should not be generalized to other immigrant groups in Norway since differences in culture, tradition, and genetic profile pose important confounders to BP, salt intake, and associated socio-demographic factors.

Many see lower representativeness as a worthy cost for the collection of internally valid, high quality data –achieved by use of the gold standard, 24-hour urine collection method (6). Although distinct, internal and external validity sometimes exhibit an inverse relationship. For this study, limiting the sample to eligible members residing in Sagene was important for minimizing selection bias. However, these confines may have limited the sample's representativeness of all Somalis in Norway. Nevertheless, due to reasons stated above, it is possible our results are generalizable to the wider population of adult Somalis in Norway.

7. IMPLICATIONS & CONCLUSIONS

It is a challenge, in globalised communities, to consume less than five grams of salt per day due to the widespread availability of processed foods containing already-added salt (36, 126, 127). As found in other HICs (125), the presence of salt in the Norwegian food supply may be ubiquitous. It suggests that current salt intake – deemed unhealthily high by many – is an issue of the pervasiveness of salt in the food industry rather than one of personal choice. The implications of this hypothesis are critical to consider for the success of current and future salt reduction interventions.

Salt reduction is an attractive and widely targetable option for improving CVD health on account of the hard-held hypothesis that the BP-lowering effects of salt reduction will translate into decreased CVD events and mortality (11). Indeed, salt reduction campaigns are routine in public health and have been implemented on almost every level of governance (53, 78, 128). Compared to other populations around the world, it is estimated that both men and women in the population sampled in this study consume under the global average of salt intake. Moreover, the evidence for the heterogeneous salt-BP relationship discussed in *Chapter 2.1.3*, throws a level of uncertainty at swooping salt reduction campaigns aiming to drastically lower population-wide salt intake. On account of the literature and our findings, implementation of a salt reduction intervention targeting Somalis in Norway would be premature action. Nevertheless, the hypothesis that salt intake exceeding 5 g/day confers health risks cannot be refuted by this study on account of its study design and power. Further research is required to assess the health benefits of sodium reduction in this Somali population in Norway. Potassium intake should likewise be further investigated on account of the low intake found in our sample population compared to other populations around the world.

The invitation of *all* eligible Somalis in the district through community-based, culturally sensitive methods is a major strength of this study. Immigrant groups can be relatively inaccessible – one reason for the number of knowledge gaps. This study reports on a specific aspect of health among Somali adults in the Sagene district of Oslo, Norway. It does not intend to pathologise, what can often become, the ‘immigrant situation’. As individuals and

equally valued members of Norwegian society, it is the responsibility of researchers and policy makers to recognize the heterogeneity and complexity of the health situation of its immigrant groups. Therefore, despite the challenges associated with assessing the representativeness of the sample population, the data is a valuable addition to the void of studies on risk factors for disease among Somalis and to Norwegian public health. It provides an important stepping stone for further comparative research such as estimation of salt intake among Somalis in other areas of Norway, other immigrants groups in Norway, and longitudinally. Building a robust health risk factor profile is critical for implementing preventative interventions as well as guiding social and policy actions to optimize health equity between groups.

REFERENCES

1. GBD 2015 Risk Factors Collaborators. Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015. *The Lancet*. 2016;388(10053):1659-724.
2. GBD 2015 Mortality and Causes of Death Collaborators. Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980–2015: a systematic analysis for the Global Burden of Disease Study 2015. *The Lancet*. 2016;388(10053):1459-544.
3. Aparicio A, Rodríguez-Rodríguez E, Cuadrado-Soto E, Navia B, López-Sobaler AM, Ortega RM. Estimation of salt intake assessed by urinary excretion of sodium over 24 h in Spanish subjects aged 7–11 years. *European Journal of Nutrition*. 2017;56(1):171-8.
4. Ortega RM, Lopez-Sobaler AM, Ballesteros JM, Perez-Farinos N, Rodriguez-Rodriguez E, Aparicio A, et al. Estimation of salt intake by 24 h urinary sodium excretion in a representative sample of Spanish adults. *The British journal of nutrition*. 2011;105(5):787-94.
5. Xu J, Wang M, Chen Y, Zhen B, Li J, Luan W, et al. Estimation of salt intake by 24-hour urinary sodium excretion: a cross-sectional study in Yantai, China. *BMC public health*. 2014;14:136.
6. Land M-A, Webster J, Christoforou A, Praveen D, Jeffery P, Chalmers J, et al. Salt intake assessed by 24 h urinary sodium excretion in a random and opportunistic sample in Australia. *BMJ open*. 2014;4(1).
7. Blaustein MP, Leenen FHH, Chen L, Golovina VA, Hamlyn JM, Pallone TL, et al. How NaCl raises blood pressure: a new paradigm for the pathogenesis of salt-dependent hypertension. *American Journal of Physiology - Heart and Circulatory Physiology*. 2012;302(5):H1031-H49.
8. Pocock G, Richards CD. *Human physiology : the basis of medicine*: Oxford University Press; 2006.
9. O'Donnell M, Mente A, Smyth A, Yusuf S. Salt intake and cardiovascular disease: why are the data inconsistent? *European heart journal*. 2013;34(14):1034-40.
10. Intersalt Cooperative Research Group. INTERSALT - An international study of electrolyte excretion and blood-pressure - results for 24 hour urinary sodium and potassium excretion. *Br Med J*. 1988;297(6644):319-28.
11. World Health Organization. *Guideline: Sodium intake for adults and children*. Geneva: World Health Organization; 2012.
12. Powles J, Fahimi S, Micha R, Khatibzadeh S, Shi P, Ezzati M, et al. Global, regional and national sodium intakes in 1990 and 2010: a systematic analysis of 24 h urinary sodium excretion and dietary surveys worldwide. *BMJ open*. 2013;3(12):e003733.
13. American Heart Association. How much sodium should I eat per day? 2017 [cited 2018 05 Apr]. Available from: https://sodiumbreakup.heart.org/how_much_sodium_should_i_eat.
14. Center for Disease Control and Prevention. *Dietary Guidelines for Americans 2015-2020*. 2015.
15. Nordic Nutrition Recommendations. *Nordic Nutrition Recommendations 2012: Integrating nutrition and physical activity 2014* [cited 2018 7 Jan]. Available from: <https://www.norden.org/en/>.

16. Norwegian Health Directorate. Salt action plan 2014-2018: reduction of salt intake in the population. 2014.
17. Mills KT, Bundy JD, Kelly TN, Reed JE, Kearney PM, Reynolds K, et al. Global Disparities of Hypertension Prevalence and Control: A Systematic Analysis of Population-Based Studies From 90 Countries. *Circulation*. 2016;134(6):441-50.
18. Addo J, Smeeth L, Leon DA. Hypertension in sub-saharan Africa: a systematic review. *Hypertension (Dallas, Tex : 1979)*. 2007;50(6):1012-8.
19. Cappuccio FP, Miller MA. Cardiovascular disease and hypertension in sub-Saharan Africa: burden, risk and interventions. *Internal and emergency medicine*. 2016;11(3):299-305.
20. He FJ, Li J, Macgregor GA. Effect of longer-term modest salt reduction on blood pressure. *The Cochrane database of systematic reviews*. 2013(4).
21. Ha SK. Dietary salt intake and hypertension. *Electrolyte & blood pressure : E & BP*. 2014;12(1):7-18.
22. Takahashi H, Yoshika M, Komiyama Y, Nishimura M. The central mechanism underlying hypertension: a review of the roles of sodium ions, epithelial sodium channels, the renin-angiotensin-aldosterone system, oxidative stress and endogenous digitalis in the brain. *Hypertension research : official journal of the Japanese Society of Hypertension*. 2011;34(11):1147-60.
23. Leenen FH. The central role of the brain aldosterone-"ouabain" pathway in salt-sensitive hypertension. *Biochimica et biophysica acta*. 2010;1802(12):1132-9.
24. Osborn JW, Jacob F, Guzman P. A neural set point for the long-term control of arterial pressure: beyond the arterial baroreceptor reflex. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*. 2005;288(4):R846-R55.
25. Huang BS, Leenen FH. Both brain angiotensin II and "ouabain" contribute to sympathoexcitation and hypertension in Dahl S rats on high salt intake. *Hypertension (Dallas, Tex : 1979)*. 1998;32(6):1028-33.
26. Takahashi H. Upregulation of the Renin-Angiotensin-Aldosterone-Ouabain System in the Brain Is the Core Mechanism in the Genesis of All Types of Hypertension. *International Journal of Hypertension*. 2012;2012:242786.
27. Mente A, O'Donnell MJ, Rangarajan S, McQueen MJ, Poirier P, Wielgosz A, et al. Association of Urinary Sodium and Potassium Excretion with Blood Pressure. *N Engl J Med*. 2014;371(7):601-11.
28. Graudal NA, Hubeck-Graudal T, Jurgens G. Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride. *Cochrane Database of Systematic Reviews*. 2017(4).
29. Le Fanu J. Intersalt data. Cross cultural studies such as Intersalt study cannot be used to infer causality. *BMJ (Clinical research ed)*. 1997;315(7106):484; author reply 7.
30. Alderman MH. Salt, blood pressure and health: a cautionary tale. *International journal of epidemiology*. 2002;31(2):311-6.
31. Asayama K, Stolarz-Skrzypek K, Persu A, Staessen JA. Systematic Review of Health Outcomes in Relation to Salt Intake Highlights the Widening Divide Between Guidelines and the Evidence. *American journal of hypertension*. 2014;27(9):1138-42.
32. Stolarz-Skrzypek K, Kuznetsova T, Thijs L, Tikhonoff V, Seidlerova J, Richart T, et al. Fatal and Nonfatal Outcomes, Incidence of Hypertension, and Blood Pressure Changes in Relation to Urinary Sodium Excretion. *JAMA-J Am Med Assoc*. 2011;305(17):1777-85.

33. Mente A, O'Donnell M, Rangarajan S, Dagenais G, Lear S, McQueen M, et al. Associations of urinary sodium excretion with cardiovascular events in individuals with and without hypertension: a pooled analysis of data from four studies. *Lancet (London, England)*. 2016;388(10043):465-75.
34. Graudal NA, Hubeck-Graudal T, Jurgens G. Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride. *Cochrane Database of Systematic Reviews*. 2011(11).
35. Adler AJ, Taylor F, Martin N, Gottlieb S, Taylor RS, Ebrahim S. Reduced dietary salt for the prevention of cardiovascular disease. *Cochrane Database of Systematic Reviews*. 2014(12).
36. Strazzullo P, D'Elia L, Kandala NB, Cappuccio FP. Salt intake, stroke, and cardiovascular disease: meta-analysis of prospective studies. *BMJ (Clinical research ed)*. 2009;339:b4567.
37. World Health Organization. Effect of reduced sodium intake on cardiovascular disease, coronary heart disease and stroke. Geneva: WHO; 2012.
38. O'Donnell M, Mente A, Rangarajan S, McQueen MJ, Wang XY, Liu LS, et al. Urinary Sodium and Potassium Excretion, Mortality, and Cardiovascular Events. *N Engl J Med*. 2014;371(7):612-23.
39. Suckling RJ, He FJ, Macgregor GA. Altered dietary salt intake for preventing and treating diabetic kidney disease. *The Cochrane database of systematic reviews*. 2010(12):Cd006763.
40. Fang X, Wei J, He X, An P, Wang H, Jiang L, et al. Landscape of dietary factors associated with risk of gastric cancer: A systematic review and dose-response meta-analysis of prospective cohort studies. *European journal of cancer (Oxford, England : 1990)*. 2015;51(18):2820-32.
41. Escribano J, Balaguer A, Roque i Figuls M, Feliu A, Ferre N. Dietary interventions for preventing complications in idiopathic hypercalciuria. *The Cochrane database of systematic reviews*. 2014(2):Cd006022.
42. Moosavian SP, Haghighatdoost F, Surkan PJ, Azadbakht L. Salt and obesity: a systematic review and meta-analysis of observational studies. *International journal of food sciences and nutrition*. 2017;68(3):265-77.
43. Franco V, Oparil S. Salt sensitivity, a determinant of blood pressure, cardiovascular disease and survival. *Journal of the American College of Nutrition*. 2006;25(3 Suppl):247s-55s.
44. Weinberger MH, Fineberg NS, Fineberg SE, Weinberger M. Salt sensitivity, pulse pressure, and death in normal and hypertensive humans. *Hypertension (Dallas, Tex : 1979)*. 2001;37(2 Pt 2):429-32.
45. Iatrino R, Manunta P, Zagato L. Salt Sensitivity: Challenging and Controversial Phenotype of Primary Hypertension. *Current hypertension reports*. 2016;18(9):70.
46. Adrogué HJ, Madias NE. Sodium and Potassium in the Pathogenesis of Hypertension. *N Engl J Med*. 2007;356(19):1966-78.
47. Mishra S, Ingole S, Jain R. Salt sensitivity and its implication in clinical practice. *Indian Heart Journal*. 2017.
48. Modesti PA, Hagi MI, Corsoni V, Ferraro A, Di Vincenzo E, Vanni S, et al. Impaired adaptation of cardiopulmonary receptors to Western diet in normotensive black immigrants. *American journal of hypertension*. 1999;12(2 Pt 1):145-50.

49. Pilic L, Pedlar CR, Mavrommatis Y. Salt-sensitive hypertension: mechanisms and effects of dietary and other lifestyle factors. *Nutrition reviews*. 2016;74(10):645-58.
50. Sacks FM, Obarzanek E, Windhauser MM, Svetkey LP, Vollmer WM, McCullough M, et al. Rationale and design of the Dietary Approaches to Stop Hypertension trial (DASH). A multicenter controlled-feeding study of dietary patterns to lower blood pressure. *Annals of epidemiology*. 1995;5(2):108-18.
51. Brunner HR, Baer L, Sealey JE, Ledingham JG, Laragh JH. The influence of potassium administration and of potassium deprivation on plasma renin in normal and hypertensive subjects. *The Journal of clinical investigation*. 1970;49(11):2128-38.
52. Aburto NJ, Hanson S, Gutierrez H, Hooper L, Elliott P, Cappuccio FP. Effect of increased potassium intake on cardiovascular risk factors and disease: systematic review and meta-analyses. *BMJ (Clinical research ed)*. 2013;346:f1378.
53. World Health Organization. Guideline: potassium intake for adults and children. World Health Organization; 2012.
54. World Health Organization. SHAKE the salt habit: the SHAKE technical package for salt reduction. Geneva: WHO; 2016.
55. United Nations General Assembly. Political declaration of the High-level Meeting of the General Assembly on the Prevention and Control of Non-communicable Diseases. New York; 2011.
56. Oyebo O, Oti S, Chen YF, Lilford RJ. Salt intakes in sub-Saharan Africa: a systematic review and meta-regression. *Popul Health Metr*. 2016;14:14.
57. Brown IJ, Tzoulaki I, Candeias V, Elliott P. Salt intakes around the world: implications for public health. *International journal of epidemiology*. 2009;38(3):791-813.
58. Muthuri SK, Oti SO, Lilford RJ, Oyebo O. Salt Reduction Interventions in Sub-Saharan Africa: A Systematic Review. *PloS one*. 2016;11(3):e0149680.
59. Modesti PA, Tamburini C, Hagi MI, Cecioni I, Migliorini A, Neri Serneri GG. Twenty-four-hour blood pressure changes in young Somalian blacks after migration to Italy. *American journal of hypertension*. 1995;8(2):201-5.
60. Meyer HE, Johansson L, Eggen A, Holvik K. Salt intake assessed by 24-hour urine excretion in the Tromso Study 2015-2016. *European journal of preventive cardiology*. 2017;24:S12.
61. Omvik P, Lund-Johansen P, Eide R. Sodium excretion and blood pressure in middle-aged men in the Sogn County: an intra- and interpopulation study. *Journal of hypertension*. 1983;1(1):77-83.
62. Mozaffarian D, Fahimi S, Singh GM, Micha R, Khatibzadeh S, Engell RE, et al. Global Sodium Consumption and Death from Cardiovascular Causes. *N Engl J Med*. 2014;371(7):624-34.
63. McLean RM. Measuring population sodium intake: a review of methods. *Nutrients*. 2014;6(11):4651-62.
64. Ortega RM, Perez-Rodrigo C, Lopez-Sobaler AM. Dietary assessment methods: dietary records. *Nutricion hospitalaria*. 2015;31 Suppl 3:38-45.
65. Prentice RL, Mossavar-Rahmani Y, Huang Y, Van Horn L, Beresford SA, Caan B, et al. Evaluation and comparison of food records, recalls, and frequencies for energy and protein assessment by using recovery biomarkers. *American journal of epidemiology*. 2011;174(5):591-603.

66. Day N, McKeown N, Wong M, Welch A, Bingham S. Epidemiological assessment of diet: a comparison of a 7-day diary with a food frequency questionnaire using urinary markers of nitrogen, potassium and sodium. *International journal of epidemiology*. 2001;30(2):309-17.
67. Gemming L, Jiang Y, Swinburn B, Utter J, Mhurchu CN. Under-reporting remains a key limitation of self-reported dietary intake: an analysis of the 2008/09 New Zealand Adult Nutrition Survey. *European journal of clinical nutrition*. 2014;68(2):259-64.
68. Freisling H, van Bakel MM, Biessy C, May AM, Byrnes G, Norat T, et al. Dietary reporting errors on 24 h recalls and dietary questionnaires are associated with BMI across six European countries as evaluated with recovery biomarkers for protein and potassium intake. *The British journal of nutrition*. 2012;107(6):910-20.
69. Subar AF, Kipnis V, Troiano RP, Midthune D, Schoeller DA, Bingham S, et al. Using intake biomarkers to evaluate the extent of dietary misreporting in a large sample of adults: the OPEN study. *American journal of epidemiology*. 2003;158(1):1-13.
70. Whelton PK, Appel LJ, Sacco RL, Anderson CA, Antman EM, Campbell N, et al. Sodium, blood pressure, and cardiovascular disease: further evidence supporting the American Heart Association sodium reduction recommendations. *Circulation*. 2012;126(24):2880-9.
71. Park Y, Dodd KW, Kipnis V, Thompson FE, Potischman N, Schoeller DA, et al. Comparison of self-reported dietary intakes from the Automated Self-Administered 24-h recall, 4-d food records, and food-frequency questionnaires against recovery biomarkers. *The American journal of clinical nutrition*. 2018;107(1):80-93.
72. Hunter D, Van Dam R. Biochemical Indicators of Dietary Intake. In: Willett W, editor. *Nutritional Epidemiology*. Oxford: Oxford University Press; 2013.
73. World Health Organization. European regional technical consultation on noncommunicable disease surveillance: Report of the Consultation. Geneva; 2012.
74. Hawkes C, Webster J. National approaches to monitoring population salt intake: a trade-off between accuracy and practicality? *PloS one*. 2012;7(10):e46727.
75. Wielgosz A, Robinson C, Mao Y, Jiang Y, Campbell NR, Muthuri S, et al. The Impact of Using Different Methods to Assess Completeness of 24-Hour Urine Collection on Estimating Dietary Sodium. *Journal of clinical hypertension (Greenwich, Conn)*. 2016;18(6):581-4.
76. Jakobsen J, Pedersen AN, Ovesen L. Para-aminobenzoic acid (PABA) used as a marker for completeness of 24 hour urine: effects of age and dosage scheduling. *European journal of clinical nutrition*. 2003;57(1):138-42.
77. John KA, Cogswell ME, Campbell NR, Nowson CA, Legetic B, Hennis AJ, et al. Accuracy and Usefulness of Select Methods for Assessing Complete Collection of 24-Hour Urine: A Systematic Review. *Journal of clinical hypertension (Greenwich, Conn)*. 2016;18(5):456-67.
78. Laatikainen T, Pietinen P, Valsta L, Sundvall J, Reinivuo H, Tuomilehto J. Sodium in the Finnish diet: 20-year trends in urinary sodium excretion among the adult population. *European journal of clinical nutrition*. 2006;60(8):965-70.
79. Uechi K, Sugimoto M, Kobayashi S, Sasaki S. Urine 24-Hour Sodium Excretion Decreased between 1953 and 2014 in Japan, but Estimated Intake Still Exceeds the WHO Recommendation. *The Journal of nutrition*. 2017.

80. Olde Engberink RHG, van den Hoek TC, van Noordenne ND, van den Born BH, Peters-Sengers H, Vogt L. Use of a Single Baseline Versus Multiyear 24-Hour Urine Collection for Estimation of Long-Term Sodium Intake and Associated Cardiovascular and Renal Risk. *Circulation*. 2017;136(10):917-26.
81. Iwahori T, Miura K, Ueshima H, Chan Q, Dyer AR, Elliott P, et al. Estimating 24-h urinary sodium/potassium ratio from casual ('spot') urinary sodium/potassium ratio: the INTERSALT Study. *International journal of epidemiology*. 2016.
82. Kawasaki T, Itoh K, Uezono K, Sasaki H. A simple method for estimating 24 h urinary sodium and potassium excretion from second morning voiding urine specimen in adults. *Clinical and experimental pharmacology & physiology*. 1993;20(1):7-14.
83. Brown IJ, Dyer AR, Chan Q, Cogswell ME, Ueshima H, Stamler J, et al. Estimating 24-hour urinary sodium excretion from casual urinary sodium concentrations in Western populations: the INTERSALT study. *American journal of epidemiology*. 2013;177(11):1180-92.
84. Tanaka T, Okamura T, Miura K, Kadowaki T, Ueshima H, Nakagawa H, et al. A simple method to estimate populational 24-h urinary sodium and potassium excretion using a casual urine specimen. *Journal of human hypertension*. 2002;16(2):97-103.
85. Huang L, Crino M, Wu JHY, Woodward M, Barzi F, Land M-A, et al. Mean population salt intake estimated from 24-h urine samples and spot urine samples: a systematic review and meta-analysis. *International journal of epidemiology*. 2016;45(1):239-50.
86. Ji C, Sykes L, Paul C, Dary O, Legetic B, Campbell NR, et al. Systematic review of studies comparing 24-hour and spot urine collections for estimating population salt intake. *Revista panamericana de salud publica = Pan American journal of public health*. 2012;32(4):307-15.
87. Polonia J, Lobo MF, Martins L, Pinto F, Nazare J. Estimation of populational 24-h urinary sodium and potassium excretion from spot urine samples: evaluation of four formulas in a large national representative population. *Journal of hypertension*. 2017;35(3):477-86.
88. Wang CY, Cogswell ME, Loria CM, Chen TC, Pfeiffer CM, Swanson CA, et al. Urinary excretion of sodium, potassium, and chloride, but not iodine, varies by timing of collection in a 24-hour calibration study. *The Journal of nutrition*. 2013;143(8):1276-82.
89. Allen NB, Zhao L, Loria CM, Van Horn L, Wang CY, Pfeiffer CM, et al. The Validity of Predictive Equations to Estimate 24-Hour Sodium Excretion: The MESA and CARDIA Urinary Sodium Study. *American journal of epidemiology*. 2017;186(2):149-59.
90. Zhou L, Tian Y, Fu JJ, Jiang YY, Bai YM, Zhang ZH, et al. Validation of spot urine in predicting 24-h sodium excretion at the individual level. *The American journal of clinical nutrition*. 2017;105(6):1291-6.
91. McLean R, Williams S, Mann J. Monitoring population sodium intake using spot urine samples: validation in a New Zealand population. *Journal of human hypertension*. 2014;28(11):657-62.
92. United Nations Populations Fund. *Population Estimation Survey 2014: for the pre-war regions of Somalia*. UNFPR; 2014.
93. Koshen HIA. Strengths in Somali Families. *Marriage & Family Review*. 2007;41(1-2):71-99.

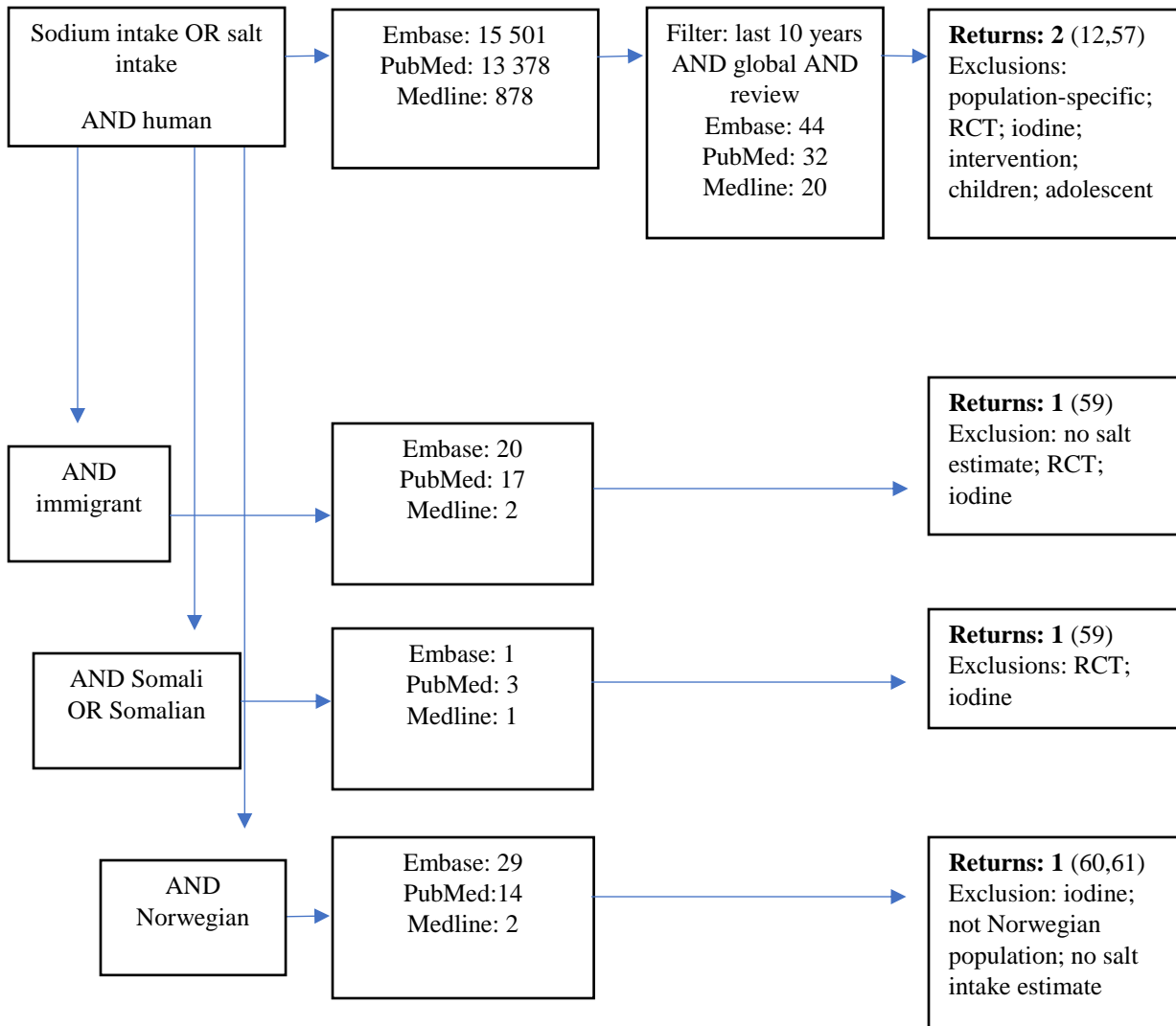
94. The UN Refugee Agency. Population Statistics: The UN Refugee Agency; 2015 [cited 2017 03 Sep]. Available from: http://popstats.unhcr.org/en/time_series.
95. United Nations Development Programme. Human Development Reports: Norway: UNDP; 2015 [cited 2017 09 Apr]. Available from: <http://hdr.undp.org/en/countries/profiles/NOR>.
96. Statistics Norway. Table 09817: Immigrants and Norwegian-born to immigrant parents, by immigration category, country background and percentages of the population 2010 - 2018: SSB; 2018 [updated 1 Jan 2018; cited 2018 27 Mar]. Available from: <https://www.ssb.no/en/statbank/>.
97. Statistics Norway. 11609: Employed immigrants, by sex, age, country background and years of residence in Norway. 4th quarter 2008 - 2017 2018 [updated 1 Jan 2018; cited 2018 16 Mar]. Available from: <https://www.ssb.no/en/statbank/>.
98. Statistics Norway. 07117: Registered unemployed 15-74 years, by country background, world region and sex. Absolute numbers and in per cent of the labour force 2001K1 - 2017K4: SSB; 2017 [cited 2017 09 Dec]. Available from: <https://www.ssb.no/en/statbank/>.
99. Statistics Norway. 09623: Immigrants 16 years and over, by level of education and country background. Numbers 2004 - 2016: Statistics Norway; 2018 [updated 1 Jan 2018; cited 2018 24 Apr]. Available from: <https://www.ssb.no/en/statbank/>.
100. Statistics Norway. Worse living conditions, yet high trust and strong belonging: SSB; 2016 [cited 2018 09 Feb]. Available from: <https://www.ssb.no/sosiale-forhold-og-kriminalitet/>.
101. Rabanal KS, Selmer RM, Igland J, Tell GS, Meyer HE. Ethnic inequalities in acute myocardial infarction and stroke rates in Norway 1994–2009: a nationwide cohort study (CVDNOR). *BMC public health*. 2015;15:1073.
102. Gele AA, Mbalilaki AJ. Overweight and obesity among African immigrants in Oslo. *BMC research notes*. 2013;6:119.
103. Gele AA, Pettersen KS, Kumar B, Torheim LE. Diabetes Risk by Length of Residence among Somali Women in Oslo Area. *Journal of diabetes research*. 2016;2016:5423405.
104. Sanou D, O'Reilly E, Ngnie-Teta I, Batal M, Mondain N, Andrew C, et al. Acculturation and nutritional health of immigrants in Canada: a scoping review. *J Immigr Minor Health*. 2014;16(1):24-34.
105. Venters H, Gany F. African Immigrant Health. *Journal of immigrant and minority health*. 2011;13(2):333-44.
106. Janson E, Bolmsjö I. Obesity in Somali migration women due to post migration dietary changes and decreasing self-esteem :: a qualitative interview study on diet, knowledge about risk of heart disease, inactivity, body image and self-esteem. *Journal Of Research In Obesity*. 2013(142971).
107. Wolf RL, Lepore SJ, Vandergrift JL, Wetmore-Arkader L, McGinty E, Pietrzak G, et al. Knowledge, barriers, and stage of change as correlates of fruit and vegetable consumption among urban and mostly immigrant black men. *Journal of the American Dietetic Association*. 2008;108(8):1315-22.
108. Pomerleau J, Ostbye T, Bright-See E. Place of birth and dietary intake in Ontario. II. Protein and selected micronutrients. *Preventive medicine*. 1998;27(1):41-9.

109. Renzaho AM. Fat, rich and beautiful: changing socio-cultural paradigms associated with obesity risk, nutritional status and refugee children from sub-Saharan Africa. *Health & place*. 2004;10(1):105-13.
110. World Health Organization. The WHO STEPwise approach to surveillance of noncommunicable diseases (STEPS). Geneva: World Health Organization; 2003.
111. Oslo Municipality Statistics Bank. Immigrant population by country background, gender, and age 2015 [cited 2018 16 Mar]. Available from: <http://statistikkbanken.oslo.kommune.no/webview/>.
112. Oslo Municipality Statistics Bank. Population by country background 2008-2016: Oslo Municipality; 2015 [cited 2018 13 Mar]. Available from: <http://statistikkbanken.oslo.kommune.no/webview/>.
113. Norwegian Institute of Public Health. The Oslo Health Study (HUBRO) Oslo: Norwegian Institute of Public Health (NIPH); 2005 [cited 2017 30 Nov]. Available from: <https://www.fhi.no/en/studies/regional-health-studies/the-oslo-health-study-hubro/>.
114. Shemin D, Dworkin LD. Sodium balance in renal failure. *Current opinion in nephrology and hypertension*. 1997;6(2):128-32.
115. Lote CJ. Disease conditions which alter renal sodium and water reabsorption. *Principles of Renal Physiology*. Dordrecht: Springer Netherlands; 1994. p. 165-82.
116. Sarstedt AG & Co. Sarstedt Diagnostic Products [cited 2018 03 May]. Available from: <https://www.sarstedt.com/en/products/diagnostic/urine/urine-monovetter/>.
117. Roche Diagnostics. 510(k) Summary - ISE Indirect Na, K, CI for Gen.2. Indianapolis: Office of In Vitro Diagnostic Devices (OIVD); 2005.
118. Takahashi H, Yokoi T, Yoshika M. Validation of the OMRON HBP-1300 upper arm blood pressure monitor, in oscillometry mode, for clinic use in a general population, according to the European Society of Hypertension International Protocol revision 2010. Dable Education. 2014.
119. Regional Committees for Medical and Health Research Ethics. Templates for Participation Information and Consent: REK; 2017 [cited 2018 06 Apr]. Available from: <https://helseforskning.etikkom.no/>.
120. Morrison TB, Wieland ML, Cha SS, Rahman AS, Chaudhry R. Disparities in preventive health services among Somali immigrants and refugees. *J Immigr Minor Health*. 2012;14(6):968-74.
121. Gele AA, Pettersen KS, Torheim LE, Kumar B. Health literacy: the missing link in improving the health of Somali immigrant women in Oslo. *BMC public health*. 2016;16(1):1134.
122. Division of Biomathematics and Biostatistics. Biomath: unpaired t-test: Columbia University Medical Center; [cited 2018 01 May]. Available from: <http://biomath.info/power/ttest.htm>.
123. Stamler J, Chan Q, Daviglus ML, Dyer AR, Van Horn L, Garside DB, et al. Relation of Dietary Sodium (Salt) to Blood Pressure and Its Possible Modulation by Other Dietary Factors: The INTERMAP Study. *Hypertension (Dallas, Tex : 1979)*. 2018.
124. Rothman KJ, Gallacher JEJ, Hatch EE. Why representativeness should be avoided. *International journal of epidemiology*. 2013;42(4):1012-4.
125. Webster JL, Dunford EK, Neal BC. A systematic survey of the sodium contents of processed foods. *The American journal of clinical nutrition*. 2010;91(2):413-20.

126. Wang J, Olendzki BC, Wedick NM, Pursuitte GM, Culver AL, Li W, et al. Challenges in sodium intake reduction and meal consumption patterns among participants with metabolic syndrome in a dietary trial. *Nutrition Journal*. 2013;12(1):163.
127. Stolarz-Skrzypek K, Bednarski A, Kawecka-Jaszcz K, Czarnecka D, Staessen JA. Will Sodium Intake Reduction Improve Cardiovascular Outcomes in the General Population? A Critical Review of Current Evidence. *Current hypertension reviews*. 2015;11(1):22-9.
128. McLaren L, Sumar N, Barberio AM, Trieu K, Lorenzetti DL, Tarasuk V, et al. Population-level interventions in government jurisdictions for dietary sodium reduction. *The Cochrane database of systematic reviews*. 2016;9:Cd010166.

APPENDIX A

The final literature search was conducted on 28 April 2018 using three databases: Embase and Medline accessed through Ovid; and PubMed. Results were few enough to review titles or titles and/or abstracts individually to look for relevant articles and then remove duplicates. The search was carried out in three databases since initial searches prior to the final, formal search returned highly varied numbers of results across the databases. All searches began with the terms in the top left box: ‘sodium intake’ OR ‘salt intake’; AND ‘human’. The search term “Somalian” was included later on since the Modesti et al. (59) article was found through the reference list of another source and was not detected in the databases by the use of the term “Somali”.



UiO : Universitetet i Oslo



Informasjonsmøte om helseundersøkelse

Alle voksne med Somalisk bakgrunn er invitert til å delta et informasjonsmøte om helseundersøkelse blant somaliere i bydel Sagene.

Dette er samarbeid mellom SKAS, Sagene Foreldregruppe, Sagene Samfunnshus og Det Medisinske fakultetet, Universitetet i Oslo.

Dato: Søndag 29 November 2015

Tid: 18.00-20.00

Sted: Kaysalen, Sagene samfunnshus

Mer informasjon kontakt til:

Hussein 97492881

Mahdi 93856002

Beverting: Det vil bli servert enkel mat og drikke



Product Specification
UriSet 24

Page 1



Product description

Order number	77.578.252
Product description	UriSet 24 - the complete set for optimal 24-hour urine collection

Product characteristics

Graduation	Yes
Type of cap	Screw cap
Light protection	Yes
Design	With stabiliser and urine-Monovette®
Application	Urine collection
Example of use	Collection, stabilisation and storage of urine
Viewing window	Yes

Size

Sample volume	3000 ml
Max. volume	3 l
Diameter of opening	83,5 mm
Width of product	130 mm
Length of product	125 mm

This is the current specification for this product. Sarstedt reserves the right to make changes, in full or in part, at any time without prior notification.

This specification is confidential and the property of Sarstedt. It is neither to be duplicated nor made available to third parties without our prior written consent.

Date of issue:
2018-04-30

This document was prepared by EDP support and is valid without signature.





**Product Specification
UriSet 24**

Length including cap 246 mm
Length excluding cap 243 mm

Material & colours

Product material Polyethylene (PE)
Colour of product Brown
Cap material Polypropylene (PP)
Colour of cap Green

Purity & certification

Product in accordance with IVD
MDD or IVDD
CE certified CE
Lot or serial number batched

Packaging

Minimum order qty. 30
Pcs. / inner box 30
Pcs. / box 30
Pcs. / pallet 270
Depth of case 643 mm
Width of case 403 mm
Height of case 515 mm
Case volume 0,1335 cbm
Weight of case 7,69 KG
EAN of inner box 4038917113878
EAN of outer box 4038917072762

This is the current specification for this product. Sarstedt reserves the right to make changes, in full or in part, at any time without prior notification.

This specification is confidential and the property of Sarstedt. It is neither to be duplicated nor made available to third parties without our prior written consent.

Date of issue:
2018-04-30

This document was prepared by EDP support and is valid without signature.



SARSTEDT

ID-nummer:

**SPØRRESKJEMA OM RISIKOFAKTORER FOR
LIVSSTILSSYKDOMMER**

Fornavn: _____

Etternavn: _____

Adresse: _____

Fødselsdato: (Dag/måned/år): _____

Telefon Nummer: _____

Dato for utfylling ut av skjemaet: _____

ID-nummer:

1. Bakgrunnsspørsmål om deg

1.1 Alder (år): _____

1.2 Kjønn Kvinne Mann Antall barn: _____

1.3 Sivilstand nå gift separert skilt Enslig eller enke/enkemann

1.4 Hvor lenge har du bodd i Norge? Antall år: _____ år

Født i Norge? Ja Nei

1.5 Bor du i bydel Sagene? Ja Nei

Hvor lenge har du bodd i bydel Sagene?: _____

2. Utdanning og arbeid

2.1. Hvor mange års skolegang/utdanning har du fullført?

Antall år.....

2.2. Hvor gode vil du si at dine norskkunnskaper er?

Svært Gode Middels Litt dårlig Dårlig

2.3. Jobber du nå?

Ja, heltid Ja, deltid Nei, jeg er arbeidsløs Jeg studerer Nei, jeg går kurs

Nei, jeg er sykemeldt Nei, jeg er i permisjon

2.4. Hvis du jobber nå, hva slags jobb har du: _____.

ID-nummer:

3. Din Helse	
3.1 Hvordan er helsen din nå?.	Dårlig <input type="checkbox"/> Ikke helt god <input type="checkbox"/> God <input type="checkbox"/> Svært god <input type="checkbox"/>
3.2 Har du diabetes?	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
Dersom ja, hva slags behandling får du:	
Tabletter	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
Insulin	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
Bare kostholdsråd	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
3.3 Har du hatt hjerteinfarkt	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
Dersom Ja, alder første gang: _____	
3.4 Har du hatt Hjerneslag/hjerneblødning	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
Dersom Ja, alder første gang: _____	
3.5 Har du angina pectoris (hjertekrampe)	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
3.6 Har du KOLS (kroniske lungesykdommer)	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
3.7 Har du høyt blodtrykk?	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
Dersom ja, hva slags behandling får du:	
Tabletter	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
Insulin	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
Bare kostholdsråd	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
3.8 Har du høyt kolesterol?	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
Dersom ja, hva slags behandling får du:	
Tabletter	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
Insulin	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
Bare kostholdsråd	Ja <input type="checkbox"/> Nei <input type="checkbox"/>
3.9 Sykdom i familien	
Kryss av for de i familien som har eller har hatt noen av sykdommene:	
	Mor Far Bror Søster Barn Ingen
Hjerneslag eller hjerneblødning	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>
Diabetes (sukkersyke)	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>
Hjerteinfarkt før 60 års alder	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>
3.10 Psykososiale forhold	
3.10.1 Hvor ofte har du følt stress (tenk i løpet av de siste 12 måneder)	
aldri opplevd stress	<input type="checkbox"/>
opplevd enkelte perioder med stress hjemme eller på jobb	<input type="checkbox"/>
opplevd flere perioder med stress hjemme eller på jobb	<input type="checkbox"/>
opplevd permanent med stress hjemme eller på jobben	<input type="checkbox"/>
3.11 Søvnproblemer	
3.11.1 Hvor ofte har du søvnproblemer?	
ingen eller få ganger i året	<input type="checkbox"/>
1-3 ganger/månd	<input type="checkbox"/>
ca engang i uken	<input type="checkbox"/>
mer enn engang i uken	<input type="checkbox"/>

3

ID-nummer:

4. KOSTHOLD

4.1 Brød, pålegg, egg

4.1.1 Hvor mye brød spiser du i løpet av en vanlig dag? (antall skiver)

Fullkorn brød eller knekkebrød _____ antall per dag antall per uke

Hvitt brød _____ antall per dag antall per uke

Halvgrovt (>50% grovhet) _____ antall per dag antall per uke

4.1.2 Hvor ofte spiser disse påleggene?

	Sjelden/aldri	1-2 ggr/uke	3-4 ggr/uke	hver dag
Ost hvit, gul	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Ost, brun	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Majones / Coleslaw	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Kjøtt pålegg	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Syltetøy	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Nugatti/sjokolade	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Fiskepålegg	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

(makrell/sardin i tomat, sardin i olje, havbris posteit, røkt laks, svolværposteit og liknende)

4.2 Egg

7.1 Spiser du vanligvis egg? (stekt, kokt, eggerøre, omlett)?

Antall egg pr. dag eller uke

Sjelden/aldri	1-3 pr. uke	4-6 pr. uke	1 pr. dag	2-3 pr. dag	4-6 pr. dag
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

4.3 Melk og melkeprodukter

4.3.1 Driker du vanligvis melk og melkeprodukter som drikke eller bruker det i matlaging?

- Ja
 Nei → gå til spørsmål

Hvis ja, ber vi deg å angi type og mengde. (sett ett kryss pr. linje)

4.3.2 Hvor ofte drikker du melk?	Antall glass pr. uke el. pr. dag (i gjennomsnitt)					
	Sjelden/aldri	1-3 pr. uke	4-6 pr. uke	1 pr. dag	2-3 pr. dag	4-6 pr. dag
H-melk	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Lettmelk	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Skummet melk	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Ekstra lett, lettmelk (med vitamin D)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Yoghurt, naturell/frukt	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Cultura, alle typer	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Kefir	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

4.3.3 Hvor mye melk drikker vanligvis

< 1/2 glass
 1/2 til 1 glass
 > 1 glass

ID-nummer:

<p>4.3.4 Melk til te/kaffe Hvor mange kopper te/kaffe med melk/fløte drikker du per dag?</p>	<p>Antall kopper pr. uke eller pr. dag Sjelden/aldri 1-3/uke 4-6/uke 1/dag 2-3/dag 4-6/dag</p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p>
<p>4.3.5 Melk til matlaging Bruker du melk/yoghurt til matlaging</p> <p><input type="checkbox"/> Ja, daglig <input type="checkbox"/> Ja, av og til <input type="checkbox"/> Nei</p>	<p>Antall ganger pr. uke el. pr. dag (i gjennomsnitt) Sjelden/aldri 1-3 pr. uke 4-6 pr. uke 1 pr. dag 2-3 pr. dag 4-6 pr. dag</p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p>
<p>4.3.6 Ost Spiser du ost (alle typer)?</p> <p><input type="checkbox"/> Ja, daglig <input type="checkbox"/> Ja, av og til <input type="checkbox"/> nei</p>	
<p>4.3.7 Hvor ofte bruker du melk til kornblanding/frokostblanding?</p> <p>H-melk Lettmelk Skummet melk Ekstra lett, lettmelk (med vitamin D) Yoghurt, naturell/frukt Cultura, alle typer Kefir</p> <p>Hvor mye melk bruker du vanligvis?</p> <p><input type="checkbox"/> < ½ glass <input type="checkbox"/> 1/1 til 1 glass <input type="checkbox"/> > 1 glass</p>	<p>Sjelden/aldri 1-3 pr. uke 4-6 pr. uke 1 pr. dag 2-3 pr. dag 4-6 pr. dag</p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p>
<p>4.4 Sukkerholdige drikker</p>	
<p>Hvor ofte drikker du juice, brus, saft?</p> <p>Eple Appelsin Mango Tropisk Saft tilsatt sukker Brus tilsatt sukker (cola, pepsi, fanta) Brus ikke tilsatt sukker (zero cola, pepsi max, zero fanta)</p> <p>Hvor mye brus, juice, saft drikker vanligvis</p> <p><input type="checkbox"/> < ½ glass <input type="checkbox"/> 1/1 til 1 glass <input type="checkbox"/> > 1 glass</p>	<p>Antall glass pr. uke el. pr. dag (i gjennomsnitt) Sjelden/aldri 1-3 pr. uke 4-6 pr. uke 1 pr. dag 2-3 pr. dag 4-6 pr. dag</p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p> <p><input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/></p>

ID-nummer:

4.5 Kaffe og te

4.4.1 Hvor mange kopper kaffe eller te drikker du daglig? (skriv 0 hvis du ikke drikker kaffe eller te daglig)

Kopper av kaffe kopper

Kopper av te kopper

4.4.2 Hvor mange teskjeer av sukker bruker du i en kopp te/kaffe-----

4.6 Matvarer og retter

4.6.1 Hvor ofte spiser du disse matvarene?

	Sjelden/aldri	1-3ggr/mnd	1-2 ggr/uke	3-4 ggr/uke	5-7ggr/uke
Ris gryte med kjøtt/kylling	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Pasta/spagetti med kjøtt/kylling	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Kebab	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Burger (hamburger, cheese burger)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Pizza	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Pommes frites	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Noodler	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Retter med bønner, linser	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Bare Kjøtt	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Kimis, moofa	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Fetfisk (laks, makrell, ørret, sild)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Vegetabilsk retter	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

4.6.2 Mengde spist for hver gang (slå opp i heftet bilde)

_____ ganger ris eller pasta per dag per uke
Porsjon størrelsen: A B C D

_____ Ganger upolert ris eller fullkorn pasta per dag per uk
Porsjon størrelsen: A B C D

_____ Ganger havregrøt per dag per uk
Porsjon størrelsen : A B C D

_____ Ganger comflakes eller komblanding med lite fiber per dag per uk
Porsjon størrelsen: A B C D

ID-nummer:

___ Ganger havregryn, müsli, frokostblandinger med mye fiber per dag per uk
Porsjon størrelsen: A B C D

___ Antall poteter per dag per uke

___ Ganger bønner, linser per dag per uke
Porsjon størrelsen : A B C D

___ Ganger Kjøtt porsjon størrelsen: per dag per uk
Porsjon størrelsen: A B C D

___ Ganger fisk porsjon størrelsen per dag per uke
Porsjon størrelsen: A B C D

4.6.3 Hvor mange måltider spiser til du i en vanlig dag

Hvor stor andel av hele måltidet er basis som for eksempler (ris, spaghetti, pasta)
 $\frac{1}{4}$ $\frac{1}{2}$ $\frac{3}{4}$ liten eller ingen

Hvis du tenker på de måltidene du vanligvis spiser til middag, hvordan kan du beskrive dem
(kryss det som passer)

Ofte, norsk retter
Blanding, men mest norsk retter
Blanding, men mest somalisk retter
Stort sett somalisk retter

5. Fett (Olje, smør, margarin)

5.1 Hva slags fett bruker du til matlaging (gryter, steking, baking.)?

Olje
Smør
Soft Margarin
Hardt Margarin
Begge
Ingen

5.2 Dersom du bruker olje til matlaging, hva slags olje bruker du til matlaging?

oliven soya mais solsikke rapsolje kokos olje Annen olje

5.3 Hvor mye olje bruker du når du lager gryter, saus eller andre retter?
_____ (ml)

5.4 Bruker du smør/margarin til brød/knekkebrød/lahooh?

Sjelden/aldri 1-2 ggr/uke 3-4 ggr/uke hver dag

ID-nummer:

5.5 Hvis ja: Antall brødkiver/knekkebrød/lahoh per dag: _____

6 Grønnsaker

6.1 Hvor mye grønnsaker spiser du vanligvis?

En porsjon er for eksempel en mellomstor gulrot, 2 tomater eller en liten bolle med salat eller grønnsaker

- ≥ 4 porsjoner/dag
- 2-3 porsjoner/dag
- 1 porsjon/dag
- 4-6 porsjon/uke
- 1-3 porsjoner/uke
- < 1 porsjon/dag

6.2 Hvor mye frukt spiser du vanligvis?

En porsjon er for eksempel et eple, en banan, en håndfull druer, 2 mandariner, 5 jordbær eller en håndfull bær eller fruktsalat.

- ≥ 4 porsjoner/dag
- 2-3 porsjoner/dag
- en del/dag
- 4-6 porsjoner/uke
- 1-3 porsjoner/uke
- < 1 porsjon/dag

7. Søtsaker og dessert

Hvor ofte spiser disse søtsakene?	Sjelden/aldri	1-2 ggr/uke	3-4 ggr/uke	5-7 ggr/uke
Sjokolade	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Kjeks	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Cookie, donut	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Pannekake, vafler	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Tørket frukt (dadler viken, rosiner)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Potetgull	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Søtsaker (halwad)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Samosa	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Iskrem	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Kaker	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Vaniljesaus (labaniyad)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

8. Sauser/dressing

Hvor ofte bruker du disse sausene/dressingene?

	Sjelden/aldri	1-2 ggr/mnd	1-2 ggr/uke	3-4 ggr/uke	5-7 ggr/uke
Thousand Island dressing	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Ketchup	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Majones	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Chili sause	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

ID-nummer:

Shigni	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
--------	--------------------------	--------------------------	--------------------------	--------------------------	--------------------------

9. Salt

9.1 Hvor ofte bruker du?

	Sjelden/aldri	1-2 ggr/uke	3-4 ggr/uke	hver dag
Bord salt	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Hvor mye salt bruker per gang _____ (gram)

10. Tran, vitamin- og mineraltilskudd (kosttilskudd)

10.1 Tar du vitamin kosttilskudd (vitaminer eller mineraler)?

- Ja nå
 Nei → gå til spørsmål

Navn på flytende kosttilskudd	Antall ganger pr. uke								Mengde pr. gang			
	7	6	5	4	3	2	1	<1	1 ts	1 bs	1 ss	
_____	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
_____	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
Navn på kosttilskudd i tablett-/kapselform	Antall ganger pr. uke								Antall pr. gang			
	7	6	5	4	3	2	1	<1	1	2	3	4+
_____	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
_____	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

ID-nummer:

11. Alkohol

Drikker du alkohol? Ja Nei

Dersom ja, omtrent hvor ofte har du i løpet av det siste året drukket alkohol?

(Lettøl og alkoholfritt øl regnes ikke med)

4-7 ggr/uka 2-3 ggr/uka ca. 1 g/uke noen få ggr siste år omtrent 1 gang/mnd

Til dem som har drukket siste år:

Når du har drukket alkohol, hvor mange glass og/ eller drinker har du vanligvis drukket?

Antall.....

Omtrent hvor mange ganger i løpet av det siste året har du drukket så mye som minst 5 glass og/eller drinker i løpet av ett døgn? Antall ganger.....

12. RØYKEVANER

12.1 Hvor lenge er du vanligvis daglig tilstede i røykfylt rom? Antall hele timer.....

12.2 Røykte noen av de voksne hjemme da du vokste opp? Ja Nei

12.3 Bor du, eller har du bodd, sammen med

noen dagligrøykere etter at du fylte 20 år? Ja Nei

12.4 Har du røykt/røyker du daglig? Ja nå Ja tidligere Nei

Hvis ALDRI: Hopp til spørsmål → 13

12.5 Hvis du røyker daglig nå, røyker du:

Sigaretter?..... Ja Nei

Sigarer/sigarillos?..... Ja Nei

Pipe..... Ja Nei

12.6 Hvis du har røykt daglig tidligere, hvor lenge er det siden du sluttet? Antall år.....

12.7 Hvis du røyker daglig nå eller har røykt tidligere:

Hvor mange sigaretter røyker eller røykte du vanligvis daglig? Antall sigaretter.....

Hvor gammel var du da du begynte å røyke daglig? Alder i år.....

Hvor mange år til sammen har du røykt daglig? Antall år.....

ID-nummer:

13. MOSJON OG FYSISK AKTIVITET

13.1 Hvordan har din fysiske aktivitet i fritiden vært det siste året?

Tenk deg et ukentlig gjennomsnitt for året.

Arbeidsvei regnes som fritid. Besvar begge spørsmålene.

	Ingen	Under 1t/uke	1-2 t/uke	3 t & mer
Lett aktivitet (Ikke svett/andpusten)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Hard fysisk aktivitet (Svett/andpusten)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

13.2 Angi bevegelse og kroppslig anstrengelse i din fritid. Hvis

aktiviteten varierer meget f.eks. mellom sommer og vinter, så ta et gjennomsnitt.

Spørsmålet gjelder bare det siste året. (Sett kryss i den ruta som passer best)

- Leser, ser på fjernsyn eller annen stillesittende beskjeftigelse?
- Spaserer, sykler eller beveger deg på annen måte minst 4 timer i uka?
(Her skal du også regne med gang eller sykling til arbeidsstedet, søndagsturer m.m.)
- Driver mosjonsidrett, tyngre hagearbeid e.l.?
(Merk at aktiviteten skal vare minst 4 timer i uka)
- Trener hardt eller driver konkurranseidrett regelmessig og flere ganger i uka?

13.3 Hvor mange minutter går du ute hver dag?

- < 10 minutter 10-30 minutter 1/2 -1 time > 1 time

ID-nummer:

14. Fysisk Målinger

VEKT, HØYDE, MIDJE OG HOFTEMÅLING

1./Høyde (i dag) cm

2.Vekt (i dag) kg

3. Midjemål cm

4. hoftemål cm

BLODTRYKKE MÅLING (Deltakere skal være i ro i 15 minutter og deretter vil blodtrykk målt tre ganger med to minutters mellomrom)

Måling1	Systolisk (mmHg)
	Diastolisk (mmHg)
	Puls (slag/min)
Måling2	Systolisk (mmHg)
	Diastolisk (mmHg)
	Puls(slag/min)
Måling3	Systolisk (mmHg)
	Diastolisk (mmHg)
	Puls (slag/min)
Gjennomsnitt:	Systolisk -----
	Diastolisk -----
	Puls -----

UNIVERSITETET I OSLO

DET MEDISINSKE FAKULTET

FORESPØRSEL OM DELTAKELSE I FORSKNINGSPROSJEKTET

KARTLEGGING AV RISIKOFAKTORER FOR LIVSSTILSSYKDOM BLANT SOMALISKE INNVANDRERE I NORGE

Det er for tiden stort fokus på forebygging av livsstilssykdommer, som diabetes, hjerte- og karsykdommer og kreft. Vi vet lite om helsesituasjonen i den somaliske gruppen i Norge og ønsker derfor å undersøke forekomsten av risikofaktorer for livsstilssykdommer i denne gruppen. Målet er å kunne fremme tiltak for god helse blant somaliske innvandrere i Norge. Studien omfatter kvinner og menn (20-67 år) med somalisk bakgrunn, bosatt i Oslo.

HVA INNEBÆRER PROSJEKTET?

Høyde, vekt, midje og hoftemål vil bli målt. En kvinnelig prosjektmedarbeider vil utføre målingene blant kvinnelige deltakere. Sammen med deg vil vi fylle ut et spørreskjema ved studiens start. Spørsmålene omhandler bl.a., kjønn, alder, utdanning, yrke, bo-lengde, selvrappertert sykdom, diabetes og andre kroniske sykdommer i familien, bruk av medisiner, røykevaner, fysisk aktivitet, mestring og kosthold. Vi vil også måle ditt blodtrykk med standardisert apparat. Deltakere skal være i ro i 15 minutter og deretter vil blodtrykket bli målt tre ganger med to minutters mellomrom.

Du vil få en rekvisisjon til å ta blodprøve hos Først Medisinsk Laboratorium i Oslo sentrum. Blodprøvene skal brukes til å bestemme konsentrasjonen av glukose og glykosert hemoglobin (HbA1c), som viser gjennomsnittlig blodsukker de siste 8-12 ukene før prøven er tatt, lipider (total kolesterol, HDL, LDL og triglyserider) i blodet og andre relevante markører.

Følgende gjelder bare et underutvalg og vil bli utelatt fra informasjonsskrivet til deg det ikke gjelder.

Vi vil også måle natrium og jod i urin. I forbindelse av dette skal du samle urin i ett døgn. Prøvene vil bli analysert med tanke på bl.a. natrium, kalium og jod, og kombinert med den totale mengden urin vil den enkeltes 24 timers natriumutskillelse kalkuleres.

|

UNIVERSITETET I OSLO

DET MEDISINSKE FAKULTET

MULIGE FORDELER OG ULEMPER

Belastningen for deg er at du skal samle urin i ett døgn og må ta blodprøve. Blodprøven blir tatt på vanlig måte i armen av en erfaren biokjemiker hos [Fūrst](#) Medisinsk Laboratorium. Du vil få vite din [høyde](#) og vekt og ditt blodtrykk rett etter måling og blodprøveresultat etter at studien er gjennomført.

FRIVILLIG DELTAKELSE OG MULIGHET FOR Å TREKKE SITT SAMTYKKE

Det er frivillig å delta i prosjektet. Dersom du ønsker å delta, undertegner du samtykkeerklæringen på siste side. Du kan når som helst og uten å oppgi noen grunn trekke ditt samtykke.

Dersom du trekker deg fra prosjektet, kan du kreve å få slettet innsamlede prøver og opplysninger, med mindre opplysningene allerede er inngått i analyser eller brukt i vitenskapelige publikasjoner.

Dersom du senere ønsker å trekke deg eller har spørsmål til prosjektet, kan du kontakte

Ahmed [Madar](#), forsker. a.a.madar@medisin.uio.no, Tel: 99486552

Haakon E. Meyer, professor. h.e.meyer@medisin.uio.no, Tel: 48082702

HVA SKJER MED INFORMASJONEN OM DEG?

Prøvene tatt av deg og informasjonen som registreres om deg skal kun brukes slik som beskrevet her. Alle opplysningene vil bli behandlet konfidensielt. Dataene som forskerne analyserer vil ikke inneholde navn og fødselsnummer eller andre direkte gjenkjenne opplysninger. En kode knytter deg til dine opplysninger og prøver gjennom en kodeliste. Denne koden blir oppbevart på Universitetet i Oslo, og bare personell med ansvar for studien har tilgang til denne. Informasjon om deg vil bli anonymisert eller slettet senest fem år etter prosjektslutt.

Det vil ikke være mulig å identifisere deg i resultatene av studien når disse publiseres.

FORSIKRING

Blod prøve tas hos [Fūrst](#) Medisinsk Laboratorium i Oslo sentrum. Blodprøvetaking for pasienter hos [Fūrst](#) er forsikret via "pasientskadeloven".

ØKONOMI

Studien er finansiert gjennom forskningsmidler fra Helsedirektoratet og Folkehelseinstituttet. Deltakeren får et gavekort som takk for deres deltakelse i studien.

GODKJENNING

Prosjektet vil søke godkjenning fra Regional komite for medisinsk og helsefaglig forskningsetikk.

INNSYNSRETT OG OPPBEVARING AV MATERIALE

Hvis du sier ja til å delta i studien, har du rett til å få innsyn i hvilke opplysninger som er registrert om deg

UNIVERSITETET I OSLO
DET MEDISINSKE FAKULTET

SAMTYKKE TIL DELTAKELSE I PROSJEKTET

JEG ER VILLIG TIL Å DELTA I PROSJEKTET

Sted og dato

Deltakers signatur

Deltakers navn med trykte bokstaver

Jeg bekrefter å ha gitt informasjon om prosjektet

Sted og dato

Signatur

Rolle i prosjektet





UNIVERSITETET I OSLO
DET MEDISINSKE FAKULTET

KA QEYBQAADO BAADHISTA CAAFIMAAD EE XAAFADA SAGENE

BAADHIS CAAFIMAAD OO KU SAABSAN SABABAHA KEENA CUDURADA SIDA WADNE XANNUUNKA, KAADI MACAANKA IYO MASKAX DHIIG FURANKA

Beryahan dambe waxa aad u soo kordhaya cudurada sida wadne xannuunka, macaanka, kansarka iwm ee ku dhaca dadka dalkan ku nool iyo caalamkaba. Hase yeeshee wax badan lagama oga xaaladaha caafimaad ee ajaanibka gaar ahaan Soomalida deggan Norway. Sidaa darteed waxa muhiim ah in wax laga ogaado sababaha keena cuduradan. Ujeedaduna waxa weeye in la keeno hawl waxqabad oo ku saabsan sidii looga hortegi lahaa cuduradan isla markaana wax looga qaban lahaa caafimaadka Soomaalida.

MAXEY KA KOOBANTEY DARAASADAN?

Waxaan cabireynaa cadaadiska dhiigaaga. Waxa la cabirayaa dhererkaaga, dhexdaada iyo miskahaaga. Dumarka waxa cabiraya dumar kale. Sidoo kale waxaad buuxbuuxineysaa foom sida waxbarashadaada, shaqada aad qabato, nooca jimicsi ee aad sameyso, xaaladaada guud ee caamifamaad, cuntada aad cunto iwm.

Waxa lagu siinayaa warqad aad dhiig tijaabo iskaga qaado oo bilaash ah. Waxaad warqada la tegeysaa Füst Medisinsk Laboratorium i Oslo sentrum. Waxa dhiigaaga laga baadhayaa sonkor, xaalada sonkorta dhiigaagu isu bedeshey 2 dii bilood ee ugu dambeeyey, subaga dhiiga ku jira iwm.

Waxa kale oo aad iska qaadeysaa tijaabo kaadi 24 saacadood ah oo laga baadhayo waxyaabo la xidhiidha dhiigkarka (Na, K) iyo aayodhiin (jod) oo wax ka sheegeysa xaalada qanjidhka cunaha. |

DHIB IYO DHEEF

Daraasadani faa'iidooyin badan ayey kuu leedahay waayo waxaad ogaaneysaa xaaladadaada caafimaad sida cadaadiska dhiigaaga, inaad halis ugu jirto macaanka, wadne xanuunka, subaga dhiigaaga ku jira, baruurta ku saaran, inaad cunto sax ah cunto, milix/cusbo badan inaad cunto iwm. Dhibta keliya ee ka iman kartaa waa ururinta kaadida 24 saacadood oo waqti kaaga baahan iyo in dhiig lagaa qaado. Hase yeeshee waxa dhiiga lagaaga qaadaya Füst Medisinsk Laboratorium ee suuqa dhexdiisa ku taalla.

WAA IKHTIYAARKAA HADDII AAD RABTO INAAD KA QAYB QAADATO

Waa ikhtiyaar ka qeybqaadashada daraasadan, sidoo kale marka aad rabtana waad joojin kartaa sabab la'aan. Haddii aad rabto inaad ka qayb gashid waxaad sexeexaysaa warqada qaybta ugu dambaysa.

Haddii aad qabto wax su'aal ah waxaad la xidhiidhaysaa masuulka daraasadan:

Ahmed Madar. a.a.madar@medisin.uio.no, Tel: 99486552 ama 22850634



UNIVERSITETET I OSLO

DET MEDISINSKE FAKULTET

MAXAA LAGU SAMEYNAYAA TIJAABOYINKA IYO MACLUUMAADKA LAGAA URURIYO?

Tijaabooyinka iyo macluumaadka lagaa diiwaan gelin doono waxa loo adeegsan doonaa sida halkan ku xusan: Dhammaan macluumaadka aad dhiibtey waa amaan oo cid aan anaga aheyn oo kale ma ogaan karto. Macluumaadkaaga laguma qori doono wax magac ama dhalasho ah ama wax kale oo lagu garan karo qofka. Lambar sir ah (ID nummer) ayaa loo sameynayaa qof kasta kaas oo ku xidhaya warka iyo tijaabooyinka qofka laga qaadey.

Lambarka sirta ah waxa lagu xafidayaa kulliyada caafimaadka ee jaamacadda Oslo waxaana keliya oo ogaan kara qofka ku shaqo leh daraasadan. Kadib marka daraasadu dhamaato ee qof kasta loo sheego maxsuulkiisa waa la tirtiri doonaa dhammaan magacyada oo dhan. Cid ogaan kartaana ma jirto magacaaga iyo maxsuulka marka natiijada la daabacayo.

CEYMISKA

Dhiiga waxaa lagaaga qaadayaa Fürst Medisinsk Laboratorium i Oslo sentrum.

MAALGELIN

Daraasadan waxa iska kaashanaya Helsedirektoratet, Folkehelseinstituttet iyo Medisinsk fakultet. Dhiigga waxaa lagaaga qaadi doonaa Fürst Medisinske Laboratorium. Qofku markuu iska soo qaadayo tijaabada dhiiga waxa laga bixinayaa kaadhka baska haddii uu u baahdo.

OGOLAANSHAHA DARAASADA

Daraasadan in la sameeyo waxa ogolaadey Regional komite for medisinsk og helsefaglig forskningsetikk.

WAXAAN RABAA INAAN KA QAYBQAATO DARAASADAN

Goobta, taariikh

Saxeexa

Waxaan caddaynayaa in ka qaybqaatuhu war buuxa helay

Goobta, taariikh

Saxeex

Hogaamiyaha daraasada



Etikett

**Dato:****Slik samler du inn døgnurin:**

Det kan være en fordel å samle inn døgnurinen en dag du er mye hjemme og bare er ute for kortere perioder. Bortsett fra dette oppfordrer vi deg til å spise/drikke som vanlig. For kvinner: ikke samle urin under menstruasjon.

Du bes om å samle inn all urin du avgir i løpet av 24 timer. Det er ikke vanskelig, slik gjør du:

Dag 1: Første gang du tisser om morgenen (morgenurinen) tisser du i toalettet. Morgenurinen første dag skal altså **ikke være med**, men tidspunktet for denne vannlatingen noteres:

Dato for første vannlatning (eks. 19 april 2015):

Dag: _____ Måned: _____ År: _____

Klokkeslett for første vannlatning (eks. kl 07:15):

Klokkeslett: _____

Heretter skal ALL urin samles inn i de neste 24 timene, både dag og natt.

Dag 2: når du står opp om morgenen samler du morgenurinen i beholderen. Så er du ferdig. Noter tidspunktet for denne siste vannlatingen, under. MERK: Om innsamlingen ikke har foregått over nøyaktig 24 timer er dette ok så lenge tidspunktene for oppstart og avslutning er nøyaktig notert.

Dato du sluttet innsamlingen (eks. 20 april 2015):

Dag: _____ Måned: _____ År: _____

Klokkeslett du stoppet innsamlingen (eks. kl 10.30):

Klokkeslett: _____

Prøven leveres til Tromsøundersøkelsen i Heilovn 6, dagen innsamlingen avsluttes. Ta gjerne kontakt på tel 776 20 700 hvis du har spørsmål om innsamling eller levering.

tromsundersokelsen.no

Heilovegen 6
+47 776 20 700
Tromso7@uit.no



Merk:

- Tiss all urin i plastmuggen du fikk utlevert, hell dette over til beholderen. Husk alltid å samle urin før evt. avføring.
- Skru lokket ordentlig på beholderen og oppbevar den kjølig, helst i kjøleskap.
- Det gjør ingenting om det ikke er nøyaktig 24 timer så lenge du har notert klokkeslett for start og slutt.

Hvis du mister en urinprøve:

Hvis du ikke får registrert en prøve av en eller annen grunn, for eksempel på grunn av avføring, skal dette noteres; under:

Tidspunkt og et grovt anslag på hvor urin som ikke ble samlet (mye/middels/lite):

Spoturin:

I løpet av dagen du samler inn urin, ber vi også om at du leverer en urinmengde på eget prøverør som du har fått utlevert. Du kan ta dette fra plastmuggen. Det er uansett når på dagen du gjør dette bare du noterer tidspunktet for denne prøven.

Klokkeslett for spoturinen (eks. kl 10.30): _____

**Prøven leveres til Tromsøundersøkelsen i Heilovn. 6, dagen innsamlingen avsluttes.
Ta gjerne kontakt på tel 776 20 700 hvis du har spørsmål om innsamling eller levering.**

Takk for at du bidrar til viktig forskning!



Ururinta kaadida 24 saacadood (døgnurin):

Waxa fiican inaad kaadida ururiso maalin aad inta badan guriga joogto. Uma baahnid inaad wax ka bedesho nolol maalmeedkaaga waqtiga aad kaadida ururineyso. Haddii aad gabadh tahey ha ururin kaadida waqtiga aad caadada leedahey (menstruasjon).

Waxa lagaa codsanayaa inaad ururiso kaadida aad kaadido muddo 24 saacadood gudahood ah. Ma adka ee u samee sidan:

Maalinta 1: Kaadida ugu horeysa subaxnimada **ha keydin** ee iska daadi. (Morgenurinen første dag skal altså ikke være med), laakiin waqtiga aad kaadidey qor:

Waqtiga ugu horeeya ee aad kaadido:

Maalinta: _____ Bisha: _____ Sannadka: _____

Saacada aad kaadidey (Tusaale. kl 07:15):

Saacada: _____

Intaa kadib ururi kaadidaada 24ka saacadood ee soo socda habeen iyo maalinba.

Maalinta 2: Maalinta labaad subixii markaad kacdo ku shub kaadidaada caaga: Qor waqtiga aad kaadidey.

Waqtiga aad joojisey ururinta kaadida:

Maalinta: _____ Bisha: _____ Sannadka: _____

Saacada ugu dambeysey eed kaadidey (subaxa labaad)

Saacada: _____

Xusuus! Marka hore ku kaadi caaga yar dababed ku shub caaga weyn. Daboolka si fiican u giiji dabadeed ku rid qaboojiyaha.

Haddii aad ilowdo inaad ururiso kaadida hal mar ama wax ka badan fadlan qor waqtiga

Maalinta: _____ Bisha: _____ Saacada: _____

Maalinta aad dhameyso uririnta kaadida la hadal:

Samsam 96983994 ama Kawsar 41353967 ama Mohamed 90940919

APPENDIX G



Region: REK sør-øst	Saksbehandler: Harsha Gajjar Mikkelsen	Telefon: 22845513	Vår dato: 24.03.2017	Vår referanse: 2015/1552 REK sør-øst B
			Deres dato: 16.03.2017	Deres referanse:

Vår referanse må oppgis ved alle henvendelser

Haakon E. Meyer
Universitetet i Oslo

2015/1552 Kartlegging av risikofaktorer for livsstilssykdom blant Somaliske innvandrere

Forskningsansvarlig: Universitetet i Oslo
Prosjektleder: Haakon E. Meyer

Vi viser til søknad om prosjektendring datert 16.03.2017 for ovennevnte forskningsprosjekt. Søknaden er behandlet av sekretariatet i REK sør-øst på delegert fullmakt fra REK sør-øst B, med hjemmel i helseforskningsloven § 11.

Prosjektleders prosjekttale

"Forekomsten av diabetes og hjerte- og karsykdommer er ofte høyere blant innvandrere fra lav- og mellom inntektsland enn i vertsbefolkningen. Somaliere er den største ikke-vestlige innvandrergroupe i Norge og det er mangel på kunnskap om deres sykdomsforekomst. Kunnskap om forekomsten av kroniske sykdommer og deres risikofaktorer trengs for å utvikle passende og effektive forebyggende tiltak. I en tverrsnittundersøkelse blant personer i Oslo med somalisk innvandrerbakgrunn vil vi: a) Beskrive forekomsten av risikofaktorer for kroniske sykdommer som diabetes type-2 og hjerte og karsykdommer b) Beskrive salt og jod inntak ved å måle 24 timers urin-utskillelse"

Endringene innebærer

Nye prosjektmedarbeidere i studien er PhD student Sohair Hassan, Masterstudent Niki Marjerisson og Mastersudent Sairah Lai Fa Chen.

Vurdering

Sekretariatet i REK har vurdert de omsøkte endringene, og har ingen forskningsetiske innvendinger til endringene slik de er beskrevet i skjema for prosjektendring.

Vedtak

Søknad om prosjektendring godkjennes med hjemmel i helseforskningsloven § 11, annet ledd.

Godkjenningen er gitt under forutsetning av at prosjektet gjennomføres slik det er beskrevet i søknad, endringsøknad, oppdatert protokoll og de bestemmelser som følger av helseforskningsloven med forskrifter.

Klageadgang

REKs vedtak kan påklages til Den nasjonale forskningsetiske komité for medisin og helsefag, jf. Forvaltningslovens § 28 flg. Eventuell klage sendes til REK Sør-øst B. Klagefristen er tre uker fra mottak av dette brevet.

Besøksadresse:
Gullhaugveien 1-3, 0484 Oslo

Telefon: 22845511
E-post: post@helseforskning.etikkom.no
Web: <http://helseforskning.etikkom.no/>

All post og e-post som inngår i saksbehandlingen, bes adressert til REK sør-øst og ikke til enkelte personer

Kindly address all mail and e-mails to the Regional Ethics Committee, REK sør-øst, not to individual staff

The manuscript begins on the following page. It employs the structure, and referencing format as requested by the *European Journal of Nutrition*. The manuscript was submitted on 23 March 2018 and is currently under review as of today, 11 May 2018.

1 **Estimation of salt intake assessed by 24-hour urinary sodium excretion among Somali**
2 **adults in Oslo, Norway**

3
4
5 Sairah L Chen¹, Cecilie Dahl¹, Haakon E Meyer^{1,2}, Ahmed A Madar¹

6
7 ¹Department of Community Medicine and Global Health, Institute of Health and Society,
8 University of Oslo.

9 ²Norwegian Institute of Public Health Division of Mental and Physical Health, Oslo, Norway

10
11
12
13
14 Corresponding author: Ahmed A Madar

15 Email: a.a.madar@medisin.uio.no

16 p, 0318 Oslo, Norway

17 Telephone: + 47 22 85 06 34, Fax: + 47 22 85 05 90

18
19
20
21 **Key words:** salt; urinary sodium and potassium excretion; Somali immigrants; 24-hour urine
22 collection; Norway

23
24
25
26
27
28
29 *Contributors*

30 A.A. Madar and H.E. Meyer designed the study. A.A. Madar carried out the data collection. S.
31 Chen performed data analysis and prepared the manuscript. A.A. Madar, C. Dahl and H.E.
32 Meyer critically reviewed the draft and contributed to the interpretations of the findings. All
33 authors approved the final version of the manuscript.

34
35 *Statement of interests*

36 On behalf of all authors, the corresponding author states that there is no conflict of interest.

37
38 *Acknowledgements*

39 This study was funded by the Norwegian Institute of Public Health and the Norwegian
40 Directorate of Health. Funders did not have a role in study design, sample, data analysis, or
41 writing of this article. The authors are grateful to all study participants, the Somali organization,
42 Sagene Healthy Living Centre, youth volunteers and the Sagene Medical Officer for their help in
43 completing this study.

44
45
46
47

ABSTRACT

Purpose: High dietary salt intake is associated with increased blood pressure (BP) and cardiovascular disease (CVD) risk. The migration of Somalis from East-Africa to Norway may have altered their dietary habits, making them vulnerable to adverse health outcomes. Since little is known about the lifestyle and health status of this population, the purpose of our study was to estimate salt intake in Somali adults in Oslo, Norway.

Methods: A cross-sectional study involving 162 Somali adults (76 men, 86 women) from the Sagene borough in Oslo, Norway. Sodium and potassium excretion was assessed through the collection of 24-hour urine. Creatinine-based exclusions were made to ensure completeness of urine collections.

Results: Sodium excretion corresponded to estimated dietary salt intake of 8.66 ± 3.33 g/24 h in men and 7.28 ± 3.59 g/24 h in women ($p = 0.013$). An estimated 72% of participants consumed > 5 g salt/day. The Na:K ratio was 2.5 ± 1.2 in men and 2.4 ± 1.1 in women ($p = 0.665$).

Conclusions: Estimated salt intake was, while above the WHO recommendation, within the lower range of estimated salt intakes globally and in Western Europe. Further research is required to assess the health benefits of sodium reduction in this Somali immigrant population.

INTRODUCTION

97
98
99 Increased dietary salt intake is widely acknowledged as a major determinant for raised blood
100 pressure and hypertension [1-3] and has been found to be associated with cardiovascular disease
101 (CVD) outcomes [4,5]. According to the Global Burden of Disease Study 2015, hypertension has
102 been the greatest contributor to mortality and morbidity over the past quarter century [6]. It is a
103 leading risk factor for cardiovascular disease (CVD), which is responsible for one-third of all
104 global deaths [7].

105
106 Globally, it is estimated that individuals consume between six and twelve grams of salt daily [8],
107 far exceeding the WHO's salt recommendation of five grams maximum [9]. However, baseline
108 data on salt intake are lacking in many populations, especially among low- and middle-income
109 communities, including immigrant populations living in high-income countries. This is
110 particularly troublesome considering growing evidence that immigrant communities bear a
111 disproportionate burden of CVD [10-12]. Estimating salt intake from dietary surveys is often
112 unreliable[13]. The 'gold standard' method for estimating salt intake is 24-hour urine collection
113 as around 90% of ingested sodium is excreted in the urine [14-16]. However, the method is
114 burdensome for researchers to conduct and participants to adhere to, often rendering 24-hour
115 urine collection unfeasible [16].

116
117 There is little information about salt intake in the Norwegian population. The Somali community
118 is Norway's largest non-European immigrant group and there is currently no knowledge about
119 salt intake in this population. This study, commissioned by the Norwegian Institute of Public
120 Health, aims to estimate dietary salt intake among Somali immigrants to Norway by the use of
121 24-hour urinary sodium excretion. The baseline measurement is of importance to informing
122 effective health promotion and for monitoring potential changes over time to meet targets in the
123 global effort to prevent hypertension and cardiovascular events.

124

125

126

METHODS

127

128

129

Population and recruitment

130

131

132 This cross-sectional study design attempted to recruit all Somali adults (>18 years) living in the
133 *Sagene* borough of Oslo, Norway. The baseline survey was conducted between December 2015
134 and October 2016. Participants were recruited through a variety of community-based methods,
135 including information meetings at activity centers, and through local Somali radio. Collaboration
136 was established with local Somali organizations, a healthy life center, and three project
137 assistants, identified by these partners, were trained to assist with recruitment and data collection.
138 Door-to-door visits were conducted in order to access every known-Somali adult in the borough.

139

140 Conventional random sampling was deemed not practical as our previous experiences in
141 recruiting immigrant populations through Statistics Norway (SSB) resulted in low response rates
142 [17]. We thus decided to limit the study to one borough in Oslo with a high population of people

143 of Somali origin. Cultural sensitivity during the recruitment process was essential to increase the
144 chance of participation. To establish trust between project leaders and potential participants oral
145 communication was preferred.

146
147

148 *Urine collection*

149

150 Each participant was given a urine collection kit, comprised of a 3.0-liter urine container with an
151 integrated unit for closed urine transfer using the vacuum system, V-Monovette®. Instruction for
152 use was given orally and in writing. First morning void should be discarded, but voids
153 throughout the rest of the day until the morning of the day after were instructed to be collected.
154 Start time, end time, and irregularities were to be noted and the container kept cool throughout
155 collection. Urine collection containers were returned, contents mixed, total volume was recorded,
156 and 2 mL samples of each participant's urine containers were extracted and stored at -20°C. All
157 2 mL samples were sent to the Medical Laboratory, University Hospital of North Norway (UNN)
158 in one batch for one-kit analysis of urine volume, creatinine, sodium, and potassium levels.
159 Urinary sodium and potassium were assessed using Roche Hitachi – an indirect ion-selective
160 electrode to determine ion concentration [18]. The Medical Laboratory at UNN was accredited
161 by Norwegian Accreditation according to the standard, *NS-EN ISO 15189 TEST 209*.

162

163

164 *Collection of other clinical and demographic factors*

165

166 Blood pressure was measured using *Omron HBP 1300* – a validated oscillometric device [19].
167 Participants were given a seated rest-period of five minutes, and then blood pressure was
168 measured three times consecutively for one-minute intervals. Blood pressure was estimated as
169 the average of the second and third measurements. Hypertension was defined as >140 mmHg
170 systolic and/or >90 mmHg diastolic and/or as being on antihypertensive treatment.

171

172 All participants were administered a questionnaire, guided by the WHO Stepwise approach to
173 chronic disease risk factor surveillance (STEPS) [20]. Project assistants were responsible for
174 helping participants complete the questionnaire on paper to ensure standard comprehension and
175 response. Gender, age (in whole years), number of years lived in Norway, education level (years
176 completed), and use of table salt (rarely, sometimes, everyday) were considered relevant
177 variables in the current analyses as assessed by directed acyclic graph (DAG) analysis and
178 literature review.

179

180

181 *Sample flow*

182

183 See *Figure 1* for flow chart of sample population. Around 272 persons were identified as
184 eligible, however 30 persons refused to participate. Participants were excluded if they were
185 pregnant, affected by kidney failure, hemorrhage, liver disease, or had begun diuretic medication
186 less than two weeks prior to recruitment (n=20) (2). A total of 222 participants were included in
187 the study and invited to participate in urine collection as well, but 53 participants did not return
188 their urine collection kits after several attempts at follow-up. The number of urine samples sent

189 to the laboratory was therefore 169; however 8 samples were not analysed for sodium and/or
 190 potassium and/or urine volume. The final study sample comprised of 161 participants (76 men
 191 and 86 women).

192
 193 Two participants were not included in the analysis as they had invalid urine collections assessed
 194 as 24-hour creatinine < 4 mmol/24h for women and < 6 mmol/24h for men, or < 500 mL urine
 195 collected /24-hour [14]. One identified woman had creatinine of 2.18 mmol/24h and one
 196 identified man had creatinine of 3.55 mmol/24h – both just over half the minimum creatinine
 197 required to be included with respect to their gender. Therefore, a total of 159 cases were used to
 198 estimate 24-hour urinary electrolytic content and included in the analysis. One participant did
 199 not complete the questionnaire, but was still included in the analysis for SBP/DBP and urine
 200 excretion.

201

202

203 *Ethics*

204

205 The study was approved by the Regional Committee for Medical and Health Research Ethics
 206 (study code: 2015/1552 REK South-East). Informed written consent was obtained from all
 207 participants.

208

209

210 *Statistical analysis*

211

212 Estimated daily sodium chloride intake (NaCl g/24 h) was calculated from sodium excretion
 213 using the formula [21]:

214

$$215 \text{ NaCl (g/24h)} = \text{Na (mmol/24h)} \cdot \frac{58.4}{1000}$$

216

217 The molar ratio of sodium to potassium was also calculated: $\frac{\text{Na (mmol/24h)}}{\text{K (mmol/24h)}}$

218

219 Mean and standard deviation (SD) were described for all continuous variables. Number and
 220 percentage were reported for categorical variables. Sodium intake is likely to differ by gender,
 221 therefore results are shown separately for men and women. Differences between gender were
 222 tested with the independent samples T-test. The Mann-Whitney U-test was used to test the
 223 difference in data sets of non-parametric distribution.

224

225 For normally distributed variables, Pearson's r was computed to assess correlation between
 226 demographic variables and 24-hour sodium excretion. Spearman's Rho correlation coefficient
 227 was used to assess correlations for non-parametric distributions. This was similarly conducted to
 228 assess the correlation between 24-hour sodium excretion and both systolic blood pressure (SBP)
 229 and diastolic blood pressure (DBP).

230

231 Multiple linear regression – shown as β , 95% Confidence Interval (CI) – was used to examine
 232 the association between demographic variables (including gender, age, length of stay in Norway,
 233 and number of years of education) and 24-hour sodium excretion. The relation between 24-hour

234 sodium excretion and SBP (also DBP) was similarly modelled using multiple linear regression,
235 adjusting for confounding variables. Statistical assumptions were checked in the final models.

236

237 Collected data were described and analysed using IBM SPSS Statistics, Version 24. Tests for
238 differences were two-sided. The significance level was set to $p < 0.05$.

239

240

241

242

243

RESULTS

244 Demographic characteristics and blood pressure data are described in *Table 1*. Mean age was
245 40.2 years. Men were, on average, older than women. However, men and women had spent
246 approximately the same number of years living in Norway. Only 2 participants (1.2%) were born
247 in Norway. Men were significantly more educated than women. SBP and DBP were
248 significantly higher in men compared to women ($p < 0.001$ for SBP and $p = 0.003$ for DBP). A
249 considerable proportion of participants were hypertensive (27.3%).

250 *Table 2* shows levels of excreted sodium and potassium in men, women, and total. Assuming all
251 sodium from dietary intake were excreted through the urine, excretion corresponded to a 24-hour
252 dietary salt intake of 8.66 g in men and 7.28 g in women. Potassium excretion was
253 approximately 12 mmol/24h higher in men compared to women. The Na:K ratio was 2.5 in men
254 and 2.4 in women, and were not significantly different (*Table 2*).

255

256 Multiple linear regression analysis between socio-demographic factors (gender, age, years lived
257 in Norway, and years of education) and 24-hour sodium excretion demonstrated that excretion
258 decreased by 1.5 mmol/24h per year increase in age, when adjusted by gender ($\beta = -1.5$; CI = -
259 2.4, -0.7). Years lived in Norway and years of education showed no significant association with
260 24-hour sodium excretion. Additionally, multiple linear regression showed that for each 100
261 mmol/24h increase in sodium excretion, SBP increased by 1.2 mmHg (CI = -3.2, 5.6) and DBP
262 decreased with 0.8 mmHg (CI = -3.2, 1.6). However these associations were not significant.

263

264

265

266

DISCUSSION

267

Summary of findings

269

270 In this first study of salt intake among adult Somalis (age 20-67) in Norway, we estimated based
271 on 24-hour urine collection that men consumed 8.66 g of salt and women consumed 7.28 g of
272 salt per day. In addition to male gender, younger age was found to be associated with increased
273 salt intake. In the current study, estimated sodium intake was not significantly associated with
274 SBP or DBP.

275

276

Context and added understanding

278

279 There have been few population-based studies measuring sodium intake among Norwegian
280 residents and only one using 24-hour urinary excretion. This previous study by Omvik et al.
281 published in 1983 [22], reported markedly higher (28%) urinary sodium excretion in ethnic
282 Norwegian men (192.5 ± 76.4 mmol/24h) compared to our results. Among Scandinavian
283 populations, a study from Finland conducted in 2002, reported an estimated sodium excretion of
284 160.2 mmol/24h in men and 124.7 mmol/24h in women [23]. Compared to our results, Finnish
285 men excreted slightly more sodium than Somali men in Norway. Differences between the
286 women were negligible. Among a randomly selected group of young Swedish men, sodium
287 excretion was reported as 198.0 ± 69 mmol/24h – much higher compared to our results [24].
288

289 In a systematic review of 24-hour sodium excretion and dietary surveys in 51 studies from
290 Western Europe, Powles et al. [25] found that estimated mean sodium intake ranged from 3.28 to
291 4.43 g/24h, corresponding to a salt intake of 8.33 to 11.25 g/day. Our results for Somali adults
292 are situated near the bottom of this range. Oyeboode et al. [26] conducted a systematic review on
293 sodium intakes in sub-Saharan Africa, finding all populations since 1990, consumed over 2
294 g/24h, with the highest reliable intake estimates to be between 5 and 6 g/24h in Tanzania, South
295 Africa, and Ghana.
296

297 Only one study exists, to our knowledge, reporting 24-hour sodium excretion from a Somali
298 population. Modesti et al. [27] reported sodium excretion of 97 mmol/24h in 25 young Somali
299 immigrants to Florence, Italy upon arrival and a significant increase to 165 mmol/24h after six
300 months of residence. This was accompanied by an 11 mmHg increase in SBP. Our results are
301 similar to the reported sodium excretion after 6 months, suggesting Somalis in Oslo may have a
302 similarly western-acclimated diet with respect to salt [27]. Health implications of this potential
303 dietary change could apply to Somalis living in Norway, making it important to continue
304 monitoring lifestyle risk factors and health outcomes [10,12].
305

306 Men were found to consume significantly more sodium than women, which is reported in the
307 majority of previous studies investigated [25,8,28], with the exception of Oyeboode et al.'s [26]
308 systematic review of sodium intake in Sub-Saharan Africa. This is perhaps due to higher food
309 consumption among men [29]. This study also suggests an inverse relationship between salt
310 intake and age. We hypothesize this could be a result of increased consumption of fast- and
311 prepared- food among younger people, which tend to have higher salt contents than home-made
312 meals.
313

314 A decreased Na:K ratio has emerged as an important factor conferring protection against
315 hypertension and CVD. Potassium has consistently shown blood pressure lowering effects by
316 interacting with salt-sensitive processes to mitigate the blood pressure increasing effects of
317 dietary sodium [1,30-32]. Fruits and vegetables are foods typically rich in potassium. Na:K
318 ratios worldwide demonstrate a wide range, predominantly higher than the unofficial WHO
319 recommendation of 1:1 [33]. Our results are reflective of this, with a large variance and values in
320 the range of other reports. The highest reported ratio from Western-European populations is, to
321 our knowledge, the 3.23 ratio, found in the Gubbio population in Italy [1]. At the lower limit,
322 Laatikainen et al. [23] reported ratios of 2.08 for men and 1.92 for women in the Finnish
323 population. The Somali population in Florence from Modesti et al. [27] demonstrated increased
324 sodium compared to potassium intake after six-months of residence in Italy – from 2.02 (at

325 arrival) to 3.0 (after six months). In comparison, our study population has an intermediate ratio.
326 While our Na:K results are similar to previous studies, from an absolute perspective, our study
327 population consumes far less potassium compared to most populations, which suggests a lower
328 fruit and vegetable intake.

329
330 There was no significant association between sodium intake and blood pressure (SBP and/or
331 DBP) in our study, which is contrary to many comparable studies, [34,1,35]. One reason for this
332 may be that there is individual day-to-day variability in the diet, which affects a given 24-hour
333 sodium excretion. While 24-hour excretion is considered the best indicator for population intake,
334 one single 24-hour excretion is not a precise indicator of a person's habitual intake [15,36]. In
335 addition, certain recent meta-analyses have demonstrated a small and/or weak relationship
336 between sodium intake and blood pressure in populations consuming under 217.39 mmol/24h of
337 sodium and in normotensive populations [30,5]. Our sample has a relatively low prevalence of
338 hypertension, with the majority of participants excreting less sodium than 217.39 mmol/24h.

339
340

341 *Strengths*

342
343 We approached this study with standardized methods – using 24-hour urinary excretion
344 (considered the gold standard) to estimate dietary intake of salt, sodium, and potassium alongside
345 a validated protocol for collection/analysis; and adherence to WHO's recommended framework
346 for NCD surveillance (STEPS) [20]. In addition, recruitment was community-based, focusing on
347 Norway's largest non-Western immigrant population, allowing us to gain understanding of their
348 demographics, sodium and potassium intake and blood pressure status, important background
349 information for effectively improving the health status of marginalised populations [37-39].

350
351

352 *Limitations*

353
354 This study has some limitations. Selection bias may be present as there were some eligible
355 members of the Somali community in the borough who declined participation or could not be
356 reached. In addition, this study selected participants from only one out of 15 districts in Oslo,
357 which may not be representative of Somalis living in other parts of the country. Still,
358 comparison of education levels and age distribution of included participants to data from
359 Statistics Norway suggests that the participants were demographically representative of Somali
360 adults in Norway [40].

361
362 Despite using gold standard methods for estimating salt intake, the collection of one sample per
363 participant is a limitation. Conducting repeated collections and analyses were not feasible.
364 Nevertheless, one-time collection of 24-hour urine is considered a highly valid method for
365 estimating salt intake at the population level [15,13,16]. Still, our results should be interpreted
366 with caution with respect to generalisability of the findings.

367
368
369
370

CONCLUSION

371
372
373 Although above the WHO recommended intake for salt, the estimated salt intake in this Somali
374 immigrant population is within the lower range of salt intakes globally and in Western Europe.
375 Younger men were found to have the highest intake. This study is the first of its kind of an
376 immigrant population in Norway, and it contributes to the small number of studies estimating
377 salt intake in immigrant groups globally. It provides important baseline data on salt intake for
378 comparison to other immigrant groups and ethnic Norwegians, and for inclusion in follow-up
379 studies of longitudinal design.

380

381

382

383

REFERENCES

384

- 385 1. Intersalt Cooperative Research Group (1988) INTERSALT - An international study of
386 electrolyte excretion and blood-pressure - results for 24 hour urinary sodium and potassium
387 excretion. *Br Med J* 297 (6644):319-328
- 388 2. Ha SK (2014) Dietary salt intake and hypertension. *Electrolyte & blood pressure : E & BP* 12
389 (1):7-18. doi:10.5049/ebp.2014.12.1.7
- 390 3. He FJ, Li J, Macgregor GA (2013) Effect of longer-term modest salt reduction on blood
391 pressure. *The Cochrane database of systematic reviews* (4).
392 doi:10.1002/14651858.CD004937.pub2
- 393 4. Mente A, O'Donnell M, Rangarajan S, Dagenais G, Lear S, McQueen M, Diaz R, Avezum A,
394 Lopez-Jaramillo P, Lanas F, Li W, Lu Y, Yi S, Rensheng L, Iqbal R, Mony P, Yusuf R, Yusoff
395 K, Szuba A, Oguz A, Rosengren A, Bahonar A, Yusufali A, Schutte AE, Chifamba J, Mann JF,
396 Anand SS, Teo K, Yusuf S (2016) Associations of urinary sodium excretion with cardiovascular
397 events in individuals with and without hypertension: a pooled analysis of data from four studies.
398 *Lancet* 388 (10043):465-475. doi:10.1016/s0140-6736(16)30467-6
- 399 5. Graudal NA, Hubeck-Graudal T, Jurgens G (2017) Effects of low sodium diet versus high
400 sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride.
401 *Cochrane Database of Systematic Reviews* (4). doi:10.1002/14651858.CD004022.pub4
- 402 6. GBD 2015 Risk Factors Collaborators (2016) Global, regional, and national comparative risk
403 assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of
404 risks, 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015. *The*
405 *Lancet* 388 (10053):1659-1724. doi:10.1016/S0140-6736(16)31679-8
- 406 7. GBD 2015 Mortality and Causes of Death Collaborators (2016) Global, regional, and national
407 life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980–
408 2015: a systematic analysis for the Global Burden of Disease Study 2015. *The Lancet* 388
409 (10053):1459-1544. doi:10.1016/S0140-6736(16)31012-1
- 410 8. Brown IJ, Tzoulaki I, Candeias V, Elliott P (2009) Salt intakes around the world: implications
411 for public health. *International journal of epidemiology* 38 (3):791-813. doi:10.1093/ije/dyp139
- 412 9. World Health Organization (2012) Guideline: Sodium intake for adults and children. World
413 Health Organization, Geneva
- 414 10. Sanou D, O'Reilly E, Ngnie-Teta I, Batal M, Mondain N, Andrew C, Newbold BK,
415 Bourgeault IL (2014) Acculturation and nutritional health of immigrants in Canada: a scoping
416 review. *Journal of immigrant and minority health* 16 (1):24-34. doi:10.1007/s10903-013-9823-7

- 417 11. Dassanayake J, Gurrin L, Payne WR, Sundararajan V, Dharmage SC (2011) Cardiovascular
418 disease risk in immigrants: what is the evidence and where are the gaps? *Asia-Pacific journal of*
419 *public health* 23 (6):882-895. doi:10.1177/1010539509360572
- 420 12. Venters H, Gany F (2011) African Immigrant Health. *Journal of immigrant and minority*
421 *health* 13 (2):333-344. doi:10.1007/s10903-009-9243-x
- 422 13. McLean RM (2014) Measuring population sodium intake: a review of methods. *Nutrients* 6
423 (11):4651-4662. doi:10.3390/nu6114651
- 424 14. Land M-A, Webster J, Christoforou A, Praveen D, Jeffery P, Chalmers J, Smith W,
425 Woodward M, Barzi F, Nowson C, Flood V, Neal B (2014) Salt intake assessed by 24 h urinary
426 sodium excretion in a random and opportunistic sample in Australia. *BMJ open* 4 (1).
427 doi:10.1136/bmjopen-2013-003720
- 428 15. Hunter D, Van Dam R (2013) Biochemical Indicators of Dietary Intake. In: Willett W (ed)
429 *Nutritional Epidemiology*. Oxford University Press, Oxford,
- 430 16. Wielgosz A, Robinson C, Mao Y, Jiang Y, Campbell NR, Muthuri S, Morrison H (2016) The
431 Impact of Using Different Methods to Assess Completeness of 24-Hour Urine Collection on
432 Estimating Dietary Sodium. *Journal of clinical hypertension (Greenwich, Conn)* 18 (6):581-584.
433 doi:10.1111/jch.12716
- 434 17. Kumar GL, Meyer HE, Søggaard AJ, Strand BH (2008) The Oslo Immigrant Health Profile.
435 *Norwegian Institute of Public Health* 2008 (7)
- 436 18. Roche Diagnostics (2005) 510(k) Summary - ISE Indirect Na, K, CI for Gen.2. Office of In
437 Vitro Diagnostic Devices (OIVD), Indianapolis
- 438 19. Cao X, Song C, Guo L, Yang J, Deng S, Xu Y, Chen X, Sapa WB, Wang K (2015) Quality
439 Control and Validation of Oscillometric Blood Pressure Measurements Taken During an
440 Epidemiological Investigation. *Medicine* 94 (37):e1475. doi:10.1097/md.0000000000001475
- 441 20. World Health Organization (2003) The WHO STEPwise approach to Surveillance
442 of noncommunicable diseases (STEPS). *Noncommunicable Diseases and Mental Health*. World
443 Health Organization, Geneva
- 444 21. Huang L, Crino M, Wu JHY, Woodward M, Barzi F, Land M-A, McLean R, Webster J,
445 Enkhtungalag B, Neal B (2016) Mean population salt intake estimated from 24-h urine samples
446 and spot urine samples: a systematic review and meta-analysis. *International journal of*
447 *epidemiology* 45 (1):239-250. doi:10.1093/ije/dyv313
- 448 22. Omvik P, Lund-Johansen P, Eide R (1983) Sodium excretion and blood pressure in middle-
449 aged men in the Sogn County: an intra- and interpopulation study. *Journal of hypertension* 1
450 (1):77-83
- 451 23. Laatikainen T, Pietinen P, Valsta L, Sundvall J, Reinivuo H, Tuomilehto J (2006) Sodium in
452 the Finnish diet: 20-year trends in urinary sodium excretion among the adult population.
453 *European journal of clinical nutrition* 60 (8):965-970. doi:10.1038/sj.ejcn.1602406
- 454 24. Hulthen L, Aurell M, Klingberg S, Hallenberg E, Lorentzon M, Ohlsson C (2010) Salt intake
455 in young Swedish men. *Public health nutrition* 13 (5):601-605. doi:10.1017/s1368980009991431
- 456 25. Powles J, Fahimi S, Micha R, Khatibzadeh S, Shi P, Ezzati M, Engell RE, Lim SS, Danaei G,
457 Mozaffarian D (2013) Global, regional and national sodium intakes in 1990 and 2010: a
458 systematic analysis of 24 h urinary sodium excretion and dietary surveys worldwide. *BMJ open*
459 3 (12):e003733. doi:10.1136/bmjopen-2013-003733
- 460 26. Oyebo O, Oti S, Chen YF, Lilford RJ (2016) Salt intakes in sub-Saharan Africa: a
461 systematic review and meta-regression. *Popul Health Metr* 14:14. doi:10.1186/s12963-015-0068-
462 7

- 463 27. Modesti PA, Tamburini C, Hagi MI, Cecioni I, Migliorini A, Neri Serneri GG (1995)
464 Twenty-four-hour blood pressure changes in young Somalian blacks after migration to Italy.
465 *American journal of hypertension* 8 (2):201-205
- 466 28. Land MA, Neal BC, Johnson C, Nowson CA, Margerison C, Petersen KS (2018) Salt
467 consumption by Australian adults: a systematic review and meta-analysis. *The Medical journal*
468 *of Australia* 208 (2):75-81
- 469 29. Ortega RM, Lopez-Sobaler AM, Ballesteros JM, Perez-Farinos N, Rodriguez-Rodriguez E,
470 Aparicio A, Perea JM, Andres P (2011) Estimation of salt intake by 24 h urinary sodium
471 excretion in a representative sample of Spanish adults. *The British journal of nutrition* 105
472 (5):787-794. doi:10.1017/s000711451000423x
- 473 30. Mente A, O'Donnell MJ, Rangarajan S, McQueen MJ, Poirier P, Wielgosz A, Morrison H, Li
474 W, Wang XY, Di C, Mony P, Devanath A, Rosengren A, Oguz A, Zatonska K, Yusufali AH,
475 Lopez-Jaramillo P, Avezum A, Ismail N, Lanas F, Puoane T, Diaz R, Kelishadi R, Iqbal R,
476 Yusuf R, Chifamba J, Khatib R, Teo K, Yusuf S, Investigators P (2014) Association of Urinary
477 Sodium and Potassium Excretion with Blood Pressure. *N Engl J Med* 371 (7):601-611.
478 doi:10.1056/NEJMoa1311989
- 479 31. Pilic L, Pedlar CR, Mavrommatis Y (2016) Salt-sensitive hypertension: mechanisms and
480 effects of dietary and other lifestyle factors. *Nutrition reviews* 74 (10):645-658.
481 doi:10.1093/nutrit/nuw028
- 482 32. O'Donnell M, Mente A, Rangarajan S, McQueen MJ, Wang XY, Liu LS, Yan H, Lee SF,
483 Mony P, Devanath A, Rosengren A, Lopez-Jaramillo P, Diaz R, Avezum A, Lanas F, Yusoff K,
484 Iqbal R, Ilow R, Mohammadifard N, Gulec S, Yusufali AH, Kruger L, Yusuf R, Chifamba J,
485 Kabali C, Dagenais G, Lear SA, Teo K, Yusuf S, Investigators P (2014) Urinary Sodium and
486 Potassium Excretion, Mortality, and Cardiovascular Events. *N Engl J Med* 371 (7):612-623.
487 doi:10.1056/NEJMoa1311889
- 488 33. World Health Organization (2012) Guideline: potassium intake for adults and children.
489 World Health Organization,
- 490 34. Aburto NJ, Ziolkovska A, Hooper L, Elliott P, Cappuccio FP, Meerpohl JJ (2013) Effect of
491 lower sodium intake on health: systematic review and meta-analyses. *Bmj* 346:f1326.
492 doi:10.1136/bmj.f1326
- 493 35. Stamler J, Chan Q, Daviglus ML, Dyer AR, Van Horn L, Garside DB, Miura K, Wu Y,
494 Ueshima H, Zhao L, Elliott P (2018) Relation of Dietary Sodium (Salt) to Blood Pressure and Its
495 Possible Modulation by Other Dietary Factors: The INTERMAP Study. *Hypertension* (Dallas,
496 Tex : 1979). doi:10.1161/hypertensionaha.117.09928
- 497 36. Olde Engberink RHG, van den Hoek TC, van Noordenne ND, van den Born BH, Peters-
498 Sengers H, Vogt L (2017) Use of a Single Baseline Versus Multiyear 24-Hour Urine Collection
499 for Estimation of Long-Term Sodium Intake and Associated Cardiovascular and Renal Risk.
500 *Circulation* 136 (10):917-926. doi:10.1161/circulationaha.117.029028
- 501 37. Gele AA, Pettersen KS, Kumar B, Torheim LE (2016) Diabetes Risk by Length of Residence
502 among Somali Women in Oslo Area. *Journal of diabetes research* 2016:5423405.
503 doi:10.1155/2016/5423405
- 504 38. Kumar R, Einstein G (2012) Cardiovascular Disease in Somali Women in the Diaspora.
505 *Current Cardiovascular Risk Reports* 6 (3):229-237. doi:10.1007/s12170-012-0233-5
- 506 39. Kinzie JD, Riley C, McFarland B, Hayes M, Boehnlein J, Leung P, Adams G (2008) High
507 prevalence rates of diabetes and hypertension among refugee psychiatric patients. *J Nerv Ment*
508 *Dis* 196 (2):108-112. doi:10.1097/NMD.0b013e318162aa51

509 40. Population's level of education, after the survey on education. (2013) Statistics Norway.
510 <https://www.ssb.no/en/utdanning/> Accessed 5 March 2018
511
512

513
514
515

TABLES

Table 1 Clinical and demographic characteristics of participants, according to gender

	Men (n=74; 46.8%)		Women (n=84; 53.2%)		Total (n=158)
	<i>Mean (SD)</i>		<i>Mean (SD)</i>		<i>Mean (SD)</i>
Age (years)	42.3 (11.3)		38.4 (10.5)		40.3 (11.1)
Years lived in Norway	13.6 (7.5)		12.1 (6.1)		12.8 (6.8)
Years of education	11.9 (4.0)		8.0 (4.9)		9.9 (4.9)
Marital status		<i>N (%)</i>	<i>N (%)</i>		<i>N (%)</i>
	<i>Married</i>	56 (75.7)	51 (60.7)		107 (67.7)
	<i>Single, separated or divorced</i>	18(24.3)	33 (39.3)		51 (32.3)
Systolic BP (mmHg)	129.9 (16.9)		118.3 (18.0)		123.8 (18.3)
Diastolic BP (mm Hg)	83.3 (10.0)		78.7 (8.6)		80.9 (9.6)

^a*p* represents asymptotic significance516
517
518
519
520**Table 2** Urine analysis laboratory data of participants who returned complete 24-hour urine samples, according to gender

	Men (n=75)		Women (n=84)		Total (n=159)
	<i>Mean</i>		<i>Mean</i>		<i>Mean</i>
Urine volume (mL / 24 h)	2061 (775)		1780 (649)		1913 (722)
Creatinine (mmol/24 h)	15.8 (3.9)		10.3 (4.8)		12.9 (5.2)
Na (mmol/24 h)	150.6 (57.0)		126.6 (62.4)		137.9 (61.3)
K (mmol/24 h)	66.9 (25.5)		54.8 (19.5)		60.5 (23.3)
Na:K	2.5 (1.2)		2.4 (1.1)		2.4 (1.1)
NaCl (g/24 h)	8.80 (3.38)		7.39 (3.64)		8.05 (3.58)

521
522
523
524

Table 3 Multiple linear regression showing the relationship between daily sodium excretion (mmol/24h) and demographic factors ;univariate (crude) and adjusted estimates are shown

	Crude β estimate	95% CI		p	Adjusted β^a	95% CI		p
		Lower	Upper			Lower	Upper	
Years lived in Norway ^b	-0.03	-1.41	1.47	0.97	0.2	-1.30	1.64	0.82
Years of education ^c	2.6	0.6	4.5	0.01	0.1	-0.7	3.6	0.19

^a β coefficients for each demographic factor was adjusted for by inclusion of relevant confounders.

^b*Years lived in Norway* was adjusted by *gender, age, and years of education*.

^c*Years of education* was adjusted by *gender, age, and years lived in Norway*.

525
526
527
528
529
530
531
532
533

FIGURES

See separate file for figures.