Salt and obesity: a systematic review and meta-analysis of observational studies

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ABSTRACT

Background: Existing research has investigated the association between sodium intake and obesity. We aim to conduct a systematic review and meta-analysis of reported evidence regarding the association between sodium intake and obesity.

Methods: Multiple electronic databases (PubMed, Scopus, and Google Scholar) were searched for observational studies published until August 2016. A systematic literature review identified 11 cohort and 21 cross-sectional studies.

Result: Among the 32 studies identified in the systematic literature search, only 18 cross-sectional reports had sufficient data to be included in the meta-analysis. Higher sodium consumption was associated with greater BMI (weighted mean difference (WMD) = 1.24 kg/m², 95%CI: 0.80, 1.67; I² = 98.4%; p < .0001), and higher sodium intake was associated with 4.75 cm (95%CI: 3.25, 6.25; 90.8%; p < .0001) greater waist circumference (WC).

Conclusion: The present meta-analysis suggests that sodium consumption was associated with greater BMI and WC.

Introduction

Obesity has become a leading global health concern over the past three decades (Wang & Lobstein 2006). Lifestyle factors including unhealthy dietary intake and sedentary lifestyles have been associated with increased risk of obesity (Fonseca-Alaniz et al. 2007). From the time of hunters and gatherers to the year 2012, considerable increases in sodium intake (from <1.0 g/d to 9–12 g/d) have been reported (US Department of Health and Human Services 2001; Brown et al. 2009). In the modern day, ~80% of total sodium intake comes from processed and restaurant food (Brown et al. 2009). Several epidemiological studies have suggested that increased sodium intake might be associated with the rises in obesity prevalence in recent decades (WHO 2014; Commission 2015). Both obesity and high salt consumption have been linked to greater risk of a variety of chronic diseases such as hypertension, diabetes, cardiovascular events, certain cancers and other health conditions (US Department of Health and Human Services 2001; WHO 2002, 2014).

Several explanations may directly link salt consumption and obesity. One hypothesis is that consumption of higher amounts of sugar-sweetened beverages follow salt consumption (He et al. 2008; He & MacGregor 2009). Specifically, higher sodium intake drives a thirst response and promotes fluid intake (Campbell 2004; Stachenfeld 2008), which might result in sugar-sweetened beverage consumption (Grimes et al. 2013a, 2013b). Furthermore, salty foods are often high in fat and energy (Azadbakht & Esmaillzadeh 2008; Rouhani et al. 2012) and are more palatable, encouraging individuals to consume greater quantities of these foods. Therefore, the direct link between sodium and obesity may be mediated by increased energy intake. However, there is evidence supporting an association of salt intake and obesity that is independent of energy intake (Ellison et al. 1980; Libuda et al. 2012; Song et al. 2013; Yoon & Oh 2013). Although such an association has
not been investigated in humans, animal models indicate that a high-sodium diet increases adiposity hypertrophy as well as plasma leptin concentrations that might be attributable to the lipogenic capacity of white adipose tissue (Fonseca-Alaniz et al. 2007). To date, a few research works have studied the association of 24-hour urinary sodium (24-UNa) and obesity (Hoffmann & Cubeddu 2009; Grimes et al. 2013b; Song et al. 2013; Yi & Kansagra 2014; Ma et al. 2015). In a representative sample of adults from New York City, after normalizing the sodium-potassium ratio as a proxy adjustment for energy, higher sodium intake was related to greater odds of obesity (Yi & Kansagra 2014). Another cross-sectional study from the UK National Diet and Nutrition Survey showed that higher salt intake was significantly associated with greater body fat mass in both children and adults, after controlling for energy intake (Ma et al. 2015). In that study, a 1 g/d increase in salt intake increased the risk of obesity by 28% in children and 26% in adults (Ma et al. 2015). Consistent with this, in two other studies, body mass index (BMI) increased proportional to incremental salt intake (Hoffmann & Cubeddu 2009; Grimes et al. 2013b). Nevertheless, the association between sodium and body fat has not been comprehensively examined (Jain et al. 2014; Lee & Kim 2016). A recent cross-sectional study among Korean children (Lee & Kim 2016) and a multiethnic cohort (Jain et al. 2014) showed direct links between sodium and body fat determined by dual-energy X-ray absorptiometry (DEXA).

Given the widespread use of sodium in commercial foods and the magnitude of their contribution to sodium intake, finding a link between salt and obesity could inform health policies with regard to the quantity of salt content permitted in processed and restaurant foods, and thereby improve cardiovascular health. Moreover, in the existing literature there have been considerable methodological differences in sodium intake assessment methods, study populations and design, which may lead to heterogeneity between studies. Therefore, in the present systematic review, we performed a meta-analysis of emerging literature to elucidate the association between salt intake and obesity.

Method and materials

Search strategy

We searched MEDLINE, Google Scholar and Scopus databases for relevant studies published until August 2016. Additional papers were obtained by hand searching of references from relevant original and review articles. Keywords from Medical Subject Headings (MeSH) were selected in the search strategy. The search terms included were equivalents of sodium and salt intake or excretion in the combination with terms for anthropometric measures, including weight, waist circumference (WC), BMI, overweight, obesity, fat mass and adiposity. No restrictions were applied regarding the time of publication, the study design or language. The present meta-analysis is based on PRISMA guidelines.

Inclusion and exclusion criteria

Two reviewers independently checked inclusion and exclusion criteria by reading the titles, abstracts, and then the full text of the articles, if necessary. For two potentially relevant studies with missing data, we contacted the research groups and asked them to provide the necessary information, but only one of them responded. Studies were included if they (i) were observational in design and (ii) reported means and standard deviation (SD) or standard error (SE) or 95% confidence intervals (CI) for anthropometric indices (weight, BMI, WC, waist-to-hip ratio) in low and high categories of salt/sodium intake or urinary sodium. Reviews, editorials, non-human studies, and letters without sufficient data were excluded.

Data extraction

The following information was extracted for each study: first author’s name, publication year, sample size, age of subjects, study design and adjusted confounders. Moreover, means and their corresponding SD or SE or 95%CI of obesity-related indices were extracted from the first and the last categories of salt/sodium. Some studies reported data stratified by gender (Hoffmann & Cubeddu 2009; Murata et al. 2010; Pfister et al. 2014). In this case, several effect sizes were extracted from one paper.

Statistical analysis

Reported SEs were converted to SDs. Since most of the studies had reported mean±SD/SE of BMI and WC, and not many included other obesity indices (e.g. fat mass, weight, and WHR) or odds ratios for obesity risk, the present meta-analysis was conducted on only means of BMI and WC. STATA 11.0 (Stata Corp., College Station, TX) software was used to run the meta-analysis. The pooled effect size was calculated using a random effects model. Heterogeneity and
between-subgroup heterogeneity were evaluated using the $I^2$ statistic and a fixed-effect model, respectively. The contribution of each study on the overall effect was estimated using sensitivity analyses. To detect the publication bias, Begg’s and Egger’s adjusted rank correlation test was run.

**Appraisal of the quality of studies**

The quality of the studies included in the meta-analysis was assessed using the Newcastle–Ottawa Scale modified for cross-sectional studies. A quality score was determined on the basis of three major components: (i) selection of study groups (0–5 points), (ii) adequacy of adjustment for confounding (0–2 points) and (iii) ascertainment of the outcome of interest (0–3 points). High-quality studies were defined as those that scored with at least seven stars on the Newcastle–Ottawa scale. Medium quality received scores of five to six stars.

**Results**

Of 9610 articles retrieved from 1952 to August 2016, 32 studies conducted between 2008 and 2016 were included in this systematic review (Verhave et al. 2004; Hu et al. 2005; Shikata et al. 2006; Radhika et al. 2007; He et al. 2008; Umesawa et al. 2008; Hoffmann & Cubeddu 2009; Jablonski et al. 2009; Visser et al. 2009; Hulthén et al. 2010; Murata et al. 2010; Libuda et al. 2012; Räisänen et al. 2012; Grimes et al. 2013a, 2013b; Larsen et al. 2013; Otsuka et al. 2013; Polonia et al. 2013; Song et al. 2013; Yoon & Oh 2013; Baudrand et al. 2014; Han et al. 2014; Jain et al. 2014; Ogna et al. 2014; Pfister et al. 2014; Zhao et al. 2014; Zhu et al. 2014; Huh et al. 2015; Ma et al. 2015; Ponzo et al. 2015; Yi et al. 2015; Lee & Kim 2016) (Figure 1). However, some of these studies had insufficient data for inclusion in the meta-analysis ($n=13$), or reported sodium density as an independent variable ($n=1$). Therefore, the meta-analysis included 18 cross-sectional reports. Eligible study characteristics are shown in Table 1. Six studies were conducted with children (He et al. 2008; Grimes et al. 2013a, 2013b; Zhu et al. 2014; Ogna et al. 2014; Lee & Kim 2016) and three studies were conducted with both children and adults (Libuda et al. 2012; Yoon & Oh 2013; Ma et al. 2015). Study designs were either cohort (Hu et al. 2005; Shikata et al. 2006; Umesawa et al. 2008; Murata et al. 2010; Libuda et al. 2012; Larsen et al. 2013; Otsuka et al. 2013; Jain et al. 2014; Pfister et al. 2014; Zhao et al. 2014; Lee & Kim 2016) or cross-sectional (Verhave et al. 2004; Radhika et al. 2007; He et al. 2008; Hoffmann & Cubeddu 2009; Jablonski et al. 2009; Visser et al. 2009; Hulthén et al. 2010; Räisänen et al. 2012; Grimes et al. 2013a, 2013b; Polonia et al. 2013; Song et al. 2013; Yoon & Oh 2013; Han et al. 2014; Ogna et al. 2014; Baudrand et al. 2014; Zhu et al. 2014; Huh et al. 2015; Ma et al. 2015; Ponzo et al. 2015; Yi et al. 2015). Three different dietary assessment tools were used: three-day dietary records (He et al. 2008; Jablonski et al. 2009; Libuda et al. 2012; Räisänen et al. 2012), food frequency questionnaires (FFQ) (Shikata et al. 2006; Radhika et al. 2014; Hulthén et al. 2010; Murata et al. 2010; Libuda et al. 2012; Räisänen et al. 2012), or 24-hour dietary recalls (He et al. 2008; Jablonski et al. 2009; Libuda et al. 2012; Räisänen et al. 2012).
| Author (Year)       | Design          | Dietary assessment tool | Country | N of subjects (male: female) | Age (year) | Sodium (lowest: highest category) | Method of calculating sodium | Reported or extracted data | Multivariable adjusted model or crude model | Result                                                                 |
|--------------------|-----------------|-------------------------|---------|-----------------------------|------------|----------------------------------|-----------------------------|---------------------------|----------------------------------------|------------------------------------------------------------------------|
| Huh et al. (2015)  | Cross-sectional | 24h Dietary recall      | Korea   | 3545 (3545/0)              | 45         | (112.28 ± 16.72: 188.49 ± 22.82) | Urinary sodium excretion (mmol/day) | BMI, WC                  | Crude                                  | Urinary sodium excretion was positively associated with both BMI and WC levels |
| Huh et al. (2015)  | Cross-sectional | 24h Dietary recall      | Korea   | 3617 (0/3617)              | 45         | (112.67 ± 17.78: 192.86 ± 22.46) | Urinary sodium excretion (mmol/day) | BMI, WC                  | Crude                                  | Urinary sodium excretion was positively associated with both BMI and WC levels |
| Ma et al. (2015)   | Cross-sectional | Record                  | UK      | 458 (240/218)              | 10         | (3.1 ± 0.8: 8.5 ± 2.2)            | Salt intake (g/day)          | BMI, WC                  | Crude                                  | Salt intake was positively associated with both BMI and WC levels       |
| Ma et al. (2015)   | Cross-sectional | Record                  | UK      | 785 (371:414)              | 49         | (4.3 ± 1.1: 11.5 ± 2.4)           | Salt intake (g/day)          | BMI, WC                  | Crude                                  | Salt intake was positively associated with both BMI and WC levels       |
| Ponzo et al. (2015)| Cross-sectional | 24h Food recall & FFQ   | Italy   | 1200 (NR/NR)               | 12         | (2.0 ± 0.8 : 3.4 ± 0.9)           | Sodium content of snacks (g/day) | BMI, BW                 | Crude                                  | Sodium content of snacks was not significantly associated with BMI but shows it was positively associated with BW levels |
| Ponzo et al. (2015)| Cross-sectional | 24h Food recall & FFQ   | Italy   | 1200 (NR/NR)               | 12         | (2.1 ± 0.7 : 3.4 ± 0.9)           | Daily frequency of salty snacks (g/day) | BMI, BW                 | crude                                 | Daily frequency of salty snacks was not significantly associated with BMI but shows a positive association with BW levels |
| Zhao et al. (2014) | Cross-sectional | By phone call follow-up | Shenyang | 243 (140:103)              | 60         | (<6:6)                            | Dietary salt intake (g/day)  | BMI                      | Crude                                  | Dietary salt intake was positively associated with BMI levels.       |
| Zhao et al. (2014) | Cross-sectional | By phone call follow-up | Shenyang | 243 (140:103)              | 60         | (141.4 ± 2.7: 142.0 ± 2.6)        | Serum sodium (mmol/L)        | BMI                      | Crude                                  | Serum was positively associated with BMI levels                       |
| Pfister et al. (2014)| Cross-sectional | 24h Dietary recall     | England | 9017(male)                 | 59         | (115 ± 17±218 ± 31)              | Urinary sodium excretion (mmol/day): | BMI                      | crude                                  | Urinary sodium excretion was positively associated with BMI levels |
| Pfister et al. (2014)| Cross-sectional | 24h Dietary recall     | England | 10840 (female)             | 59         | (110 ± 17±216 ± 32)              | Urinary sodium excretion (mmol/day): | BMI                      | crude                                  | Urinary sodium excretion was positively associated with BMI levels |

(continued)
| Author (Year) | Design | Dietary assessment tool | Country | N of subjects (male: female) | Age (year) | Sodium (lowest: highest category) | Method of calculating sodium | Reported or extracted data | Multivariable adjusted model or crude model | Result |
|--------------|--------|-------------------------|---------|-----------------------------|-----------|--------------------------------|----------------------------|----------------------------|-------------------------------|--------|
| Han et al. (2014) | Cross-sectional | NR | Korea | 5187 | ≥19 | (82.2 ± 46.54:156.8 ± 46.89) | Urine sodium (mmol/L) | BMI,WC | Age and sex. | Urinary sodium was positively associated with both BMI and WC levels. |
| Baudrand et al. (2014) | Cross-sectional | NR | Santiago | 370 (109:261) | 46.5 | (51-149: >150) | Daily urinary sodium (mEq/24 h) | BMI | Crude | Daily urinary sodium was not significantly associated with BMI levels. |
| Otsuka et al. (2013) | Cross-sectional | FFQ | Japan | 970 (897/730) | 44 | (seldom/always) | Frequency of salty food intake | BMI | Crude | Frequency of salty food intake was positively associated with BMI levels. |
| Polonia et al. (2013) | Cross-sectional | FFQ | Portugal | 154 (89/65) | 46.5 | (126 ± 15.228 ± 21) | 24h Urinary excretion (mmol/24h) | BMI | Crude | 24h Urinary excretion was positively associated with BMI levels. |
| Polonia et al. (2013) | Cross-sectional | FFQ | Portugal | 154 (89/65) | 46.5 | (7.3 ± 0.9:16.70 ± 1.3) | Dietary salt intake (g/day) | BMI | Crude | Dietary salt intake was positively associated with BMI levels. |
| Murata et al. (2010) | Cross-sectional | Self-administered questionnaire on salted food intake | Japan | 3074(male) | 50.8 | (low intake salted food: High intake salted food) | Intake of salted food | BMI | Crude | Intake of salted food was not significantly associated with BMI levels. |
| Murata et al. (2010) | Cross-sectional | Self-administered | Japan | 3756 (female) | 50 | (low intake salted food: High intake salted food) | Intake of salted food | BMI | Crude | Intake of salted food was not significantly associated with BMI levels. |
| Hoffmann and Cubeddu (2009) | Cross-sectional | NR | USA | 251(male) | 44.9 | (< 119.5: >112) | 24h Urinary excretion (mmol/24h) | BMI, WC, BW | Crude | 24h urinary excretion was not significantly associated with BMI levels but showed a positive association with WC and BW. Sodium excretion was positively associated with BMI, WC and BW. Sodium intake was not significantly associated with BMI but showed a positive association with WHR levels. Sodium excretion was not significantly associated with BMI levels but showed a positive association with body weight. |
| Hoffmann and Cubeddu (2009) | Cross-sectional | NR | USA | 515(female) | 44.9 | (< 92.4: > 158) | 24h Urinary excretion (mmol/24h) | BMI, WC, BW | Crude | Urinary sodium excretion was positively associated with BMI, WC and BW. Sodium intake was not significantly associated with BMI but showed a positive association with WHR levels. Sodium excretion was not significantly associated with BMI levels but showed a positive association with body weight. |
| Jablonski et al. (2009) | Cross-sectional | 3-Day food intake records or food-frequency questionnaires | USA | 25 (16:9) | 60.5 | (73 ± 20.74:144 ± 20.76) | Sodium intake (mmol/day) | BMI,WHR | Crude | Sodium intake was not significantly associated with BMI but showed a positive association with WHR levels. Sodium excretion was not significantly associated with BMI levels but showed a positive association with body weight. |
| Visser et al. (2009) | Cross-sectional | NR | The Netherlands | 78 (78:0) | 23 | (38 ± 25: 241 ± 68) | Sodium excretion (mmol/24h) | BMI, body weight | Crude | Sodium excretion was not significantly associated with BMI levels but showed a positive association with body weight. |

(continued)
| Author (Year)          | Design         | Country             | N of subjects (male: female) | Age (year) | Sodium (lowest: highest category) | Method of calculating sodium | Reported or extracted data | Multivariable adjusted model or crude model | Result                                                                 |
|------------------------|----------------|---------------------|-----------------------------|------------|----------------------------------|------------------------------|-----------------------------|---------------------------------------------|------------------------------------------------------------------------|
| Visser et al. (2009)   | Cross-sectional| NR                  | The Netherlands             | 78 (78:0)  | 23                               | Serum [sodium] (mmol/l)      | BMI, body weight             | Crude                         | Serum sodium was not significantly associated with BMI levels but showed a positive association with body weight. Sodium excretion was positively associated with both BMI and body weight. Serum sodium excretion was positively associated with both BMI and body weight. |
| Visser et al. (2009)   | Cross-sectional| NR                  | The Netherlands             | 78 (78:0)  | 23                               | Sodium excretion (mmol/24h)  | BMI, body weight             | Crude                         | Sodium excretion was positively associated with both BMI and body weight. Serum sodium excretion was positively associated with both BMI and body weight. |
| Visser et al. (2009)   | Cross-sectional| NR                  | The Netherlands             | 78 (78:0)  | 23                               | Serum [sodium] (mmol/l)      | BMI, body weight             | Crude                         | Serum sodium excretion was positively associated with both BMI and body weight. Serum sodium excretion was positively associated with both BMI and body weight. |
| Hulthén et al. (2010)  | Cross-sectional| FFQ                 | Sweden                      | 79 (79:0)  | 19                               | Sodium excretion (mmol/24h)  | BMI                          | Crude                         | Urinary sodium excretion was positively associated with BMI levels. Total dietary salt was positively associated with both BMI and WC levels. Total dietary salt was negatively associated with BMI levels. Urinary sodium excretion was positively associated with both BMI levels. |
| Radhika et al. (2007)  | Cross-sectional| FFQ                 | India                       | 1902 (828/1081) | 39.7 (4.9:13.8) | Total dietary salt (g/day) | BMI,WC                        | Crude                         | Total dietary salt was positively associated with both BMI and WC levels. Total dietary salt was negatively associated with BMI levels. Urinary sodium excretion was positively associated with both BMI levels. |
| Shikata et al. (2006)  | Cross-sectional| FFQ                 | Japan                       | 2476 (996/1480) | 57.3 (<10: ≥16) | Dietary salt intake (g/day) | BMI                          | Crude                         | Total dietary salt was negatively associated with BMI levels. Urinary sodium excretion was positively associated with both BMI levels. |
| Hu et al. (2005)       | Cohort/18.1    | Specific questionnaire| Finland                     | 1935 (932:1003) | 49.5 | NR:NR | Urinary sodium excretion (mmol/24h) | BMI                          | Crude                         | Urinary sodium excretion was positively associated with both BMI levels. Total dietary salt was negatively associated with BMI levels. Urinary sodium excretion was positively associated with both BMI levels. |
| Verhave et al. (2004)  | Cross-sectional| Dietary recall      | The Netherlands             | 7850 (3950/3900) | 51.5 | (79.5:220.1) | Urinary sodium excretion (mmol/24h) | BMI, WHR                     | Crude                         | Urinary sodium excretion was positively associated with both BMI and WHR levels. Total dietary salt was negatively associated with BMI levels. Urinary sodium excretion was positively associated with both BMI levels. |

BMI: body mass index; BW: body weight; FFQ: food frequency questionnaire; NR: not reported; SDS: standard deviation score; WC: waist circumference; WHR: waist-to-hip ratio.
Hu et al. 2005; Shikata et al. 2006; Radhika et al. 2007; and two 24-hour dietary recalls (Verhave et al. 2004; Grimes et al. 2013a, 2013b; Song et al. 2013; Yoon & Oh 2013; Jain et al. 2014; Pfister et al. 2014; Zhu et al. 2014; Ponzo et al. 2015; Yi et al. 2015; Lee & Kim 2016). Sodium was estimated by different methods including thorough measurement of dietary sodium/salt intake or urinary sodium excretion (Verhave et al. 2004; Hu et al. 2005; Shikata et al. 2006; Radhika et al. 2007; He et al. 2008; Umesawa et al. 2008; Hoffmann & Cubeddu 2009; Jablonski et al. 2009; Visser et al. 2009; Murata et al. 2010; Libuda et al. 2012; Räisänen et al. 2012; Grimes et al. 2013a, 2013b; Larsen et al. 2013; Polonia et al. 2013; Song et al. 2013; Yoon & Oh 2013; Baudrand et al. 2014; Han et al. 2014; Jain et al. 2014; Ogna et al. 2014; Pfister et al. 2014; Zhao et al. 2014; Zhu et al. 2014; Huh et al. 2015; Yi et al. 2015; Lee & Kim 2016), serum sodium concentrations (Visser et al. 2009; Zhao et al. 2014), and via the frequency of consuming salty-snacks (Murata et al. 2010; Otsuka et al. 2013; Ponzo et al. 2015). The direct link between higher sodium intake and obesity indices has been reported in most studies (Verhave et al. 2004; Hu et al. 2005; Shikata et al. 2006; Radhika et al. 2007; He et al. 2008; Umesawa et al. 2008; Jablonski et al. 2009; Visser et al. 2009; Hulthén et al. 2010; Murata et al. 2010; Libuda et al. 2012; Räisänen et al. 2012; Grimes et al. 2013a, 2013b; Larsen et al. 2013; Polonia et al. 2013; Song et al. 2013; Yoon & Oh 2013; Baudrand et al. 2014; Han et al. 2014; Jain et al. 2014; Ogna et al. 2014; Pfister et al. 2014; Zhao et al. 2014; Zhu et al. 2014; Huh et al. 2015; Yi et al. 2015; Lee & Kim 2016), though the magnitude of this association differs between them. Therefore, the current meta-analysis was carried out on 18 cross-sectional reports (Verhave et al. 2004; Hu et al. 2005; Shikata et al. 2006; Radhika et al. 2007; Hoffmann & Cubeddu 2009; Jablonski et al. 2009; Visser et al. 2009; Hulthén et al. 2010; Murata et al. 2010; Otsuka et al. 2013; Polonia et al. 2013; Baudrand et al. 2014; Han et al. 2014; Pfister et al. 2014; Zhao et al. 2014; Huh et al. 2015; Yi et al. 2015; Lee & Kim 2016). 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Of those that attained four, not all used validated measurement tools for ascertainment of the exposure, and the study with three stars was not truly representative of the average in the target population and also did not use a validated measurement tool for exposure ascertainment. In regard to comparability criteria, the score for all studies was zero. Regarding outcome criteria, 12 studies achieved the maximum of three stars (Hu et al. 2005; Jablonski et al. 2009; Hoffmann & Cubeddu 2009; Visser et al. 2009; Hulthén et al. 2010; Polonia et al. 2013; Baudrand et al. 2014; Han et al. 2014; Pfister et al. 2014; Huh et al. 2015; Ma et al. 2015), five studies scored four stars (Shikata et al. 2006; Jablonski et al. 2009; Murata et al. 2010; Zhao et al. 2014; Ponzo et al. 2015) and one study scored 3 stars (Otsuka et al. 2013). Of those that attained four, not all used validated measurement tools for ascertainment of the exposure, and the study with three stars was not truly representative of the average in the target population and also did not use a validated measurement tool for exposure ascertainment. In regard to comparability criteria, the score for all studies was zero. Regarding outcome criteria, 12 studies achieved the maximum of three stars (Hu et al. 2005; Jablonski et al. 2009; Hoffmann & Cubeddu 2009; Visser et al. 2009; Hulthén et al. 2010; Räisänen et al. 2012; Baudrand et al. 2014; Pfister et al. 2014; Zhao et al. 2014; Huh et al. 2015; Ponzo et al. 2015).

Higher sodium consumption was associated with greater BMI (weighed mean difference (WMD) = 1.24 kg/m², 95%CI: 0.80, 1.67; I² = 98.4%; p < .0001). Heterogeneity could not be eliminated by subgroups based on sodium assessment tools (i.e. salt/sodium intake, urinary sodium, serum sodium, salty snacks), though heterogeneity between subgroups was statistically significant (p < .0001) (Figure S1). Incorporation of salty snacks with salt/sodium intake led to a 0.81 kg/m² increment in BMI, but

| Study | Selection | Comparability | Outcome | Total score |
|-------|-----------|---------------|---------|-------------|
| Baudrand R | 5 | 0 | 3 | 8 |
| Sang Youb Han | 5 | 0 | 2 | 7 |
| Jablonski, MS | 4 | 0 | 3 | 7 |
| Radhika G | 5 | 0 | 1 | 6 |
| Hoffmann I | 5 | 0 | 3 | 8 |
| Polonia J | 5 | 0 | 1 | 6 |
| PonzoV | 4 | 0 | 3 | 7 |
| Verhave J | 5 | 0 | 3 | 8 |
| Visser FW | 5 | 0 | 3 | 8 |
| Huh, J | 5 | 0 | 3 | 8 |
| Ma, Y | 5 | 0 | 3 | 8 |
| Hulthén, L | 5 | 0 | 3 | 8 |
| Shikata K | 4 | 0 | 1 | 5 |
| Zhao X | 4 | 0 | 3 | 7 |
| Pfister M | 5 | 0 | 3 | 8 |
| Murata A | 4 | 0 | 2 | 6 |
| Otsuka T | 3 | 0 | 1 | 4 |
| Hu G | 5 | 0 | 3 | 8 |

*The tool used to determine the quality of the studies was based on the Newcastle-Ottawa scales.*
heterogeneity was high ($I^2$: 94.3%, $p < .0001) (Figure 2). In subgroups by gender (male, female and both sexes), direct associations between sodium/salt and BMI were also observed (Figure 3). There was no evidence of publication bias (Begg’s test, $p = .080$ and Egger’s test, $p = .629$).

Individuals who had higher sodium intakes in comparison with those who consumed lower amounts of sodium had 4.75 cm (95% CI: 3.25, 6.25) greater WC; however, there was significant heterogeneity ($I^2 = 90.8\%$; $p < .0001$). After subgroup analyses based on gender, a significant increment in WC was observed in females and both sexes, though this association was not significant in males. This subgroup analyses eliminated heterogeneity in females, but not in both sexes ($I^2 = 90.7\%$; $p < .0001$) and males ($I^2 = 73.4\%$; $p = .053$) (Figure 4). Subgroup analyses based on the method of sodium intake assessment showed that both urinary and dietary sodium were positively associated with WC (WMD for dietary sodium: 6.52 cm (95%CI: 5.07, 7.96) and WMD for urinary sodium: 3.63 cm (95%CI: 2.18, 5.08) (Figure 5). Subgroup analyses by sodium assessment tool eliminated heterogeneity for dietary instruments ($I^2 = 43.4\%$; $p = .171$), but not in the urinary sodium group ($I^2 = 87.5\%$; $p < .0001$) (Figure 5). No evidence for publication bias was observed (Begg’s test, $p = .711$ and Egger’s test $p = .113$).

**Discussion**

The results of this meta-analysis show that high salt/sodium intake was significantly associated with greater BMI and WC. Since heterogeneity was high and there were subgroup differences based on sex and sodium assessment tools, further studies are needed to explore associations between salt and anthropometric measures. To our knowledge, this is the first comprehensive systematic review and meta-analysis to evaluate the relation between sodium/salt intake and obesity.

**Table 1**

| Study                         | WMD (95% CI) | Weight |
|-------------------------------|--------------|--------|
| Kinaroshikat (2006)           | –0.30 (–0.33, –0.27) | 3.97   |
| Radhika, G (2007)             | 2.70 (2.10, 3.30)   | 3.70   |
| Jablonski, K. L (2009)        | 1.80 (–1.03, 4.63) | 1.49   |
| Murata, A. (2010)             | 0.10 (–0.10, 0.30) | 3.93   |
| Murata, A. (2010)             | 0.20 (–0.02, 0.42) | 3.93   |
| Polonia, J (2013)             | 2.90 (1.18, 4.62)  | 2.46   |
| T. Otsuka (2013)              | 1.00 (0.30, 1.70)  | 3.60   |
| Xin Zhao (2014)               | 0.80 (0.10, 1.50)  | 3.60   |
| - Baudrand, R (2014)          | 0.40 (–0.57, 1.37) | 3.32   |
| V Ponzo (2015)                | 0.20 (–0.37, 0.77) | 3.72   |
| V Ponzo (2015)                | 0.30 (–0.26, 0.86) | 3.72   |
| Yuan Ma (2015)                | 1.40 (0.78, 2.02)  | 3.67   |
| Yuan Ma (2015)                | 1.70 (0.52, 2.88)  | 3.07   |
| Subtotal (I-squared = 94.3%, $p = 0.000$) | 0.81 (0.39, 1.22) | 44.18 |
| Urine                         |              |        |
| J. C. VERHAVE1 (2004)         | 3.80 (3.50, 4.10) | 3.90   |
| G. Hu (2005)                  | 2.90 (2.38, 3.42) | 3.76   |
| Hoffmann, I. S (2009)         | 2.50 (–0.27, 5.27) | 1.53   |
| Hoffmann, I. S (2009)         | 2.00 (0.76, 3.24) | 3.00   |
| Visser, F. W (2009)           | 0.40 (0.12, 0.68) | 3.90   |
| Visser, F. W (2009)           | 0.40 (–0.38, 1.18) | 3.52   |
| Hulthén L (2010)              | 3.00 (1.89, 4.11) | 3.16   |
| Polonia, J. (2013)            | 2.90 (1.18, 4.62) | 2.46   |
| Roman Prüfer (2014)           | 1.20 (0.98, 1.42) | 3.93   |
| Roman Prüfer (2014)           | 1.60 (1.35, 1.85) | 3.92   |
| Han, S. Y (2014)              | 1.10 (0.82, 1.38) | 3.91   |
| Ji Hye Huh (2015)             | 0.60 (0.36, 0.84) | 3.92   |
| Ji Hye Huh (2015)             | 1.35 (1.09, 1.61) | 3.91   |
| Subtotal (I-squared = 97.0%, $p = 0.000$) | 1.73 (1.13, 2.33) | 44.80 |
| Serum                         |              |        |
| Visser, F. W (2009)           | 0.40 (0.12, 0.68) | 3.90   |
| Visser, F. W (2009)           | 0.40 (–0.38, 1.18) | 3.52   |
| Xin Zhao (2014)               | 0.80 (0.10, 1.50) | 3.60   |
| Subtotal (I-squared = 0.0%, $p = 0.581$) | 0.45 (0.20, 0.70) | 11.02 |
| Overall (I-squared = 98.4%, $p = 0.000$) | 1.24 (0.80, 1.67) | 100.00 |

*Figure 2.* Forest plot derived from random-effects models depicting the association of salt intake with BMI by incorporation of salty snacks with salt/sodium intake.
This meta-analysis of observational studies provides a more reliable conclusion than individual assessment of particular studies. Cohort studies are more indicative of causal relations because of their prospective nature and provide stronger evidence compared to cross-sectional studies. According to the ranking of evidence by study design, cohort studies are superior to cross-sectional designs, where inverse causation bias may occur. To date, only few cohort studies have directly assessed the relationship between salt intake and obesity (Libuda et al. 2012; Larsen et al. 2013; Yoon & Oh 2013), but data from these studies could not be used for our meta-analysis due to the variability of reported outcomes, their statistical analysis, or because they reported sodium density as an exposure. Overall, both cross-sectional and cohort studies have reported direct correlations between salt intake and obesity.

In the present meta-analysis, high salt/sodium intake was positively associated with greater BMI and WC. Similar to these results, several studies have shown significant correlations between sodium intake and obesity. Yoon et al. indicated that high sodium intake might be a significant risk factor for weight gain independent of calorie intake (Yoon & Oh 2013). A longitudinal study by Libuda et al. reported that BMI and percent body fat in 3–18-year-old children were directly associated with urinary sodium (Libuda et al. 2012). Larsen et al. showed that high sodium intake was associated with subsequent gain in body fat and loss of fat-free mass, while it was not associated with changes in BW or WC (Larsen et al. 2013).

At the same time, a few studies have demonstrated no significant relation or even an inverse relation between sodium and BMI (Hoffmann & Cubeddu 2009; Jablonski et al. 2009; Visser et al. 2009; Murata et al. 2010; Baudrand et al. 2014; Zhao et al. 2014; Ponzo et al. 2015). These discrepancies, as well as the substantial heterogeneity in the results of this meta-analysis, are probably related to sodium assessment method (diet or urinary sodium), the accuracy of assessment method or differences in populations (age, sex, and ethnicity) or study design type. However, due

**Figure 3.** Forest plot derived from random-effects models depicting the association of salt intake with BMI by gender.
to the limited number of studies in each subgroup, we could not determine the source of heterogeneity. This was particularly a problem regarding the determination of whether study design was a specific source of heterogeneity; as only cross-sectional effect sizes could be extracted from cohort studies (baseline or end values), it was not possible to conduct analysis on prospective effect sizes.

**Figure 4.** Forest plot derived from random-effects models depicting the association of salt intake with WC by gender.

| Study            | WMD (95% CI)            | Weight |
|------------------|-------------------------|--------|
| Both             |                         |        |
| Radhika, G (2007) | 7.70 (6.14, 9.26)       | 13.62  |
| Han, S. Y (2014)  | 3.00 (2.17, 3.83)       | 15.22  |
| Yuan Ma (2015)    | 5.90 (4.41, 7.39)       | 13.80  |
| Yuan Ma (2015)    | 5.20 (2.08, 8.32)       | 9.45   |
| **Subtotal (I-squared = 90.7%, p = 0.000)** | **5.42 (2.94, 7.89)** | **52.09** |
| **Male**          |                         |        |
| Hoffmann, I. S (2009) | 6.00 (1.62, 10.38)     | 6.77   |
| Ji Hye Huh (2015) | 1.61 (0.90, 2.32)       | 15.42  |
| **Subtotal (I-squared = 73.4%, p = 0.053)** | **3.25 (-0.91, 7.41)** | **22.19** |
| **Female**        |                         |        |
| Hoffmann, I. S (2009) | 6.20 (3.43, 8.97)      | 10.34  |
| Ji Hye Huh (2015) | 4.20 (3.46, 4.94)       | 15.38  |
| **Subtotal (I-squared = 46.5%, p = 0.171)** | **4.74 (3.00, 6.47)** | **25.72** |
| **Overall (I-squared = 90.8%, p = 0.000)** | **4.75 (3.25, 6.25)** | **100.00** |

**Figure 5.** Forest plot derived from random-effects models depicting the association of salt intake with WC by sodium assessment tools.

| Study            | WMD (95% CI)            | Weight |
|------------------|-------------------------|--------|
| Urine            |                         |        |
| Hoffmann, I. S (2009) | 6.00 (1.62, 10.38)     | 6.77   |
| Hoffmann, I. S (2009) | 6.20 (3.43, 8.97)      | 10.34  |
| Han, S. Y (2014)  | 3.00 (2.17, 3.83)       | 15.22  |
| Ji Hye Huh (2015) | 1.61 (0.90, 2.32)       | 15.42  |
| Ji Hye Huh (2015) | 4.20 (3.46, 4.94)       | 15.38  |
| **Subtotal (I-squared = 87.5%, p = 0.000)** | **3.63 (2.18, 5.08)** | **63.13** |
| **Dietary instruments** |                   |        |
| Radhika, G (2007) | 7.70 (6.14, 9.26)       | 13.62  |
| Yuan Ma (2015)    | 5.90 (4.41, 7.39)       | 13.80  |
| Yuan Ma (2015)    | 5.20 (2.08, 8.32)       | 9.45   |
| **Subtotal (I-squared = 43.4%, p = 0.171)** | **6.52 (5.07, 7.96)** | **36.87** |
| **Overall (I-squared = 90.8%, p = 0.000)** | **4.75 (3.25, 6.25)** | **100.00** |
In the assessment of study quality, most studies included in this meta-analysis were graded seven points or above. Regarding study selection, most studies used an acceptable method for selection, but confounding factors were not controlled in any study. This matter is relevant since individuals with higher salt intake are also less likely to have healthy lifestyles, e.g., they are likely to have lower physical activity levels, make less healthy food choices, and have poorer eating behaviors (Larsen et al. 2013). Therefore, these factors may influence the association between salt and obesity. However, most studies received all possible stars for the outcome criteria.

Several reasons may explain the direct link between salt and obesity: (i) higher sodium intake drives the thirst response and promotes fluid intake (Campbell 2004; Stachenfeld 2008), which might be compensated with drinking sugar-sweetened beverages (Grimes et al. 2013a, 2013b); (ii) salty foods are often high in fat and energy; (iii) salty foods are more palatable and encourage individuals to consume greater quantities of these foods.

The current meta-analysis has some strengths. First, various subgroup analyses were performed to identify sources of heterogeneity. Second, a comprehensive systematic literature search was conducted to find all relevant articles. However, some limitations should also be considered when interpreting the results. First, cross-sectional studies cannot be used to infer causality. Second, we could not determine the dose–response association between salt intake and weight gain. Third, failure to accurately assess sodium intake in some studies (e.g., that used a food intake questionnaire instead of the gold standard of urine sodium) may influence the relation between salt and obesity indices. Dietary recall and weighted diet records often underestimate intake due to under-reporting. FFQs are more useful than dietary recall and dietary records since they assess intake over a longer period; however, accurate quantification of daily food intake is difficult. Although 24-hour urine collection is known as a gold standard and the most accurate method for assessment of sodium intake, research is limited by under-collection of data using this method (McLean 2014). Fourth, BMI has limitations for estimating the amount of muscle mass and fat. Fifth, we reviewed some studies in which the associations between salt and anthropometric measures were reported as secondary outcomes. Finally, the most important limitation of using these secondary data was insufficient control for confounders.

In conclusion, the results of this meta-analysis suggest that high salt/sodium intake is directly correlated with BMI and WC. However, several gaps still remain that warrant further investigation. Future studies are needed to (1) investigate the sources of heterogeneity inherent in this association, (2) assess other anthropometric measures, especially fat mass, (3) conduct prospective studies that specifically examine the association of salt intake and obesity while taking into account the role of relevant confounders and (4) use more precise methods to measure obesity indices, such as fat mass as well as sodium intake.

**Disclosure Statement**

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**References**

Azadbakht L, Esmaillzadeh A. 2008. Fast foods and risk of chronic diseases. J Res Med Sci. 131:1–2.

Baudrand R, Campino C, Carvajal C, Olivieri O, Guidi G, Faccini G, Vöhringer PA, Cerda J, Owen G, Kalergis AM, Fardella CE. 2014. High sodium intake is associated with increased glucocorticoid production, insulin resistance and metabolic syndrome. Clin Endocrinol (Oxf). 80:677–684.

Brown JJ, Tzoulaki I, Candeias V, Elliott P. 2009. Salt intakes around the world: implications for public health. Int J Epidemiol. 38:791–813.

Campbell S. 2004. Dietary reference intakes: water, potassium, sodium, chloride, and sulfate. Clin Nutr Insight. 30:1.

Commission E [Internet]. 2015. Implementation of the EU Salt Reduction Framework. 2015; [cited 2015 Jun 15]. Available from: http://ec.europa.eu/health/nutrition_physical_activity/docs/salt_report_en.pdf

Ellison RC, Sosenko J, Harper GP, Gibbons L, Pratter FE, Miettinnen OS. 1980. Obesity, sodium intake, and blood pressure in adolescents. Hypertension. 2:78–182.

Fonseca-Alaniz MH, Brito LC, Borges-Silva CN, Takada J, Andreotti S, Lima FB. 2007. High dietary sodium intake increases white adipose tissue mass and plasma leptin in rats. Obesity (Silver Spring). 15:2200–2208.

Grimes CA, Riddell LJ, Campbell KJ, Nowson CA. 2013b. Dietary salt intake, sugar-sweetened beverage consumption, and obesity risk. Pediatrics. 131:14–21.

Grimes CA, Wright JD, Liu K, Nowson CA, Loria CM. 2013a. Dietary sodium intake is associated with total fluid and sugar-sweetened beverage consumption in US children and adolescents aged 2–18 y: NHANES 2005-2008. Am J Clin Nutr. 98:189–196.

Han SY, Hong JW, Noh JH, Kim D-J. 2014. Association of the estimated 24-H urinary sodium excretion with albuminuria in adult Koreans: the 2011 Korea national health and nutrition examination survey. PLoS One. 9:e109073.
World Health Organization (WHO). 2002. The world health report 2002: reducing risks, promoting healthy life. Switzerland: World Health Organization.

World Health Organization (WHO). 2014. Obesity: situation and trends. Geneva (CH): World Health Organization.

Yi SS, Firestone MJ, Beasley JM. 2015. Independent associations of sodium intake with measures of body size and predictive body fatness. Obesity (Silver Spring). 23:20–23.

Yi SS, Kansagra SM. 2014. Associations of sodium intake with obesity, body mass index, waist circumference, and weight. Am J Prevent Med. 46:e53–ee5.

Yoon YS, Oh SW. 2013. Sodium density and obesity; the Korea national health and nutrition examination survey 2007-2010. Eur J Clin Nutr. 67:141–146.

Zhao X, Yang X, Zhang X, Li Y, Zhao X, Ren L, Wang L, Gu C, Zhu Z, Han Y. 2014. Dietary salt intake and coronary atherosclerosis in patients with prehypertension. J Clin Hypertens (Greenwich). 16:575–580.

Zhu H, Pollock NK, Kotak I, Gutin B, Wang X, Bhagatwala J, Parikh S, Harshfield GA, Dong Y. 2014. Dietary sodium, adiposity, and inflammation in healthy adolescents. Pediatrics. 133:e635–ee42.