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Family Health Spillovers: A Multidimensional Regression Discontinuity Design

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Family Health Spillovers: A Multidimensional Regression Discontinuity Design

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Abstract

A multidimensional regression discontinuity design reveals large and asymmetric spillovers from personalized health information within Chinese families. Warning a man that he has high blood pressure (BP) is estimated to reduce his wife's BP by 7.5% and her probability of high BP by 24% points, while there is no female-to-male spillover. Warning a parent (father, particularly) reduces their adult child's BP. Identified mechanisms are reduced adiposity (spousal) and healthier behavior (intergenerational). Asymmetry can be explained by joint health production under collective but gender-unequal household decision making. We demonstrate screening even for non-communicable disease risk can generate positive spillovers.

Keywords: Health behavior | Peer effects | Information | Collective household | Screening

JEL classification: C21; I12; I15

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I. Introduction

Personalized health interventions potentially impact the health of relatives through information sharing, heightened health salience or joint health production within families. Yet evaluations typically ignore any family health spillover, not least because identification is confounded by strong health correlation within families (Meyler *et al.*, 2007; Halliday, 2023) due to shared risk exposure and assortative mating (Capatina and Kahn, 2024). We circumvent these obstacles to identification by comparing health outcomes of Chinese adults with a close relative (spouse or parent) just above a blood pressure (BP) cutoff that triggers a warning about high BP with outcomes of others whose respective relative is just below the cutoff.

Unlike other applications of regression discontinuity design (RDD) to spillover identification (Dahl *et al.*, 2014; Bouckaert *et al.*, 2020; Oral *et al.*, 2026), we do not require that individuals exposed indirectly are far from the margin of being treated directly. This restriction is usually imposed to avoid confounding the spillover with the direct effect, but it rules out identification of spillover heterogeneity by own treatment status (Vazquez-Bare, 2023). We use multidimensional RDD (Papay *et al.*, 2011) to identify flexible direct and spillover effects from outcome comparisons near the two-dimensional treatment frontier defined by each individual's BP relative to the warning cutoff.

In spousal dyads, we estimate a saturated two-dimensional local response surface over the BP (running variable) of each individual in a married couple and across four treatment states: man warned, woman warned, both warned, or neither warned. This allows direct and spillover effects to differ by sex as well as the warning status and BP of the other spouse. Distinguishing treatment states by direct and indirect exposure is consistent with advances in multidimensional RDD for causal effects with spillovers (interference) (Aronow *et al.*, 2017; Auerbach *et al.*, 2024; Borusyak and Kolerman-Shemer, 2025), including Torrione *et al.* (2024) who propose an estimator useful for networks with several peers and high-dimensional treatment boundaries. In our dyadic setting, we can estimate the two-

dimensional response surface directly and recover frontier-specific direct and spillover effects that are interdependent.

We find no evidence that a high BP warning has a direct effect on the recipient's BP about 2.5 years later but estimate large and asymmetric spillovers. Warning a man reduces his wife's systolic BP by 7.5% and her probability of having high BP by 24 percentage points (pp), while there is no evidence of a female-to-male spillover. Point estimates of male-to-female spillovers are even larger when females are not directly warned about their BP. When they are, estimated spillover effects of their husband's warning are smaller, suggesting that direct exposure attenuates the effect of indirect exposure. The results are robust to alternative RDD specifications and sample selections as well as to estimation in outcome changes rather than levels.

We show that the gendered pattern of results is consistent with joint health production under collective but unequal household decision making (Browning and Chiappori, 1998). If households prioritize male health, warning a man about a health risk can induce larger household investment in shared health inputs than the same warning to a woman. The health gain to a woman can be larger, however, if shared inputs are more productive in generating female health, possibly due to offsetting effects of male-prevalent unhealthy habits.

We use unidimensional RDD to test for unidirectional intergenerational spillovers and estimate that warning a parent about high BP causes an almost 5% reduction in their adult child's systolic BP 2.5 years later. Father-to-child spillovers are even larger: an estimated 8.8% reduction in BP and an almost 14 pp fall in the probability of high BP. Further stratification by child sex suggests these effects are driven by father-to-son spillovers, although a paucity of data on father-daughter dyads constrains the comparison. This pattern of results is again consistent with collective household behavior that is more responsive to warnings about male health and displays a son preference. It would also be compatible with a parental health warning being more salient to the sex-concordant child.

We capitalize on rich health and nutrition survey data to test for mechanisms through

diagnosis and medication of hypertension, health knowledge, diet, health behavior and adiposity. The male-to-female spousal spillover effect on BP appears to go through a healthier diet and reduced adiposity. We estimate that warning a man about high BP reduces his (unwarned) wife's body mass index by over 8%. The intergenerational spillover seems to run through an adult child's acquisition of health knowledge and adoption of a healthier lifestyle involving less smoking.

Warnings about high BP issued to survey respondents have been used to estimate direct effects of screening for hypertension (Zhao *et al.*, 2013; Ciancio *et al.*, 2021; Dai *et al.*, 2022; Pedron *et al.*, 2022; Kämpfen and Mosca, 2024; Kämpfen *et al.*, 2026). We extend the evidence by adding estimates of spillover effects on spouses and adult children. This reveals potential for a social multiplier (Glaeser *et al.*, 2003) from screening for the principal modifiable risk factor for cardiovascular disease (CVD), the main cause of death globally.

Health and longevity are known to be positively correlated between spouses (Meyler *et al.*, 2007) and between parents and children (Currie and Moretti, 2007; Halliday, 2023; Black *et al.*, 2024; Zeltzer *et al.*, 2025).[1] These correlations could arise through unobservables, endogenous selection and simultaneity, the classic impediments to identification of causal spillovers (Manski, 1993, 2013; Angrist, 2014). Studies exploiting plausibly exogenous variation in the timing of exposure to health shocks find effects on relatives' healthcare use (+), preventive behavior (+) and mental health (-) (Fadlon and Nielsen, 2019; Hodor, 2021; Glaser and Pruckner, 2023; Böckerman *et al.*, 2025). Postulated mechanisms include health salience, risk information, shared constraints and emotional strain. A limitation of the health-shock strategy is that it identifies the effect of an acute event that is not directly malleable to policy. We identify family responses to information delivered through screening: a warning about chronic disease risk potentially signaling shared exposure to genetic, behavioral or environmental risks. This advantage is shared with Hoagland (2025),

[1]Cardiovascular health (Di Castelnuovo *et al.*, 2009) and BP (Beresford, 1976) are also concordant within families.

who finds that diagnosis of a chronic condition in the U.S. increases relatives' medical spending, including on low-value preventive care.

Evidence on public health program spillovers mostly concerns communicable disease vaccination or screening (Oster, 2018; Carpenter and Lawler, 2019; Bouckaert *et al.*, 2020), where spillovers operate mainly through infection externalities. Family spillovers from screening for non-communicable disease (NCD) can operate only through information and behavior. There is some evidence of behavioral spillovers between spouses in smoking and alcohol use (Cutler and Glaeser, 2010; Fletcher and Marksteiner, 2017), although other studies attribute spousal smoking concordance to assortative mating (Clark and Etilé, 2006; Palali and Van Ours, 2017). We contribute causal evidence of family health spillovers from hypertension screening that has implications for the evaluation and design of CVD prevention programs and demonstrates that prevention even of NCD can generate positive externalities.

Testing for family spillovers from BP screening is particularly relevant in China, where hypertension is highly prevalent and poorly managed (Li and Ge, 2015; Zhang *et al.*, 2023). Family ties are close, intergenerational co-residence remains common and household decision making is often gendered (Brown, 2009; Emery *et al.*, 2019; Huang *et al.*, 2023).

We make three main contributions. First, we extend evaluation of NCD risk screening to include within-family responses, providing causal evidence on policy-malleable spillovers from China, where the NCD burden is large and family health production is likely to matter. Second, we show that spillovers can be large and asymmetric by sex, consistent with joint health production that responds more strongly to information about male health but generates larger health gains for women. Third, we introduce a practical multidimensional RDD approach for dyads that separates direct from spillover effects and allows each to vary with the other dyad member's treatment status.

II. Theoretical framework

The collective household model (Browning and Chiappori, 1998) has been shown to predict behavior of Chinese families (Brown, 2009; Huang *et al.*, 2023) and used to consider implications of family health spillovers for the cost-effectiveness of medical care (Basu and Meltzer, 2005).[2] We use this framework to show how warning someone about their high BP could affect the health behavior and outcomes of family members, and why these spillovers may differ by sex of the person warned. Details are in Appendix A.

Preliminaries.—Consider a household comprising a couple, with the two individuals distinguished by sex: male (M) and female (F).[3] Each individual, $J \in \{M, F\}$, derives utility, U^J, from their health, H^J, and (within-household) public consumption, C: $U^J = U^J\left(H^J, C; I^J\right)$, where U^J is increasing at a diminishing rate in H^J and C, and I^J is exogenous health information.[4] A warning about high BP provides information, which increases the salience of health improvement and so raises the marginal utility of health, $U^J_{HI} = \frac{\partial^2 U^J}{\partial I^J \partial H^J} > 0$. This characterization of the mechanism through which a BP warning affects behavior is pertinent because high BP is largely asymptomatic. Being warned about high BP does not correspond to revelation of a health deterioration that immediately lowers utility through bothersome symptoms. Rather, it signals that improving underlying health by lowering BP reduces the risk of serious future disease, and so health gain is more valuable than would be appreciated without a warning. There is evidence that raised health salience is an important mechanism underlying family health spillovers (Fadlon and Nielsen, 2019).

Health is produced by individual effort, e^J, and a household shared input, X, representing

[2] Guidelines recommend inclusion of family spillovers in health economic evaluations (Neumann et al., 2016; NICE, 2026), but implementation is limited by scarcity of robust evidence (Henry *et al.*, 2024).

[3] There are no same-sex couples in our data.

[4] We assume egoistical health preferences to demonstrate that (asymmetric) spillovers can emerge even without (asymmetric) altruism. See Appendix A.

joint consumption of healthy goods and activities: $H^J = H^J\left(e^J, X\right)$. This allows male and female health to respond differently to the shared input. For example, men may skip healthy home-cooked meals more often or undermine their health impact by indulging more in salty, high-fat food while drinking with friends.

The household acts as if maximizing a weighted sum of the two individuals' utilities subject to constraints imposed by health technology and (exogenous) income. Assuming utility is additively separable in health and consumption, approximating the utility of consumption and using the (binding) income constraint to substitute for consumption, the household problem can be written as

$$\max_{e^M, e^F, X} (1-\theta)u^M(H^M; I^M) + \theta u^F(H^F; I^F) - c^M(e^M) - c^F(e^F) - c(X), \tag{1}$$

where $u^J(\cdot)$ is health utility, $\theta \in [0,1]$ is a Pareto weight on the woman's welfare, which increases with her bargaining power, $c^J(\cdot)$ and $c(\cdot)$ are utility-equivalent cost functions for individual health effort and the shared health input, respectively, that are increasing and convex (see Appendix A for derivations). Evidence from China shows household bargaining power and resource allocation are shaped by gender norms, property rights and marriage market conditions (Qian, 2008; Brown, 2009; Wang, 2014; Huang *et al.*, 2023).

Behavioral response.—Subject to simplifying assumptions about preferences and health production, a high BP warning that imparts information increases both the recipient's health effort ($de^J/dI^J > 0$) and the shared health input ($dX/dI^J > 0$). The latter response can be larger. It will be if the ratio of the marginal products of the shared input and the recipient's effort is greater than the relative steepness of the loss function at the respective margins (Appendix A). In that case, whatever the balance of bargaining power, the household chooses to produce additional health for the person receiving the warning through investment in its shared health environment more than through increased recipient effort. This scenario is more likely when the recipient is less productive in generating health by their own effort or faces high opportunity costs of doing so. Higher average male earnings increase the relative costs

(to the household) of male health effort. If, compared with women, men also find exercise, self monitoring or treatment adherence more difficult, then the response through shared household health investment relative to that through recipient effort would be predicted to be greater for BP warnings to men than it is for warnings to women.

If information raises the marginal utility of health equally for men and women ($u^M_{HI} = u^F_{HI}$), then the comparative magnitudes of shared input responses to male and female BP warnings depend on relative productivity and welfare weights:

$$\frac{dX}{dI^M} > \frac{dX}{dI^F} \quad \Longleftrightarrow \quad \frac{H^M_X}{H^F_X} > \frac{\theta}{1-\theta},$$

where $H^J_X = \frac{\partial H^J}{\partial X}$. When less weight is placed on female welfare ($\theta < 0.5$), a male warning can induce greater household health investment than a female warning even if the shared input is less productive in generating male health.

Spousal spillovers.—Since information impacts the shared input, possibly more than it induces individual effort, there is a channel for a potential spillover from a high BP warning to the health of the recipient's spouse. If the magnitude of the shared input response to the warning varies with the recipient's sex or the productivity of that input is sex specific, then the spillover will be asymmetric by sex.

The spillover can be larger than the direct effect on the recipient of the warning. This may occur, for example, when warnings to men generate a stronger response through shared household health investment than through male effort, and when that shared input is less productive for male health than for female health. This combination of circumstances would increase the likelihood that warning a man about his high BP induces a larger improvement in his wife's health than in his own: $\frac{dH^F}{dI^M} > \frac{dH^M}{dI^M}$ (Appendix A).

Since giving less weight to female welfare weakens the behavioral response to a female BP warning through the household shared input, it also weakens the spillover effect on the husband's health, which is reduced further if the shared input is less productive for male health. Consequently, if women are disadvantaged in the distribution of bargaining power but advantaged in the health gained from any given level of household health investment,

then the effect from a male BP warning to female health is likely to be the stronger of the two spillovers.

Intergenerational spillovers.—A similar framework gives reason to expect spillover from a parent's BP warning to the health behavior and health of an adult child (Appendix A). There are two possible mechanisms. First, assume the parent and adult child are co-resident and the latter's health production function is $H^C = H^C(e^C, X)$, where e^C is the adult child's health effort and X is again the household shared health input. Then, any increase in that input induced by the parent's BP warning will benefit the adult child's health, even if that does not (partly) motivate the increased household joint health investment.

Second, a parental warning may also provide information about the adult child's future risk because BP is shaped by inherited traits and shared family environments. If this information is transmitted, it may raise health salience for the child and motivate their preventive effort and, in the case of co-residence, the household shared health input. Under mild assumptions, both inputs would respond positively to a parental health warning (Appendix A).

Putting the two mechanisms together, the intergenerational health spillover from warning a parent (P) about high BP, and so increasing their health information (I^P), is

$$\frac{dH^C}{dI^P} = H_e^C \frac{de^C}{dI^P} + H_X^C \frac{dX}{dI^P} > 0.$$

A parental health warning may be considered more relevant to the offspring's health when the parent and child share the same sex. With these beliefs, warning a father (mother) about high BP would have a stronger impact on the salience and marginal utility of their adult son's (daughter's) health. Consequently, the spillover effects on the child's effort and the shared input would be larger when there is parent-child sex concordance.

III. Data

A. Study design

Setting.—We use data from China between 1989 and 2015, a period of epidemiological transition toward NCD. Hypertension prevalence is estimated to have peaked at around 30% of the population aged 18-69 in 2010, before falling back to about 25% in 2018 (Zhang *et al.*, 2023). The burden of hypertension is exacerbated by low rates (in 2018) of awareness (38%), treatment (35%) and control (12%) (Zhang *et al.*, 2023). Uncontrolled hypertension accounts for approximately one third of CVD-related deaths (Lewington *et al.*, 2016). Chronic disease management is improving, however, partly through village doctor (Sun *et al.*, 2022) and mobile health interventions (Jia *et al.*, 2021; Yan *et al.*, 2021).

Sample.—We use data from the China Health and Nutrition Survey (CHNS), which is a longitudinal household panel of multiple cohorts across heterogeneous provinces (Popkin *et al.*, 2010). The sampling is based on a multistage random cluster design implemented within purposively selected provinces (nine in the initial wave), covering approximately 3,800 households and 16,000 individuals at baseline in 1989. Data are collected approximately every two years. In 1997, the sample was expanded to new households in the original communities and to include an additional province. It was further extended to three megacities in 2011 (China Health and Nutrition Survey, 2025; Atlas of Longitudinal Datasets, 2025).

In each wave, data are collected on all individuals living in each sampled household. If possible, households that change address are followed if they remain within the original community. Individuals, including adult children, who leave a household are also followed if they remain in the community.

Treatment.—In each wave and for each household, a trained enumerator follows standard procedure to measure BP of each individual aged 7 years and older on each of three consecutive days of data collection. From the three measurements obtained, mean systolic

BP and mean diastolic BP are calculated and communicated to the respondent (Zhao *et al.*, 2013; Dai *et al.*, 2022). If mean systolic BP ≥ 140 mmHg or mean diastolic BP ≥ 90 mmHg, the respondent is warned that their measured BP is high and advised to visit a clinic for confirmation and to follow medical advice (Dai *et al.*, 2022). While this warning is not a clinical diagnosis of hypertension, the thresholds correspond to those most widely used in clinical guidelines for diagnosis and treatment. No information about severity is given and no specific treatment is recommended (Zhao *et al.*, 2013; Dai *et al.*, 2022). We use the deterministic relationship between the continuous measurements of BP and the issue of a warning on crossing either the systolic or diastolic threshold at baseline to estimate causal effects of that *treatment* on subsequent BP outcomes (Zhao *et al.*, 2013; Sudharsanan *et al.*, 2020; Ciancio *et al.*, 2021; Dai *et al.*, 2022; Pedron *et al.*, 2022; Kämpfen and Mosca, 2024; Kämpfen *et al.*, 2026).

Sample selection.—We use individual level records from the publicly available Relation Master File, which records all household relationships among individuals interviewed across CHNS waves from 1989 onward. We construct spousal and intergenerational dyads comprising married couples and parent-child relationships, respectively. We restrict the analysis samples to dyads with three measurements of systolic BP and three measurements of diastolic BP for each individual in two consecutive waves. Intergenerational dyads are constructed from the household relationship roster by linking each parent to each observed child within the household.[5] For each dyad, we use only the first two waves with complete BP data. We refer to the first and second waves as *baseline* and *follow-up*, respectively. Calender dates of baseline and follow-up vary across dyads. Spousal dyads are restricted to those in which both individuals are at least 30 years old at baseline and have complete BP

[5]A dyad is a specific parent-child pair. If both parents are interviewed and linked to one child who also participated in the survey, this generates two dyads: father-child and mother-child.

information.[6] We restrict parent-child dyads to those in which the child is at least 18 years at baseline.

There is heaping in distributions of mean BP (across three measurements) at multiples of 10 mmHg (Appendix Figure B.1). Since the cutoffs for issuing a high BP warning are at such multiples, the heaping could generate discontinuities in baseline BP distributions at treatment assignment cutoffs, which would be inconsistent with the continuity assumption required in RDD. Following standard practice (Barreca *et al.*, 2011, 2016), we address this by excluding from the analysis sample observations with a mean systolic (diastolic) BP over three baseline measurements of exactly 140 (90) mmHg. We test robustness to not making this exclusion.

The analysis samples consist of 3595 spousal dyads and 2988 intergenerational dyads (Appendix Table B.1).

Primary outcomes.—Three primary outcomes measured at follow-up are: (a) *Systolic BP* - mean systolic BP from three measurements (mmHg); (b) *Diastolic BP* - mean diastolic BP (mmHg); and, (c) *High BP* - an indicator of uncontrolled BP above hypertension thresholds $(= \mathbb{1}(\textit{Systolic BP} \geq 140 \vee \textit{Diastolic BP} \geq 90))$. In supplementary analysis, we estimate effects on changes in mean systolic BP and mean diastolic BP between baseline and follow-up.

Secondary outcomes.—A warning of high BP could lower the BP of the recipient, their spouse or adult child through various mechanisms. The recipient may be induced to consult a physician, who, after further BP measurements, might diagnose hypertension, prescribe antihypertensive medication or offer lifestyle advice about diet, exercise, smoking and alcohol consumption that is passed on to relatives. A warning may also cause a spouse or adult child to go for a medical check-up, possibly leading to diagnosis, medication or lifestyle advice. It could also prompt family-wide search for information about risks of high BP and how to control BP. In response to the acquired knowledge or medical advice, healthier individual or family collective behaviors may be adopted, which could lower body weight and excess fat

[6] We test robustness to setting the age threshold at 25 and 35.

as well as BP.

We explore mechanisms by estimating effects on six secondary outcomes measured at follow-up: (i) *Diagnosis* - an indicator of reported diagnosis of high BP or hypertension (0/1); (ii) *Medication* - an indicator of reported use of antihypertensives (0/1); (iii) *Knowledge* - an index of assessed knowledge of healthy diet, exercise and body weight; (iv) *Diet* - an index of reported healthy eating; (v) *Behavior* - an index of reported exercise, smoking and alcohol consumption behavior; and, (vi) *Adiposity* - an index of measured body weight and shape. Each of outcomes iii)-vi) is a composite index constructed as a weighted average of multiple indicators, with greater weight given to any indicator that correlates less with the others (Appendix B). We use these composites, which are in standard deviation units, to deal with the multiple comparisons problem (Anderson, 2008) and to report results more concisely. We scale indicators to ensure that outcomes iii), iv) and v) are increasing with health knowledge, healthy diet and healthy behavior, respectively. In contrast, higher values of outcome vi) correspond to less healthy body weight and shape. In the analysis of intergenerational dyads, we do not estimate effects on *Diagnosis* and *Medication* because these outcomes vary little among (relatively young) adult children.[7]

B. Descriptives

Baseline sample descriptives are in Table 1. In the spousal dyads sample, mean age is about 45 years for females and is 47 years for males. The respective means of years of education are around 7 and 9 years. On average, the baseline and follow-up interviews were conducted in early 1997 and late 1999, respectively. Mean systolic BP and mean diastolic BP are both slightly higher for men than for women. Around 15% of men and 11% of women have systolic BP or diastolic BP above the respective hypertension cutoff and so were warned of high BP at baseline.

[7]Approximately 1% of adult children were diagnosed with hypertension at follow-up, with only a fraction receiving medication.

Table 1: Sample characteristics at baseline

	Spousal dyads				
	Male		Female		
	Mean	SD	Mean	SD	N
Age (years)	47.01	11.91	44.95	11.28	3594
Education (years)	9.01	4.06	7.14	4.61	3586
Systolic BP (mmHg)	120.69	17.45	116.32	18.16	3595
Diastolic BP (mmHg)	78.30	10.86	75.41	11.09	3595
High BP (0/1)	0.153	0.36	0.111	0.314	3595
Baseline year	1997.1	6.46	1997.1	6.46	3595
Follow-up year	1999.7	6.61	1999.7	6.61	3595
	Intergenerational dyads				
	Parent		Child		
	Mean	SD	Mean	SD	N
Age (years)	55.26	10.08	26.25	8.40	2988
Education (years)	5.75	4.60	10.15	3.39	2963
Systolic BP (mmHg)	122.76	20.89	112.11	12.26	2988
Diastolic BP (mmHg)	78.01	12.15	73.82	9.24	2988
High BP (0/1)	0.197	0.398	0.068	0.251	2988
Baseline year	1995.5	5.97	1995.5	5.97	2988
Follow-up year	1998.0	6.18	1998.0	6.18	2988

Notes: Spousal dyads are married couples. Each sample is restricted to dyads with complete blood pressure (BP) measurements in two consecutive waves. Only the first pair of such waves is used. *Baseline* and *Follow-up* refer to the first and second wave, respectively. See text for other sample selections. *Systolic BP* and *Diastolic BP* are means over three measurements. High BP = $\mathbb{1}(\text{Systolic BP} \geq 140 \vee \text{Diastolic BP} \geq 90)$, where each BP is the respective mean. All statistics are measured at baseline, except follow-up year. SD denotes standard deviation.

In the sample of intergenerational dyads, the average parent is aged around 55, while the average adult (18+) child is about 26 years. On average, children have almost double the years of education of their parents. Parents have higher BP: around 30% of parents are warned of high BP at baseline, compared with only 8% of adult children. On average, parents and children are observed at baseline around mid 1995, with follow-up at the beginning of

1998.[8]

Appendix Tables B.3 and B.4 give descriptives of secondary outcomes and the indicators used to construct the composite indices.

IV. Identification and estimation

A. Preliminaries

We use RDD to identify the effect of a high BP warning at baseline on each outcome at follow-up under the assumption that respondents just below and just above each BP cutoff that triggers a warning are comparable in observed and unobserved characteristics, such that the potential outcome would evolve smoothly through the cutoffs if no warning were issued. Within-individual BP fluctuation helps make location just above or just below each cutoff plausibly exogenous.[9] We estimate direct effects on the recipient of a warning and spillover effects on their spouse or adult child. Identification of spillover effects rests on the assumption that the potential outcome of the spouse/adult child would evolve smoothly with the recipient's BP through the cutoffs if the recipient were not warned on crossing them. Since spousal and intergenerational dyads form before warnings are issued, there is no concern about endogenous selection (Manski, 1993). We assume there are no between family spillovers and identify those arising from social interactions within groups (families) that are treatment invariant and non-manipulable (Manski, 2013).

At baseline, each respondent (i) has three measurements (t) of systolic BP (s_{it}) and of diastolic BP (d_{it}) from which we derive a treatment (high BP warning) indicator, $D_i = \mathbb{1}\{\bar{s}_i \geq 140 \vee \bar{d}_i \geq 90\}$, where $\bar{x}_i = \sum_{t=1}^{3} x_{it}/3$, $x \in \{s, d\}$. Since this treatment can be triggered

[8]There are 2,988 intergenerational dyads, spread across 1,539 households, corresponding to an average of 1.94 dyads per household.

[9]Regression to the mean is unlikely to invalidate the RDD because mean reversion is not expected to change discontinuously at cutoffs.

by either $\bar{s}_i$ or $\bar{d}_i$ crossing the respective cutoff, we construct a single (binding-score) running variable that determines treatment assignment, $A_i \equiv \max\{\tilde{s}_i, \tilde{d}_i\}$, where $\tilde{x}_i = {}^{(\bar{x}_i - c_x)}/_{\sigma_x}$, $c_s = 140$, $c_d = 90$ and σ_x is the standard deviation of $\bar{x}_i$.[10] Then, $D_i = \mathbb{1}\{A_i \geq 0\}$, with the treatment frontier at $A_i = 0$. This centering method of reducing a two-dimensional assignment rule to a unidimensional running variable (Wong *et al.*, 2013), which underpins binding-score RDD (Reardon and Robinson, 2012), has been used to estimate direct effects of BP warnings (Pedron *et al.*, 2022; Kämpfen and Mosca, 2024; Kämpfen *et al.*, 2026).[11]

B. Spousal spillovers

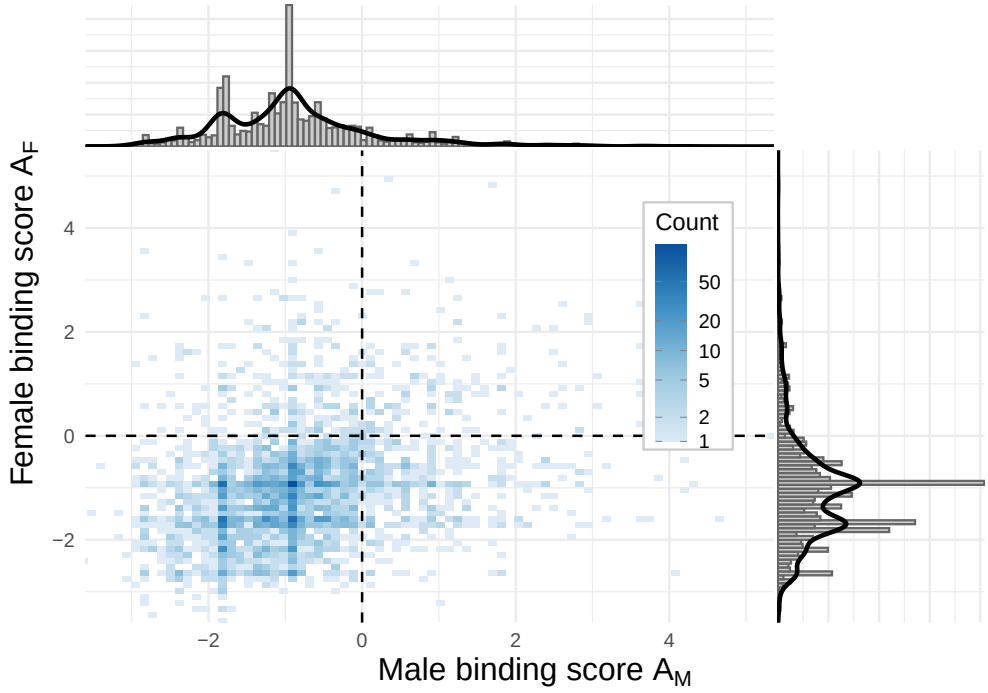
Dropping the individual index, let A_M and A_F be the male and female binding scores within a spousal dyad. Figure 1 shows the joint density of these running variables together with their marginal distributions. While most dyads are in the bottom-left quadrant, indicating that neither the male nor the female gets a BP warning, many are located in the neighborhood of each dimension of the treatment frontier. Substantial numbers are treated through warnings to the male only, the female only and both.

Exposure to a BP warning can be direct, as a recipient, or indirect, as the spouse of a recipient. RDD estimates of spillovers typically rely on a unidimensional cutoff (Dahl *et al.*,

[10] For analysis of spousal dyads, we use the standard deviation over respondents of the same sex as the respective individual. For analysis of intergenerational dyads, we use standard deviation over parents.

[11] Kämpfen *et al.* (2026) also use a multidimensional RDD that allows the effect of a BP warning due to high systolic BP to differ from that due to high diastolic BP, with each potentially varying along the other frontier. Allowing for such heterogeneity while also estimating direct and spillover effects within a dyad would substantially increase the dimensionality of the problem, complicate identification and reduce statistical power. We use a binding score to focus on effects of a warning irrespective of which component of BP triggers it.

Figure 1: Joint and marginal densities of male and female binding scores



Notes: $N = 3595$. Male (Female) binding-score running variable, A_M (A_F) on x-axis (y-axis). Dashed lines define the treatment frontier. Each dot represents one or more dyads. Darker dots indicate greater density. Histograms and (Gaussian) kernel density fits above and to the right of the scatterplot depict marginal distributions of A_M and A_F, respectively.

2014; Oral *et al.*, 2026). This requires restricting attention to individuals near an indirect exposure cutoff but sufficiently far from a cutoff at which they would be treated directly, which avoids confounding the spillover effect with the direct effect. In our application, this would identify spillovers only for spouses whose own BP is either clearly below or clearly above a cutoff that is commonly used to diagnose hypertension. Such estimates would have limited policy relevance. We avoid this limitation by using a multidimensional RDD (MRDD) approach that allows direct and indirect exposure to vary jointly around both treatment frontiers, enabling sex-specific identification of the direct effect, the spillover effect and their interaction within a single local design.

Define male and female direct treatment indicators, $D_J = \mathbb{1}\{A_J \geq 0\}$, $J \in \{M, F\}$. Let Y_J be a respective follow-up outcome. We estimate a saturated, local-linear outcome regression

with intercepts and slopes that can vary flexibly across the four quadrants of treatment exposure, $\{D_M, D_F\} \in \{0,1\}^2$ (Papay *et al.*, 2011):

$$\begin{aligned} Y_J = {} & \alpha + \alpha_M D_M + \alpha_F D_F + \alpha_{MF} D_M D_F \\ & + \beta_M A_M + \beta_F A_F + \beta_{MF} A_M A_F \\ & + \delta_M A_M D_M + \delta_F A_F D_M + \delta_{MF} A_M A_F D_M \\ & + \gamma_M A_M D_F + \gamma_F A_F D_F + \gamma_{MF} A_M A_F D_F \\ & + \eta_M A_M D_M D_F + \eta_F A_F D_M D_F + \eta_{MF} A_M A_F D_M D_F \\ & + \mathbf{Z}^\top \varphi + \upsilon, \end{aligned} \tag{2}$$

where $\mathbf{Z}$ includes age and years of schooling and υ is a random error. This fully interacted specification allows the response surface to differ flexibly on each side of the respective cutoff at which the male or female gets a warning. We estimate by weighted least squares using triangular kernel weights over a rectangular neighborhood around the treatment frontier. We set bandwidths to the average optimal univariate RDD bandwidth across the main outcomes and confirm robustness to alternatives.[12] We also confirm robustness to replacing triangular with uniform and epanechnikov weights.

To estimate male/female frontier-specific effects that depend on the spouse's location relative to their own treatment cutoff, we use strip kernels that assign weights based on distance to the relevant frontier. For strip widths $b_J > 0$, we use one-dimensional triangular kernels,

$$K_J\left(\frac{|A_J|}{b_J}\right) = \begin{cases} 1 - \dfrac{|A_J|}{b_J}, & \text{if } |A_J| \le b_J, \\ 0, & \text{if } |A_J| > b_J. \end{cases}$$

These kernels assign weights based on proximity to the frontier in dimension J. We use these weights to construct a weighted empirical distribution of the orthogonal running variable

[12] The optimal bandwidths are $h_M = h_F = 0.8$. For $h_M > 0, h_F > 0$, we define the estimation sample $\mathcal{N}(h_M, h_F) = \{(A_M, A_F) : |A_M| \le h_M,\ |A_F| \le h_F\}$ and use product triangular weights $K(A_M, A_F) = \left(1 - \frac{|A_M|}{h_M}\right)\left(1 - \frac{|A_F|}{h_F}\right) \mathbb{1}\{|A_M| \le h_M,\ |A_F| \le h_F\}$. See Appendix Figure C.1.

among observations locally close to that frontier. We summarize this local distribution by its frontier-weighted mean and evaluate the fitted response surface at this representative value. This gives the corresponding frontier-average discontinuity.

For example, along the *male* frontier, $A_M = 0$, we define the frontier-weighted empirical means of the *female* running variable on each side of the female cutoff,

$$\begin{aligned} \bar{A}_{F^-} &= \frac{\sum A_F \, K_M(|A_M|/b_M) \, \mathbb{1}\{|A_M| \le b_M, \; A_F < 0, \; |A_F| \le h_F\}}{\sum K_M(|A_M|/b_M) \, \mathbb{1}\{|A_M| \le b_M, \; A_F < 0, \; |A_F| \le h_F\}}, \\ \bar{A}_{F^+} &= \frac{\sum A_F \, K_M(|A_M|/b_M) \, \mathbb{1}\{|A_M| \le b_M, \; A_F \ge 0, \; |A_F| \le h_F\}}{\sum K_M(|A_M|/b_M) \, \mathbb{1}\{|A_M| \le b_M, \; A_F \ge 0, \; |A_F| \le h_F\}}, \end{aligned} \tag{3}$$

where h_F is the bandwidth. $\bar{A}_{F^-}$ is the weighted average of A_F over observations with husbands close to the male frontier ($|A_M| \le b_M$) and with the female untreated ($A_F < 0$). $\bar{A}_{F^+}$ is defined analogously but with the female treated ($A_F \ge 0$). We define $\bar{A}_{M^-}$ and $\bar{A}_{M^+}$ analogously along the female frontier $A_F = 0$, using weights $K_F(|A_F|/b_F)$ and restricting to $|A_M| \le h_M$, where h_M is the bandwidth. For the main estimates, we set $b_M = b_F = 0.08$, based on a bias-variance tradeoff, and test robustness to alternative values.[13]

Using these frontier-weighted values of the spouse's running variable, we evaluate the fitted response surface to obtain four frontier-specific (weighted-average) effects on the male's

[13]The strip should be narrow enough for the (weighted) averages to reflect outcomes close to the frontier, but wide enough to include sufficient observations in each of the four frontier segments. For the main estimates, we therefore set the strip width to one tenth of the (optimal) bandwidth. Appendix Figure C.1 shows the strips. As an alternative to the empirical, frontier-weighted side means, Eq.(3), we test robustness to using *deterministic* side means implied by an assumed parametric distribution for the running variables (Appendix C).

outcome and on the female's outcome (i.e. eight effects):

$$\tau_{M^-} = \lim_{A_M \downarrow 0} \mathbb{E}[Y_J \mid A_M,\, A_F = \bar{A}_{F^-},\, D_F = 0] - \lim_{A_M \uparrow 0} \mathbb{E}[Y_J \mid A_M,\, A_F = \bar{A}_{F^-},\, D_F = 0],$$

$$\tau_{M^+} = \lim_{A_M \downarrow 0} \mathbb{E}[Y_J \mid A_M,\, A_F = \bar{A}_{F^+},\, D_F = 1] - \lim_{A_M \uparrow 0} \mathbb{E}[Y_J \mid A_M,\, A_F = \bar{A}_{F^+},\, D_F = 1],$$

$$\tau_{F^-} = \lim_{A_F \downarrow 0} \mathbb{E}[Y_J \mid A_F,\, A_M = \bar{A}_{M^-},\, D_M = 0] - \lim_{A_F \uparrow 0} \mathbb{E}[Y_J \mid A_F,\, A_M = \bar{A}_{M^-},\, D_M = 0],$$

$$\tau_{F^+} = \lim_{A_F \downarrow 0} \mathbb{E}[Y_J \mid A_F,\, A_M = \bar{A}_{M^+},\, D_M = 1] - \lim_{A_F \uparrow 0} \mathbb{E}[Y_J \mid A_F,\, A_M = \bar{A}_{M^+},\, D_M = 1].$$

The estimand τ_{M^-} is the effect of a BP warning to a man when his wife remains untreated (no warning), while τ_{M^+} is the effect of the same treatment when the wife also gets a warning. Each of these estimands can give a direct effect on a male outcome or a spillover effect on a female outcome. Using (2) and (3), we estimate side-specific frontier averages of these effects,[14]

$$\tau_{M^-} = \alpha_M + \delta_F \bar{A}_{F^-}, \qquad \tau_{M^+} = \alpha_M + \alpha_{MF} + (\delta_F + \eta_F)\, \bar{A}_{F^+}. \tag{4}$$

We estimate effects of warning a woman when her husband is untreated and treated by the female-frontier averages, $\tau_{F^-} = \alpha_F + \gamma_M \bar{A}_{M^-}$ and $\tau_{F^+} = \alpha_F + \alpha_{MF} + (\gamma_M + \eta_M)\, \bar{A}_{M^+}$, respectively.

Estimation of a saturated model that distinguishes all possible treatment exposure states avoids overlooking (or attributing negative weights to) spillover effects that are heterogeneous and nonlinear (Vazquez-Bare, 2023). This is important because the running variable and (direct) treatment status of the indirectly exposed are key dimensions along which a spillover

[14] Because (2) is local linear in A_M and A_F, the implied discontinuity when crossing one individual's frontier is affine in their spouse's running variable. Holding A_F fixed, jumps in the conditional expectation of Y_J at $A_M = 0$ are $\Delta_{M^-}(A_F) = \alpha_M + \delta_F A_F$ and $\Delta_{M^+}(A_F) = \alpha_M + \alpha_{MF} + (\delta_F + \eta_F) A_F$ where $\Delta_{M^-}(A_F)$ and $\Delta_{M^+}(A_F)$ are the discontinuities when the female spouse is untreated $(D_F = 0)$ and treated $(D_F = 1)$, respectively. Evaluating these expressions at the frontier-weighted values of the female spouse's running variable given by (3) yields (4).

effect may vary (ibid.).[15]

In addition to estimating frontier-specific effects of a BP warning to one individual conditional on treatment status of their spouse, we estimate the average effect over the spouse's treatment status:

$$\tau_{\bar{M}} = \omega_{M^-} \cdot \tau_{M^-} + \omega_{M^+} \cdot \tau_{M^+}, \qquad \tau_{\bar{F}} = \omega_{F^-} \cdot \tau_{F^-} + \omega_{F^+} \cdot \tau_{F^+},$$

where ω_{J^-} and ω_{J^+} are weights.[16] Finally, we summarize treatment effects on dyads by taking a weighted average of the male-frontier and female-frontier effects,

$$\tau = \omega_M \cdot \tau_{\bar{M}} + \omega_F \cdot \tau_{\bar{F}},$$

where ω_J are weights.[17]

We report estimates of τ_{M^-}, τ_{M^+}, τ_{F^-}, τ_{F^+}, $\tau_{\bar{M}}$, $\tau_{\bar{F}}$ and τ separately for male and female primary outcomes, giving direct and spillover effects. We use a bootstrap (1000 replications) over the whole procedure to estimate standard errors simultaneously for all estimated effects,

[15]Since there are only two individuals in a dyad, each can be influenced by only one peer. Hence, the *exchangeability assumption* – the potential outcome depends on the number of peers treated, not which ones are treated (Vazquez-Bare, 2023) – that is necessary for identification of spillovers holds trivially. In the dyadic case, the Torrione *et al.* (2024) distance-based estimator would consist of a set of one-dimensional RDDs – one for each relevant frontier. We can obtain these effects jointly, and less restrictively, from a saturated two-dimensional response surface because there is only one peer in each network (dyad).

[16]We estimate the weights by first computing frontier-weighted side masses along the male frontier, $A_M = 0$, $\mu_{M^-} = \sum K_M(|A_M|/b_M)\,\mathbb{1}\{|A_M| \le b_M,\ A_F < 0,\ |A_F| \le h_F\}$, and $\mu_{M^+} = \sum K_M(|A_M|/b_M)\,\mathbb{1}\{|A_M| \le b_M,\ A_F \ge 0,\ |A_F| \le h_F\}$, which measure the weighted mass of observations near that frontier when the female is untreated ($A_F < 0$) and treated ($A_F \ge 0$), respectively. We use these measures to calculate corresponding weights, $\omega_{M^-} = \frac{\mu_{M^-}}{\mu_{M^-}+\mu_{M^+}}$ and $\omega_{M^+} = 1 - \omega_{M^-}$, and define ω_{F^-} and ω_{F^+} analogously for the female frontier.

[17]$\omega_M = \frac{\mu_{M^-}+\mu_{M^+}}{\mu_T}$, $\omega_F = 1 - \omega_M$, where $\mu_T = (\mu_{M^-} + \mu_{M^+}) + (\mu_{F^-} + \mu_{F^+})$, with μ_{J^-} and μ_{J^+} weighted masses of observations defined in footnote[16].

such that inference takes account of the multiple comparisons.

We follow the same procedure to estimate effects on secondary outcomes that are potential mediators of any effects on the primary BP outcomes. To further uncover mechanisms, in supplementary analysis, we estimate effects on the separate components of each composite index used as a secondary outcome. In this case, we correct inference for multiple comparisons by calculating False Discovery Rate (FDR) sharpened q-values (Anderson, 2008), assuming that all components (of each index) belong to one outcome family.

C. Intergenerational spillovers

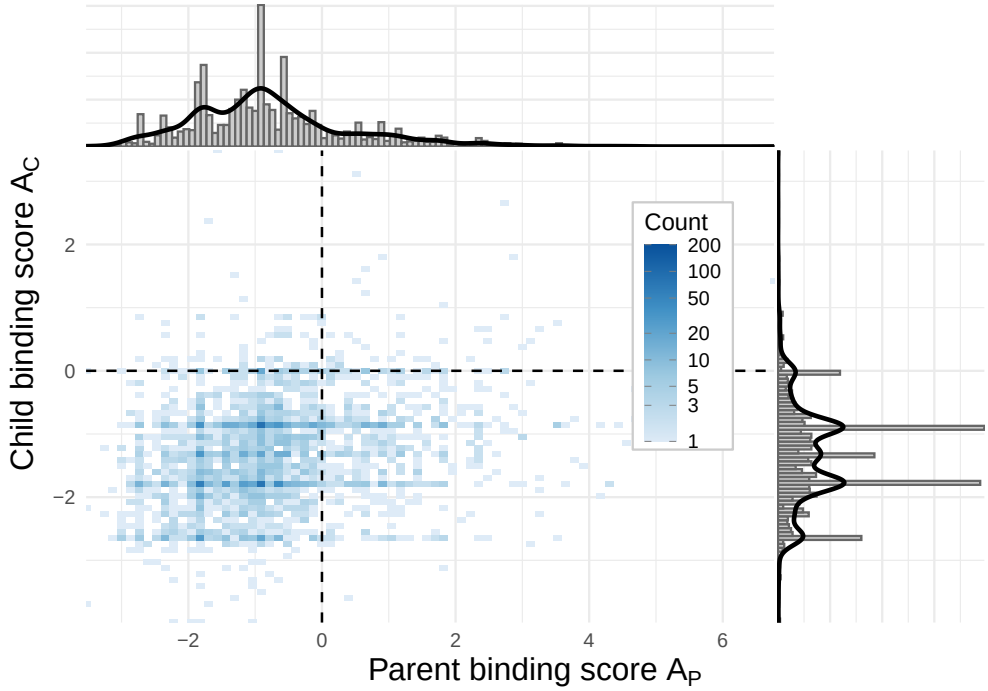
Figure 2 shows the joint density (and marginal distributions) of the binding-scores for parents, A_P, and adult children, A_C, in intergenerational dyads. The sparsity of observations above the dashed horizontal confirms that few children are warned at baseline, and so we do not attempt to estimate child-to-parent spillovers. We use unidimensional binding-score RDD to estimate parent-to-child spillovers, with little risk of confounding from direct exposure because so few children are warned. We also estimate direct effects of a parental warning on the parent.

Let $Y_G(1)$ and $Y_G(0)$ be potential outcomes when the parent does and does not receive a baseline BP warning, respectively, with $G \in \{P, C\}$ distinguishing parent from adult child outcomes. Assume expectations of these potential outcomes are continuous through the cutoff. Then, the average treatment effects of a BP warning to the parent on their outcome and on their child's outcome at the treatment cutoff are

$$\tau_G = \lim_{A_P \downarrow 0} \mathbb{E}[Y_G \mid A_P] - \lim_{A_P \uparrow 0} \mathbb{E}[Y_G \mid A_P] = \mathbb{E}[Y_G(1) - Y_G(0) \mid A_P = 0]\,. \tag{5}$$

We estimate these effects using local linear regression with triangular kernel weights that give emphasis to observations closest to the cutoff. Bandwidths are selected using the mean squared error (MSE) optimal procedure and are allowed to differ on either side of the cutoff

Figure 2: Joint and marginal densities of parent & adult child binding scores



Notes: $N = 2988$. Parent (adult child 18+) binding-score, A_P (A_C) on x-axis (y-axis). Dashed lines show treatment frontier. Each dot represents one or more observations (dyads). Darker dots indicate more observations (greater density). Histograms and (Gaussian) kernel density fits above and to the right of the scatterplot depict marginal distributions of A_P and A_C, respectively.

(Calonico *et al.*, 2014a,b, 2015). Inference is based on heteroskedasticity-robust plug-in variance estimators for local polynomial RDDs (Calonico *et al.*, 2017; Kolesár and Rothe, 2018).

Forming an intergenerational dyad for each parent-child combination within a family, pooling these dyads and estimating one parental spillover effect on an adult child relies on the exchangeability assumption (Vazquez-Bare, 2023): the effect is independent of which parent is warned. It also requires independence between (additive) spillover effects when more than one parent is warned. To relax the first assumption, we stratify by parental sex and estimate father-child and mother-child spillovers. Then, we further stratify by child sex and estimate a spillover effect for each the four parent-child sex combinations. This enables testing of the hypothesis that parent-child sex concordance increases spillover

strength. While stratification avoids pooling father and mother effects into a single average spillover effect, it stops short of estimating effects across all possible treatment exposure states defined by combinations of father and mother warnings.[18] Identification of the spillover effect of warning a parent requires that, locally around that parent's warning cutoff, the treatment status of the other parent evolves smoothly and is balanced on either side of the cutoff. Then, RDD identifies the effect of the focal parent's exposure to a warning, averaged over the local distribution of the other parent's exposure.

D. Validation analysis

As noted in section III., there is heaping in the full-sample baseline BP distributions that we deal with by dropping observations precisely at treatment assignment cutoffs and implementing a donut-hole RDD (Barreca *et al.*, 2011). This is a robust correction for heaping and potential non-random sorting (Barreca *et al.*, 2016). Even without the exclusion of observations, the appropriate density test that adjusts for mass points in the running variable (Cattaneo *et al.*, 2020) shows no discontinuity in any of those variables at any treatment cutoff (Appendix Table D.1).[19] However, there are significant discontinuities at cutoffs in some regions of the spouse's baseline binding score distribution.[20] Further, without these sample exclusions, there is some evidence of a discontinuity in age in one

[18] Vazquez-Bare (2023) recommends modeling the full vector of treatment exposures, which would comprise warning the father only, the mother only, both the father and mother, or no parent. Doing this separately by adult child sex would be infeasible with the available sample size.

[19] Without mass point adjustment, the density tests indicate large discontinuities at the cutoffs, as expected given the heaping. In that case, the t-statistics from the density tests for continuity in the male and female running variables are 6.99 ($p < 0.001$) and 7.10 ($p < 0.001$), respectively.

[20] These discontinuities, in the spouse's systolic BP, arise only conditional on the spouse's binding score being negative (Tables D.1 and D.2).

specification and in education in another (Table D.3). With the exclusions, there is no evidence of discontinuity in the density of any running variable (Figure D.1, Table D.5) nor in predetermined characteristics (Tables D.4 and D.6).

V. Results

A. Spousal spillovers

Figure 3 shows MRDD plots of each of the male and female primary outcomes against the male and female running variables. While the response surface of mean male systolic BP is uneven, it has no pronounced discontinuity that is consistent along the treatment frontier traced by a zero value of either running variable. The response surfaces for the other two male outcomes are rather flat. In contrast, there is a clear downward discontinuity in female systolic BP along the length of the frontier at which the male running variable crosses to the positive domain. This suggests that warning a man he has high BP leads to a fall in his wife's BP approximately 2.5 years later. For the other two female outcomes, downward discontinuities at the male frontier are somewhat obscured by elevation of the respective response surface beyond the frontier.

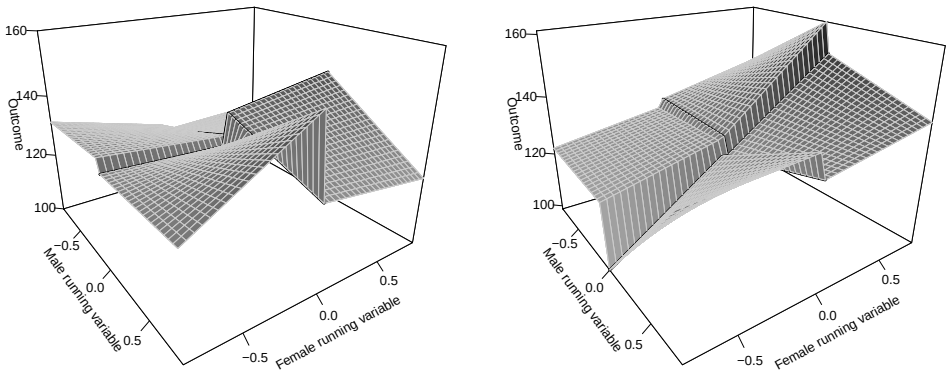
In Table 2, we report MRDD estimates of direct and spousal spillover effects of a BP warning. For parameters indexed with M, e.g. τ_{M-}, estimates for male outcomes are direct effects of warning a man he has high BP, while estimates for female outcomes are spillover effects on his wife. The reverse applies to F-indexed parameters. Consistent with Figure 3, we find no evidence of direct or spillover effects on male outcomes.

For female outcomes, there are no significant estimates of direct effects, but there appear to be substantial spillover effects: warning a man that he has high BP causes a reduction in his wife's BP at follow-up. These estimates of spillover effects, which are consistent with the discontinuities evident in Figure 3, are statistically significant when the wife is not warned that she has high BP (top row). In that case, the wife's systolic BP is estimated

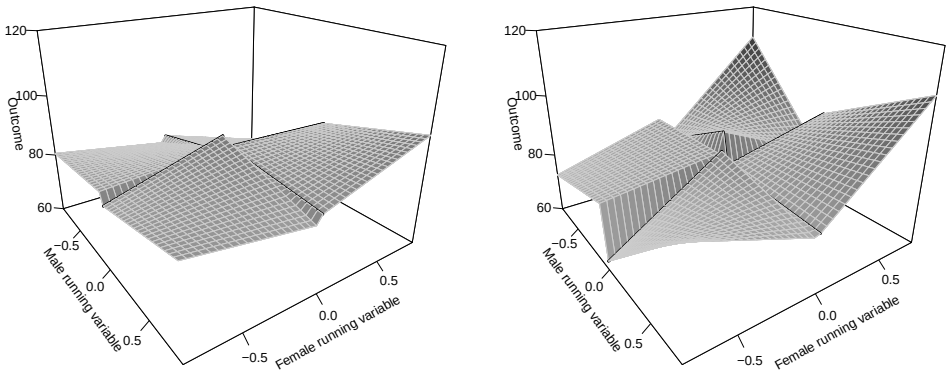
Figure 3: MRDD plots of outcomes against running variables by sex – spousal dyads

A) MALE B) FEMALE

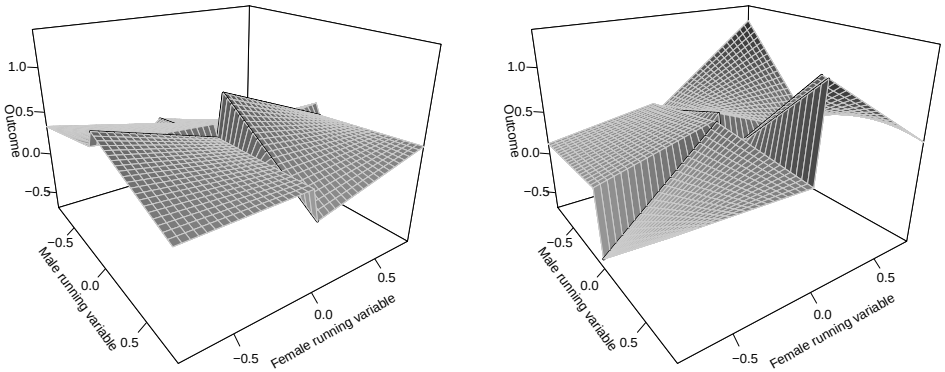
i) Systolic blood pressure



ii) Diastolic blood pressure



iii) High blood pressure



Notes: Mean outcome on z-axis against female (A_F) and male (A_M) binding-score running variables on x-axis and y-axis, respectively. At $A_J \geq 0, J \in \{F, M\}$, spouse J is warned at baseline they have high blood pressure (BP), i.e. $D_J = \mathbb{1}\{A_J \geq 0\}$ = $\mathbb{1}\{\textit{Systolic BP} \geq 140 \vee \textit{Diastolic BP} \geq 90\}$). Each point on grid is a predicted value from Eq. (2) at respective running variable values. Outcomes defined in Section III. All plots control for age and education.

to be reduced by over 10 mmHg (p-value = 0.014) – an 8.5% reduction relative to the counterfactual mean.[21] Her diastolic BP is estimated to be reduced by over 5 mmHg (p-value = 0.10) – a 6.4% reduction. We estimate that warning a man that he has high BP reduces the probability that his (unwarned) wife has high BP at follow-up by about 26 percentage points (p-value = 0.065).

In the second top row, the panel on the right gives estimates of spillovers to a wife who is warned at baseline that she has high BP herself. These estimates are slightly smaller in magnitude than those obtained when the wife is not directly treated, and they are less precise. Weighting effects of warning a man of high BP over the female running variable ($\tau_{\bar{M}}$ row) yields an estimated 9.4 mmHg reduction in his wife's systolic BP (p-value = 0.019; 7.5% decrease) and a 5.1 mmHg reduction in her diastolic BP (p-value = 0.067; 6.3% decrease), along with a 24 pp reduction in the probability she has high BP at follow-up (p-value = 0.049). Averaging the direct and spillover effects on female outcomes gives estimates (of τ, bottom row) that are smaller in magnitude than the respective spillover effects, with only the estimated effect on systolic BP being (marginally) significant.

Robustness.—The results are robust to a) varying the strip width used in (3) to calculate weighted means of the running variables along each frontier (Appendix Tables E.2 and E.3), b) calculating these means deterministically based on an assumed parametric distribution rather than empirically (Table E.4), c) varying the bandwidth (Tables E.5 and E.6),[22] d) using alternative weighting schemes to estimate the response surfaces (Tables E.7 and E.8), e) not controlling for age and education (Table E.9), f) excluding those diagnosed with hypertension at baseline (Table E.10), g) lowering the age threshold for sample selection to ≥ 25 years at baseline (Table E.11) and raising it to ≥ 35 (Table E.12), and h) estimating

[21] A counterfactual mean is computed as the average outcome among observations located "just below" each of the four frontiers, i.e. ≤ 0.1 of a standard deviation on the untreated side of the respective cutoff. Appendix Table E.1 gives estimates of these means.

[22] Narrowing the bandwidth gives less precise estimates of effects on diastolic BP.

Table 2: Direct & spousal spillover effects of high blood pressure warning on primary outcomes

		Male blood pressure			Female blood pressure		
		Systolic (mmHg)	Diastolic (mmHg)	High BP (0/1)	Systolic (mmHg)	Diastolic (mmHg)	High BP (0/1)
Male warning							
Wife unwarned	τ_{M-}	-3.292	1.21	-.046	-10.354**	-5.228*	-.257*
		(4.495)	(3.278)	(.143)	(4.203)	(3.178)	(.139)
Wife warned	τ_{M+}	9.668	3.164	.373	-7.115	-4.887	-.196
		(10.59)	(5.082)	(.247)	(9.124)	(5.379)	(.26)
Average	$\tau_{\bar{M}}$	.604	1.798	.08	-9.38**	-5.126*	-.239**
		(4.435)	(2.772)	(.124)	(3.985)	(2.798)	(.121)
Female warning							
Husband unwarned	τ_{F-}	-.063	-.852	-.117	1.174	1.515	.004
		(5.062)	(3.17)	(.164)	(5.876)	(4.575)	(.178)
Husband warned	τ_{F+}	1.504	-2.934	.29	-3.24	-4.696	-.13
		(9.539)	(6.013)	(.262)	(8.975)	(4.889)	(.313)
Average	$\tau_{\bar{F}}$	.268	-1.292	-.031	.242	.203	-.024
		(4.304)	(2.821)	(.148)	(4.985)	(3.826)	(.157)
Male-Female average	τ	.458	.456	.032	-5.201*	-2.811	-.146
		(3.118)	(1.807)	(.088)	(2.923)	(1.878)	(.089)
N		384	384	384	384	384	384

Notes: MRDD estimates of the treatment effects (discontinuity parameters) defined in Section IV.B. Based on weighted least squares estimates of local linear regression Eq. (2) using triangular kernel weights over a rectangular neighborhood around treatment frontier, with optimal univariate RDD bandwidth of 0.8. Effects derived using empirical, frontier-weighted side-means estimated with one-dimensional triangular kernel and strip width of 0.08. Systolic and diastolic blood pressure (BP) are respective means (over three measurements) at follow-up. High BP is an indicator of BP exceeding the hypertension thresholds at follow-up (= $\mathbb{1}(\textit{Systolic BP} \geq 140 \vee \textit{Diastolic BP} \geq 90)$). See section III. for detailed definitions. Bootstrapped (1000 repetitions) standard errors in parentheses. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

effects on changes in systolic and diastolic BP between baseline and follow-up rather than using follow-up levels as outcomes (Table E.13). Using unidimensional RDD to estimate effects separately at each of the male and female treatment frontiers yields a similar pattern of results: warning a man that he has high BP significantly reduces his wife's BP when she is not warned herself and there is no evidence of direct effects for either sex, nor is there evidence of female-to-male spillover effects (Table E.14).

When observations with baseline BP precisely at the treatment thresholds are retained – to move from donut-hole MRDD to MRDD – the estimated spillover effects on female diastolic BP and the probability of having high BP are similar in magnitude and precision to the main estimates, whereas the estimated male-to-female spillover effect on systolic BP

decreases in magnitude and becomes statistically insignificant (Table E.15). Using this extended sample, we estimate negative and significant effects of a warning to a woman on her husband's systolic and diastolic BP when he is also warned. However, the weighted averages of the female-to-male spillover effects are not statistically significant.

B. Intergenerational spillovers

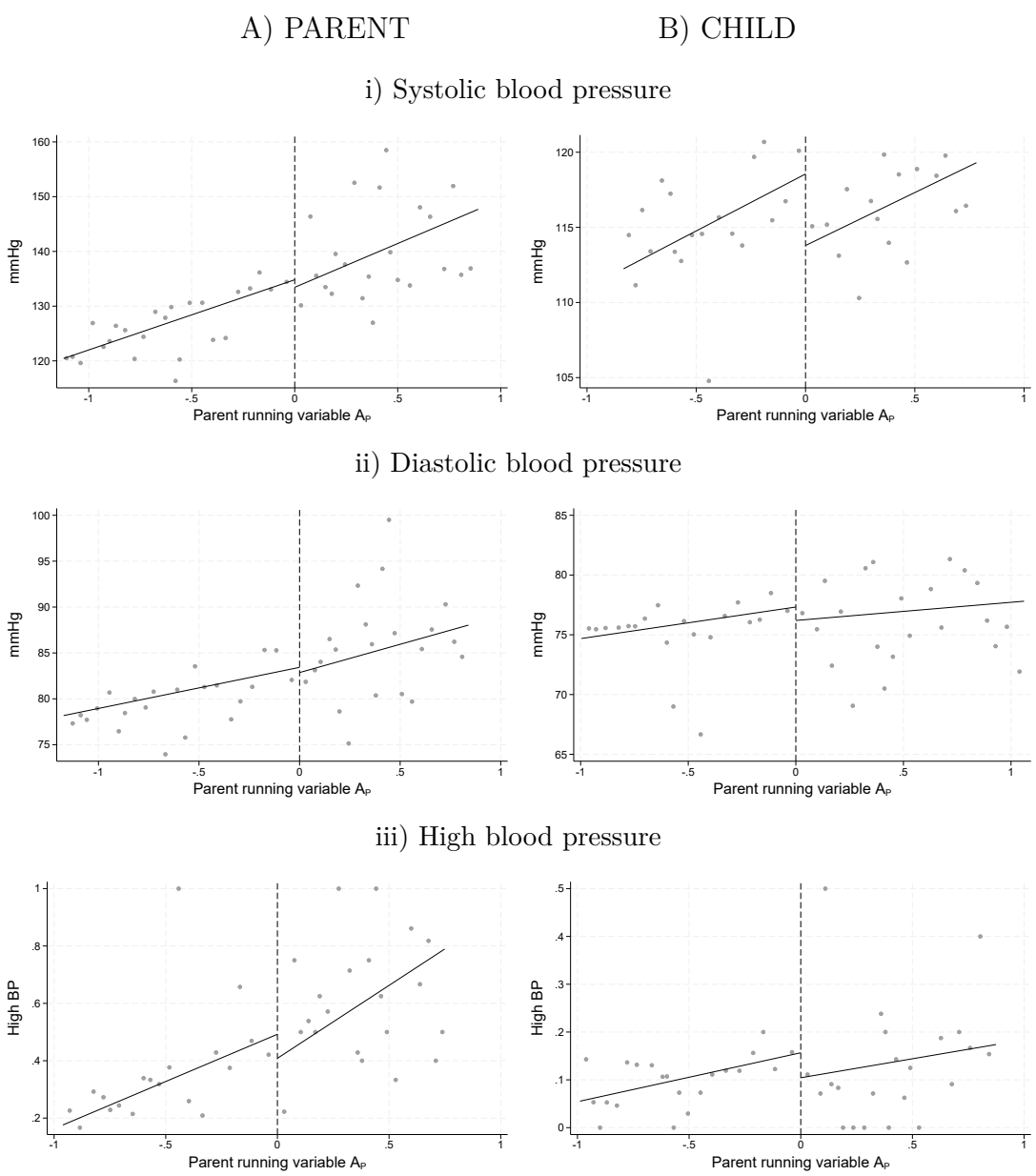
Unidimensional RDD plots in Figure 4 do not reveal a marked discontinuity in any of the primary outcomes of a parent when their baseline BP crosses the cutoff (0) at which they are warned about high BP. However, at this cutoff, there is a clear downward discontinuity in their adult child's systolic BP at follow-up, which is consistent with a parent-to-child spillover.

Table 3 presents unidimensional RDD estimates of effects of warning a parent about their high BP. Panel A gives direct effects on the parent's BP and spillover effects on an adult child's BP. Consistent with Figure 4, point estimates of all direct effects are negative but none is statistically significant.[23] Confirming the discontinuity evident in the top-right of Figure 4, we estimate that warning a parent of their high BP reduces their adult child's systolic BP by 5.8 mmHg (p-value = 0.025; 4.9%) almost 2.5 years later. Estimated spillover effects on diastolic BP and probability of the adult child having high BP at follow-up are not statistically significant.[24]

[23]Using BP change as the outcome, there is a statistically significant (p-value = 0.007) estimated 5.6 mmHg reduction in the parent's diastolic BP between baseline and follow-up (Appendix Table E.16 and Figure E.1).

[24]Estimating effects on changes in adult child BP between baseline and follow-up also reveals significant effects on systolic BP but not on diastolic BP (Appendix Table E.16 and Figure E.1). Stratifying by child sex reveals that the spillover effect of warning a parent appears to mainly reflect reduced systolic BP of sons, with the estimated effect on daughters being smaller and less precise (Table E.17, Panel A).

Figure 4: Parent & adult child primary outcomes against parent running variable



Notes: Mean outcome on y-axis against parent running variable (A_P) on x-axis. High blood pressure (BP) is an indicator of BP above hypertension thresholds (= $\mathbb{1}\{Systolic\ BP \geq 140 \vee Diastolic\ BP \geq 90\}$). At $A_P \geq 0$, the parent is warned at baseline they have high BP. Each dot is the mean outcome in a given bin. We use optimal bins obtained from the variance quantile-spaced method using spacing estimators (Calonico *et al.*, 2014a, 2017). Best-fit lines estimated with local linear regression, triangular kernels and the MSE optimal bandwidth selector. Outcomes defined in Section III.

The top row of panel B reveals that warning a father about his high BP is estimated to

Table 3: Direct & intergenerational spillover effects of high blood pressure warning to parent

	Systolic BP (mmHg)		Diastolic BP (mmHg)		High BP (0/1)	
A. Parent warning						
Parent	-2.545	(3.052)	-1.201	(1.663)	-0.127	(.096)
N	1010		1451		987	
Adult child	-5.852**	(2.614)	-1.474	(1.879)	-0.069	(.061)
N	879		1458		1341	
B. Father warning						
Adult child	-10.450***	(3.730)	-6.341**	(3.085)	-0.139**	(.070)
N	322		312		646	
Son	-10.271**	(4.077)	-5.863	(3.679)	-0.163*	(.092)
N	240		259		465	
Daughter	-5.908	(7.663)	-4.754	(5.107)	0.010	(.019)
N	103		77		51	
C. Mother warning						
Adult child	-0.587	(2.860)	2.828	(2.514)	0.018	(.093)
N	993		531		748	
Son	0.402	(2.979)	3.548	(2.766)	-0.013	(.109)
N	592		393		394	
Daughter	-1.690	(8.725)	1.185	(6.134)	0.191	(.270)
N	154		123		106	

Notes: Unidimensional RDD estimates of effects of warning a parent about high blood pressure at baseline. Panel A gives direct effects on parent and spillover effects on adult child. Panel B (C) gives spillover effects of warning a father (mother) on any adult child, a son and a daughter. Outcomes are defined in Section III. High BP is indicator of BP above hypertension thresholds ($= \mathbb{1}\{\textit{Systolic BP} \geq 140 \vee \textit{Diastolic BP} \geq 90\}$). Local linear regressions with triangular kernels and MSE optimal bandwidths selected separately on each side of the threshold. Heteroscedasticity robust standard errors reported in parentheses. N denotes the effective number of observations used in estimation. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

reduce all three adult child BP outcomes, while there is no evidence in the top row of panel C that warning a mother has any spillover effect. This mirrors the spousal spillover results. Warning a man that he has high BP results in both his spouse and adult child having lower BP about 2.5 years later. Further stratifying by the sex of the child, it appears that the latter effect mainly arises through same-sex spillover from father to son. Estimated spillovers from father to daughter BP are smaller in magnitude and less precise, with the relatively small effective samples used for these estimates contributing to their imprecision.

Robustness.—The finding that warning a parent about high BP causes their adult child to

have lower systolic BP is robust to a) estimating with local quadratic instead of local linear regression (Appendix Table E.17 Panel B), b) using a common MSE optimal bandwidth on both sides of the cutoff (Table E.17 Panel C), c) using alternative kernels (Tables E.18 Panels A and B), d) not controlling for age and education (Table E.17 Panel C), e) clustering standard errors at the household level, rather than relying on heteroskedasticity robust plug-in variance estimators (Table E.19 Panel A), f) excluding parents who were diagnosed with hypertension at baseline (Table E.19 Panel B), and g) not excluding parents with baseline BP precisely at the treatment thresholds (Table E.19 Panel C).[25] We do not test robustness of the child-sex-stratified estimates because they are based on relatively small sample sizes, which already gives cause for cautious interpretation.

C. Mechanisms

Given there is no evidence of a significant direct effect or a female-to-male spillover effect, we focus on mechanisms that may underlie the evident spillover effect from warning a man to his wife's BP. Table 4 gives estimates of spillover effects on a wife's secondary outcomes. Warning a man about high BP when his wife is not warned, which is estimated to have a significant spillover effect on her BP, leads to the wife reporting a healthier diet (p-value = 0.051) and having a less unhealthy body weight/shape (p-value = 0.003). The estimates correspond to approximately a 0.5 standard deviation (SD) improvement in the *Diet* index and a 0.75 SD reduction in the *Adiposity* index relative to no-warning counterfactuals.[26]

Estimates of effects on each component of the adiposity index suggest that warning a

[25] With this final specification, we also estimate a significant direct negative effect on the parent's systolic BP. However, this should be interpreted cautiously since the identification assumption of continuity through the threshold is not satisfied when observations that heap at this value are included.

[26] Appendix Table F.1 gives counterfactual means and SDs for each outcome computed using observations ≤ 0.1 SD from the respective treatment frontier on the untreated side.

Table 4: Spillover effects of male high blood pressure warning on wife's secondary outcomes

		Diagnosis	Medication	Knowledge	Diet	Behavior	Adiposity
Wife unwarned	τ_{M^-}	.014	-.016	-.313	.563*	.162	-.956***
		(.097)	(.08)	(.367)	(.288)	(.175)	(.324)
Wife warned	τ_{M+}	-.169	-.098	-.528	-.261	-.211	.462
		(.243)	(.257)	(.576)	(.358)	(.309)	(.679)
Average	$\tau_{\bar{M}}$	-.038	-.04	-.374	.327	.055	-.549*
		(.099)	(.093)	(.305)	(.242)	(.152)	(.319)
	N	387	390	219	395	394	391

Notes: MRDD estimates of treatment effects (discontinuity parameters) defined in Section IV.B. on secondary outcomes. Based on weighted least squares estimates of the local linear regression (2), using triangular kernel weights over a rectangular neighborhood around treatment frontier, with a bandwidth of 0.8. Effects derived using empirical, frontier-weighted side-means estimated with a one-dimensional triangular kernel and strip width of 0.08. For consistency, we use the bandwidth and strip width from the main analysis. See section III. and Appendix F for definitions of the outcomes. Bootstrapped standard errors in parentheses calculated with 1000 repetitions over all estimates simultaneously. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

man about high BP reduces his (unwarned) wife's i) body mass index (BMI) by 2.05 (FDR q-value = 0.017; 8.4%), ii) likelihood of being overweight by 23.7 pp (FDR q-value = 0.017), and iii) likelihood of having a waist-to-height ratio (WHR) above a high-risk threshold for cardiometabolic diseases by 36.6 pp (FDR q-value = 0.002) (Table F.2).[27]

Interestingly, we estimate that warning a man about his high BP significantly (p-value = 0.012) reduces his own adiposity index (when his wife is untreated) (Table F.5). This direct effect is similar in magnitude to the respective spillover effect and appears to result from reductions in BMI (-1.542 ~ 6.3%; FDR q-value = 0.103), WHR (-0.034 ~ 6.7%; FDR q-value = 0.103) and likelihood of each exceeding the respective high-risk threshold (Table F.6). However, the reduction in male body weight does not appear to feed through to reduced BP,

[27]Estimating effects of a male BP warning on components of the wife's composite diet index gives a positive estimate for the likelihood of the wife cooking for the household in the last week that is marginally significant without correcting for multiple comparisons (p-value = 0.067) but not after doing so (FDR q-value = 0.455) (Table F.2). There are no statistically significant effects on components of the wife's knowledge index (Table F.3) or her behavior index (Table F.4).

Table 5: Intergenerational spillover effects of high blood pressure warning to parent on adult child's secondary outcomes

	Knowledge		Diet		Behavior		Adiposity	
A. Parent warning								
Adult child	0.612**	(.304)	0.168	(.173)	0.323**	(.156)	0.640**	(.303)
N	355		897		843		1007	
B. Father warning								
Adult child	0.279	(.425)	0.163	(.256)	0.330	(.217)	1.033**	(.515)
N	143		375		334		734	
Son	0.085	(.412)	0.192	(.261)	0.286	(.227)	0.908	(.580)
N	102		316		239		577	

Notes: Unidimensional RDD estimates of intergenerational spillover effects of a high blood pressure (BP) warning on secondary outcomes defined in Section III. and Appendix B. Panel A gives effects of a warning to a parent on their adult child. Panel B gives effects of warning a father on any adult child and a son. Local linear regressions with triangular kernels and MSE optimal bandwidths selected separately on each side of the threshold. Heteroscedasticity robust standard errors reported in parentheses. N denotes the effective number of observations used in estimation. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

in contrast to the pattern observed for wives. This asymmetry is consistent with evidence that obesity is more strongly associated with hypertension among women than men, and that weight loss predicts lower future BP for women but not men (Kawasoe *et al.*, 2020; Nagahata *et al.*, 2025).

Table 5 gives intergenerational spillover effects of a high BP warning on secondary outcomes.[28] Estimates in panel A suggest that a parental warning improves an adult child's health knowledge and behaviors but worsens their adiposity. An explanation for the conflicting effects would be that cutting back on smoking leads to weight gain, especially for younger adults (Lycett *et al.*, 2010; Courtemanche *et al.*, 2016). Consistent with this, estimation of spillover effects on each component of the composite indices reveals apparent reductions in the probability and extent of adult child smoking (as well as a reduced probability of drinking alcohol), although the estimates lose statistical significance after

[28]Consistent with estimates of null direct effects of a high BP warning on the parent's primary BP outcomes (Table 3), there are no significant direct effects on secondary outcomes (Table F.7).

correcting for multiple comparisons (Table F.8). In any case, it appears that warning a parent about high BP reduces their adult child's BP through improved knowledge of risk factors and adoption of healthier lifestyles that dominate the negative effect of increased body size, the BP consequences of which may only become evident later in life.

Panel B shows estimates of spillover effects from warning a father. Sample sizes become smaller and estimates less precise. For this reason, and because there is no significant father-to-daughter spillover effect on BP, we do not estimate effects on a daughter's secondary outcomes. For father-to-child and father-to-son effects, magnitudes of the estimates for the behavior and adiposity indices are similar to the magnitudes of the respective parent-to-child effect estimates. This again suggests that the principal intergenerational spillover is from fathers to sons. The father spillover estimates for the knowledge index are much smaller than the parent spillover estimate and not at all close to significance, with the latter expected since the sample becomes rather small for this outcome.[29]

VI. Conclusion

This study extends accumulating evidence of family health spillovers (Fadlon and Nielsen, 2019; Hodor, 2021; Hoagland, 2025) by showing that health information can percolate through a family to influence behaviors and health risks of the spouse and adult child of the recipient of information. Screening for non-communicable disease risks may therefore generate benefits that extend beyond those screened. Ignoring these spillovers could result in underestimation of the social value of screening programs and missed opportunities to design prevention policies that lever the family as an important channel of health production. While within-family spillovers may be internalized in household decision making – if each member does not act independently and egoistically – policymakers may overlook these spillovers and so underestimate program effectiveness.

[29]The questions used to construct the knowledge index are not fielded in all waves.

We estimate large spousal and intergenerational spillovers from a warning about high blood pressure (BP) despite the apparent absence of a direct effect on the warning recipient. This pattern is consistent with heterogeneous individual behavioral responses to shared information and the collective household response impacting individuals' health differently. Information about a relative's high BP may signal shared exposure to cardiovascular risk that causes some (but not all) individuals to change their health behavior, and it may induce household investment in health that crowds out individual investments differentially across a family.

We find strong male-to-female spousal spillovers but no evidence of spillovers in the opposite direction. Estimated intergenerational effects are particularly pronounced when the warned parent is male. These asymmetries are consistent with joint household health production that is more responsive to male health warnings, possibly due to unequal bargaining power, gendered caregiving roles and greater salience of male health. A household response motivated by concern about male health may, nevertheless, disproportionately improve female health if shared inputs, such as diet, are more productive for women because they are less likely than men to undermine the collective health investment through unhealthy habits. Another study of China also finds evidence of gendered household health production, with retirement of women increasing both time allocated to that production and their husbands' health, but no such positive health spillover from male retirement (Bai *et al.*, 2024).

Methodologically, we propose a practical approach to estimate spillovers in dyads when each individual may also be directly exposed to treatment. The multidimensional regression discontinuity design framework allows direct and indirect exposure to vary jointly around two treatment frontiers, with estimation of bi-directional spillovers that depend on direct exposure. It is consistent with Manski's (2013) recommendation to define outcome response as a function of the full vector of treatment exposures to allow for within-group social interactions. This matters in our application because the male-to-female spillover appears

stronger when the wife is not directly warned about her own BP, which would be overlooked in a unidimensional design that restricted attention to one margin of treatment exposure.

While ignoring spillovers risks underestimation of health gains from screening and other prevention and healthcare programs, our results demonstrate that this need not be the case. Policymakers would likely anticipate positive direct effects on those warned about their high BP, which we do not find. For males, the absence of a direct effect may reflect individual behavior that is insufficiently responsive to the warning, with unhealthy habits offsetting collective household health investments. For females, the lack of a direct effect may be due to weak collective responses of sex-biased households to warnings about women's health. Both scenarios, which are no more than that, point to the potential centrality of the family for understanding health behavior. The broader lesson is that both direct and indirect effects need to be estimated rather than assumed.

Study limitations deserving emphasis include estimates that are local to dyads with BP near cutoffs that trigger a warning and so may not generalize to individuals with much lower or, more important, higher BP. The warning comes from BP measurement in a survey, not a clinical screening program. Warnings delivered by health workers, possibly accompanied by treatment advice, may have different, plausibly stronger, effects. Finally, the Chinese setting is particularly interesting given the high burden of hypertension, strong family ties and gendered household structures, but these features may limit generalization to other contexts.

Notwithstanding these limitations, we contribute to sparse evidence that prevention of chronic disease risks can have effects extending beyond those targeted. These spillovers may be large, asymmetric and shaped by collective household behavior. Recognizing such spillovers potentially has consequences for the evaluation and design of prevention programs. Our results support viewing health behavior as arising from interactions between individuals embedded in families that share information, resources, risks and routines. For non-communicable diseases, for which prevention and management often depends on sustained

behavioral change, recognition of this family dimension may well be essential to effective intervention.

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Appendix

A Theoretical framework - details

A. Setup

We use the collective household framework (Browning and Chiappori, 1998) with household production (Apps and Rees, 1997; Chiappori, 1997) to model household behaviors and outcomes as if arising from maximization of a weighted average of the household members' utilities. Basu and Meltzer (2005) use this framework to extend cost-effectiveness analysis of medical treatments to take account of family health spillovers.[1]

In a cohabiting/married couple household consisting of a male (M) and a female (F), each individual is assumed to derive utility from their own health, H^J, $J \in \{M, F\}$, and consumption, C, that is a public good within the household: $U^J = U^J(H^J, C; I^J)$, where I^J is exogenous information about own health that affects utility only through the value of health. Specifically, health information is assumed to increase the salience of health improvement and so raise the marginal utility of health, $U^J_{HI} = \partial^2 U^J / \partial I^J \partial H^J > 0$. This idea is consistent with evidence that family health spillovers run through health salience (Fadlon and Nielsen, 2019). A health warning, such as being told blood pressure (BP) is high, increases information.[2] We model the impact of a warning on behavior through this information

[1] Jacobson (2000) uses a unitary household model to extend Grossman's 1972 individual model of health production. Further extensions model the distribution of within-household health and consumption as if emerging from cooperative Nash bargaining (Bolin *et al.*, 2001) and non-cooperative (Cournot) strategic behavior (Bolin *et al.*, 2002). The collective model relaxes the restriction imposed in the unitary model that the household acts as if controlled by a benevolent dictator and the restriction of the cooperative game theory model that bargaining is specifically of the Nash variety (Vermeulen, 2002). The only restriction imposed is that the household's allocation is Pareto efficient, which is not guaranteed if non-cooperative behavior is assumed.

[2] Given receipt of a warning is a dichotomous event, we assume $\frac{\partial U^J}{\partial H^J}\Big|_{I^J_1} - \frac{\partial U^J}{\partial H^J}\Big|_{I^J_0} > 0$, where

mechanism.

Utility is assumed to be increasing at a diminishing rate in both endogenous arguments: $U_H^J = \partial U^J / \partial H^J > 0$, $U_{HH}^J = \partial^2 U^J / \partial H^J \partial H^J \leq 0$, $U_C^J > 0$ and $U_{CC}^J \leq 0$. This specification imposes egoistical preferences over health: each individual within a household cares only about their own health. While this simplification is unrealistic (for happily married couples!), it serves our purpose of demonstrating that health warnings to males and females can have asymmetric spousal spillover effects even when this is not hard-wired through the specification of preferences. Allowing altruistic health preferences would strengthen spillover effects because the spouse of the recipient of a warning would be motivated to respond out of direct concern about their partner's health. While this would complicate the comparative static analysis, it is unlikely to change the qualitative result derived below, provided females are not less altruistic with respect to spousal health. If we were to assume that females are more altruistic and incorporated this in the model, then it would provide another mechanism for gender asymmetry in the spillover effect.

Each individual is assumed to produce their health through own effort, e^J, and a shared household composite input, X:

$$H^J = H^J(e^J, X), \quad J \in \{M, F\}, \tag{A.1}$$

with diminishing marginal returns to each input, $H_e^J > 0$, $H_{ee}^J < 0$, $H_X^J > 0$ and $H_{XX}^J < 0$. Effort is exerted to refrain from unhealthy (out-of-home) food, alcohol and smoking, and to undertake physical exercise and adhere to medication, for example. The shared input captures joint consumption of healthy, home-cooked meals, adoption of joint exercise routines, household regimes to cut down on alcohol and smoking, and household arrangements to seek preventive health care.[3] Specifying individual-specific health production functions,

I_1^J and I_0^J indicate information with and without receipt of a health warning, respectively.

[3]Joint health production through a shared input is absent from previous models of health production within a household (Jacobson, 2000; Bolin *et al.*, 2001). These models do allow

$H^J(\cdot)$, allows any given change in the shared input to differentially affect male and female health. For example, while home-cooking is available for sharing, the male may participate less or he may avoid the healthy vegetable and fruit options. He may also be more likely to undermine the health gain from wholesome family meals by indulging more in salty and high-fat dishes while drinking with friends outside the home. He may observe family exercise regimes less.

The household's problem is

$$\max_{e^M,e^F,X,C}(1-\theta)U^M(H^M,C;I^M)+\theta U^F(H^F,C;I^F)$$

subject to the health technology, (A.1), and the budget constraint,

$$C+k^M(e^M)+k^F(e^F)+k(X)\leq Y,$$

where $\theta\in[0,1]$ is the Pareto (welfare) weight on the female's utility, which may be increasing in her bargaining power, Y is household income (assumed exogenous), and $k^J(\cdot)$ and $k(\cdot)$ are (opportunity) costs of producing health through effort and investment in the shared input. Costs include expenditures on healthy consumption, e.g. high-protein and high-vitamin foods, and time costs incurred by forgoing earnings opportunities to invest in health, e.g. time spent shopping for healthy food, cooking and exercising. We assume marginal costs are positive and non-decreasing: $k_e^J>0$, $k_{ee}^J\geq 0$, $k_X>0$ and $k_{XX}\geq 0$.

Assume utility is additively separable in health and consumption, such that $U^J(H^J,C;I^J)=u^J(H^J;I^J)+v(C)$, and that the budget constraint is binding. Then, after substituting for consumption, the maximand is

$$(1-\theta)u^M(H^M;I^M)+\theta u^F(H^F;I^F)+v\big(Y-k^M(e^M)-k^F(e^F)-k(X)\big).$$

for child health, which is treated as a public good within the household. However, child health is also assumed to be produced only through individual-specific inputs.

We approximate consumption utility by taking a first-order Taylor expansion around Y:

$$v\big(Y - k^M(e^M) - k^F(e^F) - k(X)\big) \approx v(Y) - v_Y\big[k^M(e^M) + k^F(e^F) + k(X)\big],$$

where v_Y denotes the derivative of $v(\cdot)$ with respect to its argument, evaluated at Y.[4] Dropping the constant term, $v(Y)$, which does not affect choices, the problem becomes

$$\max_{e^M,e^F,X}(1-\theta)u^M(H^M;I^M) + \theta u^F(H^F;I^F) - v_Y k^M(e^M) - v_Y k^F(e^F) - v_Y k(X),$$

Defining utility-equivalent cost functions $c^M(e^M) \equiv v_Y k^M(e^M)$, $c^F(e^F) \equiv v_Y k^F(e^F)$ and $c(X) \equiv v_Y k(X)$,[5] yields Equation (1), the maximization problem in the manuscript:

$$\max_{e^M,e^F,X}(1-\theta)u^M(H^M;I^M) + \theta u^F(H^F;I^F) - c^M(e^M) - c^F(e^F) - c(X).$$

The first order conditions are

$$f_1 \equiv (1-\theta)u_H^M H_e^M - c_e^M = 0, \tag{A.2}$$

$$f_2 \equiv \theta u_H^F H_e^F - c_e^F = 0, \tag{A.3}$$

$$f_3 \equiv (1-\theta)u_H^M H_X^M + \theta u_H^F H_X^F - c_X = 0, \tag{A.4}$$

[4]Since v_Y is evaluated at Y, the utility cost of health investments may depend on income. We focus on responses to health information, rather than income effects, and so treat Y, and hence v_Y, as fixed in the comparative statics.

[5]Since $c^J(\cdot)$ and $c(\cdot)$ simply scale the cost functions $k^J(\cdot)$ and $k(\cdot)$, respectively, assumed signs of the first and second derivatives of the latter carry over to the former. These utility-equivalent cost functions can be interpreted as capturing the full costs of health production effort, including psychic costs arising from changes in behavior, such as exercising more, reducing alcohol consumption, quitting smoking and dieting, as well as monetary, time and opportunity costs of health investment.

where $u_H^J \equiv \frac{\partial u^J(H^J;I^J)}{\partial H^J}$, $c_e^J \equiv \frac{\partial c^J(e^J)}{\partial e^J}$ etc. and signs of first, second and cross derivatives of u^J are as assumed above for U^J. Given the assumptions made about the utility, health production and cost functions, the second order condition holds under the simplifying assumptions made below to approximate the comparative statics.[6]

B. Comparative statics for a male health warning

We first derive comparative statics for effects of a health warning to a male on their own health-production effort, their spouse's effort and the household's shared input into health production. We obtain the respective comparative statics for a health warning to a female by symmetry of the problem, although the effects can be asymmetric by gender, as will become clear. The exact comparative statics derived are complex expressions containing several interaction terms, making direct interpretation difficult. To improve transparency of the economic mechanisms, we approximate the comparative statics by focusing on the model's leading forces and abstracting from terms that are of second-order importance. This gives clear insight into how a health warning can generate spillovers, and why they may depend

[6]Without these simplifying assumptions, the third leading principal minor of the Hessian is not necessarily negative. Under the diagonal approximation used below in which the off-diagonal elements of the Jacobian are approximately zero, the second-order sufficient condition for a maximum is satisfied. Alternatively, one could impose joint concavity of $H^J(e^J, X)$ in (e^J, X), rather than merely assume concavity in each argument separately, together with concavity of U^J and convexity of costs. Under this stronger assumption, the household objective is concave, so the first order conditions characterize a maximum without relying on the diagonal approximation.

on whether the warning is issued to a man or a woman. Let

$$\mathbf{z} = (e^M, e^F, X)^\top, \qquad f(\mathbf{z}; I^M) = \begin{pmatrix} f_1(\mathbf{z}; I^M) \\ f_2(\mathbf{z}; I^M) \\ f_3(\mathbf{z}; I^M) \end{pmatrix}.$$

Since f_1 does not depend on e^F and f_2 does not depend on e^M, the Jacobian is

$$J_f = \frac{\partial f}{\partial \mathbf{z}} = \begin{pmatrix} f_{1e^M} & 0 & f_{1X} \\ 0 & f_{2e^F} & f_{2X} \\ f_{3e^M} & f_{3e^F} & f_{3X} \end{pmatrix}.$$

Key derivatives are

$$f_{1I^M} = (1-\theta) u^M_{HI} H^M_e, \tag{A.5}$$

$$f_{3I^M} = (1-\theta) u^M_{HI} H^M_X, \tag{A.6}$$

$$f_{1e^M} = (1-\theta)\Big(u^M_{HH}(H^M_e)^2 + u^M_H H^M_{ee}\Big) - c^M_{ee}, \tag{A.7}$$

$$f_{2e^F} = \theta\Big(u^F_{HH}(H^F_e)^2 + u^F_H H^F_{ee}\Big) - c^F_{ee}, \tag{A.8}$$

$$f_{1X} = (1-\theta)\Big(u^M_{HH} H^M_e H^M_X + u^M_H H^M_{eX}\Big), \tag{A.9}$$

$$f_{2X} = \theta\Big(u^F_{HH} H^F_e H^F_X + u^F_H H^F_{eX}\Big), \tag{A.10}$$

$$f_{3e^M} = (1-\theta)\Big(u^M_{HH} H^M_X H^M_e + u^M_H H^M_{Xe}\Big), \tag{A.11}$$

$$f_{3e^F} = \theta\Big(u^F_{HH} H^F_X H^F_e + u^F_H H^F_{Xe}\Big), \tag{A.12}$$

$$f_{3X} = (1-\theta)\Big(u^M_{HH}(H^M_X)^2 + u^M_H H^M_{XX}\Big) + \theta\Big(u^F_{HH}(H^F_X)^2 + u^F_H H^F_{XX}\Big) - c_{XX}. \tag{A.13}$$

If J_f is nonsingular, then, by the implicit function theorem, optimal choices are differentiable functions of I^M. Writing $\mathbf{z} = \mathbf{z}(I^M)$, so that $f(\mathbf{z}(I^M); I^M) = 0$, and differentiating this

identity with respect to I^M gives

$$\frac{d}{dI^M} f(\mathbf{z}(I^M); I^M) = \frac{\partial f}{\partial \mathbf{z}} \frac{d\mathbf{z}}{dI^M} + \frac{\partial f}{\partial I^M} = J_f \frac{d\mathbf{z}}{dI^M} + f_{I^M} = 0. \tag{A.14}$$

Hence, $\frac{d\mathbf{z}}{dI^M} = -J_f^{-1} f_{I^M}$, with $\frac{d\mathbf{z}}{dI^M} = \left(\frac{de^M}{dI^M}, \frac{de^F}{dI^M}, \frac{dX}{dI^M} \right)^\top$ and $f_{I^M} = (f_{1I^M}, 0, f_{3I^M})^\top$, since I^M enters f_1 and f_3, but not f_2. Substituting these expressions in (A.14) yields

$$\begin{pmatrix} f_{1e^M} & 0 & f_{1X} \\ 0 & f_{2e^F} & f_{2X} \\ f_{3e^M} & f_{3e^F} & f_{3X} \end{pmatrix} \begin{pmatrix} \frac{de^M}{dI^M} \\ \frac{de^F}{dI^M} \\ \frac{dX}{dI^M} \end{pmatrix} + \begin{pmatrix} f_{1I^M} \\ 0 \\ f_{3I^M} \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix}.$$

Multiplying row by row gives

$$f_{1e^M} \frac{de^M}{dI^M} + f_{1X} \frac{dX}{dI^M} + f_{1I^M} = 0, \tag{A.15}$$

$$f_{2e^F} \frac{de^F}{dI^M} + f_{2X} \frac{dX}{dI^M} = 0, \tag{A.16}$$

$$f_{3e^M} \frac{de^M}{dI^M} + f_{3e^F} \frac{de^F}{dI^M} + f_{3X} \frac{dX}{dI^M} + f_{3I^M} = 0. \tag{A.17}$$

Rearranging (A.15) gives

$$\frac{de^M}{dI^M} = -\frac{f_{1I^M} + f_{1X} \frac{dX}{dI^M}}{f_{1e^M}}. \tag{A.18}$$

Similarly, from (A.16),

$$\frac{de^F}{dI^M} = -\frac{f_{2X}}{f_{2e^F}} \frac{dX}{dI^M}. \tag{A.19}$$

Substituting (A.18) and (A.19) into (A.17) and rearranging gives

$$\frac{dX}{dI^M} = -\frac{f_{3I^M} - \frac{f_{3e^M} f_{1I^M}}{f_{1e^M}}}{f_{3X} - \frac{f_{3e^M} f_{1X}}{f_{1e^M}} - \frac{f_{3e^F} f_{2X}}{f_{2e^F}}}. \tag{A.20}$$

Substituting (A.20) back into (A.18) and (A.19) yields the exact comparative static effects

of a health warning to a male on e^M, e^F and X.

$$\frac{de^M}{dI^M} = \frac{-f_{1I^M}f_{2e^F}f_{3X} + f_{1I^M}f_{2X}f_{3e^F} + f_{1X}f_{2e^F}f_{3I^M}}{f_{1e^M}f_{2e^F}f_{3X} - f_{1e^M}f_{2X}f_{3e^F} - f_{1X}f_{2e^F}f_{3e^M}}, \tag{A.21}$$

$$\frac{de^F}{dI^M} = \frac{f_{2X}\left(f_{1e^M}f_{3I^M} - f_{1I^M}f_{3e^M}\right)}{f_{1e^M}f_{2e^F}f_{3X} - f_{1e^M}f_{2X}f_{3e^F} - f_{1X}f_{2e^F}f_{3e^M}}, \tag{A.22}$$

$$\frac{dX}{dI^M} = \frac{f_{2e^F}\left(f_{1I^M}f_{3e^M} - f_{1e^M}f_{3I^M}\right)}{f_{1e^M}f_{2e^F}f_{3X} - f_{1e^M}f_{2X}f_{3e^F} - f_{1X}f_{2e^F}f_{3e^M}}. \tag{A.23}$$

To simplify these expressions and gain insight, we assume the off-diagonal terms in the Jacobian (f_{1X}, f_{2X}, f_{3e^M}, f_{3e^F}) are small relative to the own curvature terms (f_{1e^M}, f_{2e^F}, f_{3X}). Then, the Jacobian is locally dominated by its diagonal elements and the exact system is well approximated by

$$J_f^{-1} \approx \begin{pmatrix} f_{1e^M}^{-1} & 0 & 0 \\ 0 & f_{2e^F}^{-1} & 0 \\ 0 & 0 & f_{3X}^{-1} \end{pmatrix}.$$

Using this approximation to simplify (A.21), and then using (A.5) and (A.7),

$$\frac{de^M}{dI^M} \approx -\frac{f_{1I^M}}{f_{1e^M}} = \frac{(1-\theta)u_{HI}^M H_e^M}{c_{ee}^M - (1-\theta)\left(u_{HH}^M (H_e^M)^2 + u_H^M H_{ee}^M\right)}. \tag{A.24}$$

Similarly, applying the approximation to (A.23) and using (A.6) and (A.13) gives

$$\frac{dX}{dI^M} \approx -\frac{f_{3I^M}}{f_{3X}} = \frac{(1-\theta)u_{HI}^M H_X^M}{c_{XX} - (1-\theta)\left(u_{HH}^M (H_X^M)^2 + u_H^M H_{XX}^M\right) - \theta\left(u_{HH}^F (H_X^F)^2 + u_H^F H_{XX}^F\right)}. \tag{A.25}$$

Under the approximation, $\dfrac{de^F}{dI^M} \approx 0$.

Further, we assume near zero values for the terms equal to the change in the marginal utility of health multiplied by the square of the marginal health product of a) effort, $u_{HH}^M (H_e^M)^2 \approx 0$, and b) the shared input, $u_{HH}^J (H_X^J)^2 \approx 0$. A sufficient condition for these approximations to

hold is that utility is close to linear in health over warning-induced deviations from the pre-warning optimum. Marginal health products below 1, and so their squares even further below 1, would give further grounds for the approximation. Then, (A.24) and (A.25) simplify to

$$\frac{de^M}{dI^M} \approx \frac{(1-\theta)u^M_{HI}H^M_e}{c^M_{ee} - (1-\theta)u^M_H H^M_{ee}} = \frac{(1-\theta)u^M_{HI}H^M_e}{D_e}, \tag{A.26}$$

and

$$\frac{dX}{dI^M} \approx \frac{(1-\theta)u^M_{HI}H^M_X}{c_{XX} - (1-\theta)u^M_H H^M_{XX} - \theta u^F_H H^F_{XX}} = \frac{(1-\theta)u^M_{HI}H^M_X}{D_X}, \tag{A.27}$$

where $D_e > 0$ and $D_X > 0$ because each input has non-deceasing marginal cost and diminishing marginal product. D_e approximates the rate at which marginal cost of effort rises above its marginal benefit when pushed beyond its optimum. That is, the steepness of the loss function at effort margin. D_X approximates the respective steepness of the loss function at the shared input margin.[7]

Taking the ratio of (A.27) to (A.26), it follows that

$$\frac{dX}{dI^M} > \frac{de^M}{dI^M} \quad \Longleftrightarrow \quad \frac{H^M_X}{H^M_e} > \frac{D_X}{D_e}. \tag{A.28}$$

The shared input response to a warning about the man's health exceeds his own effort response if the relative marginal product of the shared input exceeds the relative steepness of the loss function at the shared input margin versus the effort margin. In this scenario, whatever the degree of prioritization of male welfare $(1-\theta)$, it is advantageous, at the margin, to produce male health by modifying the household health environment rather than increasing male effort in direct response to the warning. For example, the man may find exercise, self monitoring or treatment adherence particularly costly, inconvenient or hard to sustain, especially if the opportunity cost for the household is high because he earns a higher wage than his wife. It may be less costly per unit of health produced to adjust common

[7] See (A.7) and (A.13) while noting that we have now assumed $u^M_{HH}(H^M_e)^2 \approx 0$ and $u^J_{HH}(H^J_X)^2 \approx 0$.

household inputs, such as food purchases, meal preparation, salt use and daily routines. Thus, the shared input margin may exhibit a larger behavioral response to a male health warning, even if increasing male effort would generate a larger health improvement, holding all else constant.

Health responses to a male health warning are

$$\frac{dH^M}{dI^M} = H_e^M \frac{de^M}{dI^M} + H_X^M \frac{dX}{dI^M}, \tag{A.29}$$

$$\frac{dH^F}{dI^M} = H_e^F \frac{de^F}{dI^M} + H_X^F \frac{dX}{dI^M}. \tag{A.30}$$

Under the assumption that the off-diagonal elements of the Jacobian are approximately zero, the indirect effect on the wife's health through her effort is negligible, and so the shared input channel dominates:

$$\frac{dH^F}{dI^M} \approx H_X^F \frac{dX}{dI^M}.$$

Male health also improves through the increase in the shared input, but that gain may be modest if the man avoids exposure, e.g. by skipping family meals, or indulges in unhealthy behavior, such as eating take-away food, drinking alcohol or smoking, that undermines household-level health production. If his wife is less prone to these behaviors, then her health would be more responsive to the increased household investment in the shared input ($H_X^F > H_X^M$), despite that investment being induced by a male health warning. Further, the man's own effort response may be limited if, as argued above, the relative cost of this adjustment is high. If female health is more responsive to the shared input and the man's own effort response is modest, then it is possible that a male health warning may have a larger impact on the health of the recipient's wife, $\frac{dH^F}{dI^M} > \frac{dH^M}{dI^M}$.

C. Comparative statics for a female health warning

A health warning to the woman increases information on her health, I^F, which raises the marginal utility derived from that health. By symmetry, and under the same assumptions

invoked above to simplify comparative static effects of a male health warning, we get expressions analogous to (A.26) and (A.27),

$$\frac{de^F}{dI^F} \approx \frac{\theta u^F_{HI} H^F_e}{c^F_{ee} - \theta u^F_H H^F_{ee}}, \tag{A.31}$$

$$\frac{dX}{dI^F} \approx \frac{\theta u^F_{HI} H^F_X}{c_{XX} - (1-\theta) u^M_H H^M_{XX} - \theta u^F_H H^F_{XX}}. \tag{A.32}$$

Since both numerators are scaled by θ, a smaller value of that parameter, representing lower weight on female welfare, attenuates the responses of both the woman's health effort and the household shared input to a female health warning. Less weight on female welfare implies more weight on male welfare $(1-\theta)$ and, consequently, magnification of male effort and shared input responses to a male health warning (see (A.26) and (A.27)). Compare shared input responses to male and female warnings given by (A.27) and (A.32), respectively. The denominators are equal. If information were to impact the marginal utility of health equally for males and females ($u^M_{HI} = u^F_{HI}$), then

$$\frac{dX/dI^M}{dX/dI^F} = \frac{(1-\theta)}{\theta} \frac{H^M_X}{H^F_X}.$$

In this case, a health warning to the male induces a larger increase in the shared health input than that resulting from a female health warning provided the relative (marginal) productivity of the input in generating male health is greater than the relative welfare weight on the female:

$$\frac{dX}{dI^M} > \frac{dX}{dI^F} \quad \Longleftrightarrow \quad \frac{H^M_X}{H^F_X} > \frac{\theta}{1-\theta}.$$

If greater weight is placed on male welfare ($\theta < 0.5$), possibly because the man has more bargaining power due to an earnings advantage or biased social norms, then a male health warning can induce a larger household investment in the shared health input than would be induced by a female health warning even if the input is less productive in generating male health.

The health effects of a female health warning are analogous to (A.29) and (A.30). The female health effect falls when less weight is placed on female welfare because that lowers investment in both female health effort and the household shared input. Compare the female health effects of male and female health warnings:

$$\frac{dH^F}{dI^M} - \frac{dH^F}{dI^F} = H_e^F\Big(\frac{de^F}{dI^M} - \frac{de^F}{dI^F}\Big) + H_X^F\Big(\frac{dX}{dI^M} - \frac{dX}{dI^F}\Big).$$

If the conditions hold for a male health warning to induce a larger shared input response ($\frac{dX}{dI^M} > \frac{dX}{dI^F}$) and the productivity of that input in generating female health is sufficiently large compared with the productivity of female health effort, then a male health warning could actually impact female health more than a female health warning.

This analysis reveals that the spillover effect of a health warning on a spouse's health is potentially asymmetric by the recipient's sex. If male welfare is prioritized, then warning a man may trigger greater household collective health investment than that arising from giving the same warning to his wife. If female health is particularly responsive to that investment, then the spillover effect of a male health warning on female health can be greater than both direct effects of male/female health warnings on male/female health.

Prioritizing male welfare implies that less weight is given to female welfare, which attenuates the behavioral response to a female BP warning through the household shared input, leaving less scope for a spillover effect on the husband's health. This effect is reduced further if male health is less responsive to the shared input. Consequently, if women are disadvantaged in the distribution of bargaining power but advantaged in the health gained from household health investment, then it is likely that the male warning to female health spillover will be larger than the female warning to male health spillover. To see this formally, consider the difference between the two spillovers:

$$\frac{dH^F}{dI^M} - \frac{dH^M}{dI^F} = \left(H_e^F\frac{de^F}{dI^M} + H_X^F\frac{dX}{dI^M}\right) - \left(H_e^M\frac{de^M}{dI^F} + H_X^M\frac{dX}{dI^F}\right).$$

If, as above, we assume the behavioral spillovers on spousal health effort are negligible compared with the behavioral responses through the shared input $\left(\frac{de^F}{dI^M} = \frac{de^M}{dI^F} \approx 0\right)$, then

$$\frac{dH^F}{dI^M} - \frac{dH^M}{dI^F} \approx H_X^F \frac{dX}{dI^M} - H_X^M \frac{dX}{dI^F}.$$

This difference is positive if the shared input responds most to a male health warning, $\frac{dX}{dI^M} > \frac{dX}{dI^F}$, and is most productive in generating female health, $H_X^F > H_X^M$.

D. Comparative statics for parent health warning

The same collective household framework is used to model spillover effects of a health warning to a parent on the health behavior and health of an adult child. In this case, assume the parent (P) is altruistic with respect to the health of their offspring (H^C): $U^P = U^P(H^P, H^C, C; I^P)$, with $U^P_{H^C} > 0$, $U^P_{H^C H^C} \leq 0$, $U^P_{H^C I^P} > 0$. The rationale for the assumed positive cross derivative is that warning a parent about their own health, particularly concerning a risk factor like high blood pressure that is heritable through genes and shared habits, alerts them to the prospect that, after some time, their child becomes exposed to the same health risk, and so the marginal value of protecting their child's health increases.

Alternatively, we could maintain the assumption of egoistical preferences – the parent and adult child each care only about their own health – and allow a health warning to the parent to increase the marginal utility the child derives from their own health: $U^C = U^C(H^C, C; I^P)$, with $U^C_H > 0$ and $U^C_{HI^P} > 0$. In this case, the rationale for the positive cross derivative would be that the adult child becomes informed of the warning to their parent and this increases the salience of the child's own health prospects. Maintaining health becomes more valuable (at the margin) because it is better appreciated that doing so helps prevent development of the health problem that has afflicted the parent.

Adult child health is produced through their own effort, e^C, and a shared household

health input, X: $H^C = H^C(e^C, X)$. This health production function, along with another for parental health and the household budget, constrains the collective household maximization of a weighted average of the parent's and child's utilities.

The positive cross derivative from a parental health warning to the marginal utility of the child's health shifts the first order conditions for optimal child effort and the shared input, as in the married couple model. Under the same simplifications that are made in that model to approximate the comparative statics,[8] both the health effort exerted by the adult child and the shared input rise in response to a parental health warning: $\frac{de^C}{dI^P} > 0$ and $\frac{dX}{dI^P} > 0$.

Adult child health therefore responds according to

$$\frac{dH^C}{dI^P} = H_e^C \frac{de^C}{dI^P} + H_X^C \frac{dX}{dI^P} > 0.$$

Note that a behavioral response from the adult child is not necessary for their health to improve following a parental health warning. The spillover effect can arise entirely through the response of the shared household health input.

If the adult child no longer lives with the parent, then there is no shared input channel and the collective household model is not an appropriate framework. However, even if a unitary adult child were to maximize their utility from own health, they would still increase their health effort in response to a parental health warning if that were to increase the salience, and so marginal value, of their health.

Parent-child spillovers may depend on sex concordance of the dyad. The parent, adult child or both may consider a parental health warning to have greater relevance to the child's health when the parent and child have the same sex. With such beliefs, in a sex-concordant parent-child dyad, the warning would have a stronger impact on the salience and marginal utility of the child's health. Consequently, the spillover effect, through the child's effort and

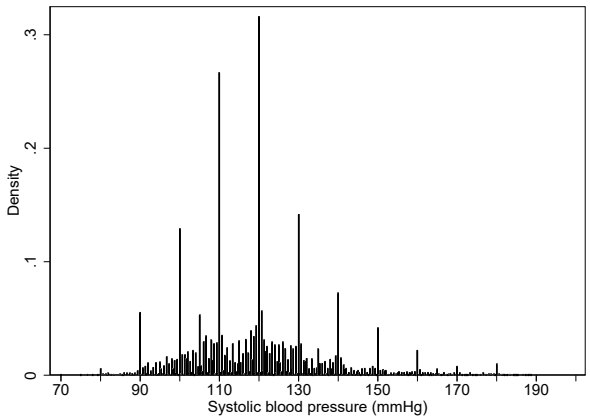
[8] That is, assuming approximately zero values for a) off-diagonal elements of the Jacobian and b) products of second derivatives of utility and squared marginal health products.

the shared input, would be larger.

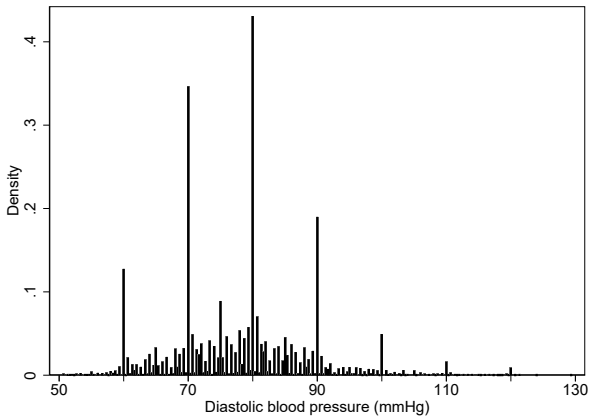
B Data - details

Figure B.1: Blood pressure distributions in the spousal dyad sample

A) Systolic blood pressure



B) Diastolic blood pressure



Notes: Histograms of A) respondent-average systolic blood pressure (mmHg) and B) respondent-average diastolic blood pressure (mmHg) at baseline in the spousal dyad sample. Average (mean) is over three measurements taken for each respondent. The sample includes observations for both spouses in each dyad, so $N = 8,378$ $(= 2 \times 4,189)$, corresponding to two individual level observations per dyad. The figure shows heaping at multiples of 10 mmHg.

Table B.1: Sample selection

	N (dyads)
Panel A: Spousal dyads	
Total	10,392
Both spouses	
have physical exam	9,835
aged 30+ at baseline	8,304
have baseline & follow-up BP	4,189
(Drop if BP = threshold)	(-594)
Analysis sample	3,595
Panel B: Intergenerational dyads	
Total	30,487
Parent & child have physical exam	27,396
Child aged 18+ at baseline	4,086
Parent & child have baseline & follow-up BP	3,380
(Drop if BP = threshold)	(-392)
Analysis sample	2,988

Notes: Dyads identified using Relation Master File published on May 10, 2019. BP = blood pressure. In each panel, "Total" gives the number of dyads observed in the Relation Master File, which recorded all household relationships among individuals interviewed from 1989 onward. "Baseline" is the first of the two consecutive waves in which a dyad is observed. "Follow-up" is the second. "BP = threshold" means that, for at least one spouse in a spousal dyad or the parent in an intergenerational dyad, either mean systolic BP over three measurements at baseline is exactly 140 mmHg or baseline mean diastolic BP = 90 mmHg. These are the values at which a high BP is issued.

Table B.2: Statements and coding used in *Knowledge* index

Item	Statement	Most correct answer
1	Choosing a diet with a lot of fresh fruits and vegetables is good for one's health.	Strongly agree
2	Eating a lot of sugar is good for one's health.	Strongly disagree
3	Eating a variety of foods is good for one's health.	Strongly agree
4	Choosing a diet high in fat is good for one's health.	Strongly disagree
5	Choosing a diet with a lot of staple foods (rice and rice products, wheat and wheat products) is not good for one's health.	Ambiguous
6	Consuming a lot of animal products daily (fish, poultry, eggs, lean meat) is good for one's health.	Ambiguous
7	Reducing the amount of fatty meat and animal fat in the diet is good for one's health.	Strongly agree
8	Consuming milk and dairy products is good for one's health.	Ambiguous
9	Consuming beans and bean products is good for one's health.	Strongly agree
10	Physical activities are good for one's health.	Strongly agree
11	Sweaty sports or other intense physical activities are not good for one's health.	Strongly disagree
12	The heavier one's body is, the healthier one is.	Strongly disagree

Notes: Respondents answer on a 5-point Likert scale: 1 = Strongly disagree, 2 = Disagree, 3 = Neutral, 4 = Agree, 5 = Strongly agree).

Secondary outcome composites

We use the weighted-average method of Anderson (2008) to construct four composite indices: (i) *Knowledge* - an index of assessed knowledge of healthy diet, exercise and body weight; (ii) *Diet* - an index of reported healthy eating; (iii) *Behavior* - an index of reported exercise, smoking and alcohol consumption behavior; and, (iv) *Adiposity* - an index of measured body weight and shape. Each of these secondary outcomes is constructed from multiple indicators. In each case, we transform each of the underlying indicators into a z-score. Then, we take the respondent-specific weighted average over all the z-scores of the indicators used for each index. The weights are constructed to give more weight to indicators that correlate less with the others, which maximizes the information content of the index (Anderson, 2008). The following paragraphs describe the components of each index.

Knowledge. Respondents rate 12 statements about health and diet, which are listed in Table B.2, on a five point Likert scale from strongly disagree (1) to strongly agree (5), with neutral coded as 3. The knowledge index is constructed from responses to 9 of the 12 statements, with statements 5, 6 and 8 excluded because of the correct answer to each is ambiguous. For statements where disagreement is the correct response, we reverse code the scale so that higher values always correspond to more correct answers. Questions about these statements were introduced in the 2004 survey wave. Consequently, the sample used to estimate effects on this index is smaller.

Diet. This index is constructed from five variables. Four are binary indicators derived from YES/NO answers to questions: (1) During the past week, have you prepared and cooked

food for the household? (NO = 0, YES = 1); (2) Do you eat fast food such as KFC, pizza, or hamburgers? (NO = 1, YES = 0); (3) Do you eat salty snacks such as potato chips, pretzels, or French fries? (NO = 1, YES = 0); and, (4) Do you drink soft fizzy drinks? (NO = 1, YES = 0). The fifth variable is average daily fat intake in grams over three days, constructed from three consecutive 24 hour dietary recalls, and converted to nutrient values using the Chinese Food Composition Table.

Behavior. This index combines 10 variables. Six capture participation in physical activities: (1) martial arts, (2) gymnastics or dancing, (3) track and field, (4) team or ball sports, such as soccer, basketball, or tennis, (5) badminton or volleyball, and (6) other activities, such as ping pong or tai chi. The other four indicate unhealthy behavior: (7) ever smoked, (8) currently smoking, (9) number of cigarettes smoked, and (10) consumed any alcohol in the previous year. We reverse code the unhealthy behavior measures so that higher values consistently reflect healthier behavior.

Adiposity. This index has four components: (1) body mass index (BMI); (2) a binary indicator of BMI $\geq$ 27.5 (NO = 0, YES = 1); (3) waist-to-height ratio (WHR); and, (4) a binary indicator of WHR $\geq$ 0.55 for men or WHR $\geq$ 0.58 for women (NO = 0, YES =1). BMI is derived from measured weight and height following standard procedures. We trim by removing the bottom and top 1 percent of the BMI distribution. BMI $\geq$ 27.5 is a threshold for high cardiometabolic risk in Asian populations that is used to guide public health and clinical action (Tan *et al.*, 2004). WHR is a widely used marker of cardiometabolic risk that outperforms waist circumference as a cardiometabolic risk factor and is recommended as a screening measure (Ashwell *et al.*, 2012). WHR thresholds of 0.55/0.58 have been shown to be optimal for detecting cardiovascular risk in Chinese adults (Peng *et al.*, 2015).

Tables B.3 and B.4 give descriptives of the variables used to construct the secondary outcomes for the spousal dyad sample and intergenerational dyad sample, respectively.

Table B.3: Descriptives of variables used for secondary outcomes - spousal dyad sample

	Male			***Female***		
	Mean	Std dev.	N	Mean	Std dev.	N
Diagnosis (hypertension) (0/1)	.087	.282	3627	.085	.279	3640
Medication (hypertension) (0/1)	.063	.243	3672	.059	.235	3695
Knowledge (1-5)						
Fruit: Lot of fresh fruits and vegetables is good	3.470	.796	1388	3.505	.787	1385
Sugar: Lot of sugar is good (reversed)	3.859	.573	1366	3.859	.581	1362
Variety: Variety of food is good	3.559	.677	1351	3.559	.692	1361
Fat: High-fat food is good (reversed)	3.870	.609	1359	3.861	.633	1360
Fatty: Eating less fatty meat is good	3.486	.722	1362	3.496	.721	1354
Beans: Beans and bean products are good	3.723	.581	1385	3.737	.5636	1376
Activity: Physical activity is good	3.674	.618	1381	3.639	.624	1364
Sweaty: Doing sweaty sports is bad (reversed)	2.890	.894	1349	2.910	.892	1328
Heavy: Being heavier is healthier (reversed)	4.033	.595	1361	4.054	.611	1371
Diet						
Prepared food for household last week (0/1)	.315	.465	3381	.903	.296	3697
Don't eat fast food (0/1)	.344	.475	1400	.320	.467	1402
Don't eat salty snacks (0/1)	.282	.450	1400	.248	.432	1402
Don't drink soft drinks (0/1)	.198	.398	1401	.169	.375	1403
Fat consumed (grams)	72.499	34.685	3647	64.658	31.761	3667
Behavior						
Does marital arts (0/1)	.009	.093	2083	.006	.076	2081
Does track and field (0/1)	.057	.232	2083	.030	.170	2083
Does gymnastics/dancing (0/1)	.011	.102	2086	.027	.162	2082
Plays soccer/basketball/tennis (0/1)	.019	.137	2087	.002	.049	2082
Plays badminton/volleyball (0/1)	.018	.132	2085	.010	.098	2082
Plays ping pong/do tai chi (0/1)	.081	.273	2085	.041	.198	2080
Ever smoked (0/1)	.651	.479	3742	.047	.212	3719
Currently smoking (0/1)	.611	.488	3683	.044	.205	3713
Cigarettes smoked daily	10.100	10.502	3655	.481	2.704	3714
Alcohol in last year (0/1)	.638	.481	3724	.108	.310	3701
Adiposity						
Body mass index (BMI)	22.803	3.003	3692	22.98	3.110	3676
$\mathbb{1}\{BMI \geq 27.5\}$	.075	.264	3692	.092	.289	3676
Waist-to-height ratio (WHR)	.492	.056	3616	.506	.060	3603
$\mathbb{1}\{WHR \geq 0.55/0.58\}$	.163	.369	3616	.133	.339	3603

Notes: Descriptives for spousal dyad sample at baseline. Top two rows give secondary outcomes: *Diagnosis* - have been diagnosed with hypertension; *Medication* - currently taking medication for hypertension. Other rows give descriptives for variables used to construct composite indices used as secondary outcomes. For *Knowledge*, see Table B.2 for the specific statements. For the other indices, see details in the text above.

Table B.4: Descriptives of variables used for secondary outcomes - intergenerational dyad sample

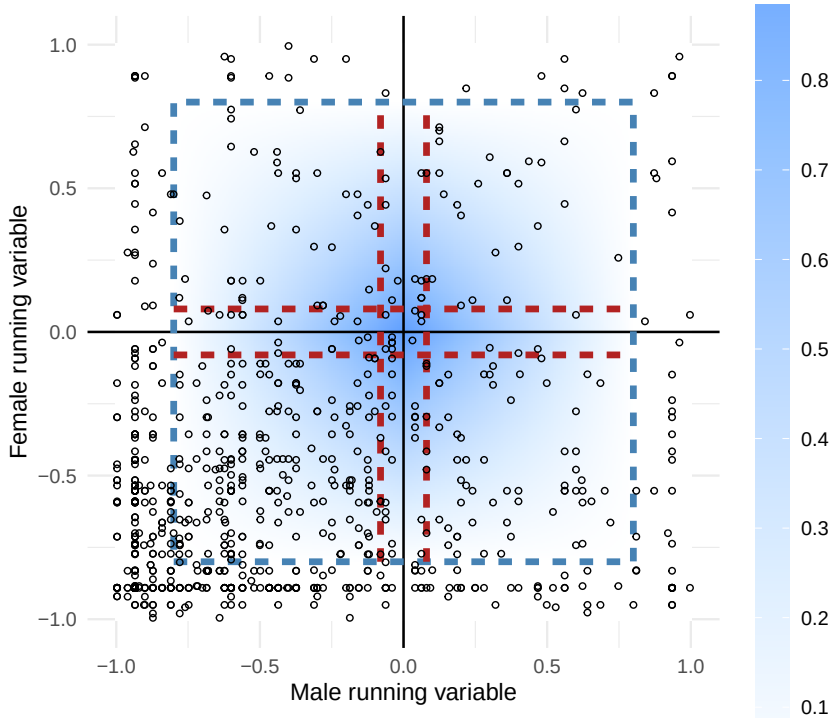
	Parent			***Child***		
	Mean	Std dev.	N	Mean	Std dev.	N
Knowledge (1-5)						
Fruit: Lot of fresh fruits and vegetables is good	3.440	.805	823	3.505	.808	832
Sugar: Lot of sugar is good (reversed)	3.819	.617	812	3.886	.531	833
Variety: Variety of food is good	3.564	.668	803	3.576	.662	816
Fat: High-fat food is good (reversed)	3.794	.652	804	3.855	.592	829
Fatty: Eating less fatty meat is good	3.491	.723	808	3.501	.730	820
Beans: Beans and bean products are good	3.732	.539	829	3.742	.562	836
Activity: Physical activity is good	3.672	.621	827	3.686	.628	838
Sweaty: Doing sweaty sports is bad (reversed)	2.898	.894	786	2.854	.903	821
Heavy: Being heavier is healthier (reversed)	4.020	.609	808	4.046	.594	828
Diet						
Prepared food for household last week (0/1)	.621	.485	2961	.219	.414	2840
Don't eat fast food (0/1)	.361	.481	859	.206	.405	859
Don't eat salty snacks (0/1)	.300	.459	859	.179	.384	859
Don't drink soft drinks (0/1)	.200	.401	858	.108	.311	859
Fat consumed (grams)	64.796	33.954	3023	67.838	34.641	2935
Behavior						
Does marital arts (0/1)	.009	.096	1390	.005	.069	1477
Does track & field (0/1)	.031	.173	1391	.047	.212	1478
Does gymnastics/dancing (0/1)	.015	.122	1394	.012	.107	1478
Plays soccer/basketball/tennis (0/1)	.003	.054	1394	.061	.240	1480
Plays badminton volleyball (0/1)	.006	.076	1393	.041	.199	1479
Plays ping pong/does tai chi (0/1)	.048	.214	1393	.093	.291	1480
Ever smoked (0/1)	.323	.468	3083	.422	.494	3081
Currently smoking (0/1)	.297	.457	3052	.412	.492	3060
Cigarettes smoked daily	4.600	8.501	3036	5.890	8.586	3035
Alcohol in last year (0/1)	.319	.466	3075	.441	.497	3071
Adiposity						
Body mass index (BMI)	22.572	3.223	3036	21.793	2.810	3071
$\mathbb{1}\{BMI \geq 27.5\}$	.076	.266	3036	.045	.207	3071
Waist-to-height ratio	.507	.062	2955	.465	.054	2966
$\mathbb{1}\{WHR \geq 0.55/0.58\}$	.246	.431	2955	.034	.181	2966

Notes: Descriptives for intergenerational dyad sample at baseline. Variables used to construct composite indices used as secondary outcomes. For *Knowledge*, see Table B.2 for the specific statements. For the other indices, see details in the text above.

C Estimation - details

A. Observations used for estimation

Figure C.1: Estimation neighborhood $\mathcal{N}(h_M, h_F)$ and orthogonal strips around frontiers



Notes: x-axis (y-axis) shows the binding-score running variable A_M (A_F) as defined in the method section IV. Black vertical and horizontal solid lines represent treatment frontier along A_M and A_F. Dashed blue lines indicate the windows of observations used in estimation ($\mathcal{N}(h_M, h_F)$). Red dashed lines indicate orthogonal strips ($K_M\left(\frac{|A_M|}{b_M}\right)$ and $K_F\left(\frac{|A_F|}{b_F}\right)$) used to compute frontier-weighted side empirical means. In blue is the product triangular weights $K(A)$ on $\mathcal{N}(h_M, h_F)$ to estimate regression (2).

B. Conditional expectation assuming joint normality

To obtain the main estimates, we use empirical, frontier-weighted side means of running variables defined in Eq.(3). We test robustness to replacing these with *deterministic* side means implied by an assumed parametric distribution for these variables. Here, we explain the parametric approach.

Assume (A_M, A_F) follows a bivariate normal distribution with means (μ_{A_M}, μ_{A_F}), standard deviations $(\sigma_{A_M}, \sigma_{A_F})$, and correlation ρ. Then the conditional distribution of A_M given $A_F = a_f$ (Maddala, 1983) is

$$A_M \mid (A_F = a_f) \sim \mathcal{N}\big(\mu_c(a_f), \sigma_c^2\big), \qquad \mu_c(a_f) = \mu_{A_M} + \rho\frac{\sigma_{A_M}}{\sigma_{A_F}}(a_f - \mu_{A_F}), \qquad \sigma_c^2 = \sigma_{A_M}^2(1-\rho^2).$$

In our sample, $\mu_{A_M} = -0.848$, $\sigma_{A_M} = 1.015$, $\mu_{A_F} = -1.094$, $\sigma_{A_F} = 1.029$, and $\rho = 0.33$. Conditioning on $A_F = 0$ yields $\mu_c(0) = -0.4919$ and $\sigma_c = 0.9581$.

We are interested in the conditional mean of A_M when we additionally restrict A_M to lie in $[a, b]$:

$$\mathbb{E}[A_M \mid A_F = 0,\ a \le A_M \le b].$$

By definition,

$$\mathbb{E}[A_M \mid A_F = 0,\ a \le A_M \le b] = \frac{\int_a^b x\, f_{A_M|A_F=0}(x)\,dx}{\int_a^b f_{A_M|A_F=0}(x)\,dx},$$

where $f_{A_M|A_F=0}(\cdot)$ is the density of $\mathcal{N}(\mu_c(0), \sigma_c^2)$.

Since $A_M \mid A_F = 0 \sim \mathcal{N}(\mu_c, \sigma_c^2)$, the conditional density is

$$f_{A_M|A_F=0}(x) = \frac{1}{\sigma_c}\,\varphi\!\left(\frac{x-\mu_c}{\sigma_c}\right).$$

Therefore,

$$\mathbb{E}[A_M \mid A_F = 0,\ a \le A_M \le b] = \frac{\int_a^b x\,\frac{1}{\sigma_c}\varphi\!\left(\frac{x-\mu_c}{\sigma_c}\right)\,dx}{\int_a^b \frac{1}{\sigma_c}\varphi\!\left(\frac{x-\mu_c}{\sigma_c}\right)\,dx}.$$

Use the change of variables $z = (x - \mu_c)/\sigma_c$ (so $x = \mu_c + \sigma_c z$ and $dx = \sigma_c\,dz$), and define

$$\alpha = \frac{a-\mu_c}{\sigma_c}, \qquad \beta = \frac{b-\mu_c}{\sigma_c}.$$

The denominator becomes

$$\int_a^b \frac{1}{\sigma_c}\varphi\!\left(\frac{x-\mu_c}{\sigma_c}\right)\,dx = \int_\alpha^\beta \varphi(z)\,dz = \Phi(\beta) - \Phi(\alpha).$$

The numerator becomes

$$\int_a^b x\,\frac{1}{\sigma_c}\varphi\!\left(\frac{x-\mu_c}{\sigma_c}\right)\,dx = \int_\alpha^\beta (\mu_c + \sigma_c z)\varphi(z)\,dz = \mu_c\int_\alpha^\beta \varphi(z)\,dz + \sigma_c\int_\alpha^\beta z\varphi(z)\,dz.$$

Using $\frac{d}{dz}\varphi(z) = -z\varphi(z)$ implies $\int z\varphi(z)\,dz = -\varphi(z)$, so

$$\int_{\alpha}^{\beta} z\varphi(z)\,dz = \varphi(\alpha) - \varphi(\beta).$$

Combining these results,

$$\int_{a}^{b} x\, f_{A_M|A_F=0}(x)\,dx = \mu_c\big[\Phi(\beta) - \Phi(\alpha)\big] + \sigma_c\big[\varphi(\alpha) - \varphi(\beta)\big].$$

Dividing numerator by denominator yields the truncated conditional mean:

$$\mathbb{E}[A_M \mid A_F = 0,\ a \le A_M \le b] = \mu_c + \sigma_c \frac{\varphi(\alpha) - \varphi(\beta)}{\Phi(\beta) - \Phi(\alpha)}.$$

We can now apply this formula for the intervals $[-0.8, 0]$ and $[0, 0.8]$. For $A_M \in [-0.8, 0]$, $\alpha_{M,1} = \frac{-0.8-\mu_c}{\sigma_c} \approx -0.322$ and $\beta_{M,1} = \frac{0-\mu_c}{\sigma_c} \approx 0.513$.

Therefore, $\mathbb{E}[A_M \mid A_F = 0,\ -0.8 \le A_M \le 0] \approx -0.406$. For $[0, 0.8]$, $\alpha_{M,2} \approx 0.513$ and $\beta_{M,2} \approx 1.348$, thus $\mathbb{E}[A_M \mid A_F = 0,\ 0 \le A_M \le 0.84] \approx 0.349$.
Analogously, conditioning on $A_M = 0$ gives $\alpha_{F,1} \approx -0.037$ and $\beta_{F,1} \approx 0.811$ and thus $E[A_F \mid A_M = 0,\ -0.8 \le A_F \le 0] \approx -0.421$ for the interval $[-0.8, 0]$. For the interval $[0, 0.8]$, one obtains $\alpha_{F,2} \approx 0.811$ and $\beta_{F,2} \approx 1.659$ and thus $E[A_F \mid A_M = 0,\ 0 \le A_F \le 0.8] \approx 0.333$.

The corresponding conditional masses are given by $\mathbb{P}(a \le W \le b) = \Phi(\beta) - \Phi(\alpha)$. For the $A_F = 0$ frontier,

$$p_1 = \mathbb{P}(-0.8 \le A_M \le 0 \mid A_F = 0) \approx 0.322, \qquad p_2 = \mathbb{P}(0 \le A_M \le 0.8 \mid A_F = 0) \approx 0.215,$$

so $p_{12} = p_1 + p_2 \approx 0.537$ and the relative shares within $[-0.8, 0.8]$ are $\frac{p_1}{p_{12}} \approx 0.600$ and $\frac{p_2}{p_{12}} \approx 0.400$.

For the $A_M = 0$ frontier, $q_1 = \mathbb{P}(-0.8 \le A_F \le 0 \mid A_M = 0) \approx 0.306$ and $q_2 = \mathbb{P}(0 \le A_F \le 0.8 \mid A_M = 0) \approx 0.160$, so $q_{12} = q_1 + q_2 \approx 0.466$ and the relative shares within $[-0.8, 0.8]$ are $\frac{q_1}{q_{12}} \approx 0.657$ and $\frac{q_2}{q_{12}} \approx 0.343$.

Finally, if we use these conditional masses to construct weights across the two frontiers, we can normalize by

$$w = p_{12} + q_{12} \approx 1.003, \qquad w_{A_M} = \frac{p_{12}}{w} \approx 0.535, \qquad w_{A_F} = \frac{q_{12}}{w} \approx 0.464.$$

Note that w is a normalization constant for the weighting scheme rather than a probability, since p_{12} and q_{12} are defined under different conditioning events.

Table C.1 summarizes the values of these different parameters.

Table C.1: Truncated conditional means and conditional masses on the two frontiers

Interval	**Truncated mean**	**Mass**	**Share**
Frontier $A_F = 0$: quantities for $A_M \mid A_F = 0$			
$[-0.8, 0]$	$\mathbb{E}[A_M \mid A_F = 0,\ -0.8 \le A_M \le 0] \approx -0.406$	$p_1 \approx 0.322$	$p_1/p_{12} \approx 0.600$
$[0, 0.8]$	$\mathbb{E}[A_M \mid A_F = 0,\ 0 \le A_M \le 0.8] \approx 0.349$	$p_2 \approx 0.215$	$p_2/p_{12} \approx 0.400$
	Total mass on $A_F = 0$ frontier:	$p_{12} = p_1 + p_2 \approx 0.537$	
Frontier $A_M = 0$: quantities for $A_F \mid A_M = 0$			
$[-0.8, 0]$	$\mathbb{E}[A_F \mid A_M = 0,\ -0.8 \le A_F \le 0] \approx -0.421$	$q_1 \approx 0.306$	$q_1/q_{12} \approx 0.657$
$[0, 0.8]$	$\mathbb{E}[A_F \mid A_M = 0,\ 0 \le A_F \le 0.8] \approx 0.333$	$q_2 \approx 0.160$	$q_2/q_{12} \approx 0.343$
	Total mass on $A_M = 0$ frontier:	$q_{12} = q_1 + q_2 \approx 0.466$	
Normalized weights across frontiers (for aggregation)			
	Normalization constant:	$w = p_{12} + q_{12} \approx 1.003$	
	Weight on $A_F = 0$ frontier:	$w_{A_M} = p_{12}/w \approx 0.535$	
	Weight on $A_M = 0$ frontier:	$w_{A_F} = q_{12}/w \approx 0.464$	

Notes: Masses are conditional probabilities on each frontier: $p_i = \mathbb{P}(\cdot \mid A_F = 0)$ and $q_i = \mathbb{P}(\cdot \mid A_M = 0)$. Shares are computed within the combined window $[-0.8, 0.8]$ on each frontier. The aggregate quantity w is a normalization constant for the weighting scheme rather than a probability, because p_{12} and q_{12} are defined under different conditioning events.

D Validation analysis

Table D.1: Manipulation tests without exclusion of observations at thresholds

	Coef.	Test stat.	p-value	N
A. Spousal dyads				
A1. Male running variable (A_M)				
Density test (A_M)		-0.124	0.901	1050
Δ Female binding score (A_F) at A_M threshold				
Unconditional	0.0425	0.406	0.685	2434
If $A_F < 0$	-0.184**	-2.152	0.031	1218
If $A_F \geq 0$	-0.114	-0.563	0.574	430
A2. Female running variable (A_F)				
Density test (A_F)		-0.106	0.915	660
Δ Male binding score (A_M) at A_F threshold				
Unconditional	-0.100	-0.909	0.363	2424
If $A_M < 0$	-0.164**	-2.016	0.044	1864
If $A_M \geq 0$	-0.281	-1.486	0.137	617
B. Intergenerational dyads				
Density test (A_P)		0.438	0.661	741
Δ Child binding score (A_C) at A_P threshold				
Unconditional	-0.091	-1.035	0.301	1864
If $A_C < 0$	-0.088	-1.145	0.252	1668
If $A_C \geq 0$	0.078	0.475	0.635	135

Notes: Baseline data. Full sample without excluding observations with precisely threshold values of baseline blood pressure. Density tests (T-statistic) for male, female and parental running variables in panels A1, A2 and B, respectively. Each is conducted using triangular weights and quadratic polynomial specifications. Results are robust to using uniform or epanechnikov weights. Other rows headed, for example, "Δ Female binding score (A_F) at A_M threshold" give unidimensional RDD estimates of "effects" of crossing the A_M running variable threshold on the A_F binding score. "Unconditional", "If $A_J < 0$" and "If $A_J \geq 0$" are obtained from all, negative and non-negative values of the respective binding score. These estimates are obtained from local linear regressions, with triangular kernels and optimal bandwidths on each side of the threshold using the MSE optimal bandwidth selector. P-values are derived from heteroscedasticity-robust standard errors. N is the effective number of observations used in estimation. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table D.2: Further manipulation tests without exclusion of observations at thresholds

	Coef.	p-value	N
A1. Male running variable (A_M)			
Δ Female BP at A_M threshold if $A_F < 0$			
Systolic BP	-4.17***	0.005	1881
Diastolic BP	-0.619	0.556	1291
A2. Female running variable (A_F)			
Δ Male BP at A_F threshold if $A_M < 0$			
Systolic BP	-3.213**	0.037	1754
Diastolic BP	-1.140	0.217	1852

Notes: This table extends analysis from panel A of Table D.1 to include unidimensional RDD estimates of separate "effects" on a spouse's systolic and diastolic blood pressure (BP), conditional on the spouse's binding score being negative. For example, Δ Female BP at A_M threshold if $A_F < 0$ gives estimates of "effects" of A_M crossing the treatment threshold on female BP, conditional on the female binding score being negative. Estimates obtained from local linear regressions, with triangular kernels and optimal bandwidths on each side of the threshold using the MSE optimal bandwidth selector. P-values are derived from heteroscedasticity-robust standard errors. N is the effective number of observations used in estimation. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table D.3: Covariate continuity tests without exclusion of observations at thresholds - spousal dyads

		Coef.	Test stat.	p-value	N
A. Unidimensional RDD					
A1. Male running variable					
Age (years) male		-.073	-0.052	0.958	1775
Age female		-.001	-0.001	0.999	1839
Education (years) male		.0813	0.182	0.856	2320
Education female		-.656	-1.209	0.227	1576
A2. Female running variable					
Age male		-1.362	-1.053	0.292	2229
Age female		-.785	-0.653	0.514	2285
Education male		-1.016**	-2.109	0.035	2070
Education female		-.232	-0.490	0.624	2448
B. Multidimensional RDD					
		Male outcomes		***Female outcomes***	
		Age (years)	Education (years)	Age (years)	Education (years)
Male warning					
Wife unwarned	τ_{M-}	.76	-.216	.561	-.865
		(2.68)	(.976)	(2.566)	(1.035)
Wife warned	τ_{M+}	-7.411**	-.509	-6.587**	-.284
		(3.382)	(1.254)	(2.768)	(1.395)
Average	$\tau_{\bar{M}}$	-3.796*	-.379	-3.424*	-.541
		(2.279)	(.835)	(1.965)	(.894)
Female warning					
Husband unwarned	τ_{F-}	.619	.052	.131	.351
		(3.005)	(1.028)	(2.742)	(.98)
Husband warned	τ_{F+}	-1.733	-.437	-1.472	.455
		(3.249)	(1.336)	(3.147)	(1.352)
Average	$\tau_{\bar{F}}$	-.745	-.232	-.799	.412
		(2.214)	(.877)	(2.161)	(.887)
Male-Female average	τ	-2.307	-.307	-2.143	-.076
		(1.551)	(.591)	(1.46)	(.621)
N		590	588	590	588

Notes: Baseline data. Full sample without dropping observations with precisely threshold values of baseline blood pressure. Panel A shows unidimensional RDD estimate of the discontinuity in age or education as the respective running variable crosses its treatment assignment threshold. Estimates obtained from local linear regressions, with triangular kernels and optimal bandwidths on each side of the threshold using the MSE optimal bandwidth selector. "Test stat." is a Z-statistic. P-values are derived from heteroscedasticity-robust standard errors. N is the effective number of observations used in estimation. Panel B reports the MRDD estimates for the discontinuity parameters defined in Section IV., evaluated on the predetermined covariates. Bootstrapped (1000 repetitions) standard errors in parentheses. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table D.4: Covariate continuity tests - intergenerational dyads

	Coef.	p-value	N
A. Without exclusion of observations at thresholds			
Age (years) parent	0.028	0.982	1447
Age child	.245	0.823	1609
Education (years) parent	-0.685	0.224	1847
Education child	-.498	0.168	2178
B. With exclusion of observations at thresholds			
Age parent	-1.302	0.479	888
Age child	0.833	0.560	1114
Education parent	-0.040	0.960	1476
Education child	0.074	0.900	1743

Notes: Baseline data. Panel A uses full sample without dropping observations with precisely threshold values of baseline blood pressure. Panel B excludes these observations. Unidimensional RDD estimate of the discontinuity in age or education as the respective running variable crosses its treatment assignment threshold. Estimates obtained from local linear regressions, with triangular kernels and optimal bandwidths on each side of the threshold using the MSE optimal bandwidth selector. P-values are derived from heteroscedasticity-robust standard errors. N is the effective number of observations used in estimation. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table D.5: Manipulation tests with exclusion of observations at thresholds

	Coef.	Test stat.	p-value	N
A. Spousal dyads				
A1. Male running variable (A_M)				
Density test (A_M)		-0.602	0.547	638
Δ Female binding score (A_F) at A_M threshold				
Unconditional	0.015	0.091	0.928	1705
If $A_F < 0$	-0.166	-1.224	0.221	964
If $A_F \geq 0$	-0.203	-0.736	0.462	164
A2. Female running variable (A_F)				
Density test (A_F)		-0.063	0.950	364
Δ Male binding score (A_M) at A_F threshold				
Unconditional	0.065	0.281	0.779	1838
If $A_M < 0$	0.056	0.385	0.701	1533
If $A_M \geq 0$	0.324	0.720	0.472	213
B. Intergenerational dyads				
Density test (A_P)		0.359	0.720	489
Δ Child binding score (A_C) at A_P threshold				
Unconditional	0.077	0.567	0.571	1484
If $A_C < 0$	-0.007	-0.055	0.956	1062
If $A_C \geq 0$	-0.009	-0.034	0.973	78

Notes: Baseline data. Analysis sample that excludes observations with precisely threshold values of baseline blood pressure. Density tests (T-statistic) for male, female and parental running variables in panels A1, A2 and B, respectively. Each is conducted using triangular weights and quadratic polynomial specifications. Results are robust to using uniform or epanechnikov weights. Other rows headed, for example, "Δ Female binding score (A_F) at A_M threshold" give unidimensional RDD estimates of "effects" of crossing the A_M running variable threshold on the A_F binding score. "Unconditional", "If $A_J < 0$" and "If $A_J \geq 0$" are obtained from all, negative and non-negative values of the respective binding score. These estimates are obtained from local linear regressions, with triangular kernels and optimal bandwidths on each side of the threshold using the MSE optimal bandwidth selector. P-values are derived from heteroscedasticity-robust standard errors. N is the effective number of observations used in estimation. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

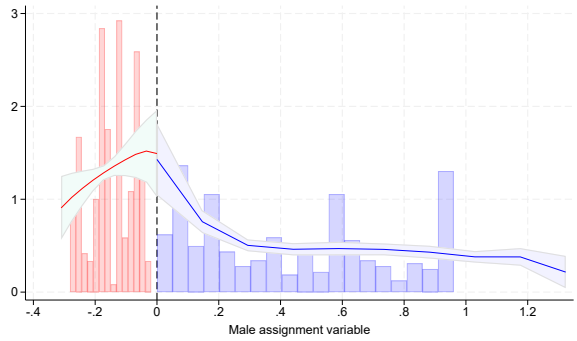
Table D.6: Covariate continuity tests with exclusion of observations at thresholds - spousal dyads

		Coef.	Test stat.	p-value	N
A. Unidimensional RDD					
A1. Male running variable					
Age (years) male		1.2799	0.6670	0.505	1171
Age female		1.2645	0.6972	0.486	1168
Education (years) male		0.8230	1.1903	0.234	1286
Education female		1.1203	1.4866	0.137	968
A2. Female running variable					
Age male		2.5252	1.1567	0.247	1514
Age female		2.8997	1.4297	0.153	1534
Education male		-0.4864	-0.6463	0.518	1502
Education female		0.2648	0.3271	0.744	1510
B. Multidimensional RDD					
		Male outcomes		***Female outcomes***	
		Age (years)	Education (years)	Age (years)	Education (years)
Male warning					
Wife unwarned	τ_{M-}	2.011	.676	2.149	-.359
		(3.652)	(1.546)	(3.429)	(1.379)
Wife warned	τ_{M+}	-3.931	1.478	-2.89	2.466
		(5.001)	(2.23)	(4.853)	(2.465)
Average	$\tau_{\bar{M}}$	.225	.917	.634	.49
		(3.017)	(1.266)	(2.729)	(1.255)
Female warning					
Husband unwarned	τ_{F-}	3.568	.116	4.032	.059
		(3.68)	(1.367)	(3.535)	(2.001)
Husband warned	τ_{F+}	-2.322	-2.312	-1.807	-.307
		(6.386)	(2.725)	(6.242)	(2.644)
Average	$\tau_{\bar{F}}$	2.323	-.397	2.799	-.019
		(3.166)	(1.313)	(3.09)	(1.672)
Male-Female average	τ	1.136	.347	1.574	.269
		(1.959)	(.825)	(1.88)	(.847)
N		386	384	386	384

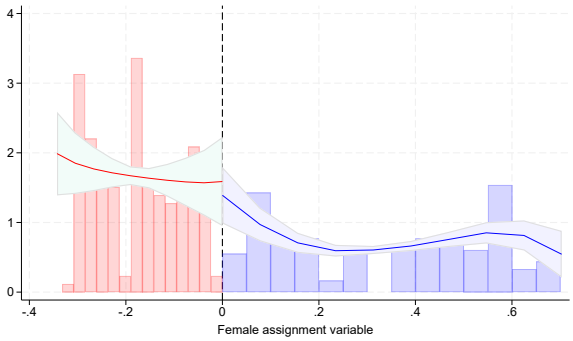
Notes: Baseline data. Analysis sample that excludes observations with precisely threshold values of baseline blood pressure. Panel A shows unidimensional RDD estimate of the discontinuity in age or education as the respective running variable crosses its treatment assignment threshold. Estimates obtained from local linear regressions, with triangular kernels and optimal bandwidths on each side of the threshold using the MSE optimal bandwidth selector. "Test stat." is a Z-statistic. P-values are derived from heteroscedasticity-robust standard errors. N is the effective number of observations used in estimation. Panel B reports the MRDD estimates for the discontinuity parameters defined in Section IV., evaluated on the predetermined covariates. Bootstrapped (1000 repetitions) standard errors in parentheses. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Figure D.1: Running variable densities

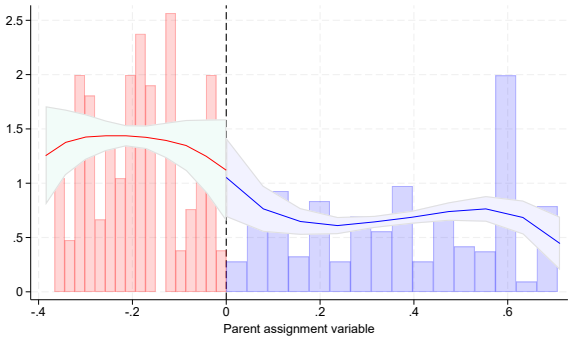
A) Male running variable, A_M



B) Female running variable, A_F



C) Parent running variable, A_P



Notes: Baseline data. Histograms of the binding-score running variables for the analysis sample that excludes observations with blood pressure precisely at threshold values. Binding scores defined in section IV. There is no statistically significant discontinuity in the density of any running variable (Table D.5). These RDD plots are generated using second order polynomial, triangular weights, and a different optimal bandwidth on each side of the threshold (based on the MSE of each density estimator separately) (Calonico *et al.*, 2015, 2017, 2018; Cattaneo *et al.*, 2018, 2020).

E Additional results - primary outcomes

Table E.1: Counterfactuals means of outcomes

A. Spousal dyads					
	Male		Female		
	Mean	SD	Mean	SD	N
Female frontier, male untreated					
Systolic BP	122.32	16.35	129.07	18.15	35
Diastolic BP	81.61	11.12	83.810	10.67	35
High BP	.229	.426	.486	.507	35
Female frontier, male treated					
Systolic BP	148.78	20.98	136.93	18.36	9
Diastolic BP	89.56	9.95	80.96	10.31	9
High BP	.556	.527	.556	.527	9
Male frontier, female untreated					
Systolic BP	127.16	14.76	118.12	13.53	51
Diastolic BP	83.74	12.15	77.61	9.45	51
High BP	.314	.469	.176	.385	51
Male frontier, female treated					
Systolic BP	135.92	22.42	154.92	31.01	12
Diastolic BP	86.28	11.47	93.25	11.40	12
High BP	0.583	0.515	0.667	0.492	12
B. Intergenerational dyads					
	Parent		Child		
	Mean	SD	Mean	SD	N
Parent frontier					
Systolic BP	134.08	17.06	118.11	12.82	50
Diastolic BP	82.42	8.618	76.25	9.802	50
High BP	.420	.499	.120	.328	50

Notes: Means and standard deviations (SD) of outcomes at follow-up for observations located within 0.1 of a standard deviations of the respective frontier on the untreated side. Each panel corresponds to a different frontier defined by treatment status.

Table E.2: Direct & spousal spillover effects of high blood pressure warning on primary outcomes - robustness to wider strips for frontier-weighted side means

		Male blood pressure			**Female blood pressure**		
		Systolic (mmHg)	Diastolic (mmHg)	High (0/1)	Systolic (mmHg)	Diastolic (mmHg)	High (0/1)
Male warning							
Wife unwarned	τ_{M-}	-3.401	.839	-.033	-11.071***	-6.049**	-.29**
		(4.456)	(3.007)	(.139)	(3.891)	(2.878)	(.122)
Wife warned	τ_{M+}	10.207	3.257	.346	-7.329	-3.83	-.148
		(9.804)	(4.746)	(.231)	(9.207)	(4.972)	(.244)
Average	$\tau_{\bar{M}}$	.844	1.593	.085	-9.904**	-5.357**	-.245**
		(4.309)	(2.552)	(.119)	(3.858)	(2.548)	(.11)
Female warning							
Husband unwarned	τ_{F-}	-.105	-.77	-.112	1.216	1.244	.002
		(4.85)	(3.036)	(.158)	(5.715)	(4.433)	(.173)
Husband warned	τ_{F+}	.38	-2.706	.261	-3.457	-4.427	-.082
		(8.662)	(5.798)	(.261)	(9.353)	(4.867)	(.29)
Average	$\tau_{\bar{F}}$	.006	-1.214	-.026	.144	-.057	-.018
		(4.064)	(2.715)	(.139)	(4.865)	(3.654)	(.15)
Male-Female average	τ	.494	.419	.039	-5.702**	-3.141*	-.15*
		(3.023)	(1.699)	(.084)	(2.849)	(1.744)	(.083)
N		384	384	384	384	384	384

Notes: MRDD estimates of the treatment effects defined in Section B. Estimation as for the main estimates in Table 2 except here empirical, frontier-weighted side means are derived using strips of 0.1 instead of 0.08. Otherwise, notes as Table 2.

Table E.3: Direct & spousal spillover effects of high blood pressure warning on primary outcomes - robustness to smaller strips for frontier-weighted side means

		Male blood pressure			**Female blood pressure**		
		Systolic (mmHg)	Diastolic (mmHg)	High (0/1)	Systolic (mmHg)	Diastolic (mmHg)	High (0/1)
Male warning							
Wife unwarned	τ_{M-}	-3.143	1.719	-.064	-9.373**	-4.105	-.213
		(4.732)	(3.746)	(.154)	(4.788)	(3.638)	(.167)
Wife warned	τ_{M+}	8.937	3.038	.409	-6.824	-6.319	-.262
		(11.964)	(5.667)	(.282)	(9.426)	(5.895)	(.288)
Average	$\tau_{\bar{M}}$	-.451	2.013	.041	-8.805**	-4.599	-.224
		(4.542)	(3.19)	(.138)	(4.323)	(3.14)	(.144)
Female warning							
Husband unwarned	τ_{F-}	.096	-1.164	-.134	1.016	2.545	.015
		(5.942)	(3.752)	(.193)	(6.722)	(5.113)	(.203)
Husband warned	τ_{F+}	6.421	-3.932	.415	-2.292	-5.872	-.34
		(12.816)	(7.163)	(.287)	(8.996)	(5.596)	(.372)
Average	$\tau_{\bar{F}}$	1.192	-1.644	-.039	.443	1.087	-.046
		(5.445)	(3.389)	(.187)	(5.775)	(4.466)	(.187)
Male-Female average	τ	.265	.42	.006	-4.776	-2.122	-.147
		(3.503)	(2.199)	(.107)	(3.429)	(2.313)	(.108)
N		384	384	384	384	384	384

Notes: MRDD estimates of the treatment effects defined in Section B. Estimation as for the main estimates in Table 2 except here empirical, frontier-weighted side means are derived using strips of 0.06 instead of 0.08. Otherwise, notes as Table 2.

Table E.4: Direct & spousal spillover effects of high blood pressure warning on primary outcomes - robustness to using deterministic side-means (see Appendix C)

		Male blood pressure			**Female blood pressure**		
		Systolic (mmHg)	Diastolic (mmHg)	High (0/1)	Systolic (mmHg)	Diastolic (mmHg)	High (0/1)
Male warning							
Wife unwarned	τ_{M-}	-3.695	-.162	.002	-13.001***	-8.259***	-.376***
		(4.843)	(2.719)	(.151)	(3.95)	(2.588)	(.103)
Wife warned	τ_{M+}	12.256	3.609	.243	-8.144	.19	.037
		(8.6)	(4.403)	(.22)	(11.681)	(4.256)	(.241)
Average	$\tau_{\bar{M}}$	1.776	1.132	.085	-11.335**	-5.361**	-.234**
		(4.358)	(2.329)	(.124)	(4.695)	(2.22)	(.106)
Female warning							
Husband unwarned	τ_{F-}	-.482	-.03	-.07	1.592	-1.198	-.024
		(4.186)	(2.525)	(.127)	(5.477)	(4.178)	(.168)
Husband warned	τ_{F+}	-4.712	-1.673	.132	-4.439	-3.209	.135
		(8.722)	(7.339)	(.324)	(13.758)	(6.641)	(.328)
Average	$\tau_{\bar{F}}$	-2.174	-.687	.011	-.821	-2.003	.039
		(4.197)	(3.404)	(.151)	(6.419)	(3.659)	(.168)
Male-Female average	τ	-.339	.158	.045	-5.699	-3.559*	-.088
		(3.054)	(2.071)	(.1)	(3.947)	(2.13)	(.102)
N		384	384	384	384	384	384

Notes: MRDD estimates of the treatment effects defined in Section B. Estimation as for the main estimates in Table 2 except here deterministic side-means are used as explained in Appendix C. Otherwise, notes as Table 2.

Table E.5: Direct & spousal spillover effects of high blood pressure warning on primary outcomes - robustness to using large windows

		Male blood pressure			**Female blood pressure**		
		Systolic (mmHg)	Diastolic (mmHg)	High (0/1)	Systolic (mmHg)	Diastolic (mmHg)	High (0/1)
Male warning							
Wife unwarned	τ_{M-}	-2.933	.731	-.002	-9.82***	-5.11**	-.264***
		(3.949)	(2.455)	(.128)	(3.287)	(2.385)	(.093)
Wife warned	τ_{M+}	10.231	4.519	.358*	-5.92	-3.116	-.104
		(8.855)	(4.543)	(.213)	(8.01)	(5.032)	(.234)
Average	$\tau_{\bar{M}}$	.292	1.659	.086	-8.864***	-4.621**	-.225**
		(3.603)	(2.159)	(.113)	(3.074)	(2.181)	(.092)
Female warning							
Husband unwarned	τ_{F-}	-1.149	-.786	-.113	2.065	1.105	-.004
		(4.306)	(2.852)	(.135)	(5.226)	(3.961)	(.149)
Husband warned	τ_{F+}	-2.6	-2.305	.156	-3.739	-2.708	-.031
		(9.347)	(5.165)	(.278)	(9.176)	(5.114)	(.258)
Average	$\tau_{\bar{F}}$	-1.478	-1.131	-.052	.748	.24	-.01
		(3.778)	(2.517)	(.128)	(4.583)	(3.274)	(.133)
Male-Female average	τ	-.424	.53	.03	-4.974*	-2.654*	-.138*
		(2.617)	(1.55)	(.082)	(2.579)	(1.597)	(.073)
N		599	599	599	599	599	599

Notes: MRDD estimates of the treatment effects defined in Section B. Estimation as for the main estimates in Table 2 except here windows of 0.9 standard deviation are used instead of 0.8. Otherwise, notes as Table 2.

Table E.6: Direct & spousal spillover effects of high blood pressure warning on primary outcomes - robustness to using small windows

		Male blood pressure			**Female blood pressure**		
		Systolic (mmHg)	Diastolic (mmHg)	High (0/1)	Systolic (mmHg)	Diastolic (mmHg)	High (0/1)
Male warning							
Wife unwarned	τ_{M-}	-2.337	1.479	-.058	-9.632**	-4.297	-.204
		(5.281)	(4.031)	(.168)	(4.68)	(3.999)	(.18)
Wife warned	τ_{M+}	9.211	1.6	.34	-8.091	-6.059	-.257
		(10.972)	(5.435)	(.255)	(9.46)	(5.741)	(.265)
Average	$\tau_{\bar{M}}$	1.446	1.519	.072	-9.127**	-4.874	-.221
		(5.037)	(3.163)	(.137)	(4.345)	(3.27)	(.144)
Female warning							
Husband unwarned	τ_{F-}	.178	-1.298	-.133	2.161	1.98	.015
		(5.351)	(3.603)	(.162)	(6.959)	(5.135)	(.207)
Husband warned	τ_{F+}	.544	-2.691	.317	-3.494	-5.606	-.134
		(9.401)	(6.881)	(.324)	(9.429)	(5.74)	(.355)
Average	$\tau_{\bar{F}}$	.259	-1.607	-.033	.907	.297	-.018
		(4.723)	(3.097)	(.154)	(5.885)	(4.214)	(.18)
Male-Female average	τ	.92	.132	.026	-4.676	-2.58	-.131
		(3.558)	(2.038)	(.097)	(3.189)	(2.269)	(.101)
N		305	305	305	305	305	305

Notes: MRDD estimates of the treatment effects defined in Section B. Estimation as for the main estimates in Table 2 except here windows of 0.7 standard deviation are used instead of 0.8. Otherwise, notes as Table 2.

Table E.7: Direct & spousal spillover effects of high blood pressure warning on primary outcomes - robustness to using uniform weights

		Male blood pressure			**Female blood pressure**		
		Systolic (mmHg)	Diastolic (mmHg)	High (0/1)	Systolic (mmHg)	Diastolic (mmHg)	High (0/1)
Male warning							
Wife unwarned	τ_{M-}	-5.169	2.529	-.031	-8.975**	-4.312	-.258*
		(4.564)	(3.083)	(.144)	(4.311)	(3.061)	(.134)
Wife warned	τ_{M+}	11.663	5.741	.43*	-2.446	-2.252	-.077
		(9.764)	(4.568)	(.232)	(9.003)	(5.364)	(.243)
Average	$\tau_{\bar{M}}$	-.109	3.494	.108	-7.012*	-3.693	-.204*
		(4.394)	(2.588)	(.124)	(4.056)	(2.733)	(.116)
Female warning							
Husband unwarned	τ_{F-}	-3.008	-1.848	-.198	-.744	.67	-.043
		(4.984)	(2.997)	(.161)	(5.939)	(4.286)	(.164)
Husband warned	τ_{F+}	2.684	-3.24	.217	.224	-.594	-.048
		(9.815)	(5.529)	(.236)	(8.012)	(4.835)	(.236)
Average	$\tau_{\bar{F}}$	-1.805	-2.142	-.11	-.54	.403	-.044
		(4.272)	(2.664)	(.141)	(4.868)	(3.573)	(.138)
Male-Female average	τ	-.846	1.046	.013	-4.201	-1.914	-.134
		(3.092)	(1.781)	(.089)	(2.906)	(1.734)	(.083)
N		384	384	384	384	384	384

Notes: MRDD estimates of the treatment effects defined in Section B. Estimation as for the main estimates in Table 2 except here uniform weights are used. Otherwise, notes as Table 2.

Table E.8: Direct & spousal spillover effects of high blood pressure warning on primary outcomes - robustness to using epanechnikov weights

		Male blood pressure			**Female blood pressure**		
		Systolic (mmHg)	Diastolic (mmHg)	High (0/1)	Systolic (mmHg)	Diastolic (mmHg)	High (0/1)
Male warning							
Wife unwarned	τ_{M-}	-3.92	1.425	-.044	-10.138**	-5.031*	-.268**
		(4.407)	(3.131)	(.141)	(4.124)	(3.003)	(.13)
Wife warned	τ_{M+}	10.938	3.803	.407*	-6.024	-3.978	-.159
		(10.201)	(4.896)	(.241)	(9.029)	(5.298)	(.256)
Average	$\tau_{\bar{M}}$	.657	2.157	.095	-8.871**	-4.706*	-.234**
		(4.381)	(2.651)	(.123)	(3.951)	(2.702)	(.116)
Female warning							
Husband unwarned	τ_{F-}	-.85	-.959	-.137	.01	.829	-.021
		(4.903)	(2.983)	(.158)	(5.751)	(4.339)	(.169)
Husband warned	τ_{F+}	1.456	-2.728	.245	-2.864	-3.693	-.074
		(9.156)	(5.928)	(.25)	(8.769)	(4.948)	(.285)
Average	$\tau_{\bar{F}}$	-.344	-1.348	-.053	-.621	-.164	-.033
		(4.144)	(2.677)	(.139)	(4.854)	(3.612)	(.146)
Male-Female average	τ	.227	.652	.031	-5.327*	-2.755	-.148*
		(3.044)	(1.752)	(.086)	(2.844)	(1.76)	(.084)
N		384	384	384	384	384	384

Notes: MRDD estimates of the treatment effects defined in Section B. Estimation as for the main estimates in Table 2 except here epanechnikov weights are used. Otherwise, notes as Table 2.

Table E.9: Direct & spousal spillover effects of high blood pressure warning on primary outcomes - robustness to using no control variables

		Male blood pressure			**Female blood pressure**		
		Systolic (mmHg)	Diastolic (mmHg)	High (0/1)	Systolic (mmHg)	Diastolic (mmHg)	High (0/1)
Male warning							
Wife unwarned	τ_{M-}	-2.441	1.472	-.02	-9.678**	-5.086	-.242*
		(4.704)	(3.171)	(.162)	(3.914)	(3.163)	(.135)
Wife warned	τ_{M+}	7.925	3.052	.293	-8.015	-5.260	-.23
		(10.328)	(5.064)	(.24)	(9.01)	(5.271)	(.241)
Average	$\tau_{\bar{M}}$	.675	1.947	.074	-9.178**	-5.139*	-.239**
		(4.549)	(2.741)	(.131)	(3.94)	(2.702)	(.12)
Female warning							
Husband unwarned	τ_{F-}	1.059	-.751	-.083	2.72	1.885	.037
		(5.109)	(3.307)	(.154)	(5.714)	(4.499)	(.178)
Husband warned	τ_{F+}	-.005	-3.463	.262	-3.918	-4.780	-.134
		(8.806)	(5.771)	(.274)	(9.496)	(4.804)	(.309)
Average	$\tau_{\bar{F}}$	.834	-1.324	-.01	1.317	.477	.001
		(4.423)	(3.789)	(.145)	(4.838)	(3.612)	(.159)
Male-Female average	τ	.745	.527	.038	-4.619	-2.699	-.135
		(3.203)	(1.811)	(.095)	(2.976)	(1.999)	(.091)
N		386	386	386	386	386	386

Notes: MRDD estimates of the treatment effects defined in Section B. Estimation as for the main estimates in Table 2 except here no control variables are used. Otherwise, notes as Table 2.

Table E.10: Direct & spousal spillover effects of high blood pressure warning on primary outcomes - robustness to keeping only undiagnosed respondents at baseline

		Male blood pressure			**Female blood pressure**		
		Systolic (mmHg)	Diastolic (mmHg)	High (0/1)	Systolic (mmHg)	Diastolic (mmHg)	High (0/1)
Male warning							
Wife unwarned	τ_{M-}	2.129	3.353	.148	-8.307**	-5.832	-.297**
		(5.627)	(3.753)	(.16)	(3.73)	(3.554)	(.132)
Wife warned	τ_{M+}	5.238	-5.027	.368	-6.406	-7.411	-.315
		(11.009)	(8.461)	(.509)	(13.417)	(9.079)	(.368)
Average	$\tau_{\bar{M}}$	2.904	1.266	.203	-7.834*	-6.226*	-.302**
		(4.894)	(3.653)	(.153)	(4.07)	(3.339)	(.132)
Female warning							
Husband unwarned	τ_{F-}	-.294	2.01	.072	.133	4.513	-.003
		(7.178)	(5.494)	(.251)	(7.97)	(7.687)	(.258)
Husband warned	τ_{F+}	-9.964	-2.115	.203	6.332	.913	.189
		(18.178)	(12.148)	(.856)	(29.713)	(13.417)	(.772)
Average	$\tau_{\bar{F}}$	-1.844	1.349	.093	1.127	3.936	.028
		(6.566)	(4.96)	(.249)	(8.106)	(6.819)	(.27)
Male-Female average	τ	.925	1.3	.157	-4.099	-1.99	-.164
		(3.662)	(2.498)	(.13)	(3.621)	(2.897)	(.118)
N		282	282	282	282	282	282

Notes: MRDD estimates of the treatment effects defined in Section B. Estimation as for the main estimates in Table 2 except here only undiagnosed respondents at baseline are used. Otherwise, notes as Table 2.

Table E.11: Direct & spousal spillover effects of high blood pressure warning on primary outcomes - robustness to keeping spouses who are at least 25 years old at baseline

		Male blood pressure			**Female blood pressure**		
		Systolic (mmHg)	Diastolic (mmHg)	High (0/1)	Systolic (mmHg)	Diastolic (mmHg)	High (0/1)
Male warning							
Wife unwarned	τ_{M-}	-2.645	2.278	-.029	-9.178**	-3.304	-.208
		(4.291)	(3.228)	(.146)	(4.245)	(3.364)	(.148)
Wife warned	τ_{M+}	9.727	3.295	.377	-7.137	-4.637	-.197
		(10.746)	(4.897)	(.247)	(8.775)	(5.589)	(.263)
Average	$\tau_{\bar{M}}$	.902	2.57	.087	-8.593**	-3.687	-.205
		(4.364)	(2.674)	(.125)	(3.904)	(2.869)	(.126)
Female warning							
Husband unwarned	τ_{F-}	-.993	-1.092	-.122	1.12	2.25	0.000
		(5.013)	(3.082)	(.153)	(6.099)	(4.736)	(.182)
Husband warned	τ_{F+}	.743	-1.991	.261	-2.668	-4.002	-.059
		(8.511)	(5.73)	(.264)	(10.079)	(4.801)	(.303)
Average	$\tau_{\bar{F}}$	-.618	-1.286	-.039	.301	.898	-.013
		(4.386)	(2.743)	(.143)	(5.242)	(3.923)	(.155)
Male-Female average	τ	.23	.866	.031	-4.662	-1.66	-.12
		(3.1)	(1.827)	(.087)	(2.857)	(1.959)	(.087)
N		409	409	409	409	409	409

Notes: MRDD estimates of the treatment effects defined in Section B. Estimation as for the main estimates in Table 2 except here spouses who are at least 25 years old at baseline are used. Otherwise, notes as Table 2.

Table E.12: Direct & spousal spillover effects of high blood pressure warning on primary outcomes - robustness to keeping spouses who are at least 35 years old at baseline

		Male blood pressure			**Female blood pressure**		
		Systolic (mmHg)	Diastolic (mmHg)	High (0/1)	Systolic (mmHg)	Diastolic (mmHg)	High (0/1)
Male warning							
Wife unwarned	τ_{M-}	-1.888	3.613	.004	-10.336**	-5.445*	-.313**
		(5.286)	(3.687)	(.167)	(4.17)	(3.057)	(.155)
Wife warned	τ_{M+}	9.464	2.12	.334	-11.16	-5.941	-.295
		(11.004)	(5.445)	(.247)	(9.255)	(5.587)	(.277)
Average	$\tau_{\bar{M}}$	1.891	3.116	.114	-10.61***	-5.61**	-.307**
		(4.961)	(2.934)	(.138)	(4.046)	(2.859)	(.137)
Female warning							
Husband unwarned	τ_{F-}	.205	-1.11	-.146	.186	.971	-.024
		(5.076)	(3.278)	(.166)	(6.405)	(4.777)	(.196)
Husband warned	τ_{F+}	4.542	-5.072	.395	1.269	-3.594	-.187
		(11.662)	(6.903)	(.307)	(8.771)	(5.413)	(.366)
Average	$\tau_{\bar{F}}$	.965	-1.804	-.051	.376	.171	-.052
		(4.732)	(2.949)	(.16)	(5.425)	(4.155)	(.174)
Male-Female average	τ	1.478	.92	.04	-5.707*	-3.03	-.193**
		(3.4)	(1.952)	(.098)	(2.971)	(1.912)	(.094)
N		338	338	338	338	338	338

Notes: MRDD estimates of the treatment effects defined in Section B. Estimation as for the main estimates in Table 2 except here spouses who are at least 35 years old at baseline are used. Otherwise, notes as Table 2.

Table E.13: Direct and spousal spillover effects of high blood pressure warning on baseline to follow-up changes in blood pressure

		Male blood pressure		**Female blood pressure**	
		Δ Systolic BP	Δ Diastolic BP	Δ Systolic BP	Δ Diastolic BP
Male warning					
Wife unwarned	τ_{M-}	-3.32	2.46	-8.149*	-5.815**
		(4.454)	(3.501)	(4.441)	(2.839)
Wife warned	τ_{M+}	12.431	3.022	-7.405	-6.217
		(10.028)	(6.295)	(9.075)	(5.827)
Average	$\tau_{\bar{M}}$	1.415	2.629	-7.926*	-5.936**
		(4.357)	(3.038)	(4.116)	(2.732)
Female warning					
Husband unwarned	τ_{F-}	-2.115	1.135	3.553	1.006
		(5.215)	(3.314)	(6.632)	(4.515)
Husband warned	τ_{F+}	-.892	.245	-1.323	-1.351
		(10.853)	(6.22)	(7.934)	(5.318)
Average	$\tau_{\bar{F}}$	-1.856	.947	2.523	.508
		(4.686)	(2.887)	(5.618)	(3.761)
Male-Female average	τ	-.006	1.899	-3.387	-3.137*
		(3.332)	(1.971)	(3.393)	(1.796)
N		384	384	384	384

Notes: MRDD estimates of the treatment effects defined in Section B. Outcomes are baseline to follow-up changes in blood pressure. Estimation and notes as in Table 2.

Table E.14: Direct & spousal spillover effects of high blood pressure warning on primary outcomes - unidimensional RDD estimates

	Male blood pressure				***Female blood pressure***			
	Male warning		Female warning		Male warning		Female warning	
	Wife unwarned	Wife warned	Husband unwarned	Husband warned	Wife unwarned	Wife warned	Husband unwarned	Husband warned
Systolic BP	3.713	3.642	2.065	-4.936	-5.559**	-12.166	2.441	-7.268
	(2.728)	(.644)	(3.406)	(7.436)	(2.742)	(8.389)	(3.959)	(6.435)
N	790	159	596	185	926	224	1502	166
Diastolic BP	2.315	-2.469	1.129	-1.756	-3.37**	-1.541	2.649	-3.359
	(1.851)	(4.706)	(2.067)	(4.137)	(1.656)	(3.867)	(2.653)	(3.716)
N	1431	128	723	166	932	176	1393	163
High BP	.041	.052	.012	.271	-.143**	-.051	.03	-.336
	(.084)	(.204)	(.089)	(.189)	(.058)	(.178)	(.116)	(.194)
N	961	107	505	162	1904	177	667	147

Notes: Unidimensional RDD estimates obtained from local linear regression, with triangular kernels and optimal bandwidths on each side of the threshold using the MSE optimal bandwidth selector. Heteroscedasticity-robust standard errors are reported in parentheses in the second rows of each outcome. The "Male blood pressure" & "Male warning" panel gives direct effects of warning a man about his high BP on his BP when his wife does not get a warning ("Wife unwarned") and does ("Wife warned"). The "Male blood pressure" & "Female warning" panel gives spillover effects of warning a woman about her high BP on her husband's BP when he does not get a warning ("Husband unwarned") and does ("Husband warned"). Estimates in the other panels interpreted analogously. N is the effective number of observations used in estimation. High BP is an indicator of blood pressure exceeding the hypertension thresholds at follow-up ($= \mathbb{1}\{\text{Systolic BP} \geq 140 \vee \text{Diastolic BP} \geq 90\}$). See section III. for detailed definitions of outcomes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table E.15: Direct & spousal spillover effects of a high blood pressure warning on primary outcomes - robustness to not using donut-hole MRDD

		Male blood pressure			**Female blood pressure**		
		Systolic (mmHg)	Diastolic (mmHg)	High (0/1)	Systolic (mmHg)	Diastolic (mmHg)	High (0/1)
Male warning							
Wife unwarned	τ_{M-}	-3.62	-2.006	-.109	-2.094	-4.884***	-.147*
		(3.122)	(2.245)	(.104)	(3.356)	(1.871)	(.087)
Wife warned	τ_{M+}	.864	.676	.024	-5.5	-3.381	-.09
		(4.129)	(2.875)	(.149)	(5.13)	(2.966)	(.148)
Average	$\tau_{\bar{M}}$	-1.12	-.511	-.035	-3.993	-4.046**	-.115
		(2.672)	(1.886)	(.093)	(3.253)	(1.891)	(.09)
Female warning							
Husband unwarned	τ_{F-}	3.36	1.158	.073	2.004	-.18	-.027
		(3.122)	(2.073)	(.101)	(3.959)	(2.741)	(.118)
Husband warned	τ_{F+}	-7.939**	-6.563**	-.107	-3.439	-1.359	-.049
		(3.787)	(2.929)	(.133)	(4.934)	(2.497)	(.132)
Average	$\tau_{\bar{F}}$	-3.195	-3.321*	-.032	-1.153	-.864	-.04
		(2.61)	(1.933)	(.09)	(3.35)	(1.88)	(.093)
Male-Female average	τ	-2.132	-1.882	-.033	-2.607	-2.493*	-.078
		(1.854)	(1.326)	(.063)	(2.229)	(1.292)	(.063)
N		588	588	588	588	588	588

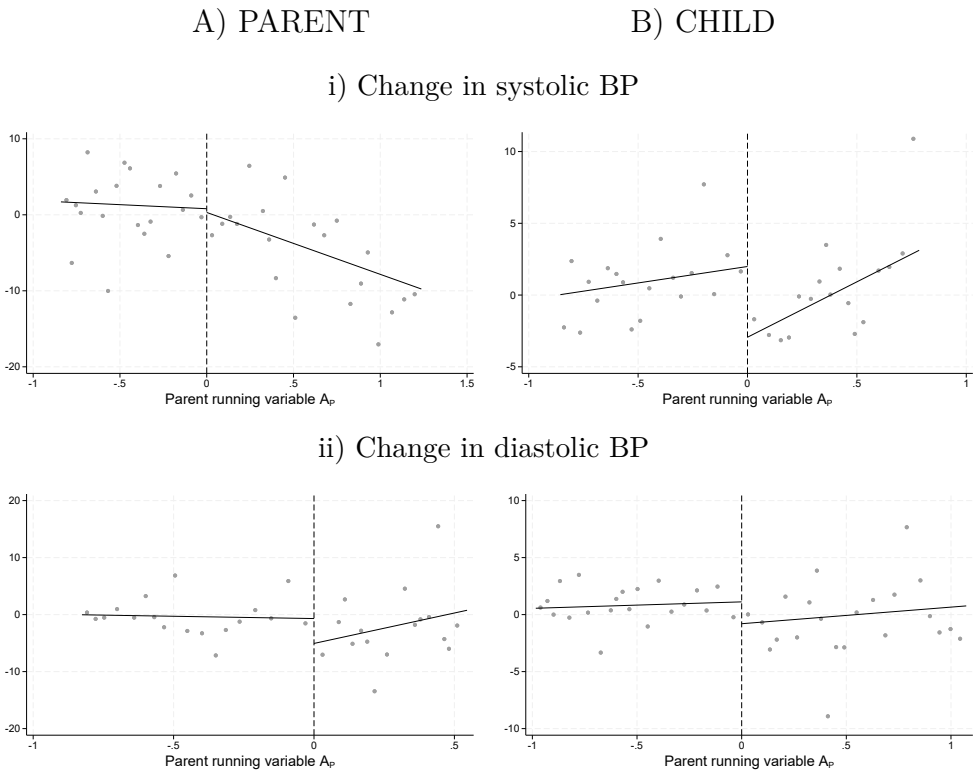
Notes: MRDD estimates of the treatment effects defined in Section B. Estimation as for the main estimates in Table 2 except here observations with blood pressure precisely at the treatment thresholds at baseline are not excluded from the sample. That is, donut-hole MRDD is not used. Otherwise, notes as Table 2.

Table E.16: Direct and parental spillover effects of warning parent of high blood pressure

	Δ Systolic BP		Δ Diastolic BP	
Parent warning				
Parent	-0.906	(2.694)	-5.560***	(2.067)
N	1106		877	
Adult child	-5.855**	(2.530)	-2.482	(1.903)
N	985		1412	

Notes: The table reports the effects of warning a parent about high blood pressure at baseline on changes in outcomes between baseline and follow up. Unidimensional RDD estimates are obtained using local linear regressions with triangular kernels and MSE optimal bandwidths selected separately on each side of the threshold. Heteroscedasticity robust standard errors reported in parenthese. *N* denotes the effective number of observations used in estimation. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Figure E.1: Parent & adult child changes in BP against parent running variable



Notes: Mean outcome on y-axis against parent running variable (A_P) on x-axis. Outcomes are baseline to follow-up changes in blood pressure. At $A_P \geq 0$, the parent is warned at baseline they have high BP. Each dot is the mean outcome in a given bin. We use optimal bins obtained from the variance quantile-spaced method using spacing estimators (Calonico *et al.*, 2014a,b, 2015, 2017). Best-fit lines estimated with local linear regression, triangular kernels and the MSE optimal bandwidth selector.

Table E.17: Effects of warning on parent and child outcomes – additional results and robustness checks

	Systolic BP (mmHg)		Diastolic BP (mmHg)		High BP (0/1)	
A. Estimation by adult child's sex						
Son	-5.578**	(2.745)	-0.481	(1.933)	-0.102	(.071)
N	703		1281		1003	
Daughter	-3.331	(5.648)	-1.876	(3.789)	0.054	(.091)
N	383		391		386	
B. Quadratic specification						
Parent	-3.430	(3.711)	-2.973	(2.181)	-0.154	(.106)
N	1612		1150		1923	
Adult child	-6.934**	(3.025)	-2.138	(2.370)	-0.070	(.068)
N	1444		1823		1943	
C. Same optimal bandwidth on both sides of the cutoff						
Parent	-1.626	(2.843)	-1.941	(1.737)	-.124	(.094)
N	1387		951		920	
Adult child	-4.793**	(2.193)	-1.728	(1.977)	-.064	(.064)
N	1398		1354		972	

Notes: Panel A: Unidimensional RDD estimates of effects of warning a parent about high blood pressure at baseline on adult son and daughter outcomes at follow up. Panel B: Unidimensional RDD estimates derived with local quadratic instead of local linear regression. Panel C: Unidimensional RDD estimates derived using a common MSE optimal bandwith on both side of the cutoff. Outcomes are defined in Section III. High BP is indicator of BP above hypertension thresholds (= $\mathbb{1}\{\text{Systolic BP} \geq 140 \vee \text{Diastolic BP} \geq 90\}$). Heteroscedasticity robust standard errors reported in parentheses. N denotes the effective number of observations used in estimation. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Table E.18: Effects of warning on parent and child outcomes using unidimensional RDD – further robustness checks

	Systolic BP (mmHg)		Diastolic BP (mmHg)		High BP (0/1)	
A. Uniform weights						
Parent	-3.015	(3.064)	-1.567	(1.670)	-0.128	(.092)
N	870		819		770	
Adult child	-4.802*	(2.477)	-1.153	(1.744)	-0.064	(.062)
N	855		899		870	
B. Epanechnikov weights						
Parent outcomes	-2.493	(3.023)	-1.129	(1.727)	-0.125	(.096)
N	1009		1284		944	
Adult Child	-5.682**	(2.593)	-1.479	(1.865)	-0.066	(.062)
N	933		1398		1288	
C. No control variables						
Parent outcomes	-2.336	(2.963)	-0.773	(1.667)	-0.129	(.096)
N	1479		1495		1296	
Adult child	-5.750**	(2.648)	-1.584	(1.943)	-0.067	(.064)
N	965		1422		1352	

Notes: Panel A: Unidimensional RDD estimates of effects of warning a parent about high blood pressure at baseline on adult child using uniform weights instead of triangular weights. Panel B: Unidimensional RDD estimates derived using epanechnikov weights instead of triangular weights. Panel C: Unidimensional RDD estimates derived without using any predetermined control variables in the specification. Outcomes are defined in Section III. *High BP* = $\mathbb{1}(SBP \geq 140 \vee DBP \geq 90)$. Heteroscedasticity robust standard errors reported in parentheses. N denotes the effective number of observations used in estimation. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Table E.19: Effects of warning on parent and child outcomes using unidimensional RDD – further robustness checks

	Systolic BP (mmHg)		Diastolic BP (mmHg)		High BP (0/1)	
A. Household cluster standard errors						
Parent	-2.580	(3.324)	-1.211	(1.820)	-0.125	(.103)
N	1026		1454		1005	
Adult child	-5.852**	(2.813)	-1.462	(2.062)	-0.069	(.066)
N	905		1458		1343	
B. Restriction to undiagnosed parents at baseline						
Parent	-0.225	(3.443)	-0.630	(1.797)	-0.072	(.101)
N	798		1234		1151	
Adults child	-6.615**	(2.665)	-1.512	(1.879)	-0.079	(.067)
N	682		1208		1172	
C. Including heaping observations						
Parent	-4.830**	(2.088)	0.408	(1.006)	-0.061	(.059)
N	1452		2030		1527	
Adult child	-4.721***	(1.561)	-1.493	(1.057)	-0.050	(.038)
N	1409		1912		1834	

Notes: Panel A: Unidimensional RDD estimates of effects of warning a parent about high blood pressure at baseline on adult child clustering standard errors at the household level. Panel B: Unidimensional RDD estimates derived from a sample restricted to parents who were undiagnosed with high blood pressure at baseline. panel C: Unidimensional RDD estimates derived from a sample that includes heaping observations, that is moving from donut-hole RDD to RDD. Outcomes are defined in Section III. $High\ BP = \mathbb{1}(SBP \geq 140 \vee DBP \geq 90)$. N denotes the effective number of observations used in estimation. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

F Additional results - secondary outcomes

Table F.1: Counterfactuals of secondary outcomes at follow-up - spousal dyads

	Male		Female		
	Mean	SD	Mean	SD	N
Husband warning, wife unwarned					
Diagnosis	.173	.382	.019	.137	52
Medication	.096	.298	.019	.137	52
Knowledge	.232	.947	.344	.832	25
Diet	.256	.720	-.003	1.010	53
Behaviors	-.056	.900	-.052	.812	53
Adiposity	.449	1.363	.205	1.268	53
Husband warning, wife warned					
Diagnosis	.25	.452	.8	.422	12
Medication	.25	.452	.583	.515	12
Knowledge	.334	.910	.150	.699	7
Diet	.099	.522	.099	.765	12
Behaviors	.159	.816	.262	.317	12
Adiposity	1.316	1.516	.846	1.272	11
Wife warning, husband unwarned					
Diagnosis	.111	.319	.105	.311	36
Medication	0	0	.079	.273	37
Knowledge	-.112	.900	.288	.673	21
Diet	.190	.830	-.050	.963	38
Behaviors	.294	1.238	.050	.5843	37
Adiposity	.727	1.445	.804	1.430	38
Wife warning, husband warned					
Diagnosis	.375	.518	.25	.463	8
Medication	.125	.354	.25	.463	8
Knowledge	-.639	.512	-.085	.724	5
Diet	.037	1.336	-1.269	1.581	9
Behaviors	.904	1.332	.114	1.378	9
Adiposity	.356	.866	.864	1.505	9

Notes: Descriptive statistics for secondary outcomes at follow-up for observations located within 0.1 of a standard deviation of the frontier on the untreated side. Each panel corresponds to a different frontier defined by treatment status. SD denotes standard deviation.

Table F.2: Spillover effects of male high BP warning on components of wife's *Diet* and *Adiposity* outcomes

		Diet					***Adiposity***			
		Prep. food	No fast food	No salty snacks	No soft drinks	Fat (grams)	BMI	BMI ≥ 27.5	WHR	WHR ≥ 0.58
Male warning										
Wife unwarned	τ_{M^-}	.154*	.206	.13	.275	-1.686	-2.05**	-.237**	-.017	-.366***
		(.083)	(.209)	(.193)	(.186)	(8.611)	(.856)	(.097)	(.016)	(.104)
FDR q-value		.455	.545	.601	.455	1	.017	.017	.076	0.002
Wife warned	τ_{M^+}	-.018	-.21	-.256	-.131	.824	.6	.329	.012	-.09
		(.112)	(.269)	(.235)	(.299)	(10.303)	(1.78)	(.229)	(.033)	(.255)
FDR q-value		1	1	1	1	1	1	1	1	1
Average	$\tau_{\bar{M}}$	.105	.087	.019	.158	-.966	-1.29	-.075	-.009	-.287***
		(.069)	(.176)	(.161)	(.177)	(6.644)	(.797)	(.107)	(.015)	(.105)
FDR q-value		1	1	1	1	1	.189	.375	.375	.027
N		388	219	219	219	387	384	384	382	382

Notes: MRDD estimates of effects (discontinuity parameters) defined in Section IV. Outcomes are components of the composite *Diet* and *Adiposity* indices. See Appendix B for definitions. BMI = body mass index. WHR = waist-to-height ratio. Bootstrapped (1000 repetitions) standard errors in parentheses. Some standard errors might be estimated with fewer replications because of small sample sizes. FDR q-value is the False Discovery Rate Sharpened q-value that corrects for multiple comparisons, treating all outcomes as belonging to one family (Anderson, 2008). Asterisks indicate statistical significance based on p-values without multiple comparisons correction: $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Table F.3: Spillover effects of male high blood pressure warning on components of wife's secondary *Knowledge* outcome

		Knowledge								
		Fruit	Sugar	Variety	Fat	Fatty	Beans	Activity	Sweaty	Heavy
Male warning										
Wife unwarned	τ_{M-}	-.502*	-.05	-.021	-.015	.017	-.412	-.005	-.13	.025
		(.301)	(.141)	(.257)	(.187)	(.308)	(.251)	(.245)	(.389)	(.286)
FDR q-value		.834	1	1	1	1	.834	1	1	1
Wife warned	τ_{M+}	.302	.095	-.11	.145	-.318	-.268	.061	-.213	-.305
		(.445)	(.283)	(.477)	(.441)	(.439)	(.456)	(.42)	(.748)	(.228)
FDR q-value		1	1	1	1	1	1	1	1	1
Average	$\tau_{\bar{M}}$	-.271	-.008	-.047	.031	-.079	-.371	.014	-.154	-.07
		(.252)	(.138)	(.229)	(.187)	(.243)	(.229)	(.207)	(.348)	(.211)
FDR q-value		1	1	1	1	1	1	1	1	1
N		218	216	215	214	214	216	216	212	215

Notes: MRDD estimates of effects (discontinuity parameters) defined in Section IV. Outcomes are components of the composite *Knowledge* index. See Appendix B for definitions. Bootstrapped (1000 repetitions) standard errors in parentheses. Some standard errors might be estimated with fewer replications because of small sample sizes. FDR q-value is the False Discovery Rate Sharpened q-value that corrects for multiple comparisons, treating all outcomes as belonging to one family (Anderson, 2008). Asterisks indicate statistical significance based on p-values without multiple comparisons correction: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table F.4: Spillover effects of male high blood pressure warning on components of wife's *Behavior* outcome

		Behavior									
		Martial arts	Track & field	Gym/ dancing	Soccer/ basket/ tennis	Badm./ volley	Ping -pong/ tai-chi	Ever smoked	Current smoker	Cigs. daily	Alcohol last year
Male warning											
Wife unwarned	τ_{M^-}	-.017	.027	-.036	-.019	<0.001	.239*	.032	.039	.521	-.073
		(.03)	(.122)	(.038)	(.017)	(<0.001)	(.125)	(.03)	(.03)	(.456)	(.072)
FDR q-value		.828	.828	.828	.828	.828	.828	.828	.828	.828	.828
Wife warned	τ_{M^+}	-.022	.026	-.05	-.003	<0.001	.094	-.015	-.017	-.502	.141
		(.065)	(.052)	(.056)	(.013)	(<0.001)	(.184)	(.049)	(.048)	(1.257)	(.183)
FDR q-value		1	1	1	1	1	1	1	1	1	1
Average	$\tau_{\bar{M}}$	-.018	.027	-.04	-.014	<0.001	.198**	.018	.023	.227	-.012
		(.029)	(.085)	(.029)	(.013)	(<0.001)	(.101)	(.025)	(.025)	(.46)	(.075)
FDR q-value		1	1	1	1	1	1	1	1	1	1
N		280	280	280	280	280	280	393	392	393	391

Notes: MRDD estimates of effects (discontinuity parameters) defined in Section IV. Outcomes are components of the composite *Behavior* index. See Appendix B for definitions. Bootstrapped (1000 repetitions) standard errors in parentheses. Some standard errors might be estimated with fewer replications because of small sample sizes. FDR q-value is the False Discovery Rate Sharpened q-value that corrects for multiple comparisons, treating all outcomes as belonging to one family (Anderson, 2008). Asterisks indicate statistical significance based on p-values without multiple comparisons correction: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table F.5: Direct effects of male high blood pressure warning on own secondary outcomes

		Diagnosis	Medication	Knowledge	Diet	Behavior	Adiposity
Male warning							
Wife unwarned	τ_{M^-}	-.11	-.016	.017	.058	-.072	-.985**
		(.116)	(.111)	(.416)	(.241)	(.264)	(.392)
Wife warned	τ_{M+}	.27	.221	-1.115	.607**	.259	.715
		(.23)	(.222)	(.709)	(.269)	(.233)	(.886)
Average	$\tau_{\bar{M}}$	-.001	.052	-.308	.215	.023	-.498
		(.109)	(.104)	(.361)	(.19)	(.203)	(.38)
N		387	390	219	395	394	391

Notes: MRDD estimates of the treatment effects (discontinuity parameters) defined in Section B. on secondary outcomes. Based on weighted least squares estimates of local linear regression 2 using triangular kernel weights over a rectangular neighborhood around treatment frontier, with optimal univariate RDD bandwidth of 0.8. Effects derived using empirical, frontier-weighted side-means estimated with with one-dimensional triangular kernel and strip width of 0.08. See section III. and Appendix F for definitions of these outcomes. Bootstrapped (1000 repetitions) standard errors in parentheses.. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table F.6: Direct effects of male high BP warning on components of own *Adiposity* outcome

		BMI	BMI ≥ 27.5	WHR	WHR ≥ 0.55
Male warning					
Wife unwarned	τ_{M^-}	-1.542*	-.249**	-.034*	-.239
		(.876)	(.1)	(.021)	(.155)
FDR q-value		.103	.054	.103	.103
Wife warned	τ_{M^+}	3.447*	.223	.034	.305
		(2.066)	(.176)	(.039)	(.246)
FDR q-value		.402	.402	.402	.402
Average	$\tau_{\bar{M}}$	-.112	-.113	-.015	-.083
		(.872)	(.094)	(.019)	(.134)
FDR q-value		1	1	1	1
N		387	383	383	381

Notes: MRDD estimates of effects (discontinuity parameters) defined in Section IV. Outcomes are components of the composite *Adiposity* index. See Appendix B for definitions. BMI = body mass index. WHR = waist-to-height ratio. Bootstrapped (1000 repetitions) standard errors in parentheses. Some standard errors might be estimated with fewer replications because of small sample sizes. FDR q-value is the False Discovery Rate Sharpened q-value that corrects for multiple comparisons, treating all outcomes as belonging to one family (Anderson, 2008). Asterisks indicate statistical significance based on p-values without multiple comparisons correction: * $p < 0.1$, ** $p < 0.05$,

Table F.7: Direct effects of a high BP warning to a parent on secondary outcomes

	Knowledge	Diet	Behavior	Adiposity
Parent warning				
Parent	0.143	-0.014	-0.044	0.225
	(.250)	(.150)	(.166)	(.242)
N	298	1577	754	1406

Notes: Unidimensional RDD estimates of direct effects of warning a parent about high blood pressure at baseline on their secondary outcomes at follow up. Outcomes are defined in Section III. and Appendix B. Local linear regressions with triangular kernels and MSE optimal bandwidths selected separately on each side of the threshold. Heteroscedasticity robust standard errors reported in parentheses. N denotes the effective number of observations used in estimation. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Table F.8: Intergenerational spillover effects of a high BP warning on components of secondary outcome composite indices

	Effect	SE	FDR q-value	N
Knowledge				
Fruit	.058	.259	1	279
Sugar	.318**	.141	.283	261
Variety	.188	.182	.575	464
Fat	.02	.173	1	307
Fatty	-.04	.216	1	331
Beans	.25	.178	.397	251
Activity	.365*	.191	.294	219
Sweaty	.158	.304	1	282
Heavy	.298*	.178	.343	272
Diet				
Prep. food	-.028	.078	1	966
No fast food	-.057	.110	1	283
No salty snacks	-.124	.099	1	333
No soft drinks	-.029	.084	1	284
Fat (grams)	-11.087	6.804	1	736
Behavior				
Martial arts	.008	.014	.499	396
Track & field	-.063	.050	.241	485
Gymnastics and dancing	-.005	.005	.310	275
Soccer, basketball, tennis	-.008	.045	.549	771
Badminton, volleyball	.037	.039	.310	545
Ping-pong, tai-chi	-.114**	.057	.171	454
Ever smoked	-.163*	.092	.186	949
Current smoker	-.184**	.092	.171	896
No. cigarettes daily	-3.73***	1.526	.171	1306
Alcohol last year	-.16*	.096	.186	885
Adiposity				
BMI	1.043*	.583	.110	1118
BMI $\geq$ 27.5	.112**	.057	.110	1708
Waist-to-height ratio (WHR)	.01	.010	.110	1405
WHR $\geq$ 0.55/0.58	.106**	.050	.110	1050

Notes: Unidimensional RDD estimates of spillover effects from parental high blood pressure (BP) warning on components of secondary outcome composite indices for adult children. Local linear regression, with triangular kernels and optimal bandwidths on each side of the threshold using the MSE optimal bandwidth selector. SE is heteroscedasticity-robust standard error. FDR q-value is the False Discovery Rate Sharpened q-value that corrects for multiple comparisons, treating all outcomes as belonging to one family (Anderson, 2008). Asterisks indicate statistical significance based on p-values without multiple comparisons correction: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. N is the effective number of observations used in estimation.

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