

1 **Supplemental Methods**

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3 **Estimation of SNAP population by strata**

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5 Estimates of the SNAP population by age-sex strata are an essential intermediate input in  
6 estimating mortality rates among SNAP participants, eligible non-participants and ineligible  
7 individuals. Briefly, we used data from the 2014 Current Population Survey (CPS) to estimate  
8 the proportion of individuals by age-sex strata who participated in SNAP in the prior 12 months,  
9 who were income-eligible (income-to-poverty ratio<1.3) but did not participate,<sup>22-23</sup> and those  
10 who were not eligible based on income. CPS data were used as opposed to NHIS, as 2014 NHIS  
11 data was not available at the time of project initiation and the large sample size of CPS  
12 (n=88,595) was sufficient to precisely estimate SNAP participation rates by age-sex strata. The  
13 proportions by age-sex strata were then applied to data from the 2014 American Community  
14 Survey population estimates by age-sex-strata to obtain estimates on the three SNAP groups  
15 within each strata that add to the 2014 official population estimates.

16

17 **Current mortality**

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19 There are no official estimates of mortality rates due to CHD, stroke and diabetes by age-sex-  
20 SNAP strata in the United States. Therefore, we developed an alternative approach to estimating  
21 mortality rates among SNAP participants using data from the mortality-linked National Health  
22 Interview Survey (NHIS, 2000-2009), the US Death Certificates (2000-2009),<sup>33</sup> and the 2014  
23 CPS and ACS (described above). The first step of this process was to estimate mortality rates

24 and relative risks of mortality in the mortality-linked NHIS files. We used data from 2000-2009  
25 to create a large sample size so that mortality rates and relative risks were statistically stable.  
26 Using data from the persons-level file (~50,000 individuals per year) we identified individuals  
27 who participated in SNAP in the prior-year, who were income-eligible but did not participate and  
28 who were not income-eligible.<sup>8b</sup> These data were then combined with age-sex strata to create  
29 age-sex-SNAP strata (n=36 total strata).

30  
31 For each stratum, we estimated the survey-weighted mortality rates due to heart disease (the  
32 following ICD-10 codes: I00-I09, I11, I13, I20-I51), stroke (I60-I69), and diabetes (E10-E14)  
33 with follow-up through December 31, 2011. For each of these causes the ratio of mortality rates  
34 comparing SNAP participants and income-eligible non-participants was then compared to the  
35 ineligible population. For each age-sex strata, we compared the mortality rates observed in NHIS  
36 to those from US death certificates data (which capture all deaths among US residents) to  
37 determine if NHIS death rates were comparable to those observed using all available data. We  
38 observed that while in most cases death rates were comparable by age-sex strata, there were  
39 some differences, particularly among younger (25-34y) and older ( $\geq 75y$ ) women, whose  
40 mortality experience was under-estimated in NHIS data. This suggests that, while NHIS is  
41 nationally representative, there may be some bias in who participates (e.g., in these strata, sicker  
42 individuals are less likely to participate). For most groups the difference between NHIS and  
43 death certificates was no more than +/-10-15%.

44

45 Because of potential biases in NHIS and death certificate estimates of mortality rates, we  
46 developed an approach to preserve the relative difference in mortality, but correct the NHIS

47 mortality estimates to be representative of the mortality rates in the US at-large. Specifically, we  
48 estimated the potential bias comparing actual mortality rates to those estimated in NHIS by age-  
49 sex strata. This adjustment factor was then applied within each strata to the NHIS mortality  
50 estimates by age-sex-strata. This approach makes the assumption that potential biases in NHIS  
51 mortality rates are equally distributed by SNAP group within each strata. For example, the NHIS  
52 observed stroke mortality rate among men  $\geq 75$ y was 744.5 per 100,000, but was corrected to be  
53 731.9 per 100,000 given evidence that stroke mortality in this age-sex group was under-  
54 estimated by 1.7% in NHIS compared to the death certificate data. This data processing step was  
55 essential, as otherwise the NHIS estimates would result in a different number of deaths than  
56 actually observed in the population.

57

58 To estimate mortality rates by strata these adjusted mortality rates were applied to estimates of  
59 the SNAP population previously described (combining data from CPS and ACS). Because NHIS  
60 data does not release data on CHD-specific mortality, we adjusted the heart disease mortality  
61 rates by estimating the proportion of heart deaths by age-sex strata there were due to CHD (I20-  
62 I24). The adjustment factor was smallest for the younger strata (e.g., 0.328 for men 25-34y) and  
63 greatest for the older groups (e.g. 0.683 for men 65-74y).

64

## 65 **Statistical analysis**

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67 For each dietary factor and price change condition  $j$ , the model calculated the change in the  
68 expected relative risk for mortality ( $M^j$ ) separately for each stratum of American adults

69 according to age (25-34, 35-44, 45-54, 55-64, 65-74, 75+ y), sex, and SNAP eligibility and  
70 participation status (3 categories):

71

$$72 \quad M^j = \int_{x=0}^m RR^j(x) P^j(x) dx,$$

73

74 where  $P^j(x)$  is the stratum-specific distribution of dietary intake of food  $x$  in the population at  
75 baseline ( $j=0$ ) or under the policy treatment ( $j=1$ );  $m$  is the maximum intake level beyond which  
76 no further change in risk occurs; and  $RR^j(x)$  is the stratum- and disease-specific relative risk at  
77 intake level  $x$ .

78

79 For each stratum, we computed the potential proportion of disease-specific mortality prevented  
80 by the price change intervention (potential impact fraction, PIF), as follows.

81

$$82 \quad PIF = \frac{M^0 - M^1}{M^0} .$$

83 The PIF was used to estimate the resulting deaths prevented or postponed per 10,000 adults in  
84 the population and as a percentage of all deaths.

85

86 For each food group, the distribution of current dietary intake ( $P^j(x)$ ) was modeled using a  
87 gamma distribution. In contrast with a normal distribution, which is symmetric, the gamma  
88 distribution allowed us to describe the right-skewed distribution observed in the NHANES data.

89 For baseline estimates, the mean ( $E[x]=\alpha/\beta$ ) and variance ( $\text{Var}[x]=\alpha/\beta^2$ ) were obtained from

90 NHANES estimates, using survey sampling weights and adjusting for complex survey design.

91 For estimates under each policy scenario, we multiplied the mean and standard deviation by the

92 intervention effect. This post-intervention distribution would arise, for example, under the  
93 assumption that the intervention effect is constant across individuals.

94

95 For food groups subject to the tax (SSBs and processed meats),  $RR(x)$  was assumed to increase  
96 exponentially as a function of the positive difference between  $x$  and a theoretical minimum risk  
97 exposure level:

$$98 \quad RR^j(x) = \exp[\beta(x-y(x))],$$

99 where  $\beta$  is the change in log relative risk per unit of exposure and  $y(x)$  is the theoretical  
100 minimum risk based on dietary recommendations. For food groups subject to the subsidy (fruit,  
101 vegetable, nuts, and whole grains), the convention was reversed, so that relative risk increased  
102 when consumption fell short of the minimum risk exposure level.

103

104 In computation, we used numerical integration to compute integrals in the PIF equation, and  
105 monte carlo simulation to quantify uncertainty. For each diet disease pair, stratum, and policy  
106 scenario, we drew randomly 1000 times from the normal distribution of the estimate of disease-  
107 specific change in the  $\log(RR)$  corresponding to a one unit increase in intake, the normal  
108 distribution of the estimate of the exposure mean, and the normal distribution of the estimate of  
109 the intervention effect. Draws of mean intake that were zero or less were changed to 0.00001.

110 Each set of random draws was used to calculate the PIFs and attributable mortality.

111

112 For SSBs, we estimated effects mediated by body mass index (BMI). Using the same procedure  
113 as Singh et al.,<sup>31</sup> we multiplied the  $\log(RR)$  per unit increase in BMI by the estimated increase in  
114 BMI in response to one unit increase in SSBs. The direct and mediated relative risks used in this  
115 computation are reported in Table S4.