Use of Spot Urine Samples in Estimation of Daily Sodium Intake in Patients with Chronic Kidney Disease

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List of Abbreviations

ACE	**Angiotensin converting enzyme**
ANOVA	**Analysis of variance**
ANP	**Atrial natriuretic peptide**
BMI	**Body Mass Index**
BNP	**Brain natriuretic peptide**
BP	**Blood pressure**
CKD	**Chronic Kidney Disease**
CVD	**Cardiovascular disease**
DBP	**Diastolic Blood Pressure**
EABV	**Effective arterial blood volume**
ECF	**Extracellular fluid**
ESC	**European Society of Cardiology**
ESRD	**End Stage Renal Disease**
FAO	**Food and Agriculture Organization**
FFQ	**Food Frequency Questionnaire**
GFR	**Glomerular filtration rate**
KDIGO	**Kidney Disease Improving Global Outcome**
LVH	**Left ventricular hypertrophy**
MBP	**Mean arterial Blood Pressure**
NCD	**Non-communicable Diseases**

NICE	**National Institute for Health and Clinical Excellence**
PASW	**Predictive Analytics SoftWare**
SBP	**Systolic Blood Pressure**
SMU	**Second morning voiding urine**
TOHP	**The Trial of Hypertension Prevention**
TONE	**The Trial of Nonpharmacologic Interventions in The Elderly**
WHO	**World Health Organization**

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Introduction

Sodium intake is an important issue for patients with chronic kidney disease (CKD). These patients are characterized by hypertension, which is thought to be predominantly salt sensitive **(Weir and Fink, 2005).**

High blood pressure carries a greater risk for cardiovascular accidents across the entire spectrum of chronic kidney disease **(De Nicola et al., 2004).**

It is increasingly apparent that individuals with chronic kidney disease (CKD) are more likely to die of cardiovascular accidents than to develop kidney failure **(Sarnak et al., 2003).**

There is also increasing evidence that high sodium intake may lead directly to the progression of renal failure **(Sanders, 2004)**, by increasing albuminuria and filtration fraction **(Jones-Burton et al., 2006).**

Moreover, increased salt intake also attenuates the antihypertensive effects of most antihypertensive drugs **(Weir and Fink, 2005).**

Almost 85% of patients with CKD under regular nephrologic care have been found to consume sodium above the recommended level **(Kutlugün et al., 2011).**

Salt restriction was proven effective in hypertension management for CKD patients especially in those with estimated daily sodium intake > 150 mmol/day. Reduction in sodium intake beyond 20 mmol/day reduced both BP and proteinuria significantly **(Koh et al., 2011).**

Consequently, salt intake must be considered a potential modifiable risk factor for the progression of kidney disease **(Weir and Fink, 2005).**

Although, the estimation of salt intake is essential, there are no easy methods to estimate real salt intake. The two most widely used methods to measure sodium intake are: 24-hour urinary sodium excretion measurement and sodium intake estimation by dietary recall.

The use of a spot urine method, or a brief timed collection, for assessing sodium excretion has previously been examined **(Kamata and Tochikubo, 2002).**

Kawasaki et al. reported a method of estimating 24-hour urinary sodium (Na) and potassium (K) excretion from second

morning voiding urine (SMU) in adults **(Kawasaki et al., 1993).**

In 2002, *Tanaka et al.* developed a simple method to estimate 24-hour urinary sodium excretion from spot urine specimens collected at any time, using 591 Japanese data items from the INTERSALT study **(Tanaka et al., 2002).**

However, there has been very little research to test the validity of these equations in evaluation of 24-hour urinary sodium in patients with chronic kidney disease.

Aim of the Work

Is to evaluate the use of spot urinary sodium concentrations to estimate daily sodium intake in patients with CKD.

Regulation of sodium and water homeostasis is one of the major functions of the kidney. Renal sodium excretion is the primary determinant of sodium homeostasis **(Simpson, 1988)**.

Sodium and Extracellular fluid homeostasis

Since sodium is a main cation of extracellular fluid (ECF), changes in sodium concentration are linked to changes in ECF volume and are associated with disorders of water balance **(Schrier, 2007).**

The sodium content of a normal adult is 55–65 mmol per kg body weight. The concentration of sodium in plasma is approximately 140 mmol/L (~152 mmol/kg). Under physiological conditions, the control of the ECF volume is through the control of functioning or *effective* arterial blood volume (EABV). Although the term "effective arterial blood volume" has been used in medical literature for decades, its definition remains unclear **(Schrier, 1990).**

Peters referred to "effective blood volume" as that portion of the total circulating volume that is "somehow sensed," and thus responds to too little or too much volume **(Peters, 1948).**

In an underfilled body fluid compartment, there must be signals, coming from extra-renal locations promoting retention