Sodium intake and high blood pressure among adults on caloric deficit: a multi-year cross-sectional analysis of the U.S. population, 2007–2018

Jorge Andrés Delgado-Ron1,2, Patricio López-Jaramillo1 and M. Ehsan Karim1,3

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Small studies have shown reduced sodium-sensitivity of blood pressure in obese adolescents on a caloric deficit. We aimed to explore the association between mean daily sodium intake and prevalent hypertension among a nationally representative sample of U.S. adults on a calorie deficit. We used a design-based regression model to explore the association between sodium intake and prevalent hypertension. We also conducted sensitivity analyses using multiple imputation chained equations and propensity score matching. We also measured the effect of a binary exposure derived from the widely recommended threshold of 2.3 grams of sodium intake per day. Among 5756 individuals, we did not detect any significant association between increased sodium and the odds of hypertension (OR: 0.97; 95% CI: 0.90; 1.05). All our sensitivity analyses are consistent with our main findings. People on a calorie deficit—a component of healthy weight loss—without malnutrition saw no benefit in reduced sodium intake to lower blood pressure. These results highlight the need to explore new population-specific strategies for sodium intake reduction, including new dietary prescription approaches to improve dietary adherence and reduce the risk associated with sodium-deficient diets.

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INTRODUCTION
Hypertension causes more death and disability than any other risk factor globally, ahead of smoking, high glucose, and obesity. Nearly 10.4 million deaths worldwide were attributable to elevated blood pressure in 2017 [1]. Ambard and Beaujard theorized a potential association between salt consumption and hypertension as early as 1904. However, contradictory evidence from observational studies using low-salt diets led to an intense debate that lasted more than a century [2]. Only recently, well-designed clinical trials provided reliable answers. The Dietary Approaches to Stop Hypertension-Sodium (DASH-Na) trial concluded that “blood pressure can be lowered … by reducing the sodium intake” [3]. Long-term cohort studies support the DASH-Na trial findings [4].

The precise mechanisms that cause dietary sodium to modulate blood pressure levels are not well defined, which often translates to uncertainty about who would benefit from such interventions. Past studies have shown the role of specific modifiers like ethnicity, even at the molecular level [5]. However, we know much less about the role of energy balance—the equilibrium between calories consumed and calories burned through physical activity [6]—as a modifier of “salt sensitivity” of blood pressure.

Energy balance is arguably a more critical modulator than diet or exercise on their own. After all, we reach metabolic tipping points that enable our bodies to regulate blood pressure through the additive interaction of energy intake and energy expenditure [7, 8]. Current studies lack this holistic understanding and, as a result, present somehow conflicting evidence. For instance, a reanalysis of the DASH-Na data found less energy intake associated with increased salt sensitivity [9]. Similarly, a community-based study in China found that participants in the highest quartile of physical activity had reduced salt sensitivity compared to the lowest quartile. However, there was not a linear association across quartiles [10].

Caloric restriction and fitness are recommended to prevent or control hypertension [11, 12]. The current European and American hypertension guidelines recommend a reduced sodium intake on top of that [13, 14]. However, it is not clear if patients would benefit from reducing sodium in their diet once they are on an energy deficit, given they consume enough micronutrients. Caloric restriction—a negative energy balance without malnutrition—reduces blood pressure levels through improved insulin sensitivity, reduced adiposity, and reduced sympathetic activity, independent of reaching an ideal body weight [15, 16]. These mechanisms might also play a role in modulating the salt sensitivity of blood pressure [12, 17]. Studies that induced weight loss have modified the relationship between sodium and blood pressure. However, they used small samples and are outdated [16, 18].

The present report used a design-based regression model to explore the association between mean daily sodium intake and prevalent hypertension among U.S. adults on a calorie deficit in the National Health Examination and Nutrition Survey (NHANES) from 2007 to 2018.
METHODS
Data source, design, and study population
This cross-sectional analysis uses data from NHANES, a four-stage stratified cluster complex survey representative of the non-institutionalized United States population. The survey gathers lifestyle and medical information, along with biological samples and a physical examination. Most of the data for individuals—the final sampling unit—are publicly available. An in-depth description of NHANES’ design, sampling, and procedures can be found elsewhere [19].

Analytic sample and study variables
We included adults aged 20–79 years from six two-year cycles (2007–2018) with a self-reported energy deficit of at least –350 calories per day. We chose this cutoff to correct potential underreporting from self-reported dietary data [20] under the assumption that these individuals would be on true caloric restriction (e.g., lessening caloric intake without depriving essential nutrients). We derived energy balance from its two essential components: energy input (mean daily intake) and energy output (basal metabolic rate plus physical activity). The basal metabolic rate was calculated using the revised Harris-Benedict equations [21]. Self-reported weekly vigorous and moderate physical activity was transformed into daily metabolic equivalents following NHANES’ suggested scores [22]. NHANES derives the total daily intake from two 24-hour recall interviews, 3–10 days apart, using a validated instrument to reduce recall bias [23]. We did not exclude participants with invalid or missing answers for physical activity; instead, we used their basal metabolic rate as total output.

We excluded pregnant women and participants with body mass index (BMI) below 18.5 (malnutrition) or an active thyroid pathology due to inherent metabolic differences in these patients. Adults over the age of 80 were excluded because their age is not publicly available due to privacy

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Fig. 1  N = Number of participants. Flow chart of the analytic sample selection for testing the association between sodium intake and high blood pressure among U.S. adults on caloric deficit aged 20–79, using data from the National Health and Nutrition Examination Survey; cycles 2007–2008 to 2017–2018.
concerns. We also excluded people who reported a “much less than usual” intake during either interview.

The primary outcome was hypertension, a binary variable indicating one of the following: self-reported use of antihypertensive medication or systolic hypertension (mean systolic blood pressure ≥ 130 mmHg) or diastolic hypertension (diastolic blood pressure ≥ 80 mmHg). Individuals without a second valid measurement for either systolic (SBP) or diastolic blood pressure (DBP) were excluded to avoid measurement error.

The exposure was self-reported mean consumption of sodium (grams per day) as a continuous measure. Two additional variables binary variables were derived for our sensitivity analyses. Other covariates included demographic data (age, gender, education, income, and ethnicity), pre-existing diseases (diabetes and obesity), and smoking. We excluded “Refused,” “Don’t know,” and “Missing” values for demographic variables. However, for diabetes, we required respondents to provide a definitive “Yes.”

The details of the analytic data creation process are shown in Fig. 1.

We used the web version of the “dagitty” R package [24] to graph the assumed causal relationships between our variables. Given we already restricted the sample by energy balance, we minimized bias adjusting for age, diabetes, education, ethnicity, gender, energy expenditure, BMI, and smoking status based solely on Pearls’ backdoor criterion [24]. Serum sodium and the renin-angiotensin-aldosterone system (unmeasured) were assumed mediators. Other unmeasured variables include alcohol consumption and fat accumulation (see online at http://dagitty.net/mqRZfJk).

Primary analysis

We compared individuals’ characteristics by hypertension status (yes/no) using survey featured tests and the Rao-Scott F-adjusted χ² test for categorical variables. We combined the survey weights from our six cross-sectional subsamples following NHANES recommendations and built our design using all survey features, subsetting only the eligible sample. We estimated the Odds Ratio (OR) and respective confidence intervals (95% CI) using a design-based multivariate logistic regression (hereafter referred to as the ‘outcome model’):

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\text{Hypertension} \sim \text{Exposure} + \text{age} + \text{diabetes} + \text{education} + \text{ethnicity} + \text{gender} + \text{energy expenditure} + \text{BMI} + \text{smoking status}
\]

We probed interactions between sodium intake and the other covariates using Bauer’s inferential and graphical techniques [25] based on findings from the previous literature [9]. Collinearity was assessed via variance inflation factors (VIF) and classified following the recommendations by Belsley [26].

The goodness of fit for all models was evaluated using weighted ROC curves and the Archer-Lemeshow test. All 95% confidence intervals (CI) and Cox-Snell pseudo-R² account for the survey design and day-to-day dietary intake variations.

Sensitivity analyses

Missing data. We applied the multivariate imputation by chained equation (MICE) method under the assumption of data missing at random. We imputed 20 datasets using all the variables from our primary analysis as predictors. We repeated our sampling procedure before estimating each dataset’s OR using our outcome model. Rubin’s rules were used to pool the estimates together.

Testing recommended intake threshold. We transformed the exposure in our outcome model to a binary variable. We used the threshold of ≥ 2.3 g/day to classify exposure as “high” or “low,” following the maximum sodium intake recommendations in current European and American hypertension guidelines. This threshold is compatible with the recommendations made by the World Health Organization [27].

We performed the analysis with both the complete-case dataset and the imputed datasets, pooling the estimates as described above. We also matched the probability of being exposed to “high” or “low” sodium intake using propensity scores for both thresholds. We performed a 1:1 nearest neighbour match (without replacement) on the propensity score’s logit with a 0.2 calliper [28]. We modeled the exposure using our original covariates—cycle and survey features were also covariates following Dugoff’s method [29]—to achieve adequate standardized mean difference balance (SMD < 0.2). The survey-featured outcome model was used to estimate the population average treatment effect from the matched subsample. Again, the exposure was binary (“high” vs. “low”).

Hypertension threshold. We conducted the same analysis described above, with different cutoffs for systolic (≥140 mmHg) and diastolic hypertension (≥ 90 mmHg).

Table 1. Participants characteristics by hypertensive status, U.S. adults who reported caloric deficit aged 20–79. National Health and Nutrition Examination Survey (NHANES) 2007–2018.

|                     | Non-hypertensive (n = 3023) | Hypertensive (n = 2733) | P value |
|---------------------|-----------------------------|-------------------------|---------|
| Sodium intake (g/day) [mean (SD)] | 3.20 (1.35) | 3.17 (1.36) | 0.462 |
| Age in years [mean (SD)] | 38.32 (15.37) | 51.28 (14.69) | <0.001 |
| Female (%) | 1 874 (47.0) | 1 352 (35.5) | <0.001 |
| Ethnicity (%) | White 1 619 (64.3) | 1 416 (65.7) | <0.001 |
| Latino | 1 107 (17.4) | 735 (11.9) |
| Black | 830 (12.2) | 1 120 (16.3) |
| Other | 382 (6.2) | 268 (6.1) |
| Education (%) | Some school 812 (13.7) | 902 (16.8) |
| High school degree | 946 (24.3) | 940 (26.5) |
| Higher education | 2 180 (62.0) | 1 697 (56.7) |
| Body mass index (kg/m²) | 29.56 (6.65) | 32.41 (7.35) | <0.001 |
| [mean (SD)] | 0.12 |
| Normal | 1 008 (27.5) | 397 (12.6) |
| Overweight | 1 263 (32.3) | 1 029 (30.0) |
| Obesity Class 1 | 914 (23.0) | 987 (28.2) |
| Obesity Class 2 | 435 (9.9) | 560 (14.4) |
| Obesity Class 3 | 318 (7.3) | 566 (14.7) |
| Diabetes (%) | 234 (3.8) | 827 (18.1) | <0.001 |
| Smoking (%) | Never smoker 2 287 (59.1) | 1 813 (51.2) |
| Past smoker | 680 (18.1) | 1 030 (28.4) |
| Current smoker | 971 (22.7) | 696 (20.4) |
| Daily energy intake (Kcal) | 1 846.56 (745.99) | 1 812.11 (711.60) | 0.222 |
| Daily energy expenditure (Kcal) | 3 450.90 (1867.56) | 3 381.06 (1809.72) | 0.314 |

All statistical analyses were performed using R 4.1.1; the code is available on request.

RESULTS

Study sample characteristics

We evaluated a total of 5756 individuals, representing a U.S. national population of 53,036,129. The weighted prevalence of hypertension in our sample was 42.6%. On average, hypertensive individuals were significantly more likely to be male, older,
Sodium intake did not significantly differ by hypertension status (Table 1).

**Association between mean daily sodium intake and prevalent hypertension**

A one-gram increase in daily sodium intake did not significantly correlate with higher odds of hypertension in the U.S. population on a caloric deficit. The estimate was imprecise around the null in both the crude (OR = 0.98, 95% CI: 0.93; 1.03) and adjusted models (Table 2). We did not find any significant interaction between the exposure and other covariates. Multicollinearity was “near weak” for all independent variables (VIF < 2.5).

**Sensitivity analysis**

_Multiple imputations for missing data._ Data for moderate and vigorous physical activity was missing for 30% of the sample. Other sampling variables with lower proportions of missing data were calorie intake (9%), height (8%), and the derived BMI (8%). Model variables with missingness were mean sodium intake (9%) and education (40.8%). Each imputed dataset contained ~6326 observations. The estimate and confidence intervals were equal to that of the primary analysis (OR = 0.97, 95% CI: 0.90; 1.05) (Table 2).

_Sodium intake of ≥ 2.3 g/day._ Consuming 2.3 grams of sodium per day or more was not significantly associated with higher hypertension odds in either the complete-case or imputed data analyses. Although both estimates found a different effect, they were imprecise around the significance threshold. Hence, we failed to find an association (Table 3). The final propensity score for the probability of being exposed to higher amounts of sodium was estimated using age, gender, ethnicity, education, BMI (categorical), history of diabetes, history of smoking, energy expenditure, and survey features. After propensity-score matching, a total of 2808 participants—representing a U.S. population of 24,340,468—were matched. Matching reduced the SMD to < 0.1 for all covariates, except for total daily energy expenditure (SMD = 0.27). Our outcome model corrected any remaining imbalances. The results, again, could not support an association between exposure and outcome (OR = 0.99, 95% CI: 0.77; 1.27) (Table 3).

**Hypertension threshold of 140/90 mmHg.** When we modified the threshold, 822 participants changed from hypertensive to non-hypertensive status. The weighted prevalence changed from

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**Table 2.** Survey-featured multivariable logistic regression model for the relationship between average daily sodium intake (grams per day) and hypertension among U.S. adults in caloric deficit, aged 20-79. National Health and Nutrition Examination Survey (NHANES) 2007–2018.

| Variable          | Units       | Complete case analysis Odds Ratio 95% CI | Multiple imputations odds ratio 95% CI |
|-------------------|-------------|-----------------------------------------|---------------------------------------|
| Sodium intake     | g/day       | 0.97†                                   | 0.97†                                 |
| Age               | Years       | 1.06                                    | 1.06                                  |
| Gender            | Male Ref    |                                         |                                       |
| Female            | 0.57        | [0.46; 0.70]                             | [0.46; 0.70]                          |
| Ethnicity         | White Ref   |                                         |                                       |
| Latino            | 0.76        | [0.62; 0.94]                            | [0.62; 0.95]                          |
| Black             | 1.58        | [1.31; 1.91]                            | [1.31; 1.92]                          |
| Other             | 1.24        | [0.90; 1.70]                            | [0.90; 1.69]                          |
| Education         | Some School Ref |                                   |                                       |
| High School       | 0.9         | [0.68; 1.19]                            | [0.68; 1.18]                          |
| Higher Education  | 0.87        | [0.68; 1.11]                            | [0.67; 1.1]                           |
| Body mass index   | Normal Ref  |                                         |                                       |
| Overweight        | 1.39        | [1.07; 1.79]                            | [1.06; 1.76]                          |
| Class 1 obesity   | 2.09        | [1.58; 2.76]                            | [1.59; 2.75]                          |
| Class 2 obesity   | 2.18        | [1.59; 2.98]                            | [1.58; 2.94]                          |
| Class 3 obesity   | 3.76        | [2.56; 5.53]                            | [2.59; 5.58]                          |
| Diabetes          | No Ref      |                                         |                                       |
| Yes               | 1.83        | [1.36; 2.47]                            | [1.37; 2.47]                          |
| Smoking           | Never smoker Ref |                                     |                                       |
| Past smoker       | 1.05        | [0.83; 1.33]                            | [0.83; 1.33]                          |
| Current smoker    | 0.88        | [0.71; 1.10]                            | [0.72; 1.10]                          |
| Energy Expenditure| Per 1 000 Kcal | 1.03                                  | 1.03                                  |
| Pseudo R² (Cox-Snell) | 0.217   |                                           |                                       |
| Archner-Lemeshow test | 0.83   |                                           |                                       |
| AUC               | 0.777       |                                         |                                       |

Caloric deficit was defined as an energy balance equal to or below −350 Kcal on an average day. Systolic and diastolic hypertension were defined as mean systolic and diastolic blood pressure values of ≥130 mmHg and ≥80 mmHg, respectively. Hypertension was defined as either systolic or diastolic hypertension or self-reported use of hypertensive medication. Confidence intervals were defined as mean confidence intervals, _AUC_ area under the curve.

*Adjusted by age, gender, ethnicity, education, body mass index, diabetes, smoking, and total daily energy expenditure.

*Pooled estimate of 20 imputed datasets using Rubin’s rules. Each dataset contained ~6326 observations.
42.6% to 26.8%. Furthermore, sodium consumption among the hypertensive population was lower than non-hypertensive (3.08 ± 1.36 vs. 3.23 ± 1.35; weighted p value: 0.007). They also ate less (1861.28 ± 743.17 vs. 1751.39 ± 693.05; 0.001) and spent less energy (3192.64 ± 1726.58 vs. 3504.62 ± 1877.39; < 0.001).

A one-gram increase in daily sodium intake significantly correlated with higher odds of hypertension neither in the crude model (OR = 0.92; 95% CI: 0.87; 0.98) but not the adjusted one (Table 4). The propensity-score model based on the 2.3 grams cutoff did not find significant differences (OR = 0.97 CI: 0.72;1.32).

**DISCUSSION**

**Main findings**

Our analysis of a multi-year nationally representative sample of U.S. adults on a calorie deficit did not detect any significant association between increased sodium consumption levels and the odds of hypertension (OR: 0.97; 95% CI: 0.90; 1.05). Our survey-featured logistic regression was adjusted by age, gender, ethnicity, education, BMI, diabetes, smoking, and total daily energy expenditure. Several sensitivity analyses yielded similar results, including the analysis of a widely recommended sodium intake threshold (< 2.3 grams per day). These results cement the need to target low-sodium interventions appropriately, given its associated risks [30, 31].

**Contextualizing the evidence**

Very few studies had been able to identify a representative sample of participants on a calorie deficit, commonly identified through healthy weight loss. Our findings suggest that people on caloric restriction would see no benefit in reducing sodium in their diet to lower blood pressure. These findings apply to the general U.S. adults and other populations with similar characteristics. Our findings align with previous reports in obese adolescents, who lost their “sensitivity of blood pressure to sodium” following a 20-week intervention to lose weight (through caloric restriction) [18].

Caloric restriction lowers blood pressure levels independent of other factors; it decreases body fat and, consequently, increases insulin sensitivity [32]. Insulin modulates the renal absorption of sodium. As a result, people on caloric restriction excrete more sodium than those in a caloric surplus, which might explain the loss of sodium sensitivity in these patients [33]. Such effects can
be seen within hours of fasting and had been recently hypothesized as mechanisms that regulate blood pressure levels in healthy and sick individuals [17, 34]. While these are acute effects, other cardiometabolic benefits of caloric restriction appear long-lasting [15]. We highlight the need for a tailored approach for eating occasions.

Strengths and limitations

Our study benefited from using a population-level sample, with an adequate representation of people from diverse ethnicities, ages, and socioeconomic characteristics. Our analysis used survey features, which allowed us to generalize our findings to all the non-institutional U.S. adult population going through a healthy weight loss. We restricted our sample to participants on a calorie deficit, therefore addressing the ambiguities of metabolically healthy obese patients and lean people who exhibit obese-like characteristics (dyslipidemia, altered inflammatory profile, and increased fat cell size). Rarely do hypertension studies account for such ambiguity. Our findings were robust under the missing at random assumption and to adjustments to the exposure to recommended intake levels by American and European current guidelines. Adjustments to the hypertension threshold to previous standards (140/90 mmHg) did not impact our results significantly. Our study has several limitations. We used self-reported data for energy intake, prone to measurement bias and underreporting. While our selected threshold [20] for energy balance accounts for such underreporting, this measurement error is not systematic. We further tried to minimize bias by only including the observations marked as reliable by NHANES—providing a detailed description of each food, including the amount, additional ingredients, and eating occasions—and excluding all participants reporting a less-than-usual intake. NHANES interviewers also double-check to elicit forgotten foods, and the survey weights account for variation between weekends and weekdays. We also used self-reported sodium consumption instead of the 24-hour urinary collection (considered by many as the gold standard). We made this decision based on two arguments. First, the Automated Multiple-Pass Method employed by NHANES

| Variable | Units | Odds ratio | 95% CI | Multiple imputations odds ratio | 95% CI |
|----------|-------|------------|-------|-------------------------------|-------|
| Sodium intake | g/day | 1.03† | [0.95; 1.12] | 1.03† | [0.95; 1.12] |
| Age | Years | 1.09 | [1.08; 1.10] | 1.09 | [1.08; 1.10] |
| Gender | | | | | |
| Male | | Ref | | | |
| Female | | 0.80 | [0.66; 0.98] | 0.80 | [0.66; 0.97] |
| Ethnicity | | | | | |
| White | | Ref | | | |
| Latino | | 0.72 | [0.55; 0.94] | 0.72 | [0.56; 0.93] |
| Black | | 1.84 | [1.50; 2.26] | 1.82 | [1.48; 2.23] |
| Other | | 1.45 | [0.97; 2.15] | 1.46 | [0.99; 2.16] |
| Education | | | | | |
| Some School | | Ref | | | |
| High School | | 1.02 | [0.76; 1.38] | 1.02 | [0.76; 1.37] |
| Higher Education | | 0.71 | [0.53; 0.96] | 0.71 | [0.52; 0.96] |
| Body mass index | | | | | |
| Overweight | | 1.25 | [0.85; 1.84] | 1.24 | [0.84; 1.81] |
| Class 1 obesity | | 1.86 | [1.58; 2.16] | 1.86 | [1.26; 2.74] |
| Class 2 obesity | | 2.24 | [1.52; 3.29] | 2.26 | [1.55; 3.31] |
| Class 3 obesity | | 4.73 | [2.67; 8.37] | 4.70 | [2.68; 8.25] |
| Diabetes | No | Ref | | | |
| Yes | | 2.53 | [1.95; 3.28] | 2.56 | [1.98; 3.30] |
| Smoking | Never smoker | | Ref | | |
| Past smoker | | 1.07 | [0.83; 1.38] | 1.05 | [0.82; 1.36] |
| Current smoker | | 0.97 | [0.74; 1.28] | 0.97 | [0.74; 1.28] |
| Energy Expenditure | Per 1 000 Kcal | 1.00 | [0.93; 1.07] | 1.00 | [0.93; 1.06] |
| Pseudo R² (Cragg-Uhler) | | 0.40 | | | |
| Archer-Lemeshow test | | 0.152 | | | |
| AUC | | 0.843 | | | |

Caloric deficit was defined as an energy balance equal to or below −350 Kcal on an average day. Systolic and diastolic blood pressure values of ≥140 mmHg and ≥90 mmHg, respectively. Hypertension was defined as either systolic or diastolic hypertension or self-reported use of hypertensive medication. Confidence intervals using sample weights provided by NHANES (account for sampling design and dietary variation), strata, and unit.

†Adjusted by age, gender, ethnicity, education, body mass index, diabetes, smoking, and total daily energy expenditure.

‡Pooled estimate of 20 imputed datasets using Rubin’s rules. Each dataset contained 6326 observations.
provides reliable sodium and intake measures [23]. Second, the hypothesized underlying mechanism (increased sodium retention) requires measuring intake and not excretion.

Self-reported data for moderate and vigorous activity is not as reliable as objectively measured physical activity. If people reported > 24 h of physical and sedentary activity, NHANES analysts set those values as missing. We dealt with this first with two methods. First, we took a conservative sampling (including the BMR as total energy expenditure whenever data was not available meant that participants who reported high activity levels were considered sedentary). Second, we used multiple imputations procedures under the assumption that these data, although not missing at random, could be predicted using the available variables. Statistically, there were no significant differences between the estimates obtained by either method. However, the direction of the effect changed with multiple imputations in both thresholds. Such a change of direction might reflect the effect of the omitted physical activity in the complete-case sample. Nevertheless, the difference was not statistically significant. Finally, our cross-sectional design did not allow us to establish a temporal relationship between exposure and outcome. It’s feasible for people to modify their diet to cope with their disease.

CONCLUSION
Our findings showed that sodium intake was not associated with higher odds of hypertension among the U.S. population on a calorie deficit. Our results were robust to missing data and different representations of the exposure, disputing a low-salt diet’s benefits for people who achieve weight loss and maintain it using caloric restriction. These results highlight the need to explore new population-specific strategies for sodium intake reduction, including new dietary prescription approaches that improve adherence and reduce the risk associated with deficient sodium diets.

Summary table
What is known about this topic
- Small studies have shown reduced blood pressure sensitivity to sodium in obese adolescents on caloric restriction. No population-level studies have looked at the effects of calorie deficit on the blood pressure sensitivity to sodium.

What this study adds
- Our design-based regression model explored the association between sodium intake and prevalent hypertension in the U.S. population (biannual cohorts from 2007 to 2018). Increased sodium intake was not associated with increased odds of hypertension among people with a calorie deficit.
- We dispute the benefit of a low-salt diet for people who achieve weight loss and maintain it using caloric restriction. Salt could create more palatable diets and increase adherence.

DATA AVAILABILITY
Datasets are publicly available from https://www.cdc.gov/nchs/nhanes/.

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AUTHOR CONTRIBUTIONS
JDR: conceptualization, methodology, formal analysis, investigation, data curation; writing—original draft. MEK: methodology, software, validation, writing—review & editing, supervision. PLJ: validation, writing—review & editing.

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ETHICS
This study is covered by item 7.10.3 in the University of British Columbia’s Policy 89 on studies involving human participants and Article 2.2 in the Tri-Council Policy Statement Ethical Conduct for Research Involving Humans.

ADDITIONAL INFORMATION
Correspondence and requests for materials should be addressed to Jorge Andrés Delgado-Ron.

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