

Sodium and Potassium in the Canadian Total Diet Study

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ABSTRACT

Sodium (Na) and potassium (K) are essential nutrients. We investigated Na and K content of foods collected from the 2007 Vancouver Total Diet Study (TDS). Our food nutrient content data showed that processed foods and soups had a high Na content while fluid milk, unprocessed meats and several fruits and vegetables had a high K content. The Na/K ratio indicated, with few exceptions, a segregation of the foods high in Na from ones high in K. Based on this data, new Na and K intakes were generated using Canadian Community Health Survey (CCHS2.2), cycle 2.2 Nutrition dietary recall. In general, the results showed a lower Na intake than indicated by CCHS2.2, but still higher than the Tolerable Upper Level of Intake (UL). K intakes were similar to CCHS2.2 data showing an intake lower than the AI.

Word count=136

RESUME

Le sodium (Na) ainsi que le potassium (K) sont des nutriments essentiels. Nous avons examiné le contenu de Na et de K d'aliments collectés pour l'Étude Canadienne sur l'alimentation totale de Vancouver conduite en 2007. Notre base de données sur le contenu nutritionnel des aliments a démontré que les aliments traités et les soupes ont un haut niveau de Na alors que le lait, les viandes non-traitées ainsi que plusieurs fruits et légumes détiennent un niveau élevé de K. Basés sur ces données, de nouvelles prises ont été générées en utilisant l'Enquête sur la santé dans les collectivités canadiennes (ESCC 2.2), cycle 2.2, Nutrition. En général, les résultats ont démontré un niveau d'ingestion de Na moins que CCHS mais plus élevé que l'apport maximum tolérable (AMT). Les ingestions de K étaient similaires aux données contenues dans l'ESCC 2.2, démontrant une ingestion inférieure à l'apport suffisant (AS).

Word count =142

CONTRIBUTION OF CO-AUTHORS TO MANUSCRIPTS

The work presented in this thesis took place in the Nutrition Research Division of the Bureau of Nutritional Sciences and in the Biostatistics and Computer Application Division at Health Canada. The design of the experimental work and the thesis structure is the result of numerous meetings between the candidate and her advisory committee (Dr. Cockell, Dr. Koski, Dr. Cooper).

The candidate performed the entire experimental analyses described in the thesis. It was the candidate's responsibility to select and collect the relevant scientific articles, to prepare tables and graphs and to write the text.

Dr. Cockell, as my in situ supervisor, provided his knowledge and expertise in nutrition. He provided the initial core design of the lab experiment and thesis protocol. Along the way, he provided me with guidance on the lab work and on the thesis writing. The numerous and successive steps of thesis writings were under his committed editorial input and written commentaries. Of major importance was his contribution on presenting within the paper 1 and 2 of the data from laboratory findings. In his lab, I worked with a remarkable technician, Philip Griffin, who is of a gentle and continuous support.

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*“Whoever travels without a guide
needs two hundred years for a two-day journey”
Rumi*

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PART 1 - INTRODUCTION

Section A Rationale and Statement of Purpose

Hypertension is a medical condition in which the prevention and control seem to be highly influenced by the Na, K and Na/K ratio in the diet. The high worldwide prevalence of hypertension (25% in 2000) will likely increase in the coming years (predicted to reach 29% by 2025), such that the global burden of this disease on society is considerable (Kearney et al 2005). In Ontario, Canada, the relative increase in hypertension prevalence was about 60% from 1995-2005 and the incidence increased by 25.7% from 1997-2004 (Tu et al 2008). The burden of hypertension is related to its direct impairment on lifestyle and its indirect medical consequences (occurrence of other associated diseases such as stroke, CVD, kidney, bone disease and/or diseases as a result of the adverse effects of antihypertensive drugs) (du Cailar and Mimran 2007, Titze and Ritz 2009). Additionally, hypertension is associated with economic difficulties for its sufferers (e.g. drug costs, impaired health, and consequently reduced work capacity) (Joffres et al 2007). In addition to causing hypertension, imbalanced Na and K intake may be related to obesity and diabetes (He et al 2008).

This background of scientific data regarding the medical and economic implications of hypertension has compelled many governments and health related organisations to initiate strategies aimed at the reduction of hypertension prevalence and incidence in the population, while promoting lifestyle changes rather than anti-hypertension drug therapy. The pharmacologic approach to lower blood pressure was shown not only to be accompanied by adverse health effects in people with hypertension (Chalmers and Arima 2009), but it was noticed that the individual blood pressure lowering effect translates at the population level to only 1 mm Hg. Although in many countries hypertensive drugs are reimbursed through medical coverage, the effect of this economic governmental effort is not supported by substantial population effects (Karppanen and Mervaala 2006).

As the pioneer-country of the salt reduction programs in the late 1970s, Finland initiated a firm and sustained campaign that recommended to decrease the intake of salt to one half of the prevailing levels (He and MacGregor 2009). This campaign contributed to a reduction of 10 mmHg in blood pressure and to a decrease of 75–80% in both stroke and CHD mortality, even as obesity and alcohol consumption rates increased (Karppanen and Mervaala 2006).

Similar initiatives have been adopted by other health-related organisations which advocate for the necessity of salt reduction. In 1996, the UK action group, Consensus Action on Salt and Health (CASH), lobbied for a reduction in salt intake to less than 6g per day and persuaded several food companies to reduce by 10-15% the amount of salt added to foods (MacGregor and Sever 1996). Interestingly, this reduction in salt in the food supply has not been noticed by consumers (Girgis et al 2003). Some national health authorities have all set the same goal of reducing salt intake to 6 g/day (The Australian Division of World Action on Salt and Health 2010, Food Safety Authority of Ireland 2009, Health Council of the Netherlands 2007). In 2006, the American Medical Association proposed to the Food and Drug Administration several strategies to reduce salt intake including the revocation of the “generally recognised as safe” (GRAS) status of salt. These strategies also included a reduction of 50% in salt intake in the next decade, improved labelling legislation, and participation in consumer education (Appel 2006).

Since 2008, the lowering of the sodium content of Canadian foods has become a main goal of a multi-stakeholder Working Group under the leadership of Health Canada. The current strategy of the Working Group includes a three-pronged approach (population education, food industry voluntary reduction of sodium levels, and research). Information sought by the Sodium Working Group includes: 1) an assessment of the effectiveness in reducing the salt content in foods; 2) a knowledge of the baseline current salt content of foods consumed by Canadians, based on population food choices presented by the Canadian Community Health Survey (CCHS 2.2 – Nutrition); and 3) a hierarchy of the main food contributors to salt intakes in Canadians and a reduction of the salt content of these top contributors (Health Canada 2010b). Additionally, an across Canada monitoring of the sodium content in foods could be a feasible enterprise that could precede large population studies focused on identifying hypertension and/or Na urinary

excretion trends. A study of foods that have already been collected for analyses of contaminants and other chemicals (as is the case for Total Diet Study) could provide scientific background for identifying a possible correlation between hypertension and the sodium content of foods.

However, in order to implement policy or regulation regarding salt intake reduction, the sodium content of food and the sodium intake in the population must be known. The sodium content in foods is monitored in many countries including the United States, Italy, New Zealand, France and Canada through a special program in which researchers collect market food samples at certain time intervals (US Food and Drug Administration 2007, Carnovale et al 2000, Food Standards Australia New Zealand 2009, Leblanc et al. 2005, Health Canada 2007). For example, through its Total Diet Study, the US has already accumulated 40 years experience in analysing the contaminants and nutrient content in market foods (Pennington, 2000).

In a population, anthropometric and nutrition data are collected from time to time to assess population health and well being. For many years the only national nutrition data was the Nutrition Canada survey conducted from 1970-1972 (Statistics Canada 1975). More recently (1990-1999), the provincial nutrition surveys collected information about the food intakes of Canadians which were used to carry out health risk assessments, to review existing nutrition programs and policies (Statistics Canada 1981). In recognition of a critical need for more extensive and recent information about the nutrition of Canadians, CCHS 2.2 – Nutrition was conducted in 2004. The primary goal of the Nutrition Survey was to provide reliable, timely information about dietary intakes, nutritional well-being, and their key determinants. One of its objectives was to estimate the distribution of usual dietary intake in terms of foods, food groups, dietary supplements, nutrients (including Na and K) and eating patterns for a representative sample of Canadians at provincial and national levels. Targeting individuals aged 0+, living in private occupied dwellings in each of the 10 provinces, but excluding the 3 territories; it covered 98% of Canadians. It had two major components: “a 24 hr recall” and “general health information” questionnaire (Statistics Canada 2004).

The Canadian Nutrient File (CNF) is Canada’s standard reference food composition database reporting the amount of nutrients found in the most commonly used

Canadian foods. The most recent (eleventh) edition is bilingual, follows the metric system, and contains data on 5516 food items. The CNF includes food composition data from the United States Department of Agriculture (USDA), scientific literature, Canadian government labs, industry documentation, universities and research laboratories (Health Canada 2010a). Even though quite homogenous with respect to analytical methods used for nutrient composition determination, CNF data could sometimes be considered not representative, not updated, and not specific when applied to Canadian foods (Health Canada 2010a). Some of those limitations include: 1) the non-representativeness of some data due to the major reliance on values from the USDA; 2) some analytical values have been generated long ago; 3) the minor reliance on data generated from analyses of Canadian foods. Some other CNF data limitations are related to different issues such as: 1) inclusion of some products that do not have a full nutritional profile listed (eg., kefir); 2) incomplete nutrient data sets for some food items; or 3) analytical values that do not represent the nutrient amounts available to the body. Even though there is similarity between the two nations' diets, it must be acknowledged that important differences do exist between American and Canadian foods and those should be taken into account when developing a national database. Some of the markedly American vs. Canadian differences are related to the heterogeneity of raw material (including differences due to soil mineral composition, plants and animal breeding and feeding systems), fortification practices, and food processing techniques (including the recipes) (Health Canada 2010a). These can all have important effects on the assessment of nutrient intakes in the population.

Utilising data that sometimes may be decades old, the CNF is not necessarily illustrating the current nutrient composition of foods available on the market. An example is offered by the Na content in white bread. In 2000 a value of 680mg Na/100g was included in the CNF. This Na content measurement is contradicted by the industry label survey of top brands in Canada done in 2008 that showed a content of only 433mg Na/100g for bread (J.Deeks, personal communication). The CNF also includes nutrient information about generic foods not brand specific products. Packaged foods are major sources of sodium and it would be helpful to have a more accurate and detailed description of Na content of those foods which could support the scientific interests of researchers and the evaluators using the CNF (Health Canada 2010a). As a result of some

of the current limitations associated with the CNF, including outdated nutrient data for foods, nutrient intake estimates from CCHS 2.2-Nutrition may be either under or overestimated. Some of these acknowledged limitations can be at least be partially addressed using data generated from the Canadian Total Diet Study, a study focused on Canadian foods.

Total Diet Studies (TDS, known in some regions as Market Basket Surveys) are promoted by the World Health Organization as an efficient means of monitoring for contaminants in the food supply. Although there are acknowledged limitations due to food selection and compositing of food samples prior to analysis, the TDS has been used for many years in the United States to track changes in food composition, including mineral nutrient levels, over time (Pennington 2000). TDS have the advantage that foods being analysed have been prepared as if for household consumption, rather than being analysed raw or as purchased (Leblanc et al. 2005). Total Diet Studies have been conducted in Canada since 1969, although the pattern of sampling and compositing of the food samples purchased has changed over time (Conacher et al. 1989). TDS samples from 1974-75, collected from Halifax, Montreal, Winnipeg and Vancouver, had been composited into 10 food groups and assayed for Na and K, showing 2- to 5-fold higher than recommended intakes for Na (depending on age/sex group), but sufficient K intakes (Shah et al. 1982). Current activity in the Canadian TDS involves collecting samples in one city per year, such that five regions across Canada (British Columbia, Prairie provinces, Ontario, Quebec, Atlantic provinces) are represented in a five-year cycle (Health Canada 2007).

The representative nature of the TDS collection (designed to represent the majority of the foods commonly purchased in Canada) could also serve some utility in monitoring selected nutrients in the food supply. This data could provide representative, updated, and specific values of Na and K in Canadian foods. This data could then be utilized along with national surveillance data to model intake distributions.

Section B Project overall objectives

- 1) To analyze Na and K content of food composite samples from a recent collection (2007) of the Canadian Total Diet Study (TDS).
- 2) To use the obtained results to model intake distributions by age and sex group for the Canadian population.

PART II - REVIEW OF THE LITERATURE

Section A Na and K as essential nutrients

1. Na as an essential nutrient

In the extracellular fluid, Na as the main cation and Cl as the main osmotically active anion play key roles in maintaining extracellular fluid volume and plasma osmolarity and in maintaining fluid and electrolyte balance. Na is required for the process of active transport of molecules across cell membranes and is also involved in electrical signalling for communication within brain, nervous system, and muscles (Institute of Medicine 2005a). Almost all (98%) of ingested sodium is absorbed in the small intestine, circulated through the blood, and filtered through Bowman's capsule in the kidney. After passing into the proximal tubule the bulk of the Na is reabsorbed through an active transport mechanism controlled by angiotensin II. Reabsorption continues in the ascending segment of the loop of Henle while the fine adjustment of reabsorption required to maintain Na balance, (and hence water content and blood pressure), takes place in the distal tubule and the collecting ducts and is closely regulated mainly by the action of the hormone aldosterone (Feraille and Doucet 2001). All of these physiological processes lead to a Na concentration in extracellular fluid of 137-147 mmol/L with disnatremias when Na concentration is below or above the regulated range (Whitmire 2008).

Due to insufficient data from dose response trials, it was not possible to determine an Estimate Average Requirement (EAR) for Na (Institute of Medicine 2005a). Instead, an Adequate Intake, defined as the recommended average daily nutrient intake level or observed mean nutrient intake by a group (or groups) of apparently healthy people that are assumed to be adequate, was set at 1.5 g (65 mmol) per day (Institute of Medicine 2005a). Three factors were considered to establish this threshold: 1) meeting the requirements for other dietary nutrients; 2) overcoming a low Na level ($< 0.7\text{g}$ [30mmol]) associated with some metabolically adverse effects; and 3) allowing for some Na losses due to sweating in un-acclimatized people exposed to warm temperatures or moderately active people. The adverse effects of high salt intake were used in setting the tolerable

upper intake level (UL). Defined as the highest average daily nutrient intake level that is likely to pose no risk of adverse health effects to almost all individuals in the general population, the UL for Na was set at 2.3g (100 mmol) Na/day. The UL was established based on blood pressure elevations as documented in dose-response trials (Institute of Medicine 2005a).

2. K as an essential nutrient

In the intracellular fluid, K is the main cation and is required for normal cellular function, for neural transmission, muscle contraction, and vascular tone. At the same time, K is recognised as the main reducer of NaCl-induced blood pressure (Institute of Medicine 2005b). The vasodilatory role of K was thought to be explained by the endothelium-dependent relaxations and by hyperpolarization of the vascular smooth muscle cell induced by K (Haddy et al 2006). However, many studies have shown that it is not the amount of K that is the determining factor for reducing blood pressure, but the cumulative effect of both Na and K intake (Geleijnse et al 2003). Another notable function of K, which actually is related to its conjugate anions, is the equilibration of acid-base balance by neutralizing the excess acid load that is a frequent result of a Western diet. By doing so, K and its anions diminish bone Ca reabsorption, reduce bone turn-over rate and bone demineralization and therefore reduce the risk of kidney stones (Morris Jr et al 2006).

The concentration of K in intracellular media (145 mmol/L) and extracellular fluid (3.8 to 5mmol/L) are the result of a constant equilibrium between K absorption and excretion (Institute of Medicine 2005b). Dietary K is well absorbed from food (85%), circulated by blood (as plasma K) and, through the means of insulin stimulated Na/K – ATPase pump, it contributes to the intracellular electrolyte pool. At the kidney, K is filtered and about 80% is reabsorbed within the proximal tubule, allowing for a fine tuning at the ascending limb of Henle's loop, the distal convoluted tubule and the cortical collecting duct; the remaining K is excreted under aldosterone control in urine (Landau 2004).

As with Na, no EAR was established for K due to insufficient data from dose-response trials. Taking into consideration factors such as maintaining low blood pressure,

reducing bone turnover and kidney stone formation, the AI for K was set at 4.7g (120 mmol)K/day for all adults. Due to a generally low intake in the population and also due to the fact that K is generally easily excreted if in excess, no UL has been established for K (Institute of Medicine 2005b).

3. Measurement of Na and K intakes

In order to assess the effects of Na and K on individual and population health, the intake of those nutrients must be known. There are two general methods for estimating sodium intake in individuals and populations: a) dietary surveys and b) measurement of urinary sodium excretion (Bentley 2006).

Many studies have used dietary surveys for estimating the sodium intake in the studied population. Dietary surveys (including food diaries, food frequency questionnaires, 24hr dietary recalls) represent a largely used method for estimating nutrient/sodium intakes in populations but they are vulnerable to some errors such as: 1) interviewee subjectivity in estimating of the quantity of food consumed as well as the quantity of salt added at the table; 2) inadequate knowledge of the salt content of ready to eat foods. Overall, these errors may lead to underestimation of sodium intake by using these methodologies (Bentley 2006). These limitations are highlighted when the data presented by these studies are not congruent with those generated by trials that use 24hr urinary sodium excretion as a method for estimating sodium intake (Liu and Stamler 1984).

Na intake can also be assessed based on overnight or double/multiple 24hr urinary sodium excretion. Overnight urine collection must be corrected to an 8-h base for estimating a single day's Na intake. Twenty-four hr urine collection captures sodium excretion around the clock, therefore taking into consideration individual diurnal variation in sodium excretion. This can provide a better estimate of Na intake. Even if putting a higher burden on participants, this last method is considered the preferred method for assessing Na intake in a population (Bentley 2006, Brown et al 2009).

With regards to classifying the accuracy of different methods for assessing individual average K intake, the method of analyses of food duplicates (the best method) seems to be associated with minimal limitations (Clark and Mossholder 1986). However,

as the high intra-individual variation noticed in assessing Na intake is also applicable to K intake estimation, some trials have been performed in order to identify the better estimator of K intake. By collecting eight 24h urine samples per subject Bingham and coworkers obtained a higher correlation ($r = 0.74-0.82$) (Bingham et al 1997) than the correlation ($r = 0.58$) between K intake estimated as food records and two to six 24h urine collection (McKeown et al 2001). The most frequently used method tends to be repeated 24hr urine collection (Siani et al 1989). This method, significantly related to blood pressure and heart rates is currently under assessment for use in detection of the quality of the diet and monitoring the effectiveness of dietary interventions (Mente et al 2009),

4. Na and K – physiological and patho-physiological interaction

Na and K as essential nutrients are interrelated. They share and mutually regulate each other's major physiological mechanisms (trans-membrane transport, renal absorption, blood pressure regulation). They also share clinical symptomatology (hypertension, cardiovascular, kidney and bone disease) (He and Whelton 1999, He and MacGregor 2008). The Na –K interaction at the cellular membrane level is manifested through their trans-membrane transport performed by Na-K ATPase (Na-K pump). This pump undergoes a successive phosphorylation- dephosphorylation in order to exchange 3 Na^+ and 2 K^+ across the cellular membrane (Skou 1992).

In general, high Na and low K intake levels are reflected by high Na and low K serum concentration levels that inhibit the Na-K pump and block K channels (Adroque and Madias 2007). Thereafter, Na begins to accumulate intracellularly. This in turn attracts Ca which is involved in actin-myosin interaction and decreases nitric oxide synthesis, while K remains at low concentrations; the result is a vascular endothelium depolarization and contraction that is transmitted to the smooth muscle. Contraction of smooth muscle around the blood vessel shrinks its lumen leading to increased blood pressure or hypertension. Increasing the K intake level will result in an increased serum K concentration that will stimulate the sodium pump, open K channels, and lead to K export from the endothelial cell into the myoendothelial space where, in addition to

hyperpolarisation and reduced Ca concentration, K will facilitate the blood vessel dilatation (Adroque and Madias 2007).

Our understanding of the magnitude of the Na/K interplay is expanded by including into discussion the K accompanying anion, largely as citrate or chloride in foods, or bicarbonate in supplements. This leads us to the second role of Na and K as acid/base metabolic regulators. As the modern Western diet is abundant in foods of animal origin and cereals (i.e. high in acid (H^+)) and low in fruits and legumes (i.e. acid buffering (HCO_3^-) and K^+ generating foods), a low grade metabolic acidosis may be produced. To buffer this acidity, metabolically the body might use calcium containing carbonates from bones which potentially could lead to osteoporosis, hypercalciuria and, combined with a relative lack of urinary citrate, to kidney stone formation.

K supplements, as KCl and K bicarbonate, have been suggested to increase urinary sodium excretion. This natriuretic effect helps to explain K antihypertensive role and also the reduced need for antihypertensive medication as a consequence of K supplementation (Haddy et al 2006, Van Buren et al 1992).

Section B Hypertension

1. Definition

During ventricular systole (contraction) a certain amount of blood (cardiac output) is ejected from the ventricular chambers through the aorta into the systemic arterial circulation. This circulation presents a certain degree of resistance (systemic vascular resistance) to the flow of blood. The result of those two factors (cardiac output and systemic vascular resistance) is a certain pressure exerted on the artery walls, called systolic blood pressure (SBP). During ventricular diastole (relaxation), when the ventricles are refilling with blood from the left auricle, the arterial pressure decreases to the lowest level. This new level is called diastolic blood pressure (DBP). In healthy individuals the normal values of SBP and DBP are around 120/80 mmHg (Chobanian et al 2003).

Elevation of the SBP/ DBP readings over 140/90 and 130/80 mmHg is considered an indicator of hypertension in most people and those with diabetes or chronic kidney disease, respectively. Elevated DBP was historically considered the principal indicator of hypertension. More recently the definition has evolved to suit the complexity of the disease and both SBP and DBP are considered important in assessing hypertension. The American Society of Hypertension Writing Group has provided within their Seventh Report of the Joint National Committee (JNC 7) on Prevention, Detection, Evaluation, and Treatment of high Blood Pressure a new classification system for defining hypertension (Table 1) (Chobanian et al 2003).

Canadian guidelines specify that a blood pressure reading of SBP 130-139 mmHg and/or DBP 85-89 mmHg is consistent with a high normal blood pressure. A reading of SBP 140 mmHg and DBP 90 mmHg requires a future visit for hypertension assessment that depends on the presence or absence of target organ damage and associated cardiovascular risk factors (Padwal et al 2009).

Table 1: Reference ranges for classification of blood pressure and hypertension

SBP/DPB (mmHg)	JNC 7 category
<120/80	Normal
120-129/80-84	Prehypertension
130-139/85-89	Prehypertension
>140/90	Hypertension
140-159/90-99	Hypertension Stage 1
160-179/100-109	Hypertension Stage 2
>180/110	Hypertension Stage 2

(adapted from the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of high Blood Pressure, Chobanian et al. 2003)

2. Hypertension and cardiovascular disease prevalence

The present pandemic of CVD and its major contribution to fatalities has propelled concerned health organisations and scientific media into developing worldwide programs regarding its causes, prevention and treatment (Food and Agriculture Organisation 2010). It has been widely recognized that even if hypercholesterolemia and obesity are very important causal factors, hypertension, with a prevalence of 128 million cases, remains a major contributor to worldwide CVD (Kaplan and Opie 2006). With 26.4% global prevalence in 2000 and an anticipated increase to 29% by 2025, hypertension is the focus of an impressive effort of research, regulatory and educational programs aimed at identifying both its aetiology and appropriate management strategies (Kearney et al 2005). In Canada, the high blood pressure prevalence increased from 11.6% to 14.4 % between 1994 and 2000 (Heart and Stroke Foundation of Canada 2003); according to the 2004 CCHS–Nutrition, the prevalence of hypertension is about 7.2 % in 35-44 year-olds, 17.2% in 45-54 year olds, 32.7% in 55-64 year olds, 46.5% in 65-74 year-olds, 56.6% in 75-84 year-olds, and 54.2 in those 85+ (Public Health Agency of Canada 2009). An investigation of a cohort of adults aged 20 years and older from Ontario showed an increase in hypertension prevalence and hypertension incidence by 60% and 25.7%, respectively from 1995-2005 (Tu et al 2008). The cost of CVD on the Canadian economy, including direct and indirect costs, is estimated to be \$18,472.9 million (11.6% of all illnesses) (Heart and Stroke Foundation of Canada 2003). In 2004, CVD was responsible for 72,743 Canadian deaths (32% of all deaths) (Public Health Agency of Canada 2009).

3. Factors involved in the onset of hypertension

Influenced by a multiplicity of genetic and lifestyle factors, hypertension is truly a multifactorial disease. Contributing factors include those over which people have almost no control (e.g. genetics), factors in which some control may be possible (e.g. different medical conditions including kidney disease leading to hypertension), and factors which should be possible to almost completely control (e.g. dietary factors such as Na and K intakes) (National Heart Lung and Blood Institute 2010).

Genetic factors in hypertension

Even if mapping of the genes involved in hypertension is still in its infancy, genetic studies have already helped in understanding the pathogenesis of hypertension. Studies done on mutated genes have shown that net renal salt balance is a very important pathway in altering blood pressure (Lifton et al 2001). However, genetic involvement in hypertension, aside from being complex and not fully understood, is not a readily modified factor by direct human manipulation. Since phenotypical expression of genes is influenced by environmental factors that are more readily modifiable, these will be discussed in further detail below.

Kidney and hypertension

The key role of the kidney in the regulation of blood pressure was demonstrated in renal cross-transplantation experiments that were performed in hereditary strains of hypertensive rats (Greene et al 1990, Rettig and Unger 1991). From the multitude of systems that are involved in blood pressure regulation, the kidney-fluid volume system is the one with the greatest impact on long term blood pressure regulation (Guyton 1991). In healthy individuals a high salt intake leads, as a consequence of increased osmolarity, to a sudden increase in blood volume and a temporary hypertension. This forces the kidney to increase pressure natriuresis and diuresis, leading to a reduced blood circulatory volume and consequently to a fall in arterial pressure. Pressure natriuresis is a mechanism to eliminate the excess of circulating Na by increasing the blood pressure and forcing Na glomerular filtration and excretion followed by a normalisation of blood pressure. All

those steps take place under the conditions of a healthy normal functioning kidney. An impaired kidney unable to excrete excess plasma Na will contribute to hypertension onset (Mayer 2008).

Until now, few mechanisms have fully described the role that the kidney plays in hypertension aetiology, but increased pre-glomerular vascular resistance seems to be relevant for hypertension onset. This increased vascular resistance is triggered by the association of angiotensin II and dietary sodium. It is known that the angiotensin II can induce pathophysiological arterial changes. Experiments performed on rats showed that, when associated with a 2% NaCl diet, the angiotensin II effect led to pathophysiological structural vascular changes (Simon et al 2003). Those vascular changes open the endothelium, reduce nitric oxide synthesis (and diminish its protective role), therefore leading to the vascular lesions frequently noted in hypertension. For example, if such a vascular damage appears in afferent glomerular arteriole, local endothelial sensitivity to sodium is altered towards increased Na absorption, by contributing to hypertension onset (Johnson et al 2002).

Lifestyle/dietary factors involved in hypertension onset

Particularly in hypertension, the term “environment” is used to encompass all non-genetic factors that contribute to hypertension onset. Certain environmental factors provide the best means by which an individual can have control over this condition. These include: alcohol intake, obesity, lack of physical activity, stress and dietary factors including sodium and potassium (Perry et al 1994).

In various combinations, lifestyle factors including the major dietary factors (Na, K, Ca, Mg, dietary fibre), have been investigated. Pooling data from studies done in 4 European countries plus the USA (Geleijnse et al 2005), yielded population attributable risk percentages (PAR %) for different factors contributing to hypertension. The top three factors were overweight (11-17%), excessive sodium intake (9-17%), and low potassium intake (4-17%), followed by physical inactivity (5-13%), and low intake of fish oil (3-16%). Other factors including low calcium and low magnesium intake, excessive coffee consumption, and excessive alcohol intake scored lower ((2-8%), (4-8%), (1-9%) and (2-3%)), respectively. Dietary patterns have emerged to be one of the most influential

factors in blood pressure onset. For the purpose of this thesis, while acknowledging the contribution of other factors, the discussion will be centered on sodium and potassium as dietary factors in hypertension onset. They have been proven as main contributors to hypertension's aetiology, and if consumed at the recommended levels and ratio, they are able to considerably lessen hypertension. There is consistent evidence that Na and K show the greatest departure from optimal consumption levels in common North American diets (Institute of Medicine 2005a, Institute of Medicine 2005b).

4. Dietary Na and K in hypertension onset

Human beings' genetic program which has been unchanged for almost 100 000 years is conditioned to handle unprocessed plant and animal foods in a proportion of 2/3 to 1/3, respectively. These proportions will provide 0.6g Na, with a maximum of 0.8 g/d if only animal food is consumed (Karppanen and Mervaala 2006). If only unprocessed natural foods are consumed it is thought that it would be impossible to get over 1.2 g Na per day (50 mmol/day). The minimum sodium requirement based on a physiological need of 0.5 g/day was adjusted, for several practical reasons, in the IOM recommendations to 1.5 g (65mmol)/day, but the current Na intake in the North American population ranges from 3.1 to 4.7 g (135 to 204 mmol)/day for men and 2.3 to 3.1 g (100 to 135mmol)/day for women, respectively (Institute of Medicine 2005a). A recent study utilizing the CCHS 2.2-nutrition survey data showed that in the Canadian population the average daily sodium intake was about 3.5 mg/ daily for men and 2.8 mg/ day for women, respectively (Fischer et al 2009).

The hypertension-inducing effects of Na have been shown in a wide range of epidemiological and intervention studies. The modern diet can be considered to be high in Na, while low in K. This low K intake is a result of both an increment in consumption of processed foods that are depleted of their natural content in K (Suter et al 2002) and an increase in salt intake, with a continuously low intake of fruits and vegetables intakes (Casagrande et al 2007). The excess in Na and deficit in K result in a high Na/ K ratio that has been correlated with a high incidence of hypertension (Suter et al 2002).

Acknowledging the vastness of the research in this area and the multitude of studies on the sodium and/or salt effect on blood pressure, only the results from meta-

analyses of major studies, intervention and epidemiological studies will be presented here.

a. High Na intake and blood pressure

Meta-analyses

The effect of chronic high salt intake on blood pressure among healthy normotensive and essential hypertensive elderly patients has been investigated in a meta-analysis of 11 randomized control trials (Alam and Johnson 1999). Five of these studies included patients ≥ 60 years of age only and six included patients with a mean age close to 60 years. When all trials were pooled, a chronic high NaCl diet significantly increased mean SBP and DBP by 5.58 mmHg (95% CI 4.31-6.85) and 3.5 mmHg (95% CI 2.62-4.38) respectively. There was a significant association between the level of salt intake and SBP ($P = 0.05$, $r^2 = 0.37$), but not DBP ($P = 0.76$, $r^2 = 0.01$). These data suggested that a chronic high salt diet in elderly patients with essential hypertension is associated with an increase in SBP and DBP, the association is significant for both SBP and DBP but more marked for SBP than DBP, the effect is more pronounced the older the patient and salt dose strongly predicts SBP in older patients.

Intervention studies

Due to ethical considerations the number of intervention studies is limited. The existing studies mainly involve a few participants, last for few days up to 4-6 weeks, and do not include high sodium loads.

In the area of the Na effect on blood pressure, dose response trials with various results have been conducted. For example, in normotensive healthy white subjects consuming increasing salt diets a consistent rise in blood pressure has not been detected although adaptative hormonal changes have been observed (Bruun et al 1990, Kirkendall et al 1976, Roos et al 1985). In normotensive healthy blacks (Luft et al 1979), in families with hypertension history/ risk (Fuchs et al 1987), in borderline hypertensives (Sullivan et al 1980) and in older subjects (Johnson et al 2001), a continuous increase in blood pressure has been shown as a response to an increase in Na load.

The correlation between sodium intake in infancy and blood pressure later in life has been examined. Geleijnse et al. exposed 2 groups of babies to a low (0.89 ± 0.26

mol) or normal salty diet (2.50 ± 0.95 mol) for 6 month and recorded a difference of 2.1 mmHg in blood pressure in favour of the low salt intake group. After 14.5 years of normal salt intake, the authors noticed that the difference between the groups' blood pressure persisted, being 2.2mmHg at follow-up (Geleijnse et al 1997).

Epidemiological studies

Data recorded in various populations have shown a correlation between salt intake and blood pressure. In Venezuela (Mancilha-Carvalho et al 1989), and Brazil (Oliver et al 1975) studies conducted with indigenous peoples showed that the mean salt intake of < 0.5 g/day was correlated with low blood pressure even in the elderly. In contrast, studies performed in Iran (Page et al 1981) or Northern Kashmir (Mir and Newcombe 1988) where subjects' mean salt intake varied between 4.4-20.5 g/day, the systolic and diastolic blood pressure were high and positively correlated with salt intake.

The sodium-blood pressure relation has been shown in studies that compared differences in sodium intake and blood pressure as it relates to different geographical areas occupied by members of the same population. Populations living close to water (eg costal and river communities of Newfoundland and the Solomon Islands) (Page et al 1974) had a sodium intake of 8 and 15 g/day respectively and had higher blood pressure than people living in communities in the center of the island. The influence of geographical location (proximity to the urban area where exposure to high Na diets is greater) was recorded in Kuna people in Central America and compared those living on islands, in the suburbs or in Panama City. For these people, the blood pressure increase varied from almost none to intermediary to 10 % of the population in all age categories (Hollenberg et al 1997).

Migratory studies emphasize the influence of salt on blood pressure by showing that if a group of individuals leave areas typified by low salt consumption (e.g. Kenya and China) and are exposed to higher Na intakes, their blood pressure rises accordingly (He et al 1991, Poulter and Khaw Hopwood 1984).

Major observational studies done by Dahl (1969) and Glibermann (1973) showed that at sodium intakes between 3 and 15 g/day there is a positive correlation between the 2 factors. However, Dahl's and Gliberman's studies have been criticised for the non-

uniformity in blood pressure measurements, urine sample collection, and participants' characteristics, as well as for not including several possible confounders (Dahl 1969, Gleibermann 1973).

The INTERSALT study (1982-1985) was designed to overcome the weaknesses of the previous studies. It was an international, cross sectional study that investigated the relation between 24 h salt excretion and SBP at the individual and inter-population levels. It enrolled 10,079 subjects from 32 countries. INTERSALT addressed the non-uniformity of previous studies by employing standard methods in blood pressure measurements and in urine collection while including many possible confounders. This study confirmed in individuals a positive correlation between 24-h sodium excretion and SBP, and with both SBP and DBP at the inter-population level. INTERSALT proposed a reduction by 100 mmol/day in sodium intake as a factor that would facilitate reduction in SBP, risk of coronary death, risk of stroke death at middle age (Stamler et al 1989) (see table 2).

In contrast to the findings of other epidemiological studies, two studies in Italy (Timio et al 1997) and Panama (Hollenberg et al 1997) identified that individuals with a salt intake at about 8 g/day didn't show the expected rise in blood pressure. The findings of the Italian study have not been confirmed (Timio et al 1997) and the data offered by the Panama study are thought to be a real opportunity for the investigation of genetic mechanism involved in salt sensitivity (Hollenberg et al 1997).

Overall, the results of meta-analyses and epidemiological studies support the hypothesis of Na as an important contributor to the aetiology of HT. The results from intervention studies are inconsistent.

Table 2: Comparison of 3 different lifestyle interventions on 3 endpoints in the INTERSALT study

Interventions	Effects		
	SBP in population	Risk of coronary death	Risk of stroke death at middle age
Reduction by 100 mmol in sodium intake (at the end of the study)	↓ by 2.2mmHg	↓ 4%	↓ 6%
Multiple dietary factors changes (reduced Na intake, increased k intake, reduced alcohol intake, less obesity)	↓ by 5mmHg	↓ 9%	↓ 14%
Reduction by 100 mmol in sodium intake (over life span)	↑* by only 9mmHg	↓ 16%	↓ 23%

*the authors estimated that in subjects that maintain a reduced sodium intake over their life span the increase in SBP will increase less (only by 9mmHg) in comparison with the increase in SBP in subjects who maintain the salt intake noted at the time of this study.

b. Reduced K intake on blood pressure

Intervention studies

The literature is scarce with respect to studies investigating the effects of dietary K depletion on hypertension. Some clinical studies have shown that in both normo- and hypertensive people consuming a normal salt diet, dietary K depletion elevates blood pressure (Krishna 1994). In healthy, normotensive, K depleted men whose salt intake was increased, a rise in blood pressure was observed; this was not observed in subjects ingesting normal amounts of K (Krishna 1990). However, Krishna (1994) noticed that in hypertensives with a low sodium diet, the hypertensive effect of K depletion or the hypotensive effect of K supplementation was not manifested.

Epidemiological studies

Numerous observational studies such as the NHANES II (Hajjar et al 2001), the Scottish Heart Health Study (Tunstall-Pedoe 1999), the CARDIA study (Liu et al 1996), and the INTERSALT study (Rose et al 1988) investigated the relationship between K intake or K urinary excretion and blood pressure. These studies have suggested that there is an inverse correlation between K and blood pressure. A few other studies such as the Nurses Health Study (Witteman et al 1989) and Health Professional Follow-up Study (Ascherio et al 1992) suggested that there is no correlation between K intake and blood pressure. The INTERSALT study highlighted the co-participation of other food nutrients, and more specifically the Na: K ratio in hypertension development (Rose et al 1988). Intervention studies targeted towards the identification of dietary K as a direct contributor to hypertension are limited. The results within the different epidemiological studies presented are inconsistent; therefore the relationship between a low potassium intake and blood pressure onset is not clear. However, as outlined by the INTERSALT study (Stamler et al 1991) and supported by studies presented in a later section, a higher K intake is a main contributor to hypertension reduction. Additionally, the Na/ K intake balance may contribute to the reduction of hypertension.

5. Na and K in hypertension reduction

a. Reduced Na intake

The idea of reducing blood pressure by reducing sodium intake has been investigated in both normo- and hypertensives.

Meta-analyses

A number of meta-analyses of the effect of reduced Na intake on hypertension have been done. A published overview of randomized clinical trials testing the effects of reducing sodium intake was conducted and included thirty-two trials with outcome data for 2635 subjects. The effects on blood pressure of lowering sodium in hypertensive and normotensive subjects, respectively (each trial weighted according to sample size), were 4.8/2.5 mmHg and 1.9/1.1 mmHg (Cutler et al 1997). The authors mentioned that there was no evidence that sodium reduction as achieved in these trials presented any safety hazards. Contradicting those findings, two other meta-analyses (Midgley et al 1996, Graudal et al 1998) analysing 56 and 58 trials in hyper- and normotensives, respectively, affirmed that reduced Na intake could be considered as adjuvant therapy in older hypertensives, but would not be a suitable approach for the general population. Midgley et al. showed that a 100 mmol/d reduction in daily sodium excretion was associated with a reduction of 3.7/0.9 mmHg in hypertensives and of 1.0/0.1 mmHg in normotensives. Additionally, Graudal et al. showed that the effect of reduced sodium intake as measured by urinary sodium excretion (118 mmol/24 h) for hyper- and normotensives was 3.9/1.9 mmHg and 1.2/0.26 mmHg, respectively. Due to limitations expressed regarding the inclusion criteria used by Midgley et al. and Graudal et al. (trials of short duration and extreme variations in salt intakes studied), a Cochrane meta-analysis was conducted that included 17 trials in hypertensives (n = 734) and 11 trials in normotensives (n = 2220); all of the trials were at least 4 weeks long (He and MacGregor 2004). A reduction of 4.97/2.74 mmHg in hypertensives with a median reduction in 24-h urinary sodium excretion of 78 mmol (equivalent to 4.6 g/day of salt), and of 2.03/0.99 mmHg in normotensives after a reduction in 24-h urinary sodium excretion of 74 mmol (equivalent

to 4.4 g/day of salt) was determined. These were considered by the authors as powerful support for a long and moderate reduction of Na at levels that can be relevant for public health initiatives concerning blood pressure in both hyper and normotensives. Supporting the results from the previous study, the same investigators showed, in a later meta-analysis of randomized double-blind crossover studies, a SBP decrease of about 10 mmHg equivalent to a reduction of about 88 mmol/d sodium urinary excretion. The authors calculated a corresponding anticipated reduction of 33% in stroke and 25% in ischemic heart disease (He et al 2005b).

A Cochrane review of 6 month to 7 year long trials reported the effect of advice to reduce salt intake in 2326 free living normotensives along with 801 treated and 387 untreated hypertensives. The small reduction in urinary Na (by 35.5 mmol/24 hours) correlated with a small reduction in blood pressure, suggesting that implementing and sustaining over a long period of time a diet low in salt is a difficult undertaking in free living individuals, but if complied with, this strategy of reducing salt intake could help maintain control over blood pressure without need for drug therapy (Hooper et al 2003).

A contrary view has been presented by Alderman, Cohen and colleagues, based on interpretation of one 24-hour dietary recall data recorded within National Health and Nutrition Examination Survey (NHANES I and II) (Alderman et al 1995, Alderman et al 1998). These authors suggest that salt reduction has negative consequences (higher cardiovascular mortality) that are apparently associated with insulin resistance and an increased activity of sympathetic and the renin-angiotensin system. Other authors (Cook et al 1995, Meltzer et al 1996) have scientifically criticised Alderman et al's data interpretation. The main criticism is related to the method used in those studies for assessing salt intake (one 24-hour dietary recall) that is considered unreliable and was replaced in more rigorous studies by twenty-four hour urinary sodium excretion measurement.

Convinced by the results from the plethora of experimental, epidemiological and interventional studies available about the positive correlation between Na intake levels and blood pressure readings, and acknowledging the WHO statement that 2% of all strokes and 49% of coronary heart disease are correlated with high blood pressure (World Health Organization 2002), other researchers went further and investigated the

relationship between Na intake and stroke and cardiovascular disease as endpoints. They conducted a meta-analysis of 13 prospective studies that took place between 1966 and 2008 on 19 adult population samples (170,000 people) and recorded 10,000 vascular events. The results of this study show a positive correlation between salt intake and stroke and cardiovascular events. Based on those findings, the authors suggested that a reduction in salt intake (of 5 g/day) that is similar to WHO recommended Na intake level (of 5 g/day) would reduce by 23% the rate of stroke and by 17% the rate of total cardiovascular disease (Strazzullo et al 2009).

Intervention studies

Trials of Hypertension Prevention (TOHP I) was a randomized, controlled trial that took place in six centers from 1987 - 1990. It investigated, in 2182 mildly obese pre-hypertensives, the feasibility and efficacy of seven non-pharmacologic interventions: three lifestyle interventions (weight loss, sodium restriction, and stress management) and four nutritional supplementations (calcium, magnesium, potassium, and fish oil) (Satterfield et al 1991). The intervention regarding the salt reduction involved advising participants on shopping, cooking and food selection strategies. Weight loss and salt reduction (expressed as urinary salt excretion of 44 mmol/24h) induced a reduction in SBP and DBP of 2.9/2.3 mmHg and of 1.7/0.9 mmHg respectively. These two lifestyle interventions were identified as the most efficient strategies in lowering BP in pre-hypertensives and were used as criteria for inclusion in TOHP II (Cutler et al 1992). In a later reanalysis of the results of TOHP I, including in the data analyses the effect of intra-person variability, the blood pressure reduction was calculated to be 4.4/2.8 mmHg per 100 mmol/24 hour respectively (Cook et al 1998).

TOHP II was conducted from 1990-1992 and tested in a multicenter, controlled clinical trial the effects of the most efficient blood pressure interventions (weight loss, reduced sodium intake) identified in TOHP I. The TOHP II study population consisted of 2382 middle-aged, moderately overweight and pre-hypertensive (with SBP blood pressure readings of <140 mmHg and DBP of 83-89 mmHg) participants. The subjects were allocated to one of the three intervention groups: weight loss, sodium reduction, and combination weight loss and sodium reduction group and received group and individual

counselling according to the group to which they belonged (Hebert et al 1995). The expected outcome for the two salt reduction interventions was to decrease the group mean of 24-hour urinary sodium to 1800 mg (80 mEq) or less per day. At the 36 month follow up, the authors noticed that while only 21% of the participants achieved the desired 80 mEq in sodium excretion in urine, the overall reduction in sodium urinary excretion was to a level 24% lower in the intervention group than in the usual care group. In the intervention group, the reductions in SBP corresponding to the three regular monitoring visits (at 6, 18, and 36 months) were of 2.9, 2.0, and 1.3 mmHg. Adherence to the study was positively related to attendance at face to face guidance meetings. The authors recommend that reduction of salt as a means for reduction of blood pressure in the population be accompanied by industry interest in reducing salt content in foods (Kumanyika et al 2005, Lasser et al 1995).

The Trials of Nonpharmacologic Intervention in the Elderly (TONE), tested the possibility of controlling blood pressure exclusively by weight reduction, or by reduced Na intake or through both interventions combined (Appel et al 1995). The study was done in pre-hypertensive patients (aged 60 to 80 years) that had previously used antihypertensive medication. Participants would be removed from the study for one of the following endpoints: a blood pressure reading higher than 150/90 mmHg, the resumption of medication, and occurrence of a cardiovascular event. After 3 mo of intervention (using behavioural approaches), medication was withdrawn. The results showed that the mean urinary sodium excretion was 40 mmol/d less in the reduced sodium intake intervention group than in the control group. One of the end points occurred in 59% of the reduced sodium group and 73% in the control group participants. Due to a mean reduction of 4.3 mmHg ($P<0.001$) and 2.0 mmHg prior to medication withdrawal, it was concluded that reduced Na intake was a broadly effective, non-pharmacologic therapy that could lower blood pressure and control hypertension in older individuals (Appel et al 2001).

The weight of evidence from studies conducted to date has resulted in the majority of the scientific community advocating for salt reduction as a safe and healthy means of reducing blood pressure, stroke and cardiovascular disease burden in the population.

b. High K intake

Meta-analyses

The fact that blood pressure response to K supplementation is heterogenic emerged from a meta-analysis on 5 randomised controlled trials (Dickinson et al 2006). However, the most comprehensive meta-analyses performed to examine the effects of K supplementation on blood pressure have identified a K blood pressure lowering effect (Cappuccio and MacGregor 1991, Whelton et al 1997). In a meta-analysis that included 33 randomized, controlled trials that enrolled 2,609 participants whose variable was potassium supplementation (mainly potassium chloride (KCl)), a reduction in systolic and diastolic blood pressure of 3.11/1.97 mmHg was induced by K supplementation. The K urinary excretion reported by the studies included in this meta-analysis varied from 12-129 mmol/day (Whelton et al 1997). Similar results (5.9/3.4 mmHg) were obtained in a meta-analysis of 19 clinical trials that enrolled 586 participants (Cappuccio and MacGregor 1991). A particular finding of Whelton's study was the observation that blood pressure seems to be lowered more in subjects that had high Na intakes. This observation has practical application for advocating the importance of increased K intake even in individuals that are not able to reduce their salt intake.

Complementary to those results, data from another study (MacGregor et al 1982) showed that, when used in moderation, essential hypertension patients already on a low Na diet but with a moderate K supplementation, can slightly reduce blood pressure. This study also proposed that an increase in K intake can be done by taking K supplements, but suggests that fruits and vegetables become the elective source of K. This strategy is thought to reduce the need for antihypertensive drugs in patients with mild to moderate hypertension (MacGregor et al 1982). These findings are supported by another report (Siani et al 1991) that also suggested that dietary K reduces the need for antihypertensive drugs.

In parallel to the Na reduction trials meta-analyses, Geleijnse and coworkers performed a meta-analysis of 27 trials of K supplementation which demonstrated a median K increase of 44 mmol/24 h in adults and found a subsequent SBP reduction of 2.42 mmHg and a DBP reduction of 1.57 mmHg. When comparing hypertensives and

normotensives, respectively a greater decrease in SBP (3.51/2.51 mmHg) compared to DBP (0.97/0.34 mmHg) was observed (Geleijnse et al 2003). In a randomized, double-blind, placebo-controlled trial in a Chinese population of borderline hypertensives who had an high intake of Na and low intake of K, the effect of K supplementation on blood pressure was investigated (Gu et al 2001). Giving 150 participants a moderate K supplement (60 mmol KCl supplement), the researchers observed after 12 weeks that the treatment group had a reduced SBP (5.00 mmHg), but not a significantly reduced DBP (0.63 mmHg).

Intervention studies

There is a multitude of intervention studies investigating K effect on blood pressure, many of which have been criticized for different methodological limitations (Russo et al 2005). Some of them have been performed using K supplements; therefore their results, sometimes contradictory, could miss some of the real strength of the association found between dietary K and blood pressure. For example, KCl was found to lower blood pressure (Cappuccio and MacGregor 1991, Whelton et al 1997), while in another study, KCl and K citrate were deemed as having no effect on blood pressure (Mullen and O'Connor 1990). The same K salts were analyzed in a short term supplementation trial, and identified as yielding an equal blood pressure lowering effect. (He et al 2005a). Comparing KCl and potassium bicarbonate (KHCO_3) a trial concluded that KHCO_3 had a positive impact on hypertension, CVD, kidney stones and osteoporosis prevention (Morris Jr et al 1999a). Braschi and Naismith (2008) showed in a double-blind randomized placebo-controlled trial in young healthy normotensive volunteers, that the changes in mean arterial pressure (6.69/4.26 mmHg) induced by K-citrate were similar to changes (5.24/4.30 mmHg) induced by KCl (Braschi and Naismith 2008).

Epidemiological studies

While some studies suggest a neutral effect of K, the majority of the large studies have supported an inverse relationship between K intake and blood pressure. In a Belgian study, K intake was found to have no effect on blood pressure (Kesteloot and Joossens 1988). Similar data have been presented by Witteman et al. after analysing data from 58,218 female registered nurses (Witteman et al 1989). In the Scottish Heart Health study a weak relationship between K and blood pressure was found (Tunstall-Pedoe 1999). Findings from those studies are counterbalanced by results from several major studies. In a cohort of 30,681 male health professionals, it was found that at 4 years of follow-up, potassium, dietary fibre, and magnesium were each significantly associated with lower risk of hypertension (Ascherio et al 1992). Similar results were found in the 3239 participants in the Rotterdam Study (Geleijnse et al 1996). In this study, blood pressure reportedly decreased by 0.9/0.8 mmHg for each 1 g/day increase in K intake. Cross sectional epidemiological surveys in Japanese, Chinese and American blacks have shown the inverse correlation between 24hr urinary K excretion and blood pressure levels (Russo et al 2005). Analysing the 24 hr. recall and blood pressure data of 17,030 participants in the Third National Health and Nutrition Examination Survey (NHANES III), Hajjar et al found a positive association between higher sodium, alcohol, and protein intakes with SBP and a negative association of K intake with both SBP and DBP (Hajjar et al 2001).

It can be concluded overall that K supplementation leads to lower blood pressure especially in hypertensives and in persons that have high salt intake and that fruits and vegetables should be the preferred source of K (He and MacGregor 2001).

6. Na/K intake

The Na/K intake emerged from some epidemiological studies (Khaw and Barrett-Connor 1990, Stamler et al 1991) as a new and more potent factor in hypertension development than the contribution of each of the electrolytes separately (Stamler et al 1991).

Intervention studies

The initial Dietary Approaches to Stop Hypertension (DASH) study was a randomized, controlled-feeding trial that enrolled 456 healthy men and women with high-normal and mildly elevated blood pressure, and aimed to compare the effects of three dietary patterns on blood pressure levels. This design which involved the testing of dietary patterns not single nutrients, while providing all participants with foods generally available on the market, was unique at the time (Sacks et al 1995). The diets available to the participants were represented by one of the following three: 1) the control diet resembled an typical American diet; 2) the “ideal” diet designed to provide a high intake of fruits and vegetables and a low intake of saturated fats and cholesterol; 3) the DASH diet also called “the combination diet” that provided saturated fats and cholesterol at levels similar to the typical diet while being rich in fruits and vegetables. Sodium content was not explicitly controlled in this study (Sacks et al 1995). The primary outcome measure of the study was blood pressure variation according to different diets (Svetkey et al 1999).

The decrease of SBP/DBP induced by the combination diet was the most important result of this study. While the overall blood pressure reduction due to the combination diet was 5.5/3.0 mmHg (SBP/DBP), in hypertensives this diet induced a reduction of 11.4/5.5 mmHg and in non-hypertensives the reduction was by 3.5/2.1 mmHg (Appel et al 1997). Lower blood pressure readings were attained within 2 weeks and lasted for the next 6 weeks with a similar patterns in different subgroups (men/women; by ethnicity groups) (Conlin et al 2000). In a sub study that measured the ambulatory blood pressure (ABP) in 354 participants it was shown that the DASH diet provided significant around-the-clock reduction in blood pressure, especially in hypertensive participants (Moore et al 1999).

Based on all those findings, the Sixth Report of the Joint National Committee (JNC-VI) recommended including the DASH diet amongst the 4 major contributors in lowering blood pressure (weight loss, reduced sodium intake, increased physical activity, and limited alcohol consumption), contributors already recognised by Joint National Committee on the Detection, Evaluation and Treatment of High Blood Pressure (JNC) and the Working Group Report on Primary Prevention of Hypertension.

The DASH diet was included in the PREMIER study that tested the efficacy of the implementation of multi-component behavioural lifestyle intervention in free living normotensives and stage 1 hypertensive individuals. The participants were randomized to 3 different groups (advice only, established intervention, established intervention plus DASH). The participants included in advice only group were advised to follow the 5 lifestyle modifications (weight loss, sodium reduction, healthy eating, physically activity, and limited alcohol intake) to reduce blood pressure. While the participants in the established intervention group as well as the ones in the established intervention plus DASH aimed to lose weight by reducing calorie intake and increasing physical activity, both were also advised to have a salt intake less than 2300 mg/day. Only the last group received instructions and counselling regarding DASH dietary recommendations (9-12 servings/day of fruits and vegetables and 2-3 servings/day low fat dairy) (Svetkey et al 2003). The primary outcome was SBP at 6 months. A comparison among the three diets showed that the established plus DASH intervention led to the highest net reduction in blood pressure (4.3 mmHg vs. 3.7 mmHg in established group) and induced the lowest hypertension prevalence of 12% (versus 26% in the advice only group, 17% in the established group). It was concluded that lifestyle modifications (including DASH diet) are an effective means of lowering blood pressure (Appel 2003). At the end of the 6 month trial, the decline in mean net blood pressure readings in the established group was 3.7 mmHg and 4.3 mmHg in established plus DASH group.

A few years after the first DASH study, the Sodium Intake and Blood Pressure Trial (DASH-Na) was conducted. It was designed, similarly to the initial DASH, as a multicenter, double blind, randomized trial with a run-in and a feeding period study that enrolled normotensives or stage 1 hypertension (defined as 120-159/80-95 mmHg) (Svetkey et al 1999). In this new trial the attempt was to identify the effect on blood

pressure of 2 dietary patterns (standard American vs. DASH diet) each with 3 different sodium levels (higher=142 mmol/d, intermediate=107 mmol/d, and lower=65 mmol/d). The 3 sodium levels corresponded to the typical US salt intake (the highest level), the current DRI recommendations (the intermediary level) and a “potentially optimal” level (the lowest one). The primary outcome measured was the comparison of SBP readings at the end of each intervention feeding period, compared across the 3 sodium levels within each diet and between the 2 diets within each sodium level (Svetkey et al 1999, Svetkey et al 2004). It was thought that the additive effect of DASH and lower Na intake could have a hypotensive effect not previously reached by non-pharmacological treatment. Therefore, in the DASH-Na study the authors investigated the effect of incremental sodium reduction within the control and combination (DASH) diet. Results of this study are summarized in Table 3.

The initial findings showed that in all participants each sodium level DASH diet corresponded to a lower SBP than the American diet. However, gradual sodium intake reduction from higher to medium and then to lower salt intake within each diet determined a higher blood pressure reduction within American diet (2.1 from higher to medium and 4.6 from medium to low) than in DASH diet (1.3 from higher to medium and 1.7 from medium to low) (Sacks et al 2001). In non-hypertensives, DASH-Na diet produced better results in older than 45 year vs. younger patients (7.0/3.8 mmHg vs. 3.7/1.5 mmHg) (Vollmer et al 2001). The effect of DASH diet with a low sodium level vs. control diet with a high sodium level was consistent with a higher decrease in blood pressure in hypertensives than in normotensives (11.5 mmHg vs. 7.1 mmHg) (Sacks et al 2001).

In regards to individual controlled blood pressure, at high salt content DASH is more effective than American diet (63% vs. 32 % of participants), and the best results (84%) in controlling blood pressure were obtained using DASH-Na diet. Also, consuming the DASH-Na diet was accompanied by a normalization of blood pressure in 77% of participants (Svetkey et al 2004). At follow up, after 12 months, the increase in blood pressure in DASH-Na consumers was less than in the control group (3.12/ 0.79 mmHg vs. 5.33/3.20 mmHg) (Ard et al 2004).

Table 3: Comparison of the effects of DASH-sodium diet vs. standard American typical diet on controlling blood pressure

Type of diet ¹	Salt content/diet (mmol/d) ²	BP reduction (mmHg) induced by lowering salt content in diet ³	Effective in controlling BP ⁴	BP became normal ⁴	BP increased after 12 month, ⁵ (SBP/DBP)mm Hg
American diet	High =142	From high to medium→ 2.1	32%	n/a	5.33/3.20
	Medium =107	From medium to low→ 4.6	(74%),	n/a	
	Low =65		n/a	71%	
DASH diet	High =142	From high to medium→1.3	63%	n/a	3.12/ 0.79
	Medium =107	From medium to low→ 1.7	n/a	n/a	
	Low =65		(84%)	77%	

¹= type of diet (Svetkey et al 1999)

American diet = diet resembling typical American diet

DASH diet = diet rich in fruits, vegetables and low-fat dairy, whole grains, poultry, fish, and low in red meats, fats, sweets and sugar containing beverages.

²= (Svetkey et al 1999)

³= (Sacks et al 2001)

⁴= (Svetkey et al 2004)

⁵= (Ard et al 2004)

The natriuretic effect of K was studied by giving a total intake of 146 mmol/day KCl to 29 men with essential hypertension who already had a 250 mmol/day Na intake. As K supplementation prevented the increase in blood pressure the authors attributed the result to the natriuretic effect of K (Fujita and Ando 1984). Altering the K intake level (25 vs. 75 vs. 175 mmol/day) in mildly or moderately hypertensive men while keeping the sodium intake constant at 260 mmol/day and thereby improving the Na/K ratio, resulted in an inverse correlation of K level to blood pressure and a similar conclusion was drawn regarding K's natriuretic effect (Iimura et al 1981).

The mostly widely discussed effect of Na/K ratio is related to salt sensitivity. Animal studies done on Dahl rats that had salt sensitive hypertension showed that both K supplements (Dahl et al 1972), and dietary K (Ganguli and Tobian 1990) had an impact on the blood pressure response after a high salt intake. Another study focused on African Americans' and Caucasian Americans' salt sensitivity and blood pressure response to a load of 250 mmol/day Na with 3 different levels of K bicarbonate intake (30, 70 or 120 mmol/day). "Salt sensitivity" was defined as a 3 mmHg increase in blood pressure and "severe salt sensitivity" as an increase of 10 mmHg. At the lowest K intake, the salt load led to salt sensitivity in 79% of African Americans vs. 36% of Caucasian Americans. While the intermediary level (70 mmol) facilitated a reduction of salt sensitivity in both groups, the highest level (120 mmol/day) led to an occurrence of severe salt sensitivity in only 25% in both African Americans and Caucasian Americans (Morris Jr et al 1999b).

A non-pharmacological approach to lowering blood pressure in older people with mild to moderate hypertension was suggested by Geleijnse et al (1994). In an interventional study the authors replaced regular sodium salt with a multi-mineral salt (sodium: potassium: magnesium 8:6:1) in a 24 week double-blind placebo-controlled trial that included 100 older people with untreated mild to moderate hypertension. In the mineral salt group, a reduction by 7.6 mmHg in SBP and by 3.3 mmHg in DBP was noted when the study was complete. However, the authors noticed that twenty five weeks after the study was complete (and the participants had ceased taking the mineral salt), that the attained difference in blood pressure readings between the groups was no longer detectable (Geleijnse et al 1994).

A study focusing on children aged 5-17 showed that the dietary Na/K ratio, estimated by Na/K urine excretion, was related to the rise in blood pressure (0.356 mmHg/year/unit) in childhood and might be important in the early pathogenesis of primary hypertension (Geleijnse et al 1990). Since hypertension is thought to be caused by different lifestyle factors starting from early childhood, and since DASH was proven an effective diet in adults, Moore et al (2005) identified a subgroup of children from the Framingham Children's Study and fed them the DASH diet, then followed them until the children reached adolescence. The comparison of the blood pressure readings at the beginning and the end of the study showed that children that consumed 4 or more servings vegetables/day or 2 or more dairy products servings/day had smaller yearly gains in systolic blood pressure throughout childhood and had a lower adjusted mean systolic blood pressure by 10.6 ± 2.9 mmHg, compared to those with lower intakes (Moore et al 2005).

Epidemiological studies

INTERSALT, an observational study that took place in 52 centres and 32 countries (10,079 individuals), investigated the effects of electrolytes and other factors on blood pressure and found a significant positive association between the Na/K ratio and SBP (Stamler et al 1991). Another observational study found that the dietary Na/K ratio was correlated significantly to the age-adjusted SBP and DBP in both men and women, with a correlation that was again better for the Na/K ratio than for each electrolyte separately (Khaw and Barrett-Connor 1990). Interpreting the results of this study the researchers suggested that the sensitivity to dietary Na/K ratio increases with age. Except for body mass index, which reduced the strength of the association in women, no other dietary variable (calories, protein, carbohydrate, saturated fat, alcohol, calcium, and fibre) was able to alter the relationship (Khaw and Barrett-Connor 1988).

In conclusion, the key points from this literature review that included meta-analyses, interventional and epidemiological studies focused on health effects of Na and K are:

- The role of excess Na in hypertension aetiology is well documented and accepted by the scientific community. K deficiency contribution to this condition could benefit from more studies;
- The role of reduced Na intake in hypertension alleviation is well documented and accepted by the scientific community. Increased K intake preferably from dietary sources could reduce hypertension;
- The interrelationship of Na and K in the aetiology and treatment of hypertension should be further examined. Na and K together potentially have a more significant effect on blood pressure than each of the nutrients separately.

In the next chapters of the thesis we present the Na and K content of food composites samples from Canadian Total Diet Study (2007) (paper 1) and the Na and K intake data as derived from merging our nutrient data with food intake data from Canadian Community Health Survey Cycle 2.2, Nutrition (2004) (paper 2)

PART III- EXPERIMENTAL WORK

Paper 1 Sodium and potassium in food composite samples from the Canadian Total Diet Study 2007: Vancouver

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Abstract

Sodium (Na) and potassium (K) are essential nutrients. Like people in many other Western societies, Canadians consume too much Na and not enough K, both of which contribute to hypertension. We analyzed the Na and K content of 154 food composites, broadly representative of the foods most commonly consumed in Canada, from the Canadian Total Diet Study collection of 2007. Foods were prepared as if for home consumption before compositing. No salt was added during food preparation. Samples prepared by aqueous extraction were analyzed by atomic emission (Na) or atomic absorption spectrometry (K). Processed foods and soups contained large amounts of Na per reference amount (serving) of the food while fluid milk, unprocessed meats and several fruits and vegetables contained large amounts of K per reference amount. Na:K molar ratios were typically either high or low, with few values near unity. Thus, with few exceptions, foods high in Na were lower in K, and vice versa. Through judicious food selection it may be possible for consumers to decrease Na intakes while increasing K, with associated health benefits. Such choices would be consistent with common nutrition advice to decrease consumption of processed foods, while increasing intakes of fresh fruits and vegetables.

Introduction

Sodium (Na) and potassium (K) are essential mineral nutrients (Institute of Medicine 2005). In North America, the Adequate Intake (AI) for Na has been set at 1500 mg/d for adults (less for children), higher than the purported physiological requirement but taking into account the requirements for other nutrients in an achievable diet and allowing for higher Na losses in unacclimatized people exposed to warm temperatures or moderately active people. The Tolerable Upper Level of Intake (UL) for Na was set at 2300 mg/d based on blood pressure elevation as documented in dose-response trials. The AI for K was set at 4700 mg/d in adults (less for children) based on lowering blood pressure, reducing bone turnover and kidney stone formation. Due to generally low intakes of K in the North American population, and its ready excretion given normal kidney function, no UL was established for K (Institute of Medicine 2005a)

Many people in Western societies consume too much Na and not enough K, both of which contribute to the high prevalence of hypertension in these populations (He and MacGregor 2001, He and MacGregor 2009). Chronic, progressive hypertension is strongly associated with adverse functional and structural vascular and cardiovascular changes leading to multi-organ damage, morbidity and death (Heart and Stroke Foundation of Canada 1999). High salt intake has been associated directly with increased risk of stroke and total cardiovascular disease in a meta-analysis of 19 independent cohort samples from 13 studies (Strazzullo et al 2009). The estimated total direct health care cost savings in Canada of Na intake reduction to a level near the AI would be \$1.38 billion; growing to \$2.99 billion if indirect costs were also included (Heart and Stroke Foundation of Canada 1999). Increasing K intake is associated with lowering of blood pressure and the effects of increasing K intake are additive to the effects of lowering Na intake (He and MacGregor 2001). Thus ongoing monitoring of both Na and K in foods is important in order to gauge progress towards optimizing dietary management of blood pressure in the population.

Total Diet Studies (TDS, known in some regions as Market Basket Surveys) are promoted by the World Health Organization as an efficient means of monitoring for contaminants in the food supply. Although there are acknowledged limitations arising from food selection and compositing of food samples prior to analysis, the TDS has been

used for many years in the United States to track changes in food composition, including mineral nutrient levels, over time (Pennington 2000). TDS have the advantage that foods being analysed have been prepared as if for household consumption, rather than being analysed raw or as purchased (Leblanc et al. 2005). The representative nature of the Canadian TDS collection, which is designed to represent the majority of the foods commonly purchased in Canada, suggested to us that these samples should also be useful for monitoring Na and K in the Canadian food supply. TDS have been conducted in Canada since 1969, although the pattern of sampling and compositing of the food samples purchased has undergone some changes over the decades (Conacher et al. 1989). TDS samples from 1974-75, collected from Halifax, Montreal, Winnipeg and Vancouver, had been composited into 10 food groups and assayed for Na and K, showing 2- to 5-fold higher than recommended intakes for Na (depending on age/sex group (Shah et al 1982). Based on those analyses, K intakes were sufficient to meet the recommendations of that time (Shah et al 1982), although the calculated intakes in that report fell below the modern AI value for K.

Current activity in the Canadian TDS involves collecting samples in one city per year such that five regions across Canada (British Columbia, Prairie provinces, Ontario, Quebec, Atlantic provinces) are represented in a five-year cycle. The present study, based on samples collected in Vancouver, Canada in 2007 is the first report of Na and K in Canadian TDS samples in several decades.

Materials and methods

Samples and preparation

Samples analysed for this study were collected in Health Canada's TDS program. The TDS collection in 2007 included 154 composites from a total of 930 foods, with different brands purchased at 4 different retail outlets (supermarkets or fast food restaurants, as appropriate) in Vancouver, Canada. Each food composite sample consisted of foods prepared as if for consumption from samples purchased from up to 6 manufacturers or brands, in a variety of packaging formats, representing the most popular brands based on supermarket shelf space. Purchased foods were delivered to the Food Laboratory at the Kemptville, Ontario campus of the University of Guelph where they

were processed as if for normal home consumption (washing, trimming, cooking, etc, as appropriate), combined in specified ratios for composites, homogenized and stored frozen (at -20°C) in 250 mL wide-mouth Nalgene bottles until analysis. Where needed, tap water from the Kemptville Food Laboratory was used for cooking, and was included as a separate composite in the analyses. No salt was added during or after food preparation.

Sodium and potassium analyses

Triplicate subsamples of each food composite were prepared by an aqueous extraction method (Health Protection Branch Laboratories 1983). Briefly, this process takes advantage of the high water solubility of Na and K in food matrices. Accurately weighed portions of approximately 1 g were mechanically homogenized and dispersed in 25 mL deionized water to achieve dissolution of Na and K. The homogenization apparatus was thoroughly cleaned, by rinsing with deionized water followed by acetone drying, between samples. After filtration using Whatman 541 filter paper, the particle-free filtrate was diluted appropriately (minimum two-fold) in dilute nitric acid containing CsCl₂ (1000 µg/mL) as matrix modifier. Sodium was determined by flame emission spectrometry (wavelength 589.0 nm, slit 0.2 nm) and potassium by flame atomic absorption spectrometry (wavelength 769.9 nm, slit 1.0 nm) on a PerkinElmer AAnalyst 400 (PerkinElmer Inc, Shelton CT). Standard curves were prepared using solutions prepared from 1000 mg/kg stocks in 4% HNO₃ (SCP Science, Montreal, QC, Canada). Quality control measures with each run included use of standards as check samples and recovery of Na and K from standard reference materials (SRMs): SRM1577b Bovine Liver (certified content: Na 0.242 ± 0.006 wt percent, K 0.994 ± 0.002 wt percent) or SRM1549 Non-fat Milk Powder (certified content: Na 0.497 ± 0.010 wt percent, K 1.69 ± 0.03 wt percent) (National Institute of Standards and Technology (NIST), Gaithersburg, MD). RM8414 Bovine Muscle (content: Na 0.210 ± 0.008 wt percent, K 1.517 ± 0.037 wt percent) and SRM1571 Orchard Leaves (certified content: Na 82 ± 6 µg/g, K 1.47 ± 0.03 wt percent) from the same supplier were also used in validation of the method in our laboratory.

Calculations and presentation of data

Detection limits (DL) were empirically determined based on three times the SD of 16 replicate blank readings for a typical 50x preparation (i.e. 1 g of sample in a 50 mL

minimum volume as analyzed). The DL determined in this way for Na was 2.4 mg/kg sample wet weight; for K the DL was 1.5 mg/kg sample wet weight. Food composite samples in total diet studies in Canada and elsewhere are commonly grouped according to similarity of source or use of the foods (though the exact groupings may differ from country to country). For calculation of group averages, samples containing less than the DL were assigned a value of one-half the DL. Results of Na and K analyses are presented for each TDS food composite group and for each individual food composite as mean $\pm$ SD based on wet weight of sample, to represent the food as consumed.

Analysed concentration values can have important limitations when comparing different foods, as people habitually eat different amounts of different foods in a “serving”. The Reference Amount of a food, as defined in Part B, Division 1 of Canada’s Food and Drug Regulations (Canada, Department of Justice 2009) can be used as a proxy for serving size. Similar definitions of typical servings exist in the United States (US Government Printing Office 2009) and in other jurisdictions. Canadian food regulations also define Reference Standard amounts of Na and K for use in food labelling: the reference standard for Na in Canada is 2400 mg and for K is 3500 mg. These definitions are used by food manufacturers in preparing the Nutrition Facts Table on the package label, and by the Canadian Food Inspection Agency in their programs for monitoring of food labelling accuracy (Canadian Food Inspection Agency 2009). Na and K concentrations per Reference Amount of food were calculated and reported in this work, and compared to the Reference Standard amounts. Because the ratio of Na to K has been shown to be important in blood pressure (Stamler et al 1989), the Na:K molar ratio was also calculated for each of the food composites in this study.

Results

The aqueous extraction, atomic spectroscopy method used in this study shows a high degree of accuracy for Na and K in a variety of sample matrices. Typical within-run recoveries from standard reference materials for Na were: SRM1577 Bovine Liver 100.2% of the certified value; RM8414 Bovine Muscle 100.2%. Typical within-run recoveries for K were: SRM1577 Bovine Liver 114.0%; RM8414 Bovine Muscle 103.5%; SRM1571 Orchard Leaves 98.5% of the certified value. Interday reliability results derived during the analyses reported in this paper ranged from 96-100% recovery of certified values for Na and 96-101% recovery for K (Table 4).

In examining the TDS group averages for similar foods (Table 5), the ingredients and sauces group emerged as having the highest average Na concentration on a sample wet weight basis, followed by the soups and fast foods group. Taking into account the Reference Amount of a food, the soups and fast foods clearly emerged on top in terms of potential exposure to Na per serving. Similarly, TDS group averages showed the highest K concentration in ingredients and sauces, but this group fell to the bottom of the list when serving sizes (Reference Amounts) were factored in. However, as the standard deviation terms associated with each group mean value clearly indicated, there was enormous variability within each of these groupings of “similar foods” in both Na and K concentrations.

Sodium concentration values for individual food composites (Table 6) ranged from below the detection limit of 2.4 mg/kg (5 composites: apple sauce, bananas, blueberries, cooking fats and salad oils, sugar) to over 50,000 mg/kg (2 composites: soya sauce, baking powder). Expressed as Na content per reference amount of food, 10 composites contained greater than 25% of the Canadian Na reference standard per reference amount of food (i.e. >600 mg/kg), and 8 of these were in the soups and fast foods composite group: all four composites of soups, plus pizza, chicken burger, hot dog and chicken nuggets. The other two composites providing this high amount of Na per reference amount were cured pork and baked beans. The remaining members of the soups and fast foods group provided at least 10% of the Canadian Na reference standard per reference amount of food (i.e. >240 mg/kg), as did cottage cheese, processed cheese,

lunch meats, poultry liver pate, shellfish, soya sauce, canned vegetable juice, several bread composites, cake, rice & bran cereal, pancakes & waffles and mixed pasta dishes.

Potassium concentration values for individual food composites ranged from below the detection limit of 1.5 mg/kg (4 composites: cooking fats & salad oils, tap water from Vancouver, natural spring water and natural mineral water) to over 15,000 mg/kg (1 composite: herbs & spices). Expressed as K content per reference amount of food, only the apricots composite contained greater than 25% of the Canadian K reference standard per reference amount of food (i.e. >875 mg/kg). Fluid milks, including buttermilk and chocolate milk, beef steak, beef roast, veal, organ meats, freshwater fish, bananas, citrus juices, plums & prunes, kiwi fruit, baked beans, potato chips and potatoes (baked with skin) provided at least 10% of the Canadian K reference standard per reference amount of food (i.e. >350 mg/kg).

Discussion

As a group, the soups and fast foods (all examples of highly processed foods) were the highest in Na content per Reference Amount (serving) in this study, followed by the meat, fish and poultry group and the breads and cereals group. Similar observations were made in the first French TDS (Leblanc et al. 2005), where the highest contents of Na were found in processed meat products, processed cereal products, cheeses and “composed foods” (i.e. processed foods) generally. In the report of the 2003-04 New Zealand TDS, processing of food products was specifically noted to raise the Na content, e.g. tomato had 36 mg Na/kg whereas tomato sauce had 7,070 mg Na/kg and cream had 250 mg Na/kg whereas cheese had 6,300 mg Na/kg (Thompson et al 2008). Results such as these, and those of the present study, support the general statement that much of the Na intake (approximately three-quarters of it) from a Western diet comes from processed foods (Andersen et al 2009, James et al 1987, Mattes and Donnelly 1991). It has been suggested that if the Canadian diet consisted of only unprocessed foods, the Na content would be about 80% lower, but the K content would be about 60% higher (Shah and Belonje 1983).

Commonly cited sources of dietary K include fruits and vegetables, especially leafy greens and vine fruits (tomatoes, squashes, melons) and root vegetables (He and

MacGregor 2001, Institute of Medicine 2005b). Results of the present investigation generally support these conclusions, but also suggest that milk and dairy products can provide relatively large amounts of K per serving.

With few exceptions, food composites that were high in Na per reference amount of food in this survey were low in K, and vice versa. This is reflected in the Na:K molar ratios shown in Table 6. The highest Na:K molar ratios were found for cheeses and butter, processed meats, soups and fast foods generally, bread and cereal products (but not the minimally-processed grains such as oatmeal or white flour), and ingredients and sauces generally. The lowest ratios were found for fluid milks, unprocessed meats, minimally-processed grains, fruits, minimally-processed vegetables, and baby foods. Of the food composites that stood out by having >10% of the pertinent reference standard per Reference Amount of food, only baked beans surpassed this threshold for both Na and K. Thus, with judicious food selection, it may be feasible for individuals to arrive at a diet that is lower in Na and higher in K. Such choices would be consistent with common nutrition advice to decrease consumption of processed foods, while increasing intakes of fresh fruits and vegetables. A similar conclusion was recently reported by researchers in France (Meneton et al. 2009). However, the major contribution of processed foods to sodium intakes speaks clearly to the necessity for reduction of the sodium content of these foods, as excess sodium consumption is linked to adverse health effects including hypertension (He and MacGregor 2009). An ongoing program such as the TDS can help to monitor such change.

It is noted that there is wide variation in Na and K content between food composites within a TDS group of “similar foods”. In some cases, there would also be important differences in Na and K content of foods that are combined within an individual food composite. The naming conventions of, and the selections of specific foods for, food composites in the Canadian TDS are long-established and reflect the historical emphasis of this TDS on contaminant residues. This helps to explain the choices of foods included in some of the food composites, which would seem highly unusual if Na or K were the primary focus of the TDS. Thus, for example, the cucumber composite included both raw cucumber (with very little Na) and dill pickles (higher in Na), and the corn composite included frozen corn (very little Na), canned corn (higher in

Na) and creamed corn (higher yet). Beans, beets and peas composites similarly included both fresh and canned or pickled components. Despite these unusual features, the TDS provides a convenience sample overview of common foods in the Canadian diet. Specific unexpected analytical results can be followed up with additional investigation of the components within a given composite.

The food supply is constantly changing, in the composition of basic food commodities (e.g. due to growing conditions), and through changing recipes and food habits of the population (Pennington et al 2007). In addition, the Na content of processed foods can vary by as much as an order of magnitude between competing brands (Thompson 2009). With few exceptions, the Na and K concentrations found in food composites in the present study fell within, or close to, the ranges reported over 14 years of the US FDA's TDS (US Food and Drug Administration 2007). As the marketplace changes, the Na and K content of foods may change, speaking to the need for ongoing monitoring. Continuing to evaluate Na and K content of samples from the Canadian TDS can contribute to monitoring of these changes in food composition over time.

Revisions to food composition data can also have a significant impact on estimation of nutrient intakes. For example, updating U.S. intake estimates for K from the Continuing Survey of Food Intakes of Individuals (CSFII 1994-96, 98) using the Food and Nutrient Database for Dietary Studies (FNDDS 1.0) resulted in small but significant decreases in K intake estimates (Ahuja et al 2006). We intend to apply the Na and K composition values reported here in modelling of Na and K intakes in the Canadian population, which will be presented in a companion paper.

It is a practical impossibility to analyse the composition of every food in the marketplace. Nevertheless, it is important to be aware of the potential variation in food composition in deciding what database values or specific monitoring program will best suit the intended purpose of a given investigation (Champagne and Lastor 2009). Given the central roles of dietary Na and K in hypertension, ongoing monitoring through the Canadian TDS can be an important contribution to evaluation of dietary management of hypertension in the Canadian population.

Table 4: Interday recoveries of sodium and potassium from two standard reference materials (SRMs) included as quality control samples in analytical sets from the present study.

Standard Reference Material	Measured (mg/kg)	SD (mg/kg)	Certified (mg/kg)	Uncertainty* (mg/kg)	Recovery (%)
Sodium					
Bovine Liver SRM1577b (n=8) ¹	2312	103	2420	60	96
Non-fat Milk Powder SRM1549 (n=6)	4981	376	4970	100	100
Potassium					
Bovine Liver SRM1577b (n=6)	10017	511	9940	20	101
Non-fat Milk Powder SRM1549 (n=9)	16250	878	16900	300	96

* Uncertainty term for SRMs from product certificates. This value is typically derived from 95% confidence intervals with allowance for systematic error.

¹ n is the number of analytical runs (days) where the specified standard reference material was analysed.

Table 5: Sodium and potassium concentrations in food composite groups from the Canadian Total Diet Study 2007, in mg/kg sample wet weight, and mg per Reference Amount of food

Food composite group	Sodium			Potassium		
	Measured (mg/kg)	SD (mg/kg)	Per Reference Amount (mg) ¹	Measured (mg/kg)	SD (mg/kg)	Per Reference Amount (mg) ¹
Milk and dairy products (n=12) ²	2497	3929	141	1912	973	323
Meat, poultry and fish (n=18)	3715	4423	232	2905	1331	247
Soups and fast foods (n=13)	5147	2328	837	1671	1119	228
Bread and cereals (n=21)	3577	2617	202	1160	695	81
Vegetables (n=26)	965	1240	103	2371	2413	212
Fruit (n=21)	28 ³	36	4	2302	1940	316
Snacks and sweets (n=12)	912 ³	1043	42	1969	2469	80
Ingredients and sauces (n=9)	15907 ³	24843	70	3599 ³	5663	7
Baby foods (n=9)	196	176	17	1415	591	103
Beverages (n=10)	75	138	21	357 ³	431	79

¹ group average value based on sodium or potassium per Reference Amount of food as shown in Table 3, see also footnotes to Table 3

² n is the number of individual food composites in the group

³ this food group contains some composites that were below the detection limit; assigned value of one-half the detection limit for group average calculation

Table 6: Sodium and potassium concentrations in individual food composite samples (sorted by composite group) from the Canadian Total Diet Study 2007, in mg/kg sample wet weight and mg per Reference Amount¹ (Ref Amt) of the food, with Na:K molar ratio in the sample as assayed.

Composite sample ID	Reference Amount	SODIUM			POTASSIUM			Na:K
		Mean	SD	mg/Ref	Mean	SD	mg/Ref	molar ratio
		(mg/kg)	(mg/kg)	Amt	(mg/kg)	(mg/kg)	Amt	
<i>Milk and dairy products group (n=14)</i>								
Milk, whole	250 ml	299	7	75	3180	163	795	0.16
Milk, 2%	250 ml	326	3	81	1539	70	385	0.36
Milk, 1%	250 ml	336	13	84	3191	207	798	0.18
Milk, skim	250 ml	325	3	81	3474	286	869	0.16
Evaporated milk, canned	15 ml	877	3	13	2833	181	43	0.53
Cream	15 ml	440	31	7	1357	16	20	0.55
Ice cream	125 ml	656	24	82	2148	144	269	0.52
Yogurt	175 ml	369	6	65	1743	143	305	0.36
Cheese	30 g	6257	161	188	710	31	21	15.0
Cheese, cottage	125 ml	3796	70	475	1073	64	134	6.01
Cheese, processed	30 g	14050	682	422	2025	83	61	11.8
Butter	10 g	5839	474	58	197	16	2	50.3
Chocolate milk, 1%	250 ml	796	21	199	1576	17	394	0.86
Buttermilk, 1%	250 ml	591	37	148	1723	129	431	0.58
<i>Meat, poultry and fish group (n=18)</i>								
Beef, steak	100 g	654	14	65	4596	93	460	0.24
Beef, roast	100 g	545	12	55	3524	53	352	0.26
Beef, ground	60 g	642	13	39	3380	60	203	0.32
Pork, fresh	100 g	636	14	64	3421	130	342	0.32
Pork, cured	55 g	14974	676	824	4519	246	249	5.64

Composite sample ID	Reference Amount	SODIUM			POTASSIUM			Na:K molar ratio
		Mean (mg/kg)	SD (mg/kg)	mg/Ref Amt	Mean (mg/kg)	SD (mg/kg)	mg/Ref Amt	
Veal, cutlets	100 g	820	9	82	4919	13	492	0.28
Lamb	100 g	908	12	91	3452	75	345	0.45
Luncheon meats, cold cuts	55 g	10170	140	559	2191	69	121	7.89
Luncheon meats, canned	55 g	9733	802	535	1418	49	78	11.7
Organ meats	100 g	999	6	100	3583	22	358	0.47
Wieners & sausages	55 g	9199	523	506	1573	122	87	9.95
Eggs	50 g	1121	101	56	917	26	46	2.08
Poultry, chicken & turkey	100 g	714	20	71	3127	82	313	0.39
Poultry, liver pate	55 g	6078	247	334	1352	116	74	7.65
Fish, marine	100 g	1696	145	170	3060	283	306	0.94
Fish, fresh water	100 g	474	18	47	4384	231	438	0.18
Fish, canned	55 g	3832	290	211	2319	90	128	2.81
Shellfish	100 g	3666	258	367	563	7	56	11.1
<i>Soups and fast foods group (n=13)</i>								
Soups, meats, canned	250 ml	3665	176	916	538	29	134	11.6
Soups, creamed, canned	250 ml	3700	86	925	1070	49	268	5.88
Soups, broth, canned	250 ml	4256	319	1064	251	4	63	28.8
Soups, dehydrated	250 ml	8955	676	2239	336	23	84	45.3
Popcorn, microwave	50 g	5900	1016	295	2676	249	134	3.75
Frozen entrees	195 g	1966	158	383	1705	39	333	1.96

Composite sample ID	Reference Amount	SODIUM			POTASSIUM			Na:K molar ratio
		Mean (mg/kg)	SD (mg/kg)	mg/Ref Amt	Mean (mg/kg)	SD (mg/kg)	mg/Ref Amt	
Pizza	140 g	4427	120	620	1634	115	229	4.61
French fries	70 g	7199	257	504	4470	396	313	2.74
Hamburger	140 g	4214	376	590	1994	49	279	3.59
Chicken burger	140 g	5655	181	792	2166	11	303	4.44
Hot dog	140 g	8940	416	1252	1508	53	211	10.1
Chicken nugget	140 g	6470	181	906	2108	136	295	5.22
Beef chow mein, carry-out	250 ml	1571	98	393	1266	88	316	2.11
<i>Bread and cereals group (n=21)</i>								
Bread, white	50 g	6012	60	301	1214	19	61	8.42
Bread, whole wheat	50 g	5773	150	289	2130	62	107	4.61
Bread, rye	50 g	5062	70	253	1613	36	81	5.34
Cake	80 g	3511	161	281	1639	63	131	3.64
Cereal, cooked wheat	250 ml	851	25	213	440	16	110	3.29
Cereal, corn	30 g	5100	413	153	828	27	25	10.5
Cereal, oatmeal	250 ml	24	2	6	461	16	115	0.09
Cereals, rice & bran	30 g	8035	489	241	3133	297	94	4.36
Cookies, chocolate chip	30 g	3515	49	106	1771	73	53	3.37
Crackers	20 g	9272	317	185	1207	24	24	13.1
Danish, donuts & croissant	55 g	4156	363	229	1068	38	59	6.62
Flour, white	30 g	3	1	0#	992	43	30	0.01
Muffins	55 g	3596	35	198	1217	44	67	5.02
Pancakes & waffles	75 g	4034	38	303	1477	48	111	4.65
Pasta, mixed	195 g	2869	150	560	1609	128	314	3.03

Composite sample ID	Reference Amount	SODIUM			POTASSIUM			Na:K molar ratio
		Mean (mg/kg)	SD (mg/kg)	mg/Ref Amt	Mean (mg/kg)	SD (mg/kg)	mg/Ref Amt	
dishes								
Pasta, plain	215 g	4	2	1	271	3	58	0.03
Pie, apple	110 g	1726	35	190	482	13	53	6.09
Pie, other	110 g	1762	51	194	487	38	54	6.15
Rice	140 g	30	1	4	260	4	36	0.19
Buns & rolls	55 g	4818	33	265	957	11	53	8.56
Breads, other	55 g	4960	241	273	1110	67	61	7.60
<i>Vegetables group (n=26)</i>								
Baked beans, canned	250ml	2892	35	723	2542	67	635	1.93
Beans, string	85 g	1704	145	145	1550	46	132	1.87
Beets	85 g	2008	24	171	1724	68	147	1.98
Broccoli	85 g	153	9	13	1647	77	140	0.16
Cabbage	85 g	95	9	8	1694	153	144	0.10
Carrots	85 g	163	10	14	2182	89	185	0.13
Cauliflower	85 g	148	4	13	1266	37	108	0.20
Celery	85 g	626	15	53	1627	110	138	0.62
Corn	85 g	1519	26	129	1827	25	155	1.41
Cucumbers	85 g	1614	37	137	1382	43	118	1.99
Lettuce	85 g	127	6	8	1520	30	99	0.14
Mushrooms	85 g	38	4	3	1844	162	157	0.04
Onions	85 g	33	2	3	826	79	70	0.07
Peas	85 g	1083	25	92	822	33	70	2.24
Peppers	85 g	4	0	0#	1698	164	144	0.01
Potatoes, peeled and boiled	110 g	20	2	2	2032	137	224	0.02
Potatoes, chips	50 g	1942	71	97	13282	1105	664	0.25
Rutabagas	85 g	35	0	3	519	18	44	0.11
Vegetable juice, canned	250 g	2219	23	555	1604	23	401	2.35

Composite sample ID	Reference Amount	SODIUM			POTASSIUM			Na:K molar ratio
		Mean (mg/kg)	SD (mg/kg)	mg/Ref Amt	Mean (mg/kg)	SD (mg/kg)	mg/Ref Amt	
Tomatoes	85 g	26	1	2	2599	175	221	0.02
Tomatoes & tomato sauce, canned	60 ml	3338	56	200	2281	33	137	2.49
Spinach	85 g	619	14	53	4007	241	341	0.26
Asparagus	85 g	40	4	3	2081	202	177	0.03
Brussels sprouts	85 g	64	1	6	2946	266	250	0.04
Potatoes, baked with skins	110 g	41	4	5	4921	408	541	0.01
Corn chips	50 g	4531	316	227	1231	86	62	6.26
<i>Fruit group (n=21)</i>								
Apple juice, canned	250 ml	50	1	13	957	92	239	0.09
Applesauce, canned	150 ml	--a	--	0#	778	72	117	0.00
Apples, raw	140 g	18	1	3	1138	55	159	0.03
Bananas	140 g	--	--	0#	3234	73	453	0.00
Blueberries	140 g	--	--	0#	729	50	102	0.00
Cherries	140 g	11	1	2	1468	74	206	0.01
Citrus fruit, raw	140 g	8	0	1	1728	134	242	0.01
Citrus juice, frozen	250 ml	25	1	6	2142	198	536	0.02
Citrus juice, canned	250 ml	33	0	8	2141	180	535	0.03
Grape juice, bottled	250 ml	28	0	7	831	28	208	0.06
Grapes	140 g	43	1	6	2148	117	301	0.03
Melons	150 g	144	1	22	1587	8	238	0.15
Peaches	140 g	8	0	1	1602	79	224	0.01
Pears	140 g	3	0	0#	1160	52	162	0.00*

Composite sample ID	Reference Amount	SODIUM			POTASSIUM			Na:K molar ratio
		Mean (mg/kg)	SD (mg/kg)	mg/Ref Amt	Mean (mg/kg)	SD (mg/kg)	mg/Ref Amt	
Pineapple, canned	150 ml	7	2	1	1378	25	207	0.01
Plums & prunes	140 g	3	1	0#	4087	95	572	0.00*
Raisins	40 g	95	9	4	8291	618	332	0.02
Raspberries	140 g	10	4	1	1626	133	228	0.01
Strawberries	140 g	12	10	2	1885	122	264	0.01
Kiwi fruit	140 g	14	6	2	2624	185	367	0.01
Apricot	140 g	65	4	9	6803	375	952	0.02
<i>Snacks and sweets group (n=12)</i>								
Chocolate bars	40 g	606	2	24	4044	151	162	0.25
Candy	15 g	1375	39	21	118	8	2	19.8
Gelatin dessert	125 ml	1037	60	130	16	1	2	112
Honey, bottled	20 g	41	4	1	252	20	5	0.28
Jams	15 ml	135	8	2	864	66	13	0.27
Peanut butter	15 g	3800	32	57	5210	402	78	1.24
Puddings	125 ml	1121	90	140	924	56	116	2.06
Sugar, white	4 g	--a	--	0	7	1	0	0.23
Syrup	30 ml	544	42	16	428	37	13	2.16
Seeds, shelled	50 g	750	52	38	4515	236	226	0.28
Nuts	50 g	1418	57	71	6907	391	345	0.35
Chewing gum	3 g	114	1	0#	340	26	1	0.57
<i>Ingredients and sauces group (n=9)</i>								
Cooking fats & salad oils	10 ml	--a	--	0	--b	--	0	1.70
Margarine	10 g	7909	765	79	455	40	5	29.6
Mayonnaise	15 ml	6993	291	105	188	16	3	63.2
Condiments	15 ml	8258	680	124	2462	193	37	5.70
Baking powder	0.6 g	62614	6056	38	1610	72	1	66.2
Yeast	0.6 g**	1334	111	1	10429	673	6	0.22
Vanilla extract	5 ml**	109	1	1	117	4	1	1.59

Composite sample ID	Reference Amount	SODIUM			POTASSIUM			Na:K molar ratio
		Mean (mg/kg)	SD (mg/kg)	mg/Ref Amt	Mean (mg/kg)	SD (mg/kg)	mg/Ref Amt	
Herbs & spices	0.5 g	156	5	0#	15939	1277	8	0.02
Soya sauce	5 ml	55792	866	279	1188	30	6	79.8
<i>Baby foods group (n=9)</i>								
Cereal, mixed (prepared with whole milk)	110 g	394	15	43	1952	108	215	0.34
Desserts	60 g	70	3	4	922	59	55	0.13
Dinners, cereal+vegetable +meat	60 g	42	4	3	1156	70	69	0.06
Dinners, vegetable+meat or poultry	60 g	80	7	5	1393	139	84	0.10
Formula, milk base	100 ml	356	10	36	1031	18	103	0.59
Formula, soya base	100 ml	343	30	34	641	39	64	0.91
Fruit, apple or peaches	60 g	32	2	2	2561	94	154	0.02
Meat, poultry or eggs	60 g	423	21	25	1767	91	106	0.41
Vegetables, peas	60 g	26	2	2	1315	131	79	0.03
<i>Beverages group (n=10)</i>								
Alcoholic drinks, beer	355 ml*	27	2	10	446	14	158	0.10
Alcoholic drinks, wine	150 ml*	44	5	7	835	56	125	0.09
Coffee	175 ml	49	3	9	695	44	122	0.12
Soft drinks, canned	355 ml	34	3	12	48	2	17	1.20

Composite sample ID	Reference Amount	SODIUM			POTASSIUM			Na:K molar ratio
		Mean (mg/kg)	SD (mg/kg)	mg/Ref Amt	Mean (mg/kg)	SD (mg/kg)	mg/Ref Amt	
Tea	175 ml	44	2	8	328	15	57	0.23
Soy beverage, fortified	250 ml	467	10	117	1208	68	302	0.66
Tap water, kitchen	500 ml	26	5	13	4	0#	2	9.94
Tap water, sample area	500 ml	32	1	16	--b	--	0	54.3
Water, natural spring	500 ml	5	0	3	--	--	0	9.06
Water, natural mineral	500 ml	24	0	12	--	--	0	40.35

¹ Reference Amounts of foods as specified in Canada's Food and Drug Regulations (Canada, Department of Justice 2009), except baby foods category – cereals and jarred foods from 21 CFR 101.12 (US Government Printing Office 2009)

* Reference Amount not available from Canada's Food and Drug Regulations, value is from Canada's Food Guide to Healthy Eating

** Reference Amount not available from Canada's Food and Drug Regulations, value is from the Canadian Nutrient File (J. Deeks, personal communication)

value of zero is within rounding

^a less than the detection limit for sodium (2.4 mg/kg); assigned value of 1 mg/kg for group average calculation

^b less than the detection limit for potassium (1.5 mg/kg); assigned value of 1 mg/kg for group average calculation

CONNECTING STATEMENT

Na and K content data of food composites from TDS 2007- Vancouver were reported in paper 1. These Na and K content data were merged with food intake data from CCHS 2.2- Nutrition in order to generate new estimates of Na and K intakes in Canadian population. The new Na and K intake models and the comparison with AI and UL are presented in paper 2.

Paper 2 Sodium and potassium intakes estimated from the Canadian Total Diet Study 2007: Vancouver

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Running title: Na and K intakes in Canada

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Abstract

Sodium (Na) and potassium (K) are essential nutrients. Excessive intakes of Na and insufficient intakes of K are known dietary factors in hypertension. Intakes of Na and K have been calculated based on recent analyses of Na and K content of food composites from the Canadian Total Diet Study (TDS), and compared to intakes presented from the Canadian Community Health Survey, cycle 2.2, Nutrition (CCHS 2.2). Na intakes calculated from the Canadian TDS for 2007 were significantly lower than those from the CCHS 2.2 for all demographic groups except for 1-3 year olds. As was the case with CCHS 2.2, results of the present work clearly show that Na intakes by most Canadians in all demographic groups exceed the Adequate Intake (AI), and in most cases also exceed the Tolerable Upper Level of Intake (UL). Only for women aged 71+ were mean Na intakes approximately equal to the UL. K intakes by most Canadians in all demographic groups were below the AI, and not significantly different between the TDS-based intakes and those from CCHS 2.2.

Major food category sources of Na in young children were milk and dairy products, bread and cereals, soups and fast foods. In each category, it was the more processed foods that provided the majority of the Na. With increasing age into adulthood, milk and dairy products contributions to Na intakes decreased. Major food category sources of K in young children included milk and dairy products and fruit. With increasing age into adulthood, milk and dairy products contributions to K intakes decreased, while vegetable contributions increased and fruit (including juices) continued to be important sources of K. Beverages (mostly coffee and tea) also made a substantial contribution to K intakes of adult Canadians. Continuing analysis and monitoring of Na and K intakes of Canadians through the Canadian TDS can help to inform public health activities designed to improve the nutritional health of the population.

Introduction

Sodium (Na) and potassium (K) are essential mineral nutrients (Institute of Medicine 2005a,b). Both are well absorbed, and Na and K are metabolically linked in many of their biological functions. In North America, the Adequate Intake (AI) for Na has been set at 1500 mg/d for adults (less for children), higher than the purported physiological requirement but taking into account the requirements for other nutrients in an achievable diet and allowing for higher Na losses in unacclimatized people exposed to warm temperatures or moderately active people (Institute of Medicine 2005a). The Tolerable Upper Level of Intake (UL) for Na was set at 2300 mg/d based on blood pressure elevation as documented in dose-response trials. The AI for K was set at 4700 mg/d in adults (less for children) based on lowering blood pressure, reducing bone turnover and kidney stone formation. Due to generally low intakes of K in the North American population, and its ready excretion given normal kidney function, no UL was established (Institute of Medicine 2005a,b).

Many people in Western societies consume too much Na and not enough K, both situations which contribute to the high prevalence of hypertension in these populations (He and MacGregor 2001, He and MacGregor 2009). Chronic, progressive hypertension is strongly associated with adverse functional and structural vascular and cardiovascular changes leading to multi-organ damage, morbidity and death (Heart and Stroke Foundation of Canada 1999). High salt intake has also been associated directly with increased risk of stroke and total cardiovascular disease in a meta-analysis of 19 independent cohort samples from 13 studies (Strazzullo et al. 2009).

Hypertension affects 20% of adults in Canada, with another 20% having blood pressure in the pre-hypertension range (Wilkins et al. 2010). It has been estimated that if the Na intake of Canadians were to be decreased by 1840 mg/d (to bring adult men to about the AI level of intake), hypertension prevalence could be decreased by 30%, with direct health care cost savings of \$430 million per year (Joffres et al. 2007). The same decrease in Na intake could prevent 23,500 cardiovascular disease events per year in Canada, a 13% decrease (Penz et al. 2008), for a further \$949 million per year of direct savings. The estimated total direct health care cost savings of Na intake reduction by this

extent would thus be \$1.38 billion; growing to \$2.99 billion if indirect costs were also included (Heart and Stroke Foundation of Canada 1999). Increasing K intake is associated with lowering of blood pressure and the effects of increasing K intake are additive to the effects of lowering Na intake (He and MacGregor 2001). There is an ongoing need to monitor Na and K in foods, in order to gauge progress towards the desired contribution of diet modification to lowering blood pressure in the population.

Total Diet Studies (TDS, known in some regions as Market Basket Surveys) are promoted by the World Health Organization as an efficient means of monitoring for contaminants in the food supply. Although there are acknowledged limitations due to food selection and compositing of food samples prior to analysis, the TDS has been used for many years in the United States to track changes over time in food composition, including levels of mineral nutrients such as Na and K (Pennington 2000). TDS have the advantage that foods being analysed have been prepared as if for household consumption, rather than being analysed raw or as purchased (Leblanc et al. 2005). The nature of the collection, designed to represent the majority of the foods commonly purchased in Canada, suggested to us that these samples should also be of some utility in monitoring Na and K in the Canadian food system. Current activity in the Canadian TDS involves collecting samples in one city per year, such that five regions across Canada (British Columbia, Prairie provinces, Ontario, Quebec, Atlantic provinces) are represented in a five-year cycle (Health Canada 2007). The 2007 collection in the Canadian TDS took place in Vancouver, Canada, providing the food composite samples analyzed and used in deriving the intake estimates reported here.

Materials and methods

Food sample acquisition and analysis

Sodium and potassium levels were analysed in food composite samples collected in the Canadian Total Diet Study in 2007 in Vancouver, Canada. Sample collection and processing and details of the analytical results have been reported previously (Tanase et al. 2010). Briefly, the 2007 Canadian TDS collection included 154 composites from a total of 930 foods, with different brands purchased at 4 different retail outlets (supermarkets or fast food restaurants, as appropriate). Each food composite sample was made up of foods prepared as if for home consumption, from samples purchased from up to 6 manufacturers or brands, in a variety of packaging formats, representing the most popular brands based on supermarket shelf space. Food samples were processed as if for home consumption at the Food Laboratory of the Kemptville, Ontario campus of the University of Guelph. Laboratory tap water was used in food preparation as needed, and included as a separate sample in the analyses. No salt was added to the foods during or after cooking, so that Na content did not include any “discretionary” salt.

Each food composite sample was analysed for Na and K content by aqueous extraction, atomic spectroscopy techniques (atomic emission for Na, atomic absorption for K) on a PerkinElmer AAnalyst 400 (PerkinElmer Inc, Shelton CT). Triplicate portions of each composite were analysed, yielding a single nutrient concentration value for each sample. A few composites contained levels below the detection limits of the respective techniques (for sodium: apple sauce, bananas, blueberries, cooking fats and salad oils, and white sugar; for potassium: cooking fats and salad oils, tap water, natural spring water and natural mineral water were below detection limits). For these few samples, an assigned value equal to the one half detection limit (1 mg/kg each for Na and K) was used for purposes of intake modelling. Standard reference materials SRM1577b Bovine Liver or SRM1549 Non-fat Milk Powder (National Institute of Standards and Technology, Gaithersburg, MD) were analysed as a check on the accuracy of the analyses (Tanase et al. 2010).

Food intake data and matching to food composites

Food intake data used in this modelling work were from the Canadian Community Health Survey, Cycle 2.2 (CCHS 2.2 wave 3), conducted between January 2004 and January 2005 (Health Canada 2006). CCHS 2.2 consisted of over 35,000 24-hour recalls of food intakes by Canadians, with a second 24-hour recall from a random subset of individuals to permit statistical adjustment for within-individual variation and derivation of usual intakes of the population groups. Some persons were excluded from the analyses. Those excluded were all persons under one year of age, persons who were fed only breast milk, lactating and pregnant women, and persons who did not have a valid recall in the survey. Consequently, survey results from approximately 33,000 persons were included in these analyses. These are the same conditions that were used in deriving nutrient intakes for the CCHS 2.2 survey reports (Health Canada 2008a, Health Canada 2008b). The Na and K concentration values for foods and recipes in the CCHS 2.2 survey reports came from a supplemented 2001b version of the Canadian Nutrient File (Health Canada 2010a), the national food composition database in Canada.

The matching of the Total Diet Study composites to the CCHS recall data was done by biostatisticians from the Bureau of Food Policy and Science Integration and vetted by nutrition professionals from the Bureau of Nutritional Sciences, both at Health Canada. Matching was evaluated on the basis of weight (g) of matched foods cited as being consumed in the CCHS 2.2 survey, and on the basis of calories of food energy provided by the consumed foods. The reason for this was that some foods (e.g. water) contributed to weight of foods consumed, but provided little food energy. Where no match was possible for a given food or recipe, the corresponding value from the CCHS survey was used, which was based on the Canadian Nutrient File, Canada's national database of food composition (Health Canada 2008a, Health Canada 2008b). This was done to ensure that all foods corresponded to some Na and K value, in order to facilitate comparison between the CCHS 2.2 and the TDS results for Na and K intakes in the Canadian population.

The degree of matching of foods and recipes to the TDS composites was evaluated as a proportion of the amount of food consumed and the total energy consumed. These measures were selected: (i) to determine the share of the food eaten, i.e. the amount

consumed; and (ii) since some foods such as water give mass but little energy, therefore as an indirect measure of the amount contributed to nutrient intake, the total energy that is represented by the TDS composites was calculated.

Statistical Methodology

The distribution of Na and K usual intakes was constructed by using the SIDE (Dodd 2001) software called from within a SAS environment. SIDE uses the method described by Nusser et al. (1996). The distribution was represented by output of a selection of percentiles (i.e. the 5th, 10th, 25th, Median, 75th, 90th, and 95th percentiles) and the mean. For each of these percentiles the standard error was computed by the bootstrap methodology (Manly 2007). Also, the estimated percentage, with its standard error, of the population above the AI for both Na and K were calculated. Similarly the estimated percentage of intakes above the Tolerable Upper Level of Intake (UL) for Na was calculated. These values were calculated for each of the DRI age groups for ages 1 and up. The bootstrap methodology is based on the premise that the distribution of the observed values from a sample is an approximation of the distribution of the population. Therefore repeatedly sampling, that sample with replacement, should result in multiple samples that mirror the distribution of the population. These multiple samples are used to estimate the uncertainties (standard error) of the point estimate of interest, such as the median. Because the survey design for CCHS 2.2 is complex, and to simplify matters and make it convenient for users, Statistics Canada has supplied the bootstrap weights that were used in these analyses. These bootstrap weights are used in the SIDE software to generate estimates of the uncertainties of the percentile estimates.

Mean Na and K intakes derived from the TDS analysed values for these two nutrients were compared to the corresponding CCHS 2.2 values using t-test with Bonferroni correction (Snedecor and Cochrane 1989).

Results

Matching of TDS composites to foods cited in the CCHS 2.2 nutrition survey yielded 86.0% match by weight, with 75.2% match for the energy provided by the matched foods. Thus most of the Canadian diet could be represented by the food

composites in the TDS. Examination of the list of foods unmatched for Na and K revealed that these were typically consumed by few individuals, made only very small contribution (<0.5%) to the Na and K intakes, and were widely distributed across the TDS food groups (data not shown). As noted above, where no match was obtained, the default value from the CCHS 2.2 survey results was retained, to allow estimation of the total Na and K intakes of Canadians, based to the greatest extent possible upon the new analytical values from the TDS.

Distributions of Na intakes across selected percentile estimates by demographic group have been calculated and compared to the AI and UL values by age and sex group (Table 7). Most Canadians, in practically all age and sex groups, exceeded their respective UL values for Na. Only for women 71+ years of age was less than 50% of the population subgroup above the UL, although the proportion was still greater than 30%. For adolescent and young adult males, the proportion with Na intakes exceeding the UL was greater than 90% of the population subgroup.

Distributions of K intakes across selected percentile estimates by demographic group have also been calculated (Table 8). Most Canadians in all age/sex groups were below their respective AI for K. The proportion below the AI ranged from 68.5% for children aged 1-3 years up to 98.8% for women aged 71+. Males were more successful in meeting the AI for K than their female counterparts in all age groups where the sexes were considered separately.

Mean Na intakes of almost all demographic groups exceeded both the AI and the UL (Figure 1). Na intakes based on TDS 2007 were, across almost age and sex groups, significantly lower than those reported in CCHS 2.2, which are shown for comparison. These results were statistically significant for all demographic groups except 1-3 year olds.

Mean K intakes of all demographic groups were much lower than the AI (Figure 2). No UL value has been established for K in North America, so no such comparison is possible. K intakes based upon TDS 2007 were similar to those reported in CCHS 2.2, with values across demographic groups slightly higher or lower than the corresponding CCHS 2.2 values. None of the differences were statistically significant.

For children 1-3 years old, foods included in the milk and dairy products category made the highest contribution to Na intakes (29.4%), followed by foods in the breads and cereals category (22.7%) (Table 9). With increasing age through adolescence the pattern was reversed with breads and cereals providing 25-28% of Na intakes and milk and dairy products providing 19-23%. In adults, breads and cereals continued to be top contributors to Na intakes (25-30%), with foods in the soups and fast foods category attaining second rank (18-22%). At the level of individual food composites, cheese, processed cheese, cured meats, bread (white or whole wheat), soups and salty sauces (such as soya sauce) were among the foods that contributed the most to Na intakes of Canadians (data not shown).

For children 1-3 years old, milk and dairy products made the highest contribution to K intakes (55.4%), followed by fruit (22.3%), while other food categories provided less than 10% each (Table 10). With increasing age through all age groups the degree of contribution by milk and dairy products declined, and contributions by vegetables and meat, poultry and fish increased. In adults, the beverage category made a much larger contribution to K intakes (14-16%) than in younger age groups, largely due to increased coffee and tea consumption (data not shown). At the level of individual food composites, fluid milks, citrus juice, meat, poultry, potatoes and bananas were among the foods that contributed the most to K intakes of Canadians (data not shown).

Discussion

Total Diet Studies have been conducted in Canada since 1969, although the pattern of sampling and compositing of the food samples purchased has changed over time (Conacher et al. 1989). TDS samples from 1974-75, collected from Halifax, Montreal, Winnipeg and Vancouver, had been composited into 10 food groups and assayed for Na and K, showing 2- to 5-fold higher than recommended intakes for Na (depending on age/sex group), but sufficient K intakes to meet intake recommendations that were current at that time (Shah et al. 1982).

In contrast, results of the present work showed mean Na intakes continued to exceed the AI, and exceeded the UL for almost all age and sex groups, while mean K intakes failed to meet the AI for any age and sex group. These results are similar to those

reported for the CCHS 2.2, which relied on food composition data from the Canadian Nutrient File (CNF) (Health Canada 2008a, Health Canada 2008b), and in publications arising from those reports (Garriguet 2007), and continue to emphasize a significant public health issue.

Across the full spectrum of Na intake distribution percentile estimates, the values generated from the current TDS-based project were consistently lower than those in the CCHS 2.2 report (Health Canada 2008a), which is a hopeful trend in the context of improving the (sodium) nutritional health of Canadians. As the present work was based on only one city and year of sample collection, additional monitoring and estimation of Na intakes based on subsequent TDS collections should be done to establish the validity of this trend. Since these Na intake estimates did not include any contribution from salt added in cooking or at the table (i.e. “discretionary” salt addition), the actual Na intakes could be up to 10-15% higher (James et al. 1987, Mattes and Donnelly 1991). The same was true for the Na intakes calculated for the CCHS 2.2 report (Health Canada 2008a). The Minister of Health in Canada recently formed a multi-stakeholder working group to address the need to reduce Na intakes in the Canadian population (Health Canada 2009).

K intake estimates across the full distribution of percentiles were not consistently above or below those in the CCHS 2.2 report (Health Canada 2008b), suggesting that K intakes have not changed much over the past few years. Improving K intakes in the population remains a task for dietitians and public health nutritionists.

The general pattern of food category sources for Na and K in the Canadian diet is similar to patterns published from other countries. In a report from the Italian TDS, cereals accounted for 15-18% of Na, but the total of the food categories reported was only 45-48% of Na consumed (Lombardi-Boccia et al. 2000, 2003). K intakes of Italians were principally from vegetables (26%) and meat & meat products (19%), with fruits or cereals/cereal products accounting for ~10% each. In the French TDS, the total of bread & cereal products accounted for roughly 30% of Na for adults and 44% for children (Leblanc et al. 2005). In a separate report, using intake estimates based on a French national food composition database, breads & cereals accounted for almost 30% of Na for adults and 23% for children (Meneton et al. 2009). The latter report noted that top contributors to K intakes in France were similar in adults and children: vegetables

(19.5%/18% for adults/children respectively), dairy products & cheese (13%/21.5%), meats (18.8%/16.5%), with fruits (9.3%/7.7%) also contributing. Results from the New Zealand TDS showed that the total of breads, cereals & pasta, and biscuits & cake accounted for 37% of Na (Thompson et al. 2008). No data on K were reported in that study. In a survey of samples collected in the United States in 1993-1995, for all groups except infants the primary Na source was grain products (21-26%); for infants it was milk & cheese (29%) (Hunt and Meacham 2001). K intakes in that survey were derived principally from milk & cheese for infants, toddlers, adolescents (28-42%), but from meat/fish/poultry for adults and seniors (18-22%).

In a report based on the Canadian CCHS 2.2 dataset, intake modelling data indicated that breads, breakfast cereals, cookies & bars and cakes accounted for 19-21% of Na intakes (Fischer et al. 2009). Some of the components of the bread and cereals group from the present study (e.g. pasta dishes, rice, pies, pancakes, muffins) were not included in that grouping, and may account for some of the differences between these two reports. Three decades ago, breads and cereals provided 33% and meat, poultry, fish and eggs provided 21% of Na in the Canadian diet, while dairy products (23%), potatoes (16%), fruit and fruit products (15%) and meat, poultry, fish and eggs (14%) were predominant sources of K (Shah et al. 1982). It has been calculated that, if the Canadian diet were made up of only unprocessed foods, Na content could be 80% lower while K content could be 60% higher (Shah and Belonje 1983).

One limitation of the TDS approach derives from the process of food compositing, as a limited number of samples can be analysed on a recurring basis. The food composition results, on which the present calculations are based, represent a single point in space and time, and do not take into account variation in the marketplace, particularly for a large country such as Canada. Although the majority of foods in the CCHS 2.2 food intake survey could be matched to TDS food composites, the remaining 14-24% of foods for which default composition values were used in the present study can be also seen as a limitation.

However, one of the great strengths of the TDS approach is the opportunity to monitor changes in nutrient intakes patterns over time (Pennington 1996). All of the food composition values in a TDS study are from the same place and time, as opposed to food

composition database values, which vary in provenance. Marketplace changes can affect the accuracy of food composition databases, which may become dated (Pennington et al. 2007). Recalculation of intake values based on new food composition data resulted in significant changes in K intakes in a recent paper from the U.S. (Ahuja et al. 2006). In the present work, K intakes were not significantly altered while Na intakes were, suggesting that the difference in Na intakes was not simply an artefact of the food matching process. It will be interesting to see how the results of food analyses from other city and year collections of the Canadian TDS affect the calculated Na and K intakes of Canadians. Continuing analysis and monitoring of Na and K intakes through the Canadian TDS can help to inform the success of public health activities designed to improve the nutritional health of the population.

Figure 1: Mean sodium intakes of Canadians, by demographic group, based on analyses in the Canadian Total Diet Study 2007 (Vancouver)

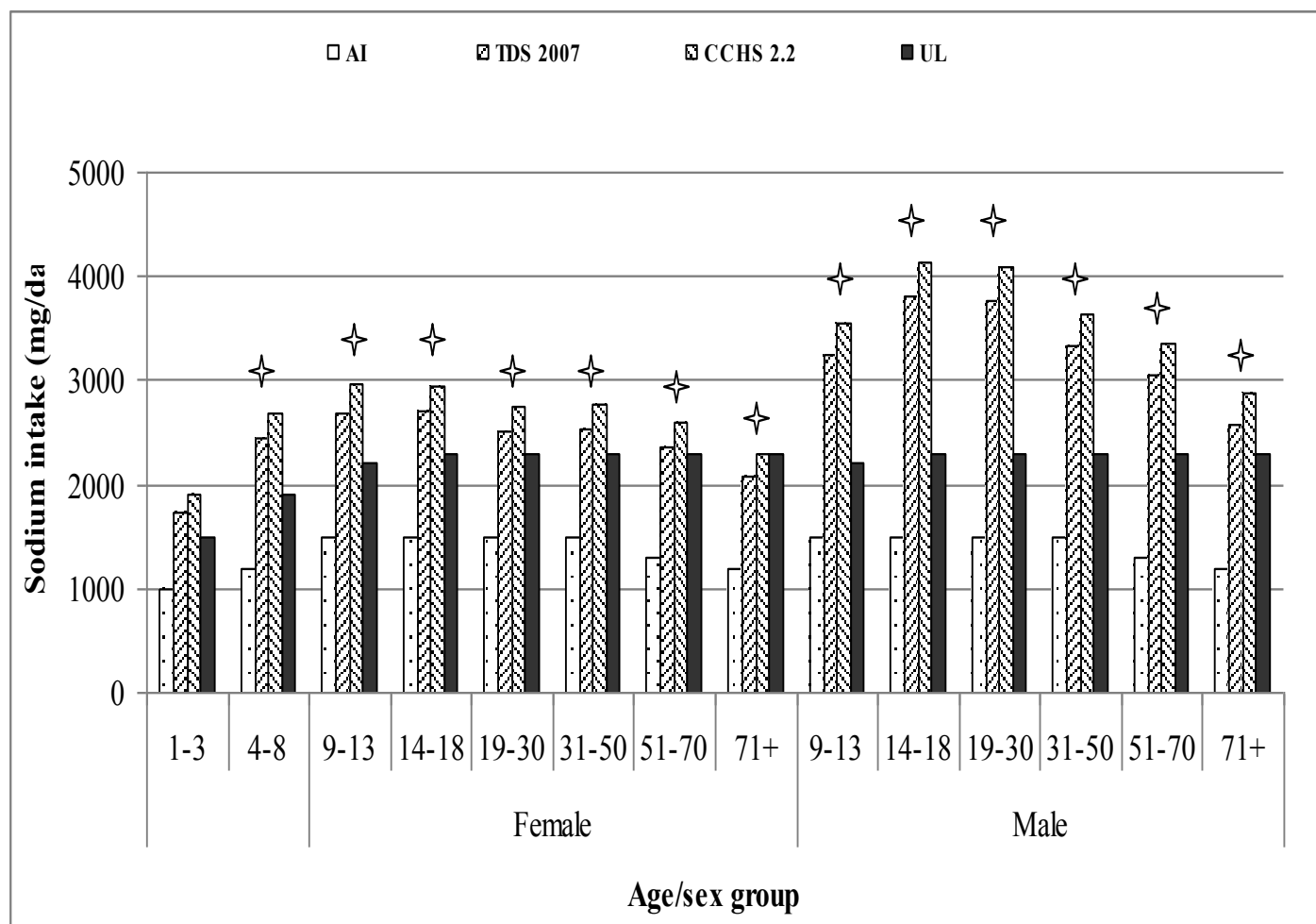


Figure 2: Mean potassium intakes of Canadians, by demographic group, based on analyses in the Canadian Total Diet Study 2007 (Vancouver)

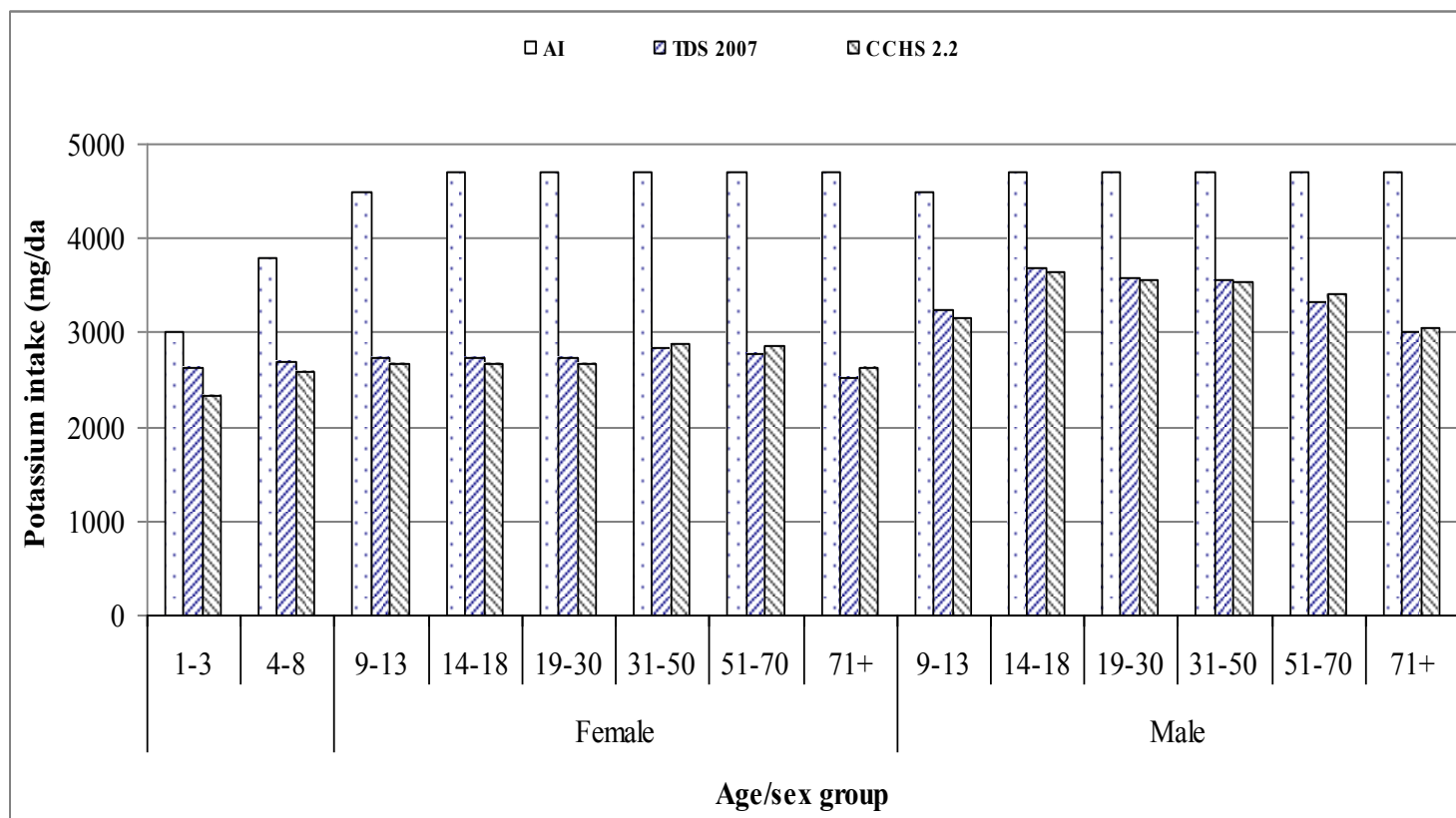


Table 7: Percentile estimates of sodium intakes by age and sex groups, based on analyses in the Canadian Total Diet Study 2007 (Vancouver)

Sodium (mg) adjusted one day intake data from Total Diet Study results using CCHS 2.2 data when there were no TDS data																								
Sex	Years	N	Mean	(SE)	5th	(SE)	10th	(SE)	25th	(SE)	50th	(SE)	75th	(SE)	90th	(SE)	95th	(SE)	AI	%>AI	(SE)	UL	%>UL	(SE)
Both	1-3	2114	1731	36	983	51	1126	48	1388	45	1725	46	2121	55	2528	75	2796	92	1000	94.5	1.5	1500	66.9	3.4
Both	4-8	3235	2437	38	1725	84	1864	73	2117	55	2428	47	2779	71	3134	114	3367	147	1200	100	0.1	1900	88.3	3.3
Female	9-13	1980	2677	58	1664	70	1847	66	2180	60	2601	63	3098	82	3631	120	3997	154	1500	97.7	0.9	2200	73.9	3.3
Female	14-18	2256	2711	47	1773	77	1961	73	2307	64	2741	62	3228	78	3713	109	4028	136	1500	98.7	0.6	2300	75.4	3.4
Female	19-30	1854	2513	64	1689	126	1836	111	2100	87	2421	76	2774	101	3121	150	3344	187	1500	98.4	1.2	2300	59.7	6.7
Female	31-50	2686	2522	49	1460	67	1652	64	2010	60	2473	61	3022	78	3603	111	3996	141	1500	94.1	1.5	2300	59.4	3.3
Female	51-70	3200	2364	42	1467	68	1628	64	1929	56	2321	51	2785	64	3272	97	3602	129	1300	98	0.8	2300	51.3	3.3
Female	71+	2610	2074	41	1230	48	1377	47	1652	46	2004	50	2417	64	2857	89	3159	113	1200	95.8	1.1	2300	31	3.3
Male	9-13	2080	3247	64	2135	95	2333	88	2704	78	3187	74	3755	91	4355	134	4765	175	1500	99.9	0.1	2200	93.6	2
Male	14-18	2288	3816	80	2401	122	2690	114	3224	102	3888	98	4633	122	5394	176	5908	224	1500	99.9	0.1	2300	96.2	1.3
Male	19-30	1804	3758	110	2450	180	2696	164	3150	139	3728	135	4396	182	5082	273	5536	347	1500	100	0.1	2300	97	1.7
Male	31-50	2596	3336	73	1882	123	2150	113	2645	98	3281	89	4022	109	4797	161	5315	204	1500	98.7	0.6	2300	86.2	2.9
Male	51-70	2550	3045	59	1728	83	1958	77	2386	69	2948	68	3596	88	4302	143	4799	194	1300	99.1	0.4	2300	78.5	2.7
Male	71+	1520	2586	59	1533	78	1722	76	2073	74	2532	78	3071	96	3632	131	4012	161	1200	99.1	0.4	2300	62.8	4.1

Table 8: Percentile estimates of potassium intakes by age and sex groups, based on analyses in the Canadian Total Diet Study 2007 (Vancouver)

Potassium (mg) adjusted one day intake data from Total Diet Study results using CCHS data when there were no TDS data																					
Sex	Years	N	Mean	(SE)	5th	(SE)	10th	(SE)	25th	(SE)	50th	(SE)	75th	(SE)	90th	(SE)	95th	(SE)	AI	%>AI	(SE)
Both	1-3	2114	2619	42	1367	52	1598	52	2040	50	2576	51	3191	62	3878	87	4355	111	3000	31.5	2.1
Both	4-8	3235	2684	37	1599	51	1801	45	2165	41	2633	43	3176	58	3745	84	4129	106	3800	9.1	1.4
Female	9-13	1980	2739	54	1529	53	1739	50	2135	48	2644	55	3234	74	3839	101	4236	123	4500	3.1	0.8
Female	14-18	2256	2730	44	1496	52	1715	50	2131	50	2665	53	3308	67	4004	93	4465	117	4700	3.4	0.7
Female	19-30	1854	2728	54	1596	68	1800	65	2176	61	2657	63	3218	78	3798	107	4184	133	4700	1.9	0.6
Female	31-50	2686	2842	39	1568	48	1798	47	2238	44	2772	49	3381	61	4025	89	4473	123	4700	3.5	0.8
Female	51-70	3200	2786	38	1604	54	1829	50	2230	45	2730	44	3306	54	3903	72	4299	89	4700	2.3	0.5
Female	71+	2610	2525	35	1436	48	1635	47	2002	46	2465	46	3005	51	3578	65	3968	79	4700	1.2	0.3
Male	9-13	2080	3239	62	1881	76	2118	74	2571	69	3150	71	3830	88	4568	122	5076	152	4500	10.9	1.7
Male	14-18	2288	3696	74	2058	87	2375	83	2956	77	3692	85	4567	111	5528	159	6205	205	4700	22.2	2.3
Male	19-30	1804	3580	77	2022	97	2294	94	2809	89	3481	91	4270	118	5096	165	5655	208	4700	15.8	2.5
Male	31-50	2596	3550	58	2040	76	2320	73	2835	66	3490	68	4242	86	5038	120	5578	149	4700	15	1.8
Male	51-70	2550	3322	49	1934	61	2191	57	2656	53	3225	59	3886	74	4595	102	5088	128	4700	8.7	1.3
Male	71+	1520	3008	89	1591	83	1834	82	2289	79	2890	87	3604	112	4368	163	4919	205	4700	6.6	1.6

Table 9: Sources of sodium intake, by food category and demographic group, based on analyses in the Canadian Total Diet Study 2007 (Vancouver)

[illegible]

Table 10: Sources of potassium intake, by food category and demographic group, based on analyses in the Canadian Total Diet Study 2007 (Vancouver)

[illegible]

PART IV - DISCUSSION

Hypertension and CVD are medical conditions of concern to health organisations worldwide. High Na intake is linked to their onset. At the same time, abundant scientific data supports the positive correlation between lowered Na intake and reduction of hypertension and CVD (He and MacGregor 2009). Also, the hypotensive effect of K and the beneficial effect of Na/K ratio seem to be gaining in attention. However, due to the current Na and K content of foods as well as population food choices, the current Na and K intakes in the population are well above the UL and well below the AI, respectively. This situation is being addressed by various governmental health organisations through programs targeted towards the reduction of salt intake and to increasing fruits and vegetables consumption (World Health Organization 2002). The overall effect of diet on population health can be judged through large epidemiological studies, while the effectiveness of government regulation implementation and food industry compliance with government regulations can be most easily assessed by studying the Na and K food content and intake modelling of those nutrients. The latter approach was the focus of this thesis.

Description of the process

The desire to analyze the Na and K content of food composite samples from a recent collection (2007) of the Canadian Total Diet Study (TDS) in order to model intake distributions by age and sex group for the Canadian population prompted this research. The TDS is a food monitoring program run by Health Canada's Food Directorate that has been dedicated to monitoring the level of contaminants (since 1969) and the level of contaminants and nutrient intake levels (since 2000) in the Canadian food supply. The analyses are performed on food samples collected from across Canada from at least four different stores within a city per year by inspectors of the Canadian Food Inspection Agency (CFIA). As part of this ongoing monitoring process, the food samples from the

Total Diet Study - 2007 Vancouver, represented by 154 food composites from 930 foods, were chosen for analyses for this thesis.

Following the laboratory analyses of the TDS samples a process of statistical analyses that matched, by weight and energy, the TDS sample ID with the foods consumed within the CCHS 2.2-Nutrition Survey was undertaken. This process led to a high matching percent (86.0% by weight and 75.2% match for energy). Using SIDE and bootstrap methodology, the analytical results of the matched TDS samples were used to generate intakes distributions of Na and K of Canadians. In this process, where the matching was not possible, the existing CCHS2.2 values were incorporated.

The results of these analytical and statistical analyses are presented within this thesis in the form of two manuscripts: paper one presents the food composites and food groups Na and K concentrations; paper 2 presents the Na and K intake modeling based on those food composition data and compares the results to intake modelling based on CCHS 2.2 merged with CNF data. This paper also presents the list of the top Na and K food contributors. The attached appendix presents the results of a comparison between TDS 2007 Na and K composition values and CNF values (see Fig 3 and 4 in appendix).

The main findings of the analyses of the TDS food sample collection (2007 Vancouver) coupled with the modelling exercise with CCHS 2.2-Nutriton data are:

- The breads and cereals and processed foods groups contributed the most to Na intake of the total population, while the milk group and fruits group contributed the most to K intake of the total population.
- Merging TDS Na and K food content with CCHS 2.2-Nutrition 24-hour recall data resulted in calculated sodium intakes in the population that were statistically significantly lower than those originally reported from CCHS 2.2-Nutrition which had utilized Na and K values from the CNF. Using either methodology, the K intake values did not vary significantly. This suggests that Na intakes of Canadians generally declined in 2007 compared to earlier estimates.

Findings

Paper 1 Sodium and potassium in food composites samples

Results from this study suggested that the food composites providing the most dietary Na were represented by soups, fast foods, cured pork and baked beans. The hierarchy of food groups with the highest Na content per reference amount was represented by: soups and fast foods, followed by the meat fish and poultry group, and by the breads and cereal group. Processed foods generally had the greatest Na content. The foods with the highest K content were milk, potato chips, baked beans, potatoes with skins, citrus juice, and veal. The food groups with the highest K content were milk and dairy products, fruits groups, meat, poultry and fish group.

Comparison to TDS in other countries

The New Zealand TDS (2003–4 NZTDS) included 110 foods representing approximately 70% of the most commonly consumed foods in New Zealand. The samples were prepared as ‘table-ready’ and analysed by Inductively Coupled Plasma-Optical Emission Spectroscopy. The highest Na concentration was calculated in processed foods while the unprocessed counterparts had a much lower Na content (Thomson et al 2008). Also, a comparative analyses of the mean sodium concentration (mg/kg) of some processed foods analysed and reported in the NZTDS and Australian and United States Department of Agriculture food composition tables have shown that bacon, corned beef, sausages and cheese, tomato sauce have the highest Na content (Thomson 2009). K content of foods in New Zealand was not investigated, but is proposed as a new entry in the 2009 New Zealand TDS (New Zealand Food Safety Authority (NZFSA) 2009).

The ongoing American Market basket study (1991-2005) identified pork bacon pan-cooked, American processed cheese, salami sliced and tomato catsup as having the highest Na content. In regards to K, sweet potatoes fresh, potatoes chips, peanut butter, pork chop pan cooked were found to be the foods with the highest content (U.S Food and Drug Administration, 2007).

Reports from TDS in some other countries have not included Na and K analytical results, reporting instead only derived intakes (Leblanc et al 2005, Turrini and Lombardi-Boccia 2002).

As can be seen, even with methodological differences, formulations of the market study and different analytical methods for nutrient analyses, this TDS studies identified in common with those of other countries that the processed foods had the highest Na content while fruits and legumes had the highest K content. However, the identity of the foods that have been incorporated in a food composite and their percent contribution as well as the processing technology is seldom reported; therefore, the similarity of a food composite between TDS in different countries can only be assumed and the comparison of the data done with some caution. Also, due to different national eating habits, in various countries the foods with highest Na and K content do not become the highest contributor to the nutrient intake. Except for the Italian TDS, the approach of investigating not just Na, but also K and the Na/ K ratio has not been very frequent.

Paper 2 Sodium and potassium intakes estimated from Canadian Total Diet Study 2007: Vancouver

Although Canadians' Na intake have been observed to be higher than both the AI and UL (Institute of Medicine 2005a), the results from the TDS 2007 food samples combined with CCHS2.2 food intake data identified lower Na intakes than previously shown. This is a difference that is statistically significant for all age/sex groups except 1-3 years old. This lower Na intake could be seen as a positive sign of a reduction of Na content of foods from the Vancouver 2007 area and is encouraging for continuing the monitoring of the sodium food content in order to make a distinction between a real negative trend or variation associated with that one year.

Across all of the age and sex groups, our results showed that the major contributors to sodium intake were breads and cereals (25.9%), soups and fast foods (19.1%) and milk and dairy products (16.8%). In other words, the foods with moderate Na content but frequently eaten and the foods with high Na content but less frequently consumed topped the food contributors list. Also from the detailed analyses of the current data, it was noticeable across all food groups that foods that were subject to processing had higher sodium content than the raw material they originated from.

In regards to potassium intakes, TDS food samples showed across all the age and sex groups a lower intake than the AI. K intake from TDS 2007 was slightly higher than ones from CCHS2.2 in some groups and lower in others (e.g. female older than 31 and

male older than 51, respectively); however, the differences were not statistically significant. The major contributors to K intakes appear to be milk and dairy products, fruit (including juices), green leafy vegetables, and beverages (mostly coffee and tea).

Comparison to TDS in other countries

In the French TDS, the food groups including bread-rusk, and prepared dishes were considered the foods contributing most (respectively 20-40% and 10%) to the Na exposure of the populations; other foods (meat products, fishing products, cheeses) contributed less than 6% of the total food exposure. Potassium intake was not investigated in this study (Leblanc et al 2005). A later study classified breads, soups, cooked pork meats, convenience foods as the main contributors to sodium intake, with vegetables, dairy products, and meats being the main contributors to potassium intake (Meneton et al, 2009)

In the New Zealand TDS, grain products collectively accounted for 33-48 % of Na exposure followed by processed meats (bacon, ham, corned beef and sausages) that contributed 10-14% of total Na exposure (Thomson et al 2008).

In the Italian TDS, the main sources of Na in the Italian total diet were the cereals (18%), meat-and-meat-products food groups (13 %), and milk-and-dairy (7%) and fish groups (6%). Main K contributors were vegetables (26 %), meat and meat products (19 %) and the fruits and cereals (10% each). When evaluated against Italian RDA, in general, the dietary patterns were considered consistent with dietary recommendations (Lombardi-Boccia et al 2003).

Comparison to Canadian data

In a recent study aiming to provide information on the current sources of dietary sodium in the Canadian food supply, the data were obtained through the merging of CCHS 2.2 data with the CNF data. The intake results, presented as 4 age/sex groups (1-8, 9-18, >19 males, >19 females) identified the main food contributors as being the breads, processed meats and pasta dishes (Fischer et al 2009). In our study we modelled the intake of Na by Canadians by using the same CCHS 2.2 food intake data but merged this with Na values generated from analyses of the recent Canadian TDS collection

(Vancouver 2007). In using CNF data, one should be aware that the data represents only one value (mean) without standard errors. The data in the CNF were generated from analyses of mostly American foods whose recipes or detailed descriptions are sometimes not available or not complete. Since our data, based on analyses of Canadian food samples, showed a statistically different lower Na intake than the one reported by CCHS 2.2, we recommend continuing the analyses of Canadian TDS food samples for verifying the descending trend in Na intake indicated by our study. While the study by Fischer et al included 4 age/sex groups, the data from this study was segregated into 16 age/sex groups. The advantage of looking at more groups is gaining a better understanding of the intake patterns and main Na contributors and therefore, increasing the precision of salt reduction recommendations once they are targeted to specific age and sex groups.

Na/K ratio

With respect to the Na and K ratio, our study showed that high levels of Na and high levels of K were generally found in different foods. These data are supported by a French cross-sectional dietary survey that used 7-day food records to show that the highly processed foods (cheeses, cooked pork meats, pastries and sugary products, breakfast cereals, breads, soups and fast foods) had the highest sodium/potassium intake ratios while less processed or raw foods (fruits, hot beverages, meats, vegetables and dairy products) displayed the lowest ratios (Meneton 2009).

This nutrient concentration segregation suggested that judicious food choices are possible to be done by an informed consumer. Also, the results of our study re-iterate the idea that processed foods are sources of Na and vegetables and fruits are sources of K. Na intake reduction in the population will depend to a large extent on the food industry's effort to reduce sodium added during food processing, while increasing K intake in the population seems to be more a matter of educating the consumer towards healthier food choices (consuming more fruits and vegetables). Consumer education and food industry efforts should be combined to have the greatest impact on reducing Na and increasing K intakes in the population.

Strengths

- The Canadian TDS is a convenient source of food samples and, as it is used by several laboratories for the analyses of a wide range of chemicals and nutrients, it makes efficient use of the resources involved in the collection of foods. This type of sampling protocol has been used for many years in other jurisdictions.
- Nutrient intake is routinely established based on food intake combined with the nutrient content of that food. This process presents two major sources of errors: one generated from the nutrient content of a food (data can be old, imported, not updated to the market changes); the second source of error occurs due to errors that are inherent to any food recall method; this sub- or over- reporting of foods is carried on into nutrient intake estimation. Therefore, through continuous analyses of currently available foods on the market foods, one source of error is eliminated.
- The Canadian Total Diet study is one of the very few research initiatives that reports both the Na and K content of foods.
- Analysing foods “as for consumption”, with no salt added during cooking, resulted in estimation of sodium content of foods and sodium intake in the Canadian population based on salt added to a product by the food industry; eliminating the variability of household added salt, a source of variation in overall sodium content of foods and in data interpretation. Also, in analysing the data in this manner, the results are comparable to other TDS studies performed in other countries.
- The analytical method is available and feasible, giving accurate and precise results.
- The analysis of Na and K from foods could be an efficient method for monitoring changes in the food supply and could contribute to monitoring of a salt reduction policy implementation.
- The process of matching of TDS samples to CCHS 2.2 sample ID demonstrated an 86.0% by weight and 75.2% match for energy indicating that the majority of foods could be represented by those analyzed in this thesis, which provides many new values for these nutrients.

Limitations

- In the context of a diverse, vast and very dynamic food supply it is expected that not all the foods available at a certain time and location can be accurately sampled or represented by grouping some related foods into one food composite, nor that all foods on the market can be analysed. A better option than grouping foods may be analyses of core foods. This implies analyses of individual, prototypical foods, but this strategy is associated with an increased cost and labour, still holds the inconvenience of not taking into account regional and cultural differences in food consumption, and is susceptible to non-representativeness of the variation of an individual market food (Pennington, 2000). The analysis of grouped foods as is done in the TDS is considered a convenience strategy.
- In composing TDS foods composites, foods with various sodium contents (e.g., cheddar:mozzarella:partially skimmed mozzarella (2:1:1)) were combined. However, to compensate for this variation the participation ratio of each component was taken into account when performing the comparison with the CNF data. This means that the CNF value for Na or K concentration of a specific composite has been calculated by weighted average (i.e. according to the proportion of each food component).
- While the Reference Amount of a food can be used as a proxy for serving size, it may not accurately reflect the amount of food that an individual eats at one occasion. Therefore, due caution should be used when applying the analytical results to real life situations.
- The TDS food sample analysed in the current study originated from one city and one year and therefore only analytical variation can be reported from the analyses. In order to obtain sample variation, a repeated TDS Vancouver food collection would be needed.

Contributions

- This study presents a new approach and design that has rarely been used (i.e. to include reporting of both Na and K and comparisons of their intakes). Thus far, the physiological association between the Na /K ratio and health effects has been largely ignored in this type of study.
- This research represents the start of a series of TDS studies aimed at monitoring Na and K trends in the Canadian food supply. The next analyses will also begin to allow for assessment of within product variation.
- This research could help refine the TDS sampling protocols by providing information to develop more homogeneous composites in future sampling.
- By presenting some of the CNF liabilities, an increased awareness among the CNF users regarding the appropriate use of CNF data is sought.
- The current findings reinforce dietary recommendations (e.g. From Canada's Food Guide) that emphasize a lower sodium intake through decreasing consumption of processed foods and higher potassium intake through consumption of more fruits and vegetables.

Future directions

- A new and interesting research area is represented by the dietary Na/K ratio and its effect on health. A new project should investigate and bring new information on this promising topic.
- Following this first study, a later replica of the Vancouver 2007 will be necessary in order to investigate Na and K foods content and intake trends over time. Those data could be correlated with data from duplicate TDS collections from other cities across Canada for investigating the role of location in Na content of Canadian foods.
- For the identified main Na and K contributors, future analyses could be done on individual foods in order to avoid highly unusual composites and to obtain direct Na and/or K concentration of specific foods.

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APPENDIX 1

Comparison between Na and K concentrations in TDS 2007 vs. CNF foods

Introduction

The purpose of this section is to present the ratio between TDS 2007 vs. CNF foods for Na and K concentrations.

Methods

The ratio was obtained by dividing the TDS calculated value by the CNF reported value. Where the composition of foods from TDS and CNF was identical, the ratio was directly calculated. However, TDS includes some food composites that have been obtained through mixing in different proportions of 2 or more foods (e.g., the Cheese composite contains cheddar (sharp or mild), mozzarella (25% MF) and partially skimmed mozzarella (17% MF) in a ratio of 2:1:1; Fish marine contains frozen haddock, cod and sole (1:1:1)). In such instances, the CNF value for Na or K concentration has been calculated by weighted average (i.e. according to the proportion of each food component). The TDS calculated value was then divided by CNF value to generate TDS/CNF ratio.

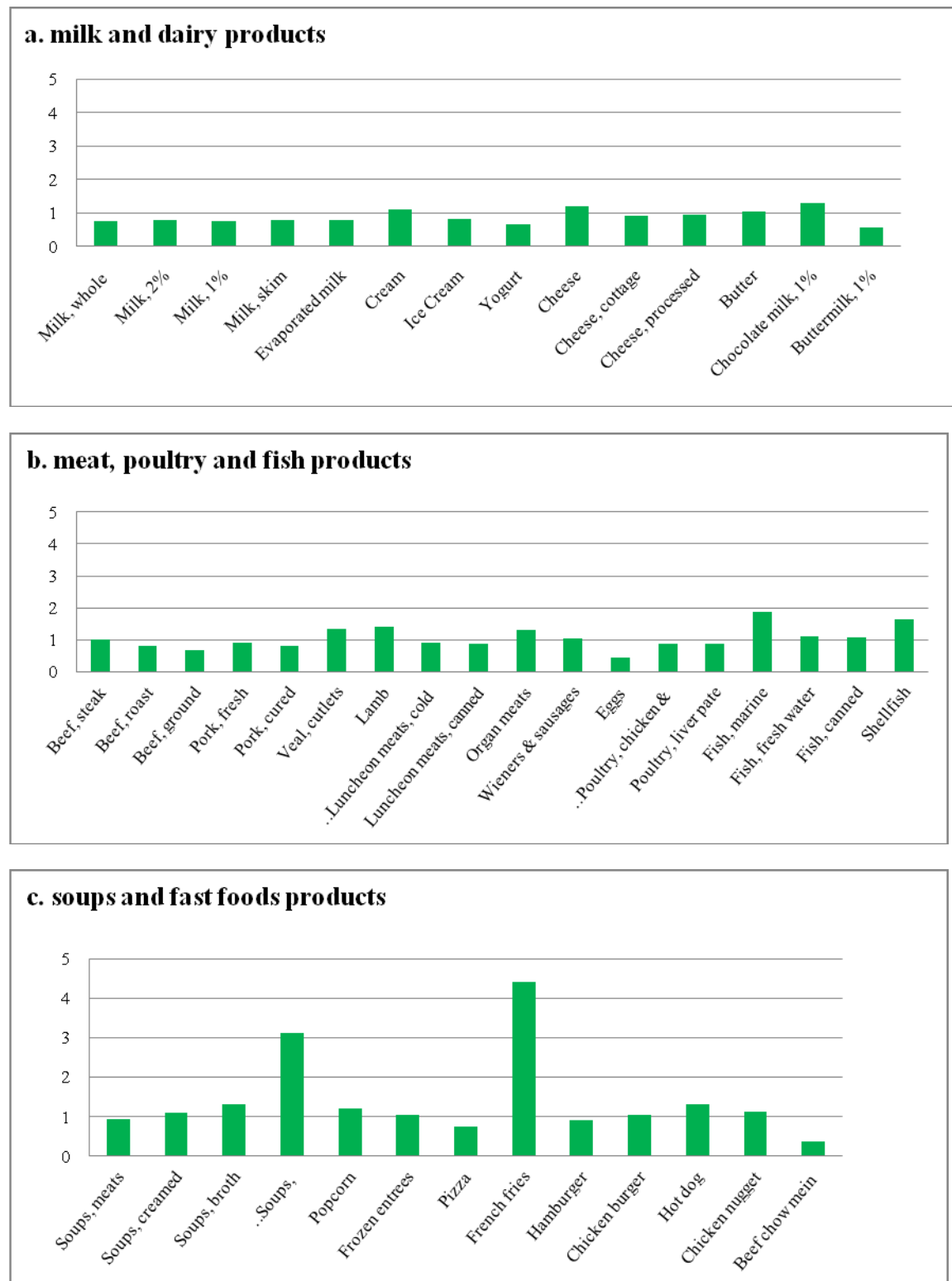
Results and Discussion

Overall, in case of both Na and K, most of the food samples' ratio averaged around 1 with various degrees of departure in case of some individual food composites (Figure 3 and 4). The statistical significance of the differences could not be calculated from two reasons: 1) the TDS Vancouver food composites were single, unreplicated samples (although each was analysed in triplicate, as reported in paper 1) which didn't allow for evaluation of the sample variability. This limitation can be addressed in the future by replication of the sampling of Vancouver market foods; 2) CNF data include only the mean of a nutrient content, with no variation term reported.

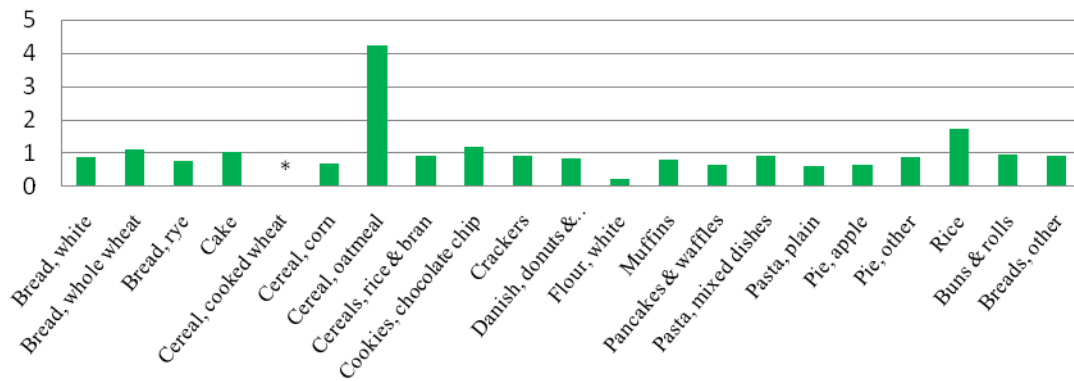
Continuing TDS sampling for nutrient content analyses (including Na and K) would contribute to understanding the variation in nutrient data and to the evaluation of trends in both food content and intakes of Na and K by Canadians.

Figure 3: Sodium ratio of food composite samples from TDS2007 vs. CNF, by food composite group. *, value listed in CNF was zero or “trace”, no calculation possible.

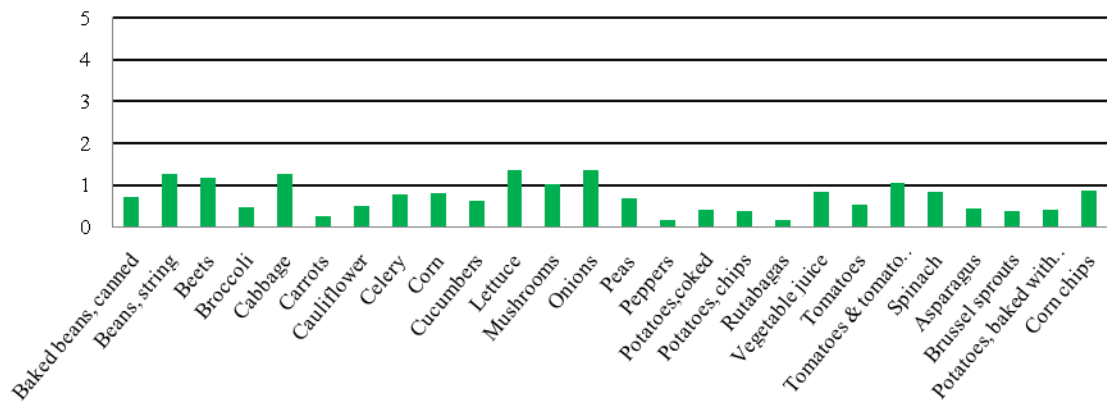
<, analytical result from TDS 2007 was below the detection limit for the element



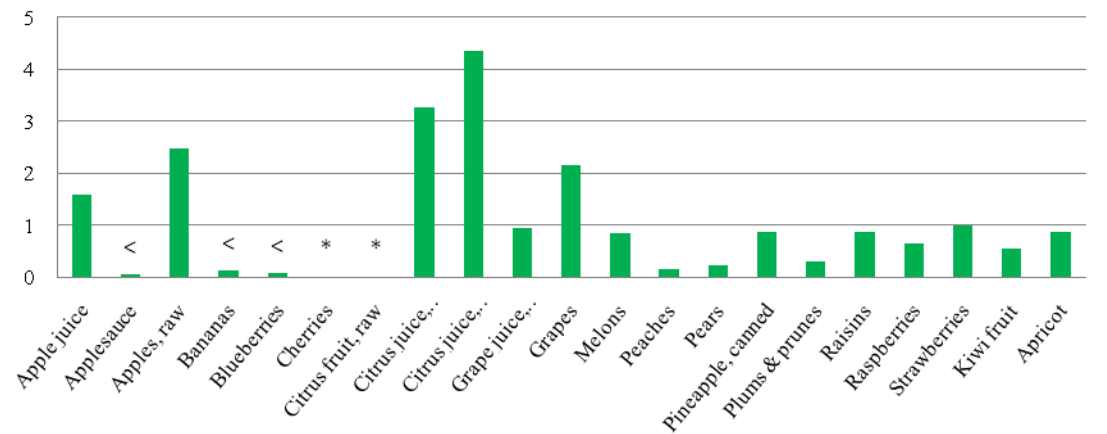
d. breads and cereal products



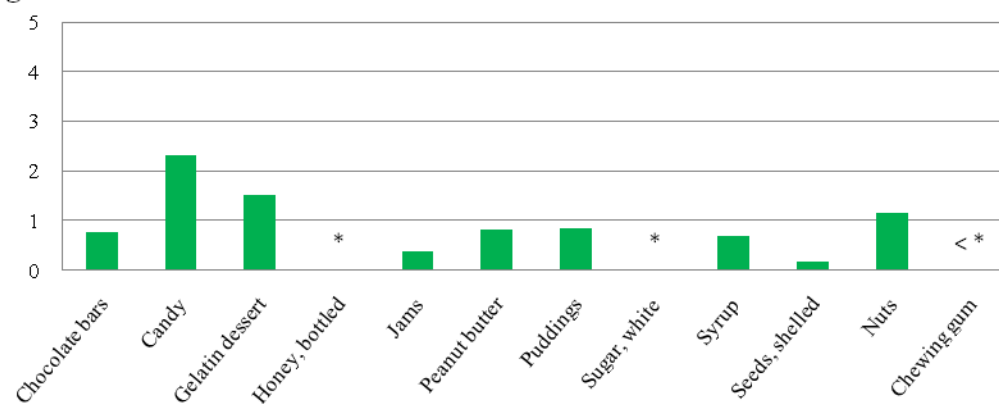
e. vegetables



f. fruits



g. snacks



h. ingredients

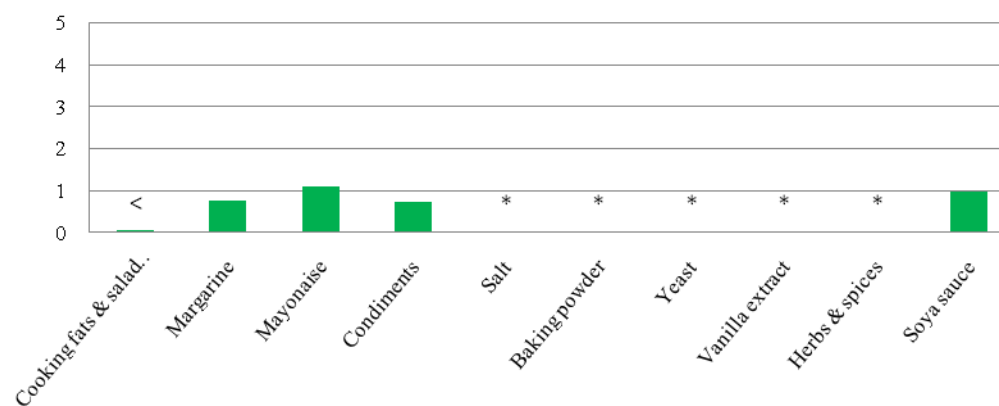
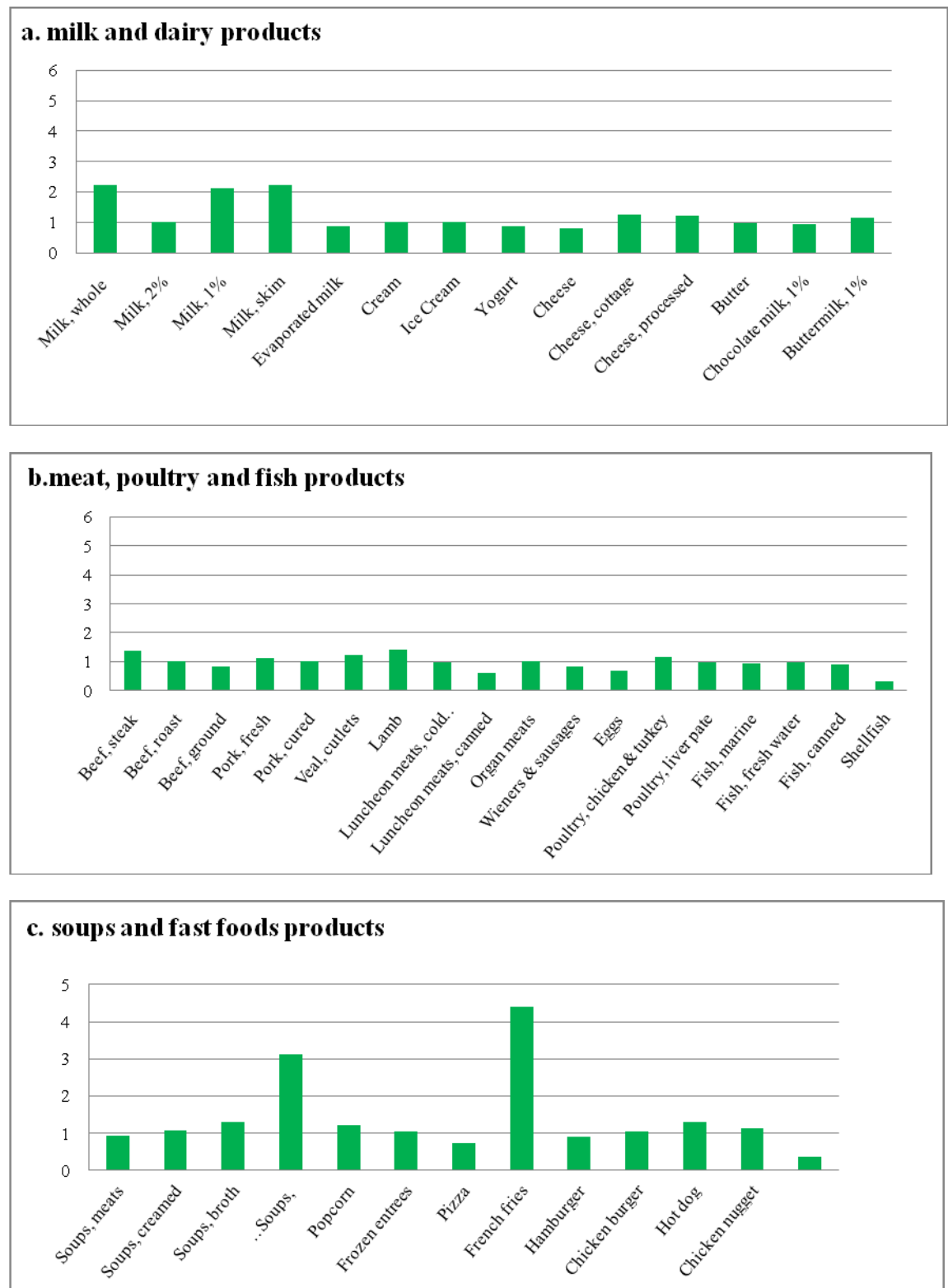
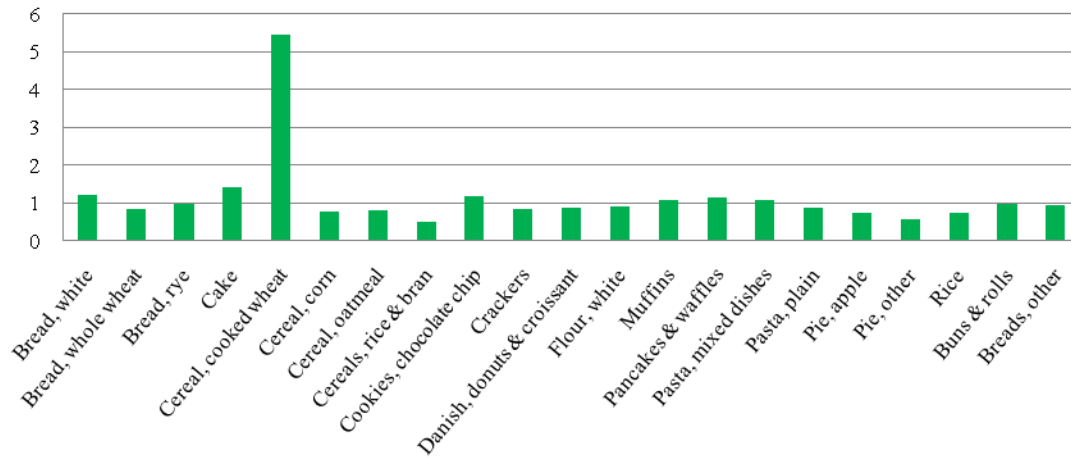


Figure 4: Potassium ratio of food composites from TDS2007 vs. CNF, by food composite group. *, value listed in CNF was zero or “trace”, no calculation possible.

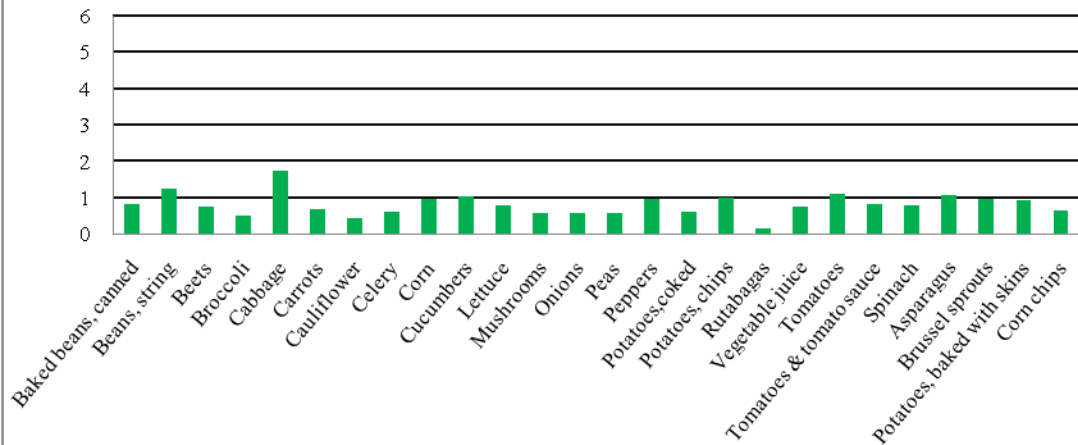
<, analytical result from TDS 2007 was below the detection limit for the element



d.breads and cereal products



e.vegetables



f.fruits

