PEACE OF MIND: HEALTH INSURANCE REDUCES STRESS AND CORTISOL LEVELS – EVIDENCE FROM A RANDOMIZED EXPERIMENT IN KENYA*

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Abstract

We show that the provision of health insurance reduces levels of self-reported stress and the stress hormone cortisol using a randomized controlled trial among informal workers in Nairobi, Kenya. The effects of health insurance on cortisol and stress levels are larger than those of equally valued unconditional cash transfers, and are not mediated by changes in economic outcomes, health or healthcare usage, or variables that confound cortisol. These results suggest that insurance may reduce stress and cortisol levels through a "peace of mind" effect. This hypothesis receives support from the fact that the median insurance taker does not use the insurance; that insurance has larger effects on more vulnerable individuals; and that insurance improves sleep.

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I. Introduction

Stress has significant welfare costs. First, stress is an undesirable outcome in its own right; individuals are generally averse to stress and in the cross-section, stress correlates with other undesirable psychological outcomes such as unhappiness and depression (Haushofer and Shapiro 2016). Second, stress is associated with adverse physiological consequences; repeated stressful situations results in wear and tear on the body ("allostatic load"), which prematurely ages the immune system and is associated with a spectrum of conditions including cardiovascular disease, HIV progression, and cancer (McEwen BS and Stellar E 1993; Cohen S, Janicki-Deverts D, and Miller GE 2007). Third, stress may have economic costs as a result of its link with depression. This relationship is intimate: roughly 80 percent of depression diagnoses are preceded by major stressful life events (Hammen 2005), and one of the most prominent accounts of the neurobiology of depression is dysfunction of the hormonal system that regulates the release of the stress hormone cortisol (Holsboer 2000). Depression, in turn, has significant economic costs; current estimates hover around 1-1.5 percent of GDP per year in terms of lost productivity alone (Sobocki et al. 2006). Lastly, there is recent evidence that stress may also have economic consequences by affecting individual economic choice. Laboratory studies have demonstrated that stress increases risk aversion and impatience, and reduces the effort people expend on learning about economic decisions (Porcelli and Delgado 2009; Cornelisse et al. 2013; Delaney, Fink, and Harmon 2014).

Together, these facts suggest that there may be an economic case for reducing stress as a matter of policy. How can this be achieved? One approach is to reduce stress directly, e.g. through psychotherapy-type interventions. Field experiments in developing countries have provided evidence of the efficacy of such approaches (Bolton et al. 2003; Blattman, Jamison, and Sheridan 2015). The present study takes a different approach by asking whether economic interventions can reduce stress. In assessing which kinds of interventions might be most effective to this end, we begin with two observations. First, stress is strongly associated with environmental uncertainty and volatility, usually referred to as "unpredictability" and

"uncontrollability" in the neurobiology literature (Miller 1979; Koolhaas et al. 2011). If this relationship is partially causal, we would expect that interventions that reduce volatility should also be effective at reducing stress. Indeed, expected utility theory predicts welfare gains for risk-averse individuals who experience a reduction of income volatility in expectation. Second, programs that increase the level of consumption, such as cash transfers, have recently been shown to reduce self-reported stress and depression symptoms, as well as, in some cases, cortisol levels (Fernald and Gunnar 2009; Haushofer and Shapiro 2016). Together, these relationships suggest that both decreases in exposure to shocks and volatility, and increases in consumption levels, might lead to reductions in stress.

This study tests these two hypotheses by asking whether health insurance and unconditional cash transfers (UCT) reduce levels of self-reported stress and the stress hormone cortisol. We conducted a randomized controlled trial with 789 informal workers in Nairobi, Kenya, in which one group received a free health insurance policy for themselves and their families for one year, a second group received an unconditional cash transfer worth the retail price of the insurance, and a third group received no intervention. The comparison between the insurance group and the unconditional cash transfer group is important because it allows us to distinguish the effect of being insured from the income effect of receiving free insurance; our research design thus allows us to empirically evaluate the benefits of insurance without resorting to structural assumptions about the utility function. We report treatment effects on a variety of health and welfare outcomes one year after the beginning of the intervention. To measure stress, our endline questionnaire included a battery of measures of psychological well-being such as self-reported stress, depression, and happiness. In addition, we collected saliva samples to analyze levels of the stress hormone cortisol. All analyses were pre-registered in a pre-analysis plan, available at `https://www.socialscienceregistry.org/trials/647`.

A year after the beginning of the intervention, we find a 0.29 SD decrease in self-reported stress as measured by the Perceived Stress Scale (Cohen, Kamarck, and Mermelstein 1983), and a 16 percent reduction in cortisol levels in the insurance group relative to control, with no such reduction in the cash group, and a significant difference in cortisol levels between

the insurance and cash groups . The effect on cortisol accounts for roughly 60 percent of the difference in morning cortisol levels between depressed and non-depressed individuals reported by a recent meta-analysis (Knorr et al. 2010). These results complement a small number of existing studies on the effect of health insurance and cash transfers on stress and cortisol: Baicker et al. (2013) find a 30.5 percent reduction in depression among insurance beneficiaries in the Oregon Health Insurance Lottery; Haushofer and Shapiro (2016) find a decrease in cortisol levels of similar magnitude as the one we observe for insurance after a USD 1,520 PPP cash transfer, but no effect with USD 400 PPP cash transfers. In line with this literature, we find no reduction in stress with a USD 338 PPP cash transfer.

What might be the channel through which insurance, but not the cash equivalent, reduces self-reported stress and cortisol levels? We consider several candidate mechanisms that might underlie the effect. First, we find no evidence that insurance improved economic outcomes such as asset holdings or consumption, arguing against the possibility that improvements in economic outcomes led to decreased stress and cortisol levels. Second, insurance provision did not improve health care utilization or health status: The cash and control groups visited health facilities just as often as the insurance group and were equally likely to be sick one year after the interventions. The reduction in stress also does not depend on usage of the insurance product: we find an equivalent reduction in stress for both users and non-users, using matching to create comparable groups. Third, variables that can confound cortisol levels, such as eating, drinking, and exercise, also do not account for the treatment effect of insurance on cortisol levels, as controlling for these variables does not change our estimates.

The most plausible mechanism for the effect of insurance on stress and cortisol levels is a "peace of mind" effect that results from merely having coverage, and that is not produced by receiving a cash transfer of equal magnitude. In this sense, our findings are in line with the core insight of expected utility theory: risk-averse individuals dislike variance, and insurance reduces variance and thereby increases utility. Further in line with this view, we find that the reduction in cortisol levels is larger for those more vulnerable to adverse shocks: households with children, low-income earners, those in worse health at baseline, and

those with higher scores on the CES-D depression scale. A tantalizing complement to this argument is the post-hoc discovery that insurance recipients sleep significantly longer than the control group.

This study extends an emerging literature on the psychological consequences of poverty and poverty alleviation. It has recently been argued that poverty may have adverse psychological consequences, which could have negative effects on economic choice and thereby perpetuate poverty (Shah, Mullainathan, and Shafir 2012; Mani et al. 2013; Haushofer and Fehr 2014). One crucial element in providing evidence for this feedback loop is to demonstrate causal effects of poverty alleviation on psychological well-being. Haushofer and Shapiro (2016) shows that large cash transfers of USD 1,520 PPP reduced cortisol levels. Similar effects have been shown for other cash transfer programs (Baird, McIntosh, and Özler 2011; Baird et al. 2012; Baird, Hoop, and Özler 2013; Baird et al. 2014), the provision of piped water (Devoto et al. 2012), the Moving to Opportunity Program (Kling, Liebman, and Katz 2007), and lotteries (Gardner and Oswald 2006; Cesarini et al. 2016). We extend this literature by showing that insurance can reduce stress and cortisol levels, and this effect is larger than that observed for a cash transfer of equal nominal value.

The rest of the paper is structured as follows. Section II describes the insurance policy and cash transfer. Section III outlines the experimental design. Section IV lays out the econometric framework. Section V presents the main results, and Section VI concludes.

II. Interventions

To identify the causal impact of health insurance and cash transfers on health and welfare outcomes, we randomized a sample of informal workers into two treatment groups and one control group. The first treatment group received an insurance product free of charge, while the second treatment group received an unconditional cash transfer equal to the cost of the insurance. Comparing the two treatment arms controls for any income effect of insurance, and allows us to evaluate the impact of providing insurance relative to a cash transfer. The control group received no intervention.

A. Health insurance

Respondents in the insurance group were enrolled in the *Afya Bora* plan, a combined inpatient and outpatient family health insurance policy offered by the Cooperative Insurance Company (CIC). These treated households were eligible for inpatient benefits of up to USD 6,437 PPP per family that covered the costs of a broad array of services, including hospital accommodation, doctor's fees, routine lab tests, UCI charges, medications, and maternity services. Chronic and pre-existing conditions were covered up to USD 1,931 PP. Households were also eligible for outpatient benefits of up to USD 1,287 PPP per family that covered routine outpatient consultations, medication (including ARVs), laboratory services, pre- and post-natal care, oncology, and psychiatry and psychotherapy. Both inpatient and outpatient covers included chronic and pre-existing conditions, including HIV/AIDS, up to USD 515 PPP, but excluded treatment outside Kenya, cosmetic treatment, treatment by non-qualified persons, infertility, self-inflicted injury, experimental treatment, and dental treatment unless occasioned by accidental injury. Beneficiaries could access these benefits through CIC's network of providers that included 26 mission and faith-based hospitals in Nairobi. Full details of the insurance cover are given in Appendix A. Beneficiaries paid KES 100 (USD 2.60 PPP) co-pay for each outpatient visit.

The plan provided benefits to principals and spouses under 72 years of age, and children dependents younger than 25 years with proof of enrollment in school or college. Respondents were enrolled in the *Afya Bora* plan free of charge for one year, a value of USD 328 PPP for the principal, spouse and up to five dependents. Each additional child dependent increased the annual premium by USD 52 PPP per child. The project fully covered households for the base cost and any added premium for more than five dependents. The average cost of the policy in our sample was USD 338 PPP.

B. Unconditional cash transfer

Respondents in the second treatment group received an unconditional cash transfer equal to the annual premium they would have had to pay had they enrolled in the *Afya Bora* plan. The magnitude of this transfer was USD 328 PPP for households with up to five dependents,

with an additional USD 52 PPP for each dependent beyond the first five, for an average of USD 338 PPP across the sample. The transfer was delivered to recipients electronically using the M-Pesa mobile money service. M-Pesa is a mobile money system offered by *Safaricom*, the largest Kenyan mobile phone operator. Using M-Pesa requires a registered SIM card and a valid Kenyan national ID card. The money was transferred from Innovations for Poverty Action Kenya's M-Pesa account to that of the recipient. To facilitate the transfers, we encouraged recipients to sign up for M-Pesa and helped them obtain, where necessary, the documents required for registration.[1] The money was transferred to the registered SIM card and the recipient could withdraw the balance at any of the large number of M-Pesa agents in Kenya by putting the SIM card into the agent's cell phone or by using their own phone.

III. Experimental Design

A. Setting

We conducted the study with workers in Kenya's informal sector, commonly known as *jua kali*, meaning "under the hot sun". Employment in *jua kali* accounts for over 70 percent of non-farm employment in Kenya (Adams, Silva, and Razmara 2013). The artisans, vendors, and mechanics in the *jua kali* sector face extreme vulnerability to illness, economic dislocation, and natural disasters. *Jua kali* workers supply goods to local markets using predominantly manual labor, little capital, and often handmade tools. The *jua kali* area in Kamukunji, Nairobi, where our study takes place, consists mostly of metalworkers and vendors working in hazardous conditions with minimal safety equipment. In our sample, 21 percent of the control group were sick or injured in the month prior to being surveyed. Less than 4 percent of our sample were hospitalized at baseline, and average monthly medical expenses for the respondents were USD 17 PPP. Less than 7 percent of respondents had any type of insurance policy.

[1] As a consequence, encouragement to sign up for M-Pesa should be considered part of the UCT treatment.

B. Sampling strategy

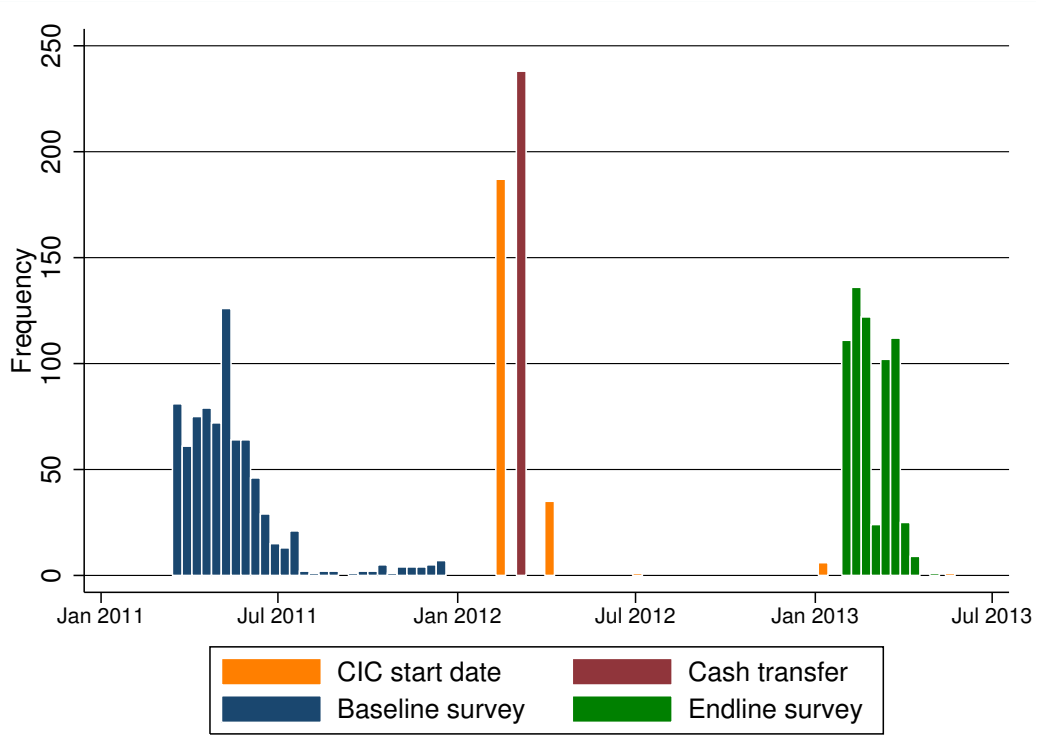
We studied a randomly selected sample of metalworkers of the Kamukunji Jua Kali Association (JKA) in Nairobi, an organization of an estimated 4,000 *jua kali* workers. All adult JKA members working in an area of Kamukunji that made him or her eligible for voting rights in the JKA (those who were over age 18 and working) were eligible to participate in the study. We randomly selected 900 participants for the randomized controlled trial. These respondents were stratified into three groups by weekly household income: 313 respondents with a weekly income greater than USD 103 PPP comprised the high income group; 300 participants with a weekly income between USD 52 PPP and USD 103 PPP comprised the middle income group; and 242 respondents with a weekly income under USD 52 PPP comprised the low income group. Within each income stratum, we randomly selected a third of respondents for one of the two treatment arms and the control group. Because only individuals with national ID cards could receive insurance, we faced differential attrition across treatment arms. We therefore exclude from our analysis respondents who did not have a valid national ID by the time we conducted the baseline survey, for a final sample size of 789 individuals. The motivation for this choice is explain in greater detail in Section C.

C. Data collection

Data collection for baseline occurred between March 2011 and December 2011. Endline data collection occurred between January 2013 and April 2013, at least one year after the baseline. Figure I presents a timeline of the experiment. Trained interviewers used netbooks to administer the surveys in an office at JKA or at the respondent's place of work. Respondents received KES 200 (USD 5.2 PPP) as payment for participating in each interview, in addition to further payouts determined by responses in the time and risk preferences section of the survey. To ensure data quality, we performed back-checks on 10 percent of all interviews, focusing on non-changing information. This procedure was known to field officers *ex ante*.

Respondents were informed of their treatment status after completing the baseline sur-

Figure I: Project timeline



Notes: This figure plots the number of respondents who were surveyed and received treatment during each phase of the project. Data collection for baseline occurred between March 2011 and December 2011. Endline data collection occurred between January 2013 and April 2013. The interventions were delivered in early 2012.

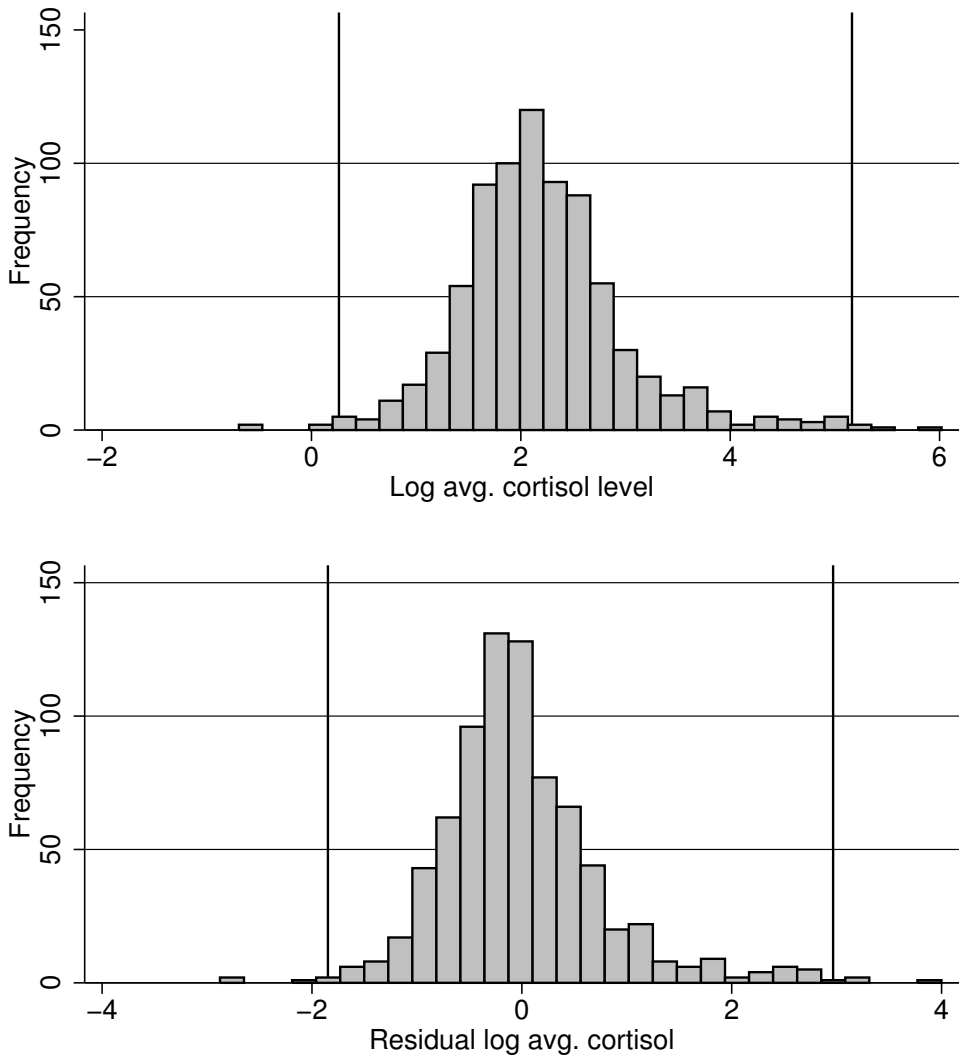
vey. In March 2012, respondents in the cash transfer group who completed the baseline and had a registered SIM card received an unconditional cash transfer via M-Pesa equal to the amount of the annual premium they would have had to pay under CIC *Afya Bora*. Respondents in the insurance group were offered to enroll in CIC's *Afya Bora* insurance free of cost for one year. Project staff assisted this group with preparing required documents, and submitted the applications to CIC on their behalf. Beneficiaries then received an ID card from CIC which they could use to claim benefits in CIC's network of 26 providers across Nairobi.

The survey instruments asked respondents about household characteristics, consumption, asset holdings, workplace, insurance usage, health, self-reported well being, and time and risk preferences. An important feature of this study is that, in addition to questionnaire measures of psychological well-being, we also obtained saliva samples from all respondents, which were assayed for the stress hormone cortisol. Cortisol has been used extensively in psychological and medical research (Kirschbaum and Hellhammer 1989), and more recently in randomized trials in developing countries similar to the present study (Fernald and Gunnar 2009; Haushofer and Shapiro 2016). Cortisol has several advantages over other outcome variables. First, it is an objective measure not prone to survey effects such as social desirability bias and it has several practical advantages which make it attractive to analyze in field studies. Second, cortisol is a useful indicator of both acute stress (Kirschbaum and Hellhammer 1989) and more permanent stress-related conditions such as major depressive disorder (Holsboer 2000; Hammen 2005). Third, cortisol is a good predictor of long-term health through its effects on the immune system. To measure cortisol levels, we collected saliva samples using the Salivette (Sarstedt, Germany). Respondents chewed on the cellulose swab for two minutes, and it was then centrifuged, stored at -20° C, and analyzed for salivary cortisol. Field officers collected a total of two saliva samples from each respondent at both baseline and endline, one each before and after the survey.[2]

We first obtained the average cortisol level in each participant by averaging the values of

[2]In addition, we collected blood samples from respondents at the end of each survey. Trained phlebotomists took blood draws in the JKA office. These samples have not been fully analyzed and are not included in this paper.

Figure II: Raw and residual log average cortisol with boundaries at the 1 and 99 percentiles



Notes: Top panel: distribution of cortisol levels at baseline, averaged across the two samples taken from each respondent, in $log(nmol/L)$. Bottom panel: baseline distribution of the residuals after regressing the raw cortisol values on control variables. Vertical lines denote the 1^{st} and 99^{th} percentiles.

the two samples. Because cortisol levels in population samples are usually heavily skewed, it is established practice to log-transform them before analysis. We follow this standard approach here. Salivary cortisol is subject to a number of confounds; it is affected by food and drink, alcohol and nicotine, medications, and strenuous physical exercise. Cortisol levels also follow a diurnal pattern: they rise sharply in the morning, and then exhibit a gradual decline throughout the rest of the day. To control for these confounds, but at the same time avoid the risk of "cherry-picking" among different measures of cortisol, we present results for several versions of the cortisol variable in the analysis, which were pre-specified in our pre-analysis plan. First, we use the log-transformed raw cortisol levels without the inclusion of control variables. Second, we construct a "clean" version of the raw cortisol variable, which consists of the residuals of an OLS regression of the log-transformed cortisol levels on dummies for having ingested food, drinks, alcohol, nicotine, or medications in the two hours preceding the interview, for having performed vigorous physical activity on the day of the interview, and for the time elapsed since waking, rounded to the next full hour. Third, we analyze the same variables after trimming at 100 $nmol/L$ and/or winsorization at the 99 percent level to account for outliers. Figure II displays the distribution of log cortisol for the sample at baseline.

IV. Econometric Strategy[3]

To capture the impact of health insurance and cash transfers, we estimate the following model:

$$y_{i,t=1} = \alpha_s + \beta_1 INS_i + \beta_2 UCT_i + \delta y_{i,t=0} + \varepsilon_i \tag{1}$$

Here, $y_{i,t=1}$ is the outcome of interest for individual i measured at endline. INS_i indicates assignment to the insurance group. UCT_i indicates assignment to the cash transfer group. ε_i is the idiosyncratic error term. α_s captures stratum-level fixed effects. β_1 is the average treatment effect of free health insurance, β_2 is the average treatment effect of a

[3]We registered a pre-analysis plan written and published before the beginning of data analysis: `https://www.socialscienceregistry.org/trials/647`

cash transfer equal to the value of the insurance policy, and $\beta_1 - \beta_2$ captures the differential effect between health insurance and the cash equivalent. $\beta_1 - \beta_2$ thus quantifies the income-smoothing value of health insurance.[4] We estimate equation 1 using intent-to-treat.

Following McKenzie (2012), we condition on the baseline level of the individual outcome, $y_{i,t=0}$, where available, to improve statistical power. We also estimate variants of Equation 1 that include a vector of covariates measured at baseline.[5] When baseline covariates are missing for an observation, we include an indicator variable for missing observations, and replace the missing observations with 0. We test joint significance across outcome variables with seemingly unrelated regression (SUR) (Zellner 1962).

To assess heterogeneous treatment effects, we test whether the impact of health insurance and cash transfers varies with pre-specified respondent characteristics measured at baseline and denoted by $X_{i,t=0}$ in the following equation:

$$y_{i,t=1} = \alpha_s + \beta_1 INS_i + \beta_2 UCT_i + \beta_3 X_{i,t=0} + \beta_4 (INS_i \times X_{i,t=0}) + \beta_5 (UCT_i \times X_{i,t=0}) + \delta y_{i,t=0} + \varepsilon_i \quad (2)$$

A. Minimum detectable effect sizes

To determine whether our null findings identify the absence of a true effect or signify a lack of statistical power, we report the minimum detectable effect size (MDE) for each outcome:

$$MDE_{\hat{\beta}} = (t_{1-\kappa} + t_{\alpha/2}) \times SE(\hat{\beta})$$

This metric is the smallest effect that would have been detectable given our current sample size. Commonly used in experimental design, we calculate MDEs with $\alpha = 0.05$ and 0.80 power for each pairwise comparison of our treatments and for comparisons of the treatment effect across dimensions of heterogeneity (Haushofer and Shapiro 2016).

[4] We model the utility gains from insurance in Appendix B.

[5] We include as control variables the number of members in the respondent's household, the respondent's age, gender, marital status, an indicator for co-habitation, and years of schooling.

B. Accounting for multiple inference

Because our interventions are likely to impact a large number of economic behaviors and dimensions of welfare and given that our survey instrument often included several questions related to a single outcome, we account for multiple inference in three ways. First, we pre-defined primary outcome groups in the pre-analysis plan before the beginning of analysis. Second, for each of these outcome groups, we construct a summary index following the procedure proposed by Anderson (2008).[6] For each outcome, we invert scores where necessary so that the positive direction always indicates a "better" outcome. We demean all outcomes and convert them to effect sizes by dividing each outcome by its control group standard deviation. Finally, we weight each outcome by the sum of the entries in the row of the inverted covariance matrix corresponding to that outcome to create a single index.

Third, because combining individual outcome variables in indices as described above still leaves us with multiple index variables, we additionally control for the family-wise error rate (FWER) using the free step-down resampling method to compute adjusted p-values. (Westfall and Young 1993).[7] This approach sets the size of the test to exactly the desired crticial value. For each index variable, we report both unadjusted p-values as well as p-values corrected for multiple inference.

[6]See Online Appendix Section C.1 for details on the procedure.
[7]See Online Appendix Section C.2 for details on the procedure.

Table I: Treatment group by survey participation

	Participation		
	Baseline	Attrited	Endline
Control	282	46	236
Insurance	259	60	199
UCT	248	41	207
Total	789	147	642

Notes: This table displays a cross-tabulation of treatment assignment and participation status. The first column includes all respondents surveyed at baseline who had national IDs. The second column includes respondents who attrited between baseline and endline surveys. The third column includes the respondents who successfully completed the endline survey.

C. Assessing potential attrition bias

Three factors made attrition a concern in this study: the high mobility among informal workers in Kenya, the collection of biomarkers, and the requirement for a national ID to obtain insurance or an M-Pesa account. In a pilot study which did not involve biomarkers nor national IDs, we found attrition rates of over 20 percent. To mitigate the attrition, we therefore compensated each respondent who completed both baseline and endline with USD 26 PPP in addition to the individual fees for each survey. Moreover, we conducted a lottery in which three respondents among those who had completed all surveys won prizes of USD 515 PPP, USD 258 PPP, and USD 129 PPP, respectively.

Despite these efforts, rates of attrition were relatively high, at 16 percent in the control group, 23 percent in the insurance group, and 17 percent in the cash group. Some factors that may have contributed to higher dropout rates include tracking issues and unwillingness to provide saliva and blood samples. In addition, due to a miscommunication with the field team, respondents were not interviewed in the endline if they were assigned to the insurance group but did not enroll in the insurance, or assigned to the cash transfer group but did not receive the cash transfer. This occurred mainly for respondents without a national ID card; these respondents could not be enrolled in insurance, or receive the cash, because CIC requires a valid ID to register insurance recipients, and Safaricom requires a national ID to register an M-Pesa account. To mitigate potential selection bias arising from this issue, we exclude from our analysis respondents who did not have a valid national ID during baseline. Table I reports attrition rates between baseline and endline surveys for this sample. Note that differential attrition due to non-compliance is reduced to 14 individuals after restricting the sample to individuals with national ID at baseline, reducing the worry about differential attrition. Online Appendix Tables 26–42 show that attriters differed little from non-attriters on the whole; in particular, they are not significantly different in terms of self-reported stress and cortisol levels. Online Appendix Tables 43–59 show that attriters were similar in terms of outcomes at baseline across the three experimental groups; again this is also true for self-reported stress and cortisol levels.

Table II presents summary statistics of selected baseline variables, separately for each of

the treatment arms and the control group with t-tests to compare means. We find that the baseline characteristics are largely similar across all three groups, with only three of the 48 pairwise comparisons significant, all at the 10 percent level.

Table II: Summary statistics – Baseline variables by treatment group

	Mean (SD, N)			Difference p-value		
	Control	Insurance	UCT	Ins. - Control	UCT - Control	Ins. - UCT
Female	0.10 (0.29)	0.10 (0.30)	0.13 (0.34)	0.96	0.18	0.20
	282	258	248			
HH Size	3.88 (2.03)	4.08 (2.00)	4.23 (2.20)	0.24	0.06*	0.45
	282	259	248			
Years of education	8.57 (2.50)	8.60 (2.50)	8.33 (2.74)	0.88	0.29	0.24
	282	258	248			
Total weekly HH inc. last week (USD PPP)	126.95 (180.35)	148.97 (311.25)	164.85 (352.18)	0.31	0.12	0.60
	277	251	244			
Total expenditure past mo. (USD PPP)	996.66 (1057.34)	1039.83 (1526.33)	945.96 (1077.18)	0.70	0.59	0.43
	282	258	248			
Total savings (USD PPP)	405.42 (984.40)	324.25 (689.49)	424.41 (1021.32)	0.29	0.83	0.21
	262	244	230			
Informal group savings (USD PPP)	21.89 (42.13)	28.36 (100.28)	23.24 (51.95)	0.32	0.74	0.48
	280	257	244			
Sick/injured (1 month)	0.21 (0.41)	0.20 (0.40)	0.21 (0.41)	0.74	0.90	0.66
	282	258	248			
Nights hospitalized (1 year)	0.44 (4.00)	0.35 (2.35)	0.33 (3.85)	0.76	0.76	0.95
	282	258	248			
Contribution to hosp. costs (USD PPP)	69.50 (288.74)	91.20 (519.89)	44.70 (144.68)	0.55	0.22	0.18
	279	258	247			
Ownership of any insurance	0.06 (0.23)	0.07 (0.26)	0.08 (0.27)	0.43	0.36	0.90
	282	258	248			
WTP for inpatient insurance (USD PPP)	12.79 (25.72)	13.93 (29.66)	9.98 (13.63)	0.63	0.12	0.06*
	282	258	248			
WTP for outpatient insurance (USD PPP)	7.79 (11.61)	8.90 (19.01)	8.04 (12.09)	0.41	0.81	0.55
	282	258	248			
Subjective well-being index	-0.00 (1.00)	-0.13 (1.00)	0.04 (1.01)	0.13	0.62	0.05*
	282	259	248			
Perceived stress	23.73 (6.42)	23.92 (6.20)	23.69 (5.97)	0.73	0.94	0.67
	282	259	248			
Log avg. cortisol level	2.18 (0.71)	2.25 (0.83)	2.19 (0.88)	0.30	0.84	0.47
	281	255	245			

Notes: The first three columns report means of each row variable for each treatment group. Standard deviations are in parentheses, and the number of observations N is displayed on the second line. The last three columns report the p-value for a difference of means t-test between pairs of groups. * denotes significance at 10 pct., ** at 5 pct., and *** at 1 pct. level.

V. Results

A. Average treatment effects on stress and cortisol

The main motivation for this project was to ask whether health insurance affects stress levels, and we therefore begin by analyzing the effects of insurance on psychological well-being and cortisol. Table III shows detailed results for variables measuring psychological well-being, as well as the subjective well-being (SWB) index, which is a weighted standardized average of the individual variables. Columns 1--3 present results without control variables, and columns 4–6 with a set of control variables. Columns 7–8 list the control group mean and standard deviation for the outcome variable in question and the number of observations, respectively.

We find no significant effects of insurance or UCTs on the index variable of psychological well-being, and the coefficients for the insurance and UCT arms are not jointly significant using seemingly unrelated regression (SUR). However, insurance leads to a significant and relatively large 0.26–0.27 SD (without and with controls, respectively) decrease in scores on self-reported stress, measured by the Perceived Stress Scale, compared to the control group. The decrease in self-reported stress is also significantly larger than that in the cash group, which itself does not differ from control. The remainder of the coefficients are non-significant after including control variables. The results are robust to controlling for attrition using a Heckman selection model instead of restricting the sample to those with national ID at baseline (Online Appendix Tables 89–90) and Lee bounds (Online Appendix Table 91).

The treatment effect of health insurance of about 0.26 SD for on average USD 338 PPP worth of insurance, or 0.08 SD for each USD 100 PPP of insurance, is of comparable absolute magnitude, but higher relative magnitude, to the effect on self-reported stress obtained in Haushofer and Shapiro (2016). That study found a reduction of 0.26 SD in the same Perceived Stress scale for the average transfer of USD 709 PPP, or a 0.04 SD for USD 100 PPP of cash transfers. Thus, the effect per dollar spent on insurance in the present study is about twice as large as the effect per dollar spent on cash transfers in Haushofer and Shapiro (2016).

These results are also broadly consistent with those reported by Baicker et al. (2013),

who conducted one of the few evaluations to examine the effect of health insurance provision on mental health outcomes. They find that insurance coverage reduced rates of depression by 30.5 percent (measured by the Patient Health Questionnaire, PHQ-8; $p < 0.05$) and increased overall self-reported mental health. It should be noted, however, that we find no effects of either insurance or cash transfers on self-reported depression and indicators of psychological well-being other than stress.

To corroborate the results obtained from self-reports, Table IV reports treatment effects on the stress hormone cortisol. Columns 1 and 2 show that cortisol decreases by between 0.13–0.16 log units in the insurance group relative to control. This effect is statistically significant and robust to different transformations of the cortisol variable and to the inclusion of a full set of baseline control variables. Cash transfers do not impact cortisol in any of the specifications. Moreover, we reject the null hypothesis of equality between the insurance and cash transfer groups for specifications that test non-transformed and winsorized cortisol, and in the specification that tests self-reported stress. This result suggests that insurance reduces stress above and beyond the income effect from receiving free insurance coverage. The results are robust to controlling for attrition using a Heckman selection model instead of restricting the sample to those with national ID at baseline (Online Appendix Tables 81–82) and Lee bounds (Online Appendix Table 83); to excluding the small number of individuals in the insurance group who took up insurance very late in the project (cf. Figure I) and who were therefore treated closer to endline (Online Appendix Table 79); to excluding individuals who took medicine (Online Appendix Table 80).

These results complement those of Haushofer and Shapiro (2016), who analyze the effects of unconditional cash transfers on cortisol levels, and find a significant 1.63 $nmol/L$ decrease in cortisol levels nine months after a USD 1,520 PPP cash transfer (or 0.11 $nmol/L$ for every USD 100 PPP on average), but no effect with USD 400 PPP cash transfers. Here, we find a reduction in stress levels with health insurance, but not with a USD 338 PPP cash transfer. The reduction in cortisol levels by 0.15 log units we observe in the insurance group corresponds to 1.56 $nmol/L$, or a 0.48 $nmol/L$ reduction for every USD 100 PPP worth of insurance. This effect is almost as large in absolute terms as the cortisol reduction reported

after USD 1,520 PPP cash transfers in Haushofer and Shapiro (2016), and amounts to 60 percent of the difference of 2.58 $nmol/L$ between depressed and non-depressed individuals reported by a recent meta-analysis (Knorr et al. 2010). The reduction in cortisol levels from our USD 338 PPP cash transfer in the UCT group is not significant, and the average point estimate relative to control is 0.04 log units, corresponding to a 0.47 $nmol/L$ reduction for a USD 338 PPP transfer, or 0.14 $nmol/L$ for every USD 100 PPP of transfers. Thus, we find similar magnitudes for the impact of cash transfers across these studies, with a 0.14 $nmol/L$ reduction per USD 100 PPP in the present study and a 0.10 $nmol/L$ reduction per USD 100 PPP in Haushofer and Shapiro (2016). With 0.48 $nmol/L$ cortisol reduction per USD 100 PPP of insurance, health insurance provision appears to be more successful in lowering cortisol levels than cash transfers.

Table III: Treatment effects – Subjective well-being

	No Controls			With Controls			Sample	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Insurance	UCT	Difference p-value	Insurance	UCT	Difference p-value	Control Mean (SD)	N
Subjective well-being index	0.07	0.03	0.73	0.07	0.05	0.89	0.00	640
	(0.10)	(0.10)		(0.10)	(0.10)		(1.00)	
Perceived stress	-0.26**	-0.01	0.03**	-0.27**	-0.04	0.04**	0.00	640
	(0.10)	(0.10)		(0.11)	(0.10)		(1.00)	
Optimism	0.02	0.15	0.21	0.02	0.17*	0.16	0.00	640
	(0.10)	(0.09)		(0.10)	(0.10)		(1.00)	
Self-esteem	-0.02	-0.04	0.84	-0.02	-0.03	0.90	-0.00	640
	(0.10)	(0.09)		(0.10)	(0.10)		(1.00)	
Depression	-0.08	-0.07	0.95	-0.10	-0.12	0.90	0.00	640
	(0.10)	(0.09)		(0.10)	(0.09)		(1.00)	
Internal locus of control	-0.08	-0.17*	0.37	-0.07	-0.15	0.47	0.00	640
	(0.10)	(0.10)		(0.10)	(0.10)		(1.00)	
Happiness	0.01	0.02	0.94	-0.01	0.01	0.85	0.00	640
	(0.09)	(0.09)		(0.09)	(0.09)		(1.00)	
Life satisfaction	0.05	0.03	0.88	0.05	0.02	0.77	-0.00	640
	(0.10)	(0.10)		(0.10)	(0.10)		(1.00)	
Joint p-value	0.12	0.44	0.11	0.11	0.37	0.13		

Notes: This table reports the estimated treatment effect of insurance and UCTs on each row variable. Columns 1–2 report estimates from an intent-to-treat analysis, correcting for selection by restricting the sample to individuals who owned national IDs at baseline. Columns 4–5 report OLS estimates controlling for baseline covariates. We include as control variables the number of members in the respondent's household, the respondent's age, gender, marital status, an indicator for co-habitation, and years of schooling. Columns 3 and 6 report the p-values for tests of the equality of the UCT and insurance coefficients. The bottom row reports the p-value for a test of the treatment effect across models using SUR. * denotes significance at 10 pct., ** at 5 pct., and *** at 1 pct. level.

Table IV: Treatment effects – Cortisol

	No Controls			With Controls			Sample	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Insurance	UCT	Difference p-value	Insurance	UCT	Difference p-value	Control Mean (SD)	N
Log avg. cortisol level	-0.14**	-0.02	0.04**	-0.13**	-0.02	0.05*	2.48	579
	(0.06)	(0.07)		(0.06)	(0.06)		(0.66)	
Residual log avg. cortisol	-0.14**	-0.03	0.06*	-0.14**	-0.03	0.08*	0.03	578
	(0.06)	(0.07)		(0.06)	(0.06)		(0.64)	
Log avg. cortisol less 100	-0.15**	-0.07	0.16	-0.14**	-0.07	0.20	2.48	576
	(0.06)	(0.06)		(0.06)	(0.06)		(0.66)	
Residual log avg. cortisol less 100	-0.15**	-0.07	0.16	-0.14**	-0.07	0.22	0.03	576
	(0.06)	(0.06)		(0.06)	(0.06)		(0.64)	
Log avg. cortisol (.99 Wins.)	-0.14**	-0.03	0.05**	-0.13**	-0.02	0.06*	2.48	579
	(0.06)	(0.06)		(0.06)	(0.06)		(0.66)	
Residual log avg. cortisol (.99 Wins.)	-0.14**	-0.03	0.07*	-0.13**	-0.04	0.10*	0.03	578
	(0.06)	(0.06)		(0.06)	(0.06)		(0.64)	

Notes: This table reports the estimated treatment effect of insurance and UCTs on each row variable. Columns 1–2 report estimates from an OLS intent-to-treat analysis, correcting for selection by restricting the sample to individuals who owned national IDs at baseline. Columns 4–5 report OLS estimates controlling for baseline covariates. We include as control variables the number of members in the respondent's household, the respondent's age, gender, marital status, an indicator for co-habitation, and years of schooling. Columns 3 and 6 report the p-values for tests of the equality of the UCT and insurance coefficients. The number of observations reflects the restriction of the sample to individuals with national ID at baseline and for whom endline cortisol could be analyzed. * denotes significance at 10 pct., ** at 5 pct., and *** at 1 pct. level.

B. Potential mechanisms

What might be the channel(s) through which insurance reduces self-reported stress and cortisol levels? In this section, we consider several possibilities. First, insurance may have led to changes in economic variables, such as labor mobility, productivity, or job risk, which could account for the effect on stress and cortisol levels. Second, insurance might have led to changes in health and healthcare utilization. Such changes could lead to a decrease in stress and cortisol levels through two mechanisms: first, cortisol levels are correlated with inflammatory processes, and thus might be lower if insurance recipients are physically healthier. Second, even in the absence of improved physical health, getting treatment might reduce the stress of being ill. Third, cortisol is affected not only by psychological stress, but also by other variables such as eating, drinking, nicotine and alcohol consumption, and physical exertion, all of which could plausibly be affected by treatment. It will emerge in this section that none of these channels can account for the effect of insurance on cortisol levels. We therefore argue that the most likely explanation for the effect of insurance on cortisol levels is that insurance confers "peace of mind", i.e. reduces the stress associated with *potential* health problems.

B.1 Changes in economic outcomes

First, we consider the possibility that insurance affects economic outcomes, which would raise the possibility that the effect of insurance on stress and cortisol levels operate through this mechanism. To address this question, we analyze the impact of insurance provision on a set of pre-specified summary indices. In addition to subjective well-being, these variables include indices for insurance ownership (weighted standardized number of different insurance products owned by the household); willingness to pay for insurance (weighted standardized average of WTP for the same insurance products); asset ownership (weighted standardized average of the number of assets owned from a list of common assets); labor mobility (weighted standardized average of variables measuring intentions to change jobs or leave the Jua Kali area); labor productivity (weighted standardized average of several measures of income, hours worked, and number of goods produced); and job risk (weighted

standardized average of "objective" and perceived and job risk, i.e. risks faced and worried about).[8]

Table V presents estimates from Equation 1 with and without control variables. We find no evidence of an impact of insurance on any of our summary indices. One possible reason for these null findings is lack of power; Table VI reports MDEs for the main outcome variables and shows that we were powered to detect effect sizes between 0.21–0.32 SD for these outcomes, and it is therefore possible that we missed smaller effects; however, very few of the coefficients show treatment effect estimates in excess of 0.1 SD.

Consumption was not among the pre-specified indices, but a short consumption module was included in the questionnaire, and results are reported in Online Appendix Table 127. We find no significant effects on consumption, with the exception of a USD 28.60 PPP (23 percent) decrease in social expenditure in the UCT group, significant at the 5 percent level, but not jointly significant with the other coefficients using SUR.

The Online Appendix extends this analysis to a large number of other economic outcome variables. We find no effects of insurance ownership on a large number of variables related to savings and credit (Online Appendix Table 135), labor mobility and labor conditions (Online Appendix Table 143), detailed variables on production (Online Appendix Table 151), a number of variables related to enterprise ownership and operation (Online Appendix Table 159), and food security (Online Appendix Table 183).

Thus, it appears that in this sample, both health insurance and cash transfers had no large overall effects on asset ownership, labor mobility, income and productivity, job risk, or other economic outcomes. The effects of insurance on stress and cortisol levels are therefore unlikely to arise from large changes in these variables.

[8]See Online Appendix Section A for exact definition and composition of the indices as well as analysis on the constituent outcomes.

Table V: Treatment effects – Summary indices

	No Controls			With Controls			Sample	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Insurance	UCT	Difference p-value	Insurance	UCT	Difference p-value	Control Mean (SD)	N
Subjective well-being index	0.07	0.03	0.73	0.07	0.05	0.89	0.00	640
	(0.10)	(0.10)	[1.00]	(0.10)	(0.10)	[0.90]	(1.00)	
	[0.99]	[0.96]		[0.99]	[0.90]			
Insurance ownership index	-0.03	0.04	0.39	-0.03	0.07	0.27	-0.00	640
	(0.08)	(0.09)	[0.86]	(0.08)	(0.09)	[0.86]	(1.00)	
	[0.99]	[0.96]		[0.99]	[0.86]			
Insurance WTP index	-0.09	-0.11	0.77	-0.08	-0.10	0.81	0.00	640
	(0.09)	(0.08)	[0.98]	(0.09)	(0.08)	[0.73]	(1.00)	
	[0.92]	[0.64]		[0.95]	[0.73]			
Asset ownership index	0.02	0.04	0.85	0.00	0.01	0.85	0.00	640
	(0.08)	(0.08)	[1.00]	(0.08)	(0.08)	[0.97]	(1.00)	
	[0.99]	[0.96]		[0.99]	[0.97]			
Labor mobility index	0.06	0.06	0.96	0.07	0.06	0.96	-0.00	640
	(0.11)	(0.10)	[1.00]	(0.11)	(0.11)	[0.90]	(1.00)	
	[0.99]	[0.96]		[0.99]	[0.90]			
Labor productivity index	-0.03	-0.12	0.45	-0.04	-0.13	0.47	0.00	638
	(0.11)	(0.09)	[0.98]	(0.11)	(0.10)	[0.83]	(1.00)	
	[0.99]	[0.84]		[0.99]	[0.83]			
Job risk index	-0.01	-0.13	0.21	-0.01	-0.11	0.28	0.00	640
	(0.09)	(0.09)	[0.90]	(0.09)	(0.09)	[0.77]	(1.00)	
	[0.99]	[0.66]		[0.99]	[0.77]			
Joint p-value	0.95	0.44	0.87	0.96	0.45	0.88		

Notes: This table reports the estimated treatment effect of insurance and UCTs on each row variable. Columns 1–2 report estimates from an OLS intent-to-treat analysis, correcting for selection by restricting the sample to individuals who owned national IDs at baseline. Columns 4–5 report OLS estimates controlling for baseline covariates. We include as control variables the number of members in the respondent's household, the respondent's age, gender, marital status, an indicator for co-habitation, and years of schooling. Columns 3 and 6 report the p-values for tests of the equality of the UCT and insurance coefficients. The bottom row reports the p-value for a test of joint significance of the treatment effect across models using seemingly unrelated regression (SUR). * denotes significance at 10 pct., ** at 5 pct., and *** at 1 pct. level.

Table VI: Minimum detectable effects – Summary indices

	No Controls		With Controls		Sample	
	(1)	(2)	(3)	(4)	(5)	(6)
	Insurance	UCT	Insurance	UCT	Control Mean (SD)	N
Subjective well-being index	0.28	0.28	0.29	0.29	0.00 (1.00)	628
Insurance ownership index	0.24	0.25	0.23	0.24	-0.00 (1.00)	628
Insurance WTP index	0.26	0.22	0.25	0.22	0.00 (1.00)	628
Asset ownership index	0.21	0.22	0.21	0.23	0.00 (1.00)	628
Labor mobility index	0.30	0.29	0.31	0.31	-0.00 (1.00)	628
Labor productivity index	0.32	0.27	0.31	0.28	0.00 (1.00)	626
Job risk index	0.26	0.26	0.26	0.27	0.00 (1.00)	628

Notes: Columns 1 and 3 report the minimum detectable effect sizes of insurance compared to control on the row variables with $\alpha = 0.05$ and 0.8 power. Columns 2 and 4 report the minimum detectable effect sizes for the UCT. The last columns report the control group means and SDs and the number of observations, respectively.

B.2 Changes in health and healthcare utilization

Another potential mechanism through which insurance may have affected stress levels are changes in health and healthcare utilization. Cortisol is an inflammatory marker and therefore could respond to changes in objective health status; more broadly, if the insurance allows recipients to receive treatment for previously untreated conditions, this effect could either reduce cortisol directly through objective health status, or indirectly through the relief of receiving treatment.

However, our results indicate that health and healthcare utilization are unlikely to be the mechanisms through which insurance reduced stress. First, it should be noted that even cash transfer recipients in principle had improved access to healthcare because the transfer made it easier for them to pay for treatment, but we do not observe a stress reduction in this group. Of course this finding is not conclusive – it might simply be the case that UCT recipients prioritized other spending over spending on healthcare, while insurance recipients did not have this choice and received improved healthcare due to the in-kind nature of their treatment.

We therefore analyze in detail the effects of our treatments on health outcomes and healthcare utilization in Table VII. The outcome variables tested include measures of health status of the respondent and their children, including whether they or other household members were sick or injured last month, and how many days the of work respondent missed for this reason; whether the respondent or a family member was or should have been hospitalized in the previous year, and associated costs; whether children are vaccinated; and whether the respondent or household members had medical consultations in the previous six months. The coefficients on the insurance arm for these outcome variables are not jointly significant using SUR, although we find a reduction in the number of nights the respondent should have been hospitalized in the previous year. This reduction is quantitatively large (a 0.69 night reduction relative to a control group mean of 0.75 nights), but statistically weak (significant at the 10 percent level). None of the other outcome variables are significantly different from control in the insurance treatment. In the UCT arm, the variables are jointly

significant at the 10 percent level, driven by a 9 percentage point reduction in the proportion of children being sick in the previous month (a 39 percent reduction relative to a control group mean of 23 percent), and weakly significant reductions in the likelihood of children having gotten a checkup in the previous six months, or any household member having been hospitalized. Thus, the cash transfer arm appears to have some effects on health outcomes and healthcare utilization, while the insurance arm appears not to affect these outcomes. This finding suggests that changes in health status or healthcare utilization are unlikely to be the drivers of the reduction in stress levels we find in the insurance treatment.

Our finding that health insurance does not strongly affect health outcomes is consistent with a number of previous randomized evaluations of health insurance in developed and developing countries, which found limited effects of health insurance provision on healthcare utilization and health: Thornton et al. (2010) randomized the price of health insurance among informal workers in Nicaragua (a similar setting to the one we use here), and find no significant impact of health insurance on visits to health facilities; out-of-pocket health care expenditures are reduced, but by an amount less than the cost of the insurance premium, underscoring the argument that insurance provision has to be distinguished from its income effect as we do here. Ansah et al. (2009) find no effect of subsidized insurance in Ghana on anemia and mortality in children; and King et al. (2009) find no impact of health insurance on self-reported health in Mexico. Gertler and Solon (2002) show that insurance provision in the Philippines did not reduce out-of-pocket health expenditure of insurance beneficiaries, mainly because providers price-discriminated between insured and uninsured patients. Dow and Schmeer (2003) analyze the effect of the rollout of health insurance for children in Costa Rica in the 1970s, and find little effect on health that cannot be explained by pre-existing trends. Brook et al. (1983) find no effect of free care versus copay on eight measures of health status and health habits in the RAND health insurance experiment. Finally, in the Oregon Health Insurance experiment, insurance had a positive impact on self-reported health, but not physical health (Baicker et al. 2013; Finkelstein et al. 2012). In sum, the effects of health insurance provision on health outcomes are limited; Acharya et al. (2012) survey 34 experimental and non-experimental studies on the effects of health insurance, and

conclude that "there is little evidence on the impact of social health insurance on changes in health status".[9] Together with the findings from the present study, this lack of improvement on health outcomes after insurance provision may partly explain the low take-up of health insurance in developing countries (Morduch 2006; Jowett, Contoyannis, and Vinh 2003).

Similar to most of the studies cited above, we measure endline outcomes one year after the provision of health insurance, which may be too soon to observe changes in health status. However, Baicker et al. (2013) find no significant effects of health insurance on objective health outcomes (hypertension, high cholesterol levels, or diabetes) even after a two year evaluation period.

An alternative account for our finding that stress and cortisol levels are affected by insurance, but not cash transfers, is that our study was differentially powered to detect treatment effects in the insurance relative to the cash arm. However, Table VIII shows only minor differences in MDEs between the insurance and cash treatments, with the exception of the number of nights individuals were hospitalized. Thus, it is unlikely that differential power prevented us from detecting treatment effects in the insurance group.

[9] A small number of papers report positive health effects as a result of health insurance provision. Alcaraz et al. (2012) find that public provision of health insuracne in Mexico increases standardized test scores among primary school children. Bloom et al. (2006) find that Colombia's *Régimen Subsidiado*, in which government health services were expanded by contracting with NGOs, resulted in increased use of government clinics relative to traditional healers, and concomitant improvements in health outcomes. The Accelerated Benefits (AB) Demonstration funded by the U.S. Social Security Administration in 2006, which provided previously uninsured individuals with Social Security Disability Insurance (SSDI) benefits, increased health care usage during the first year (Michalopoulos et al. 2012).

Table VII: Treatment effects – Health and healthcare use

	No Controls			With Controls			Sample	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Insurance	UCT	Difference p-value	Insurance	UCT	Difference p-value	Control Mean (SD)	N
Sick/injured (1 month)	-0.04	0.01	0.31	-0.05	-0.01	0.48	0.28	640
	(0.04)	(0.04)		(0.04)	(0.04)		(0.45)	
Days missed due to sickness (1 month)	0.06	-0.10	0.41	0.02	-0.17	0.35	0.46	567
	(0.20)	(0.16)		(0.19)	(0.16)		(1.58)	
Prop. of household sick (1 month)	-0.02	-0.03	0.70	-0.01	-0.02	0.61	0.26	642
	(0.04)	(0.03)		(0.03)	(0.03)		(0.37)	
Prop. children in household sick (1 month)	-0.04	-0.09**	0.20	-0.04	-0.08**	0.31	0.23	526
	(0.04)	(0.04)		(0.04)	(0.04)		(0.35)	
Consulted for illness/injury (1 month)	0.02	-0.02	0.28	0.01	-0.04	0.22	0.16	640
	(0.04)	(0.03)		(0.04)	(0.03)		(0.37)	
Any HH member hospitalized (1 year)	-0.03	-0.08*	0.32	-0.03	-0.06	0.41	0.30	640
	(0.04)	(0.04)		(0.04)	(0.04)		(0.46)	
Children vaccinated	-0.02	0.01	0.26	-0.02	0.02	0.17	0.93	517
	(0.03)	(0.03)		(0.03)	(0.03)		(0.26)	
Child check-up (6 months)	-0.03	-0.10*	0.22	-0.03	-0.09*	0.22	0.39	517
	(0.06)	(0.05)		(0.06)	(0.05)		(0.49)	
Contribution to hosp. costs (USD PPP)	50.14	-6.42	0.45	56.28	9.85	0.49	55.88	637
	(75.20)	(15.11)		(78.90)	(20.43)		(148.81)	
Nights hospitalized (1 year)	-0.00	-0.29*	0.20	-0.01	-0.29*	0.20	0.40	640
	(0.27)	(0.16)		(0.28)	(0.16)		(2.39)	
Nights should have been hospitalized (1 year)	-0.69*	-0.71*	0.65	-0.69*	-0.67*	0.71	0.75	640
	(0.39)	(0.40)		(0.38)	(0.35)		(6.15)	
Took medicine today	0.01	-0.02	0.36	-0.01	-0.04*	0.16	0.10	640
	(0.03)	(0.03)		(0.03)	(0.03)		(0.30)	
Joint p-value	0.49	0.06*	0.15	0.43	0.05**	0.15		

Notes: This table reports the estimated treatment effect of insurance and UCTs on each row variable. Columns 1–2 report estimates from an OLS intent-to-treat analysis, correcting for selection by restricting the sample to individuals who owned national IDs at baseline. Columns 4–5 report OLS estimates controlling for baseline covariates. We include as control variables the number of members in the respondent's household, the respondent's age, gender, marital status, an indicator for co-habitation, and years of schooling. Columns 3 and 6 report the p-values for tests of the equality of the UCT and insurance coefficients. The bottom row reports the p-value for a test of the treatment effect across models using SUR. The differing number of observations across variables reflects sample restrictions based on being employed and having children. * denotes significance at 10 pct., ** at 5 pct., and *** at 1 pct. level.

Table VIII: Minimum detectable effects – Health and healthcare use

	No Controls		With Controls		Sample	
	(1)	(2)	(3)	(4)	(5)	(6)
	Insurance	UCT	Insurance	UCT	Control Mean (SD)	N
Sick/injured (1 month)	0.12	0.12	0.12	0.12	0.28 (0.45)	628
Days missed due to sickness (1 month)	0.55	0.44	0.54	0.44	0.46 (1.58)	508
Prop. of household sick (1 month)	0.10	0.09	0.10	0.09	0.26 (0.37)	630
Prop. children in household sick (1 month)	0.10	0.10	0.10	0.10	0.23 (0.35)	451
Consulted for illness/injury (1 month)	0.10	0.10	0.10	0.10	0.16 (0.37)	628
Any HH member hospitalized (1 year)	0.12	0.12	0.12	0.12	0.30 (0.46)	628
Children vaccinated	0.09	0.08	0.09	0.08	0.93 (0.26)	438
Child check-up (6 months)	0.16	0.15	0.16	0.15	0.39 (0.49)	437
Contribution to hosp. costs (USD PPP)	211.42	42.48	222.90	57.71	55.88 (148.81)	622
Nights hospitalized (1 year)	0.76	0.45	0.80	0.47	0.40 (2.39)	628
Nights should have been hospitalized (1 year)	1.10	1.13	1.07	0.99	0.75 (6.15)	628
Took medicine today	0.08	0.07	0.08	0.07	0.10 (0.30)	628

Notes: Columns 1 and 3 report the minimum detectable effect sizes of insurance compared to control on the row variables with $\alpha = 0.05$ and 0.8 power. Columns 2 and 4 report the minimum detectable effect sizes for the UCT. The last columns report the control group means and SDs and number of observations, respectively. The differing number of observations across variables reflects sample restrictions based on being employed and having children.

B.3 Confounds of cortisol

As described above, cortisol levels are affect not only by stress, but also by recent food and drink, physical labor, and drugs such as *miraa* (*khat*) and standard medications. Cortisol also has a diurnal profile, rising in the early morning and then declining throughout the rest of the day. Could these variables have confounded our estimates? In our view, this possibility is unlikely, for three reasons. First, note that if the reduction in cortisol were only due to these confounds, we would not predict a concomitant reduction in *self-reported* stress levels; in contrast, we find that cortisol and self-reports move together, suggesting that at least part of the cortisol effect may result from changes in perceived stress. Second, the impact of insurance on cortisol levels in not dependent on the inclusion of the vector of control variables described in Section C, suggesting that confounds from eating, drinking, and the diurnal profile did not strongly affect results. Finally, we include the indicator for whether or not the respondent took medication prior to the interview as an outcome variable in the table of health outcomes.[10] We find no change in the likelihood of respondents having taken medication as a result of the insurance or cash treatments. This result suggests not only that the reduction in cortisol was not mediated by a direct influence of drug residuals in saliva on the cortisol assay, but also that it was not mediated by e.g. an increase in the consumption of mental health drugs, such as anxiolytics or selective serotonin reuptake inhibitors (SSRIs).

B.4 A "peace of mind" effect

Together, these results suggest that insurance did not reduce stress and cortisol levels through changes in economic variables, health status, healthcare usage, or variables that affect cortisol levels and might plausibly be affected by health insurance. In our view, these findings leave one possible channel through which the effect of insurance on stress and cortisol levels might operate, namely "peace of mind": the knowledge of having insurance may reduce the fear of incurring catastrophic health expenditures, and thereby reduce stress. Two additional pieces of evidence support this view.

[10] This addition was not contained in the pre-analysis plan.

First, if the "peace of mind" hypothesis is true, we would expect reductions in stress and cortisol levels even among insurance recipients who do not use the insurance. We therefore restrict the sample to insurance takers who did not make claims during the entire period in which they were covered by insurance. By restricting the sample in this way, we introduce potential confounds that might simultaneously determine insurance usage and differences in stress, such as baseline health status. We address this issue by employing propensity score matching to estimate the effect of insurance among non-users. We match non-users in the insurance group with UCT and control group members of a similar propensity of not making a claim, using a radius matching algorithm with a radius of 0.01. We calculate this propensity score within the subsample who successfully enrolled in the policy and assign predicted values to the UCT and control groups. This procedure creates comparable samples in all treatment groups who did not use insurance (in the insurance group), or are similar to those who did not use insurance (in the cash and control groups). We then re-estimate our main specifications of interest to assess if the treatment effects on insurance persist when restricting the sample in this fashion.

The results are presented in Table IX. We still find significant effects of insurance on cortisol, which are similar in magnitude to those found for the entire sample. Thus, we observe a reduction in cortisol levels even among individuals who never use the insurance product, supporting our claim of a "peace of mind" effect of insurance. This reduction in cortisol levels in the insurance group, even in the absence of changes in health status or healthcare usage, is in line with the core insight of expected utility theory: risk-averse consumers prefer insurance to the cash equivalent. Online Appendix Tables 84–86 show that this result holds regardless of whether we perform nearest neighbor matching, radius matching, or kernel matching. In addition, Online Appendix Tables 92–94 show that the effect of insurance on self-reported stress also persists when we exclude non-users using the same matching approach, independently of the choice of matching algorithm.

Second, we find an additional piece of evidence in support of the "peace of mind" hypothesis: insurance recipients sleep more than the control group. In particular, Online Appendix Table 207 shows an increase of 23 minutes (0.39 hours) of sleep in the insurance

group, relative to a control group mean of 7 hours 14 minutes (7.23 hours), significant at the 1 percent level. This effect is also significantly larger than the smaller 9 minute (0.15 hour) increase in the cash group, which is not itself significant. We stress that this result is a post-hoc discovery, and analysis of this variable was not laid out in our pre-analysis plan. However, it lends additional credibility to our "peace of mind" hypothesis: stress and good sleeping outcomes are strongly related, and thus the increase in sleep is an additional piece of evidence supporting the view that insurance reduces stress by instilling peace of mind.

A final line of evidence for the "peace of mind" effect comes from heterogeneous treatment effects on number of children, income, recent illness, and depression, which are reported in Section C.

Table IX: Treatment effects on cortisol for non-users by matching

	(1) Insurance v. control	(2) Insurance v. UCT	(3) Control Mean (SD)	(4) N
Log avg. cortisol level	-0.19***	-0.19**	2.49	511
	(0.07)	(0.08)	(0.67)	
Residual log avg. cortisol	-0.18**	-0.18**	0.04	510
	(0.07)	(0.08)	(0.65)	
Log avg. cortisol less 100	-0.17**	-0.17**	2.48	507
	(0.07)	(0.07)	(0.65)	
Residual log avg. cortisol less 100	-0.18**	-0.18**	0.04	508
	(0.07)	(0.07)	(0.65)	
Log avg. cortisol (.99 Wins.)	-0.19***	-0.19**	2.49	511
	(0.07)	(0.07)	(0.66)	
Residual log avg. cortisol (.99 Wins.)	-0.18**	-0.18**	0.04	510
	(0.07)	(0.07)	(0.65)	

Notes: This table reports the estimated treatment effect of holding insurance and UCTs on each row variable, restricting to sample to individuals who did not make insurance claims. To identify comparable individuals in the UCT and control groups, we match by radius matching with 0.01 radius. Standard errors are in parentheses. The number of observations reflects restriction of the sample to individuals who did not use the insurance, and their matched counterparts in the other experimental groups. * denotes significance at 10 pct., ** at 5 pct., and *** at 1 pct. level.

B.5 Why are these effects observed after the end of insurance coverage?

One important question about the effects of health insurance on stress and cortisol levels reported above is why we observe these effects *after* the end of insurance coverage. The answer to this question is two-fold. First, for self-reported stress, we asked respondents about their experience in the month prior to the survey, i.e. when most of them still had insurance; the answers should thus reflect respondents' stress levels while covered by insurance. Second, the lower cortisol levels in insurance recipients than control group members and UCT recipients reflect the fact that cortisol levels reflect not only acute, but also *cumulative* stress. For instance, socioeconomic status in 7 month old children predicts cortisol levels at 4 years of age, suggesting that the experience of poverty may have cumulative effects on cortisol levels (Blair et al. 2013). The fact that insurance coverage had ended by the time endline cortisol levels were measured implies that the treatment effect we observe is a lower bound of the effect of insurance coverage on cortisol levels.

C. Heterogeneous treatment effects

A further line of evidence in favor of the "peace of mind" hypothesis outlined above comes from analysis of heterogeneous treatment effects. If insurance alleviates stress by conferring peace of mind, individuals more vulnerable to risk should benefit more from owning insurance. Table X reports the results of analysis for heterogeneous treatment effects on cortisol estimated with Equation 2. In line with the prediction outlined above, Column 1 shows that the cortisol-reducing effect of insurance is significantly larger for those who have children. Similarly, Column 2 shows that those with weekly income below the median experience larger reductions in cortisol levels, although the interaction term is only significant at the 10 percent level. In column 3, we find no significant interaction term between having insurance and having been sick last month; however, the sum of the coefficients on being sick last month and its interaction with insurance is significant. This finding implies that among individuals who were sick last month, insurance reduced cortisol levels; this was not the case for individuals who received insurance but were not sick last month; although the difference in differences between these groups is not significant. Finally,

column 4 shows that cortisol was also reduced to a greater extent among respondents with above median scores on the CES-D, a scale measuring depressive symptomatology. Together, these findings suggest that more vulnerable individuals experience larger effects of having insurance on cortisol levels, lending credibility to our "peace of mind" account. Note, however, that we do not find similar heterogeneous effects for self-reported stress, or with other dimensions of heterogeneity; these results are reported in the Online Appendix Tables 217–220.

Table X: Heterogeneous effects – Log average cortisol level

	(1)	(2)	(3)	(4)
Ins. × Interactant	-0.440***	-0.200*	-0.251	-0.354***
	(0.146)	(0.121)	(0.156)	(0.119)
Insurance	0.201	-0.0178	-0.0895	0.0181
	(0.131)	(0.0911)	(0.0631)	(0.0756)
UCT × Interactant	-0.285**	-0.118	-0.459***	-0.378***
	(0.136)	(0.128)	(0.159)	(0.132)
UCT	0.192*	0.0349	0.0765	0.145
	(0.108)	(0.0889)	(0.0733)	(0.0908)
Interactant	0.144	-0.0495	0.204	0.223**
	(0.0979)	(0.154)	(0.134)	(0.0949)
Constant	2.205***	2.392***	2.282***	2.226***
	(0.122)	(0.175)	(0.104)	(0.106)
Interactant	Have children	Below median income	Sick last month	Above median CES-D
Adjusted R^2	0.04	0.03	0.04	0.05
H_0: Ins. + Interaction = 0	0.00	0.01	0.02	0.00
H_0: UCT + Interaction = 0	0.24	0.37	0.01	0.01
Observations	566	546	566	566

Notes: This table reports the coefficient estimates of the interaction between the treatment and a baseline variable. Each column corresponds to a model with a unique interacting variable. Standard errors are in parentheses. The number of observations reflects sample restrictions based on being employed and individuals for whom cortisol could be analyzed at endline. * denotes significance at 10 pct., ** at 5 pct., and *** at 1 pct. level.

D. Why is insurance demand low?

Above we showed that health insurance confers "peace of mind", even in the absence of changes in health, healthcare utilization, or economic outcomes. At the same time, we find that demand for health insurance is low, in three respects. First, no members of the UCT or control groups bought the health insurance product, even though it was available to them. This finding is especially salient for the UCT group, who could have afforded the insurance because their transfer amounted exactly to the cost of a policy for their particular family. Although we did not explicitly make aware to the participants in the UCT or control group that this study's insurance policy was available to them, the intervention was relatively well-known among our study population. Second, we find that no members of the insurance group continue their insurance coverage after the study period. Finally, additional evidence for low demand comes from data on willingness to pay for insurance collected at endline: Online Appendix Table 111 shows that willingness to pay (WTP) for the type of insurance provided by the CIC product we studied here is USD 26 PPP in the control group, and slightly but not significantly lower in the UCT and insurance groups. In all three groups, WTP for insurance is thus dramatically lower than the average cost of the policy, USD 338 PPP.

Why is demand for health insurance so low, despite its stress-reducing effects? At endline, we asked respondents in the insurance group why they discontinued their insurance coverage. Table XI shows that the two most frequently reported reasons for dropping the product were unaffordability and low trust in insurance companies. In our view, cost is unlikely to be sufficient to account for the lack of take-up, since none of the respondents receiving the cash transfer purchased the insurance product despite being aware of it due to the relatively public profile of the study in the area. However, it remains possible that experience with insurance and liquidity interact to generate demand for insurance; in other words, if insurance recipients had received a cash transfer at the end of the study, it is conceivable that they might have bought insurance.

The second most frequently cited reason against buying insurance was low trust in insurance companies. This factor has previously been identified as a major concern for

the take-up of health insurance (Dercon, Gunning, and Zeitlin 2015); if insurance takers do not trust that companies will reimburse their claims, demand for the product is likely to be low. We find that providing free health insurance significantly increases trust in insurance companies by 0.50 SD (Online Appendix Table 103). Interestingly, this increase in trust occurs despite the fact that most insurance recipients do no use the product; thus, the simple interaction with the insurance company at signup appears to be sufficient to generate this effect. This increase was not sufficient to increase insurance purchase after one year, but raises the possibility that longer exposure to the product, or the addition of a cash transfer to relieve liquidity constraints, might increase trust to the point where the product becomes attractive.

Taken together, our results suggest that affordability and trust are obstacles to insurance take-up, and that experience with insurance can increase trust in insurance companies.

Table XI: Reasons for discontinuing insurance

	Reason for not buying ins.	
	Freq.	Percent
Too expensive	139	64.7
Not useful	16	7.4
Mistrust ins. companies	37	17.2
Already own	3	1.4
Never considered	5	2.3
Lack information	11	5.1
Hassle to use	4	1.9
Total	215	100.0

Notes: This table tabulates reasons for not continuing the health insurance policy for respondents in the insurance group.

VI. Conclusion

In this study we provided free health insurance to a randomly chosen group of informal metal-workers in Nairobi, Kenya. We find large reductions in cortisol levels and self-reported stress, relative to both a control group and a group that received an equivalent cash transfer. We observe these effects despite the fact that insurance does not lead to improvements in economic outcomes, increased healthcare utilization or health status, or variables that might bias cortisol measurements. In addition, the effect is observed even for individuals who never use the insurance, and is stronger for households with children, the poor, those with worse baseline health, and those with more severe depressive symptoms. We argue that the most likely mechanism that accounts for our findings is that insurance conveys "peace of mind": even when health insurance does not improve health outcomes, it may increase overall welfare by conferring "peace of mind" from reduced risk exposure, particularly among vulnerable populations. An important question for future work is how the effects of insurance on stress levels compare to those of psychological interventions, such as psychotherapy (Bolton et al. 2003; Blattman, Jamison, and Sheridan 2015).

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References

Acharya, Arnab, Sukumar Vellakkal, Fiona Taylor, Edoardo Masset, Ambika Satija, Margaret Burke, and Shah Ebrahim. 2012. "The Impact of Health Insurance Schemes for the Informal Sector in Low- and Middle-Income Countries: A Systematic Review." *The World Bank Research Observer*, November, lks009.

Adams, Arvil V., Sara Johansson de Silva, and Setareh Razmara. 2013, July. *Improving Skills Development in the Informal Sector: Strategies for Sub-Saharan Africa.* World Bank Publications.

Alcaraz, Carlo, Daniel Chiquiar, María José Orraca, and Alejandrina Salcedo. 2012. "The Effect of Publicly Provided Health Insurance on Academic Performance in Mexico." Working Paper 2012-10, Banco de México.

Anderson, Michael L. 2008. "Multiple Inference and Gender Differences in the Effects of Early Intervention: A Reevaluation of the Abecedarian, Perry Preschool, and Early Training Projects." *Journal of the American Statistical Association* 103 (484): 1481–1495.

Ansah, Evelyn Korkor, Solomon Narh-Bana, Sabina Asiamah, Vivian Dzordzordzi, Kingsley Biantey, Kakra Dickson, John Owusu Gyapong, Kwadwo Ansah Koram, Brian M Greenwood, Anne Mills, and Christopher J. M Whitty. 2009. "Effect of Removing Direct Payment for Health Care on Utilisation and Health Outcomes in Ghanaian Children: A Randomised Controlled Trial." *PLoS Med* 6 (1): e1000007 (January).

Baicker, Katherine, Sarah L. Taubman, Heidi L. Allen, Mira Bernstein, Jonathan H. Gruber, Joseph P. Newhouse, Eric C. Schneider, Bill J. Wright, Alan M. Zaslavsky, and Amy N. Finkelstein. 2013. "The Oregon Experiment - Effects of Medicaid on Clinical Outcomes." *New England Journal of Medicine* 368 (18): 1713–1722 (May).

Baird, Sarah, Ephraim Chirwa, Jacobus De Hoop, and Berk Özler. 2014. "Girl Power: Cash Transfers and Adolescent Welfare. Evidence from Cluster-Randomized Experiment in Malawi." In *African Successes: Human Capital.* University of Chicago Press.

Baird, Sarah, Jacobus de Hoop, and Berk Özler. 2013. "Income Shocks and Adolescent Mental Health." *Journal of Human Resources* 48 (2): 370–403.

Baird, Sarah, Craig McIntosh, and Berk Özler. 2011. "Cash or condition? Evidence from a cash transfer experiment." *The Quarterly Journal of Economics* 126 (4): 1709–1753.

Baird, Sarah J., Richard S. Garfein, Craig T. McIntosh, and Berk Özler. 2012. "Effect of a cash transfer programme for schooling on prevalence of HIV and herpes simplex type 2 in Malawi: a cluster randomised trial." *The Lancet* 379 (9823): 1320–1329.

Blair, Clancy, Daniel Berry, Roger Mills-Koonce, Douglas Granger, and FLP Investigators. 2013. "Cumulative effects of early poverty on cortisol in young children: moderation by autonomic nervous system activity." *Psychoneuroendocrinology* 38 (11): 2666–2675 (November).

Blattman, Christopher, Julian Jamison, and Margaret Sheridan. 2015, May. "Reducing Crime and Violence: Experimental Evidence on Adult Noncognitive Investments in Liberia." SSRN Scholarly Paper ID 2594868, Social Science Research Network, Rochester, NY.

Bloom, Erik, Indu Bhushan, David Clingingsmith, Rathavuth Hong, Elizabeth King, Michael Kremer, Benjamin Loevinsohn, and J. Brad Schwartz. 2006. *Contracting for health: evidence from Cambodia.* Brookings Institution.

Bolton, Paul, Judith Bass, Richard Neugebauer, Helen Verdeli, Kathleen F Clougherty, Priya Wickramaratne, Liesbeth Speelman, Lincoln Ndogoni, and Myrna Weissman. 2003. "Group interpersonal psychotherapy for depression in rural Uganda: a randomized controlled trial." *JAMA: the journal of the American Medical Association* 289 (23): 3117–3124 (June).

Brook, Robert H., John E. Ware, William H. Rogers, Emmett B. Keeler, Allyson R. Davies, Cathy A. Donald, George A. Goldberg, Kathleen N. Lohr, Patricia C. Masthay, and Joseph P. Newhouse. 1983. "Does Free Care Improve Adults' Health?" *New England Journal of Medicine* 309 (23): 1426–1434 (December).

Cesarini, David, Erik Lindqvist, Robert Ostling, and Bjorn Wallace. 2016. "Estimating

the causal impact of wealth on health: Evidence from the Swedish lottery players." *The Quarterly Journal of Economics*.

Cohen, Sheldon, Tom Kamarck, and Robin Mermelstein. 1983. "A global measure of perceived stress." *Journal of health and social behavior*, pp. 385–396.

Cohen S, Janicki-Deverts D, and Miller GE. 2007. "PSychological stress and disease." *JAMA* 298 (14): 1685–1687 (October).

Cornelisse, Sandra, Van Ast, Vanessa, Johannes Haushofer, Maayke Seinstra, and Marian Joels. 2013, July. "Time-Dependent Effect of Hydrocortisone Administration on Intertemporal Choice." SSRN Scholarly Paper ID 2294189, Social Science Research Network, Rochester, NY.

Delaney, Liam, Günther Fink, and Colm P. Harmon. 2014, April. "Effects of Stress on Economic Decision-Making: Evidence from Laboratory Experiments." SSRN Scholarly Paper ID 2420705, Social Science Research Network, Rochester, NY.

Dercon, Stefan, Jan Willem Gunning, and Andrew Zeitlin. 2015. *The demand for insurance under limited trust: Evidence from a field experiment in Kenya."*. NBER Summer Program.

Devoto, Florencia, Esther Duflo, Pascaline Dupas, William Parient?, and Vincent Pons. 2012. "Happiness on Tap: Piped Water Adoption in Urban Morocco." *American Economic Journal: Economic Policy* 4 (4): 68–99.

Dow, William H., and Kammi K. Schmeer. 2003. "Health insurance and child mortality in Costa Rica." *Social science & medicine* 57 (6): 975–986.

Fernald, L. C. H., and M. R. Gunnar. 2009. "Poverty-alleviation program participation and salivary cortisol in very low-income children." *Social Science & Medicine* 68 (12): 2180–2189.

Finkelstein, Amy, Sarah Taubman, Bill Wright, Mira Bernstein, Jonathan Gruber, Joseph P. Newhouse, Heidi Allen, Katherine Baicker, and the Oregon Health Study Group. 2012. "The Oregon Health Insurance Experiment: Evidence from the First Year +." *The Quarterly Journal of Economics*, May, qjs020.

Gardner, Jonathan, and Andrew J. Oswald. 2006. "Money and Mental Wellbeing: A Longitudinal Study of Medium-Sized Lottery Wins." IZA Discussion Paper 2233, Institute for the Study of Labor (IZA).

Gertler, Paul, and Orville Solon. 2002. "Who benefits from social health insurance? Evidence from the Philippines." *Unpublished Manuscript, University of California, Berkeley and the University of the Philippines*.

Hammen, C. 2005. "Stress and depression." *Annu. Rev. Clin. Psychol.* 1:293–319.

Haushofer, Johannes, and Ernst Fehr. 2014. "On the psychology of poverty." *Science* 344 (6186): 862–867 (May).

Haushofer, Johannes, and Jeremy Shapiro. 2016. "The Short-Term Impact of Unconditional Cash Transfers to the Poor: Experimental Evidence from Kenya." *The Quarterly Journal of Economics*, July, qjw025.

Holsboer, F. 2000. "The corticosteroid receptor hypothesis of depression." *Neuropsychopharmacology* 23 (5): 477–501.

Jowett, M, P Contoyannis, and N. D Vinh. 2003. "The impact of public voluntary health insurance on private health expenditures in Vietnam." *Social Science & Medicine* 56 (2): 333–342 (January).

King, Gary, Emmanuela Gakidou, Kosuke Imai, Jason Lakin, Ryan T Moore, Clayton Nall, Nirmala Ravishankar, Manett Vargas, Martha María Téllez-Rojo, Juan Eugenio Hernández Ávila, and Maurici Ávila. 2009. "Public policy for the poor? A randomised assessment of the Mexican universal health insurance programme." *The Lancet* 373 (9673): 1447–1454 (April).

Kirschbaum, C, and D H Hellhammer. 1989. "Salivary cortisol in psychobiological research: an overview." *Neuropsychobiology* 22 (3): 150–169.

Kling, Jeffrey R., Jeffrey B. Liebman, and Lawrence F. Katz. 2007. "Experimental Analysis of Neighborhood Effects." *Econometrica* 75 (1): 83–119.

Knorr, Ulla, Maj Vinberg, Lars V. Kessing, and Jørn Wetterslev. 2010. "Salivary cortisol

in depressed patients versus control persons: a systematic review and meta-analysis." *Psychoneuroendocrinology* 35 (9): 1275–1286.

Koolhaas, J. M., A. Bartolomucci, B. Buwalda, S. F. de Boer, G. Flügge, S. M. Korte, P. Meerlo, R. Murison, B. Olivier, P. Palanza, G. Richter-Levin, A. Sgoifo, T. Steimer, O. Stiedl, and van Dij. 2011. "Stress revisited: A critical evaluation of the stress concept." *Neuroscience & Biobehavioral Reviews* 35 (5): 1291–1301 (April).

Mani, Anandi, Sendhil Mullainathan, Eldar Shafir, and Jiaying Zhao. 2013. "Poverty Impedes Cognitive Function." *Science* 341 (6149): 976–980 (August).

McEwen BS, and Stellar E. 1993. "Stress and the individual: Mechanisms leading to disease." *Archives of Internal Medicine* 153 (18): 2093–2101 (September).

McKenzie, David. 2012. "Beyond baseline and follow-up: The case for more T in experiments." *Journal of Development Economics* 99 (2): 210–221.

Michalopoulos, Charles, David Wittenburg, Dina A. R. Israel, and Anne Warren. 2012. "The Effects of Health Care Benefits on Health Care Use and Health: A Randomized Trial for Disability Insurance Beneficiaries." *Medical Care* 50 (9): 764–771 (September).

Miller, Suzanne M. 1979. "Controllability and human stress: Method, evidence and theory." *Behaviour Research and Therapy* 17 (4): 287–304.

Morduch, Jonathan. 2006. "Microinsurance: The next revolution." *Understanding poverty*, pp. 337–356.

Porcelli, Anthony J, and Mauricio R Delgado. 2009. "Acute stress modulates risk taking in financial decision making." *Psychological Science* 20 (3): 278–283 (March).

Shah, Anuj K., Sendhil Mullainathan, and Eldar Shafir. 2012. "Some Consequences of Having Too Little." *Science* 338 (6107): 682–685 (November).

Sobocki, P., B. Jönsson, J. Angst, C. Rehnberg, et al. 2006. "Cost of depression in Europe." *The journal of mental health policy and economics* 9 (2): 87.

Thornton, Rebecca L., Laurel E. Hatt, Erica M. Field, Mursaleena Islam, Freddy Solís Diaz, and Martha Azucena González. 2010. "Social security health insurance for the informal

sector in Nicaragua: a randomized evaluation." *Health Economics* 19 (S1): 181–206 (September).

Westfall, Peter H., and S. Stanley Young. 1993, January. *Resampling-Based Multiple Testing: Examples and Methods for P-Value Adjustment*. John Wiley & Sons.

Zellner, Arnold. 1962. "An Efficient Method of Estimating Seemingly Unrelated Regressions and Tests for Aggregation Bias." *Journal of the American Statistical Association* 57 (298): 348–368 (June).

Appendix

A. CIC *Afya Bora* Details

Participants receiving insurance enrolled in the CIC *Afya Bora* plan, a combined inpatient and outpatient family health insurance policy. These treated households received inpatient benefits of up to USD 6,437 PPP per family that covered the costs of:

1. Hospital accommodation charges for a general ward bed in contracted hospitals
2. Doctor and healthcare professional fees
3. Prescribed routine lab tests
4. X-ray and ultrasound tests
5. ICU, HDU, and theatre charges
6. Prescribed medicines, dressings, and internal surgical appliances
7. Routing diagnostic lab tests
8. Day care surgery
9. Maternity including non-elective caesarean section with 6 mo. waiting period
10. Chronic and pre-existing conditions up to USD 1,931 PPP

Households also received outpatient benefits of up to USD 1,287 PPP per family that covered:

1. Routine outpatient consultation
2. Diagnostic laboratory and radiology services
3. Prescribed medicine and dressings

4. HIV/AIDS related conditions and prescribed ARVs
5. Routine immunizations
6. Routine prenatal check ups
7. Postnatal care up to six weeks after delivery
8. Pre-existing and chronic conditions up to KES 20,000
9. Outpatient oncology
10. Psychiatry and psychotherapy

Beneficiaries paid around USD 2.60 PPP for each outpatient visit. Both covers included chronic and pre-existing conditions, including HIV/AIDS but excluded treatment outside Kenya, cosmetic treatment, treatment by non-qualified persons, infertility, self-inflicted injury, experimental treatment, and dental treatment unless occassioned by accidental injury.

B. Theoretical Framework

In this section, we present a simple model for the value of insurance that illustrates the contribution of this study, namely to isolate the "peace of mind" effect of insurance by controlling for the income effect of providing free healthcare through a separate treatment arm that delivers cash transfers. Let y be income, p the probability of an accident, c the cost of medical treatment, I the insurance premium, and B the benefit paid by the insurance company.

The expected utility of having no insurance is

$$EU_{NoInsurance} = (1-p)u(y) + pu(y-c) \tag{3}$$

while the expected utility of having insurance is

$$EU_{Insurance} = (1-p)u(y-I) + pu(y-I-c+B). \tag{4}$$

The value of insurance is thus $EU_{Insurance} - EU_{NoInsurance}$, which can be shown to be positive with a concave utility function. In practice, however, this value is impossible to estimate for two reasons. First, the exact form of the utility function is unknown. Structural models can be used, but must assume a functional form for u. Second, the decision to take up insurance is endogenous: one cannot compare people who choose to purchase insurance or not to estimate the causal impact of health insurance. However, random assignment to health insurance versus a cash transfer makes it possible to estimate this difference, as we illustrate below.

Denote by INS the treatment group that receives free health insurance for one year, and by UCT the group that receives an unconditional cash transfer amounting to the market value I of this insurance product. This experimental design allows us to control for the income effect of receiving free health insurance, as both groups receive a transfer of the same amount.[11] Thus, this design measures the "peace of mind" effect of obtaining health

[11] This analysis assumes that individuals value the health insurance at market rates. If this is not the case, our estimates would provide either upper or lower bounds on the impact of insurance. Participants in our

insurance, since compared to the UCT group, the INS group has health insurance and a lower income by an amount I. Formally:

$$EU_{UCT} = (1-p)u(y+I) + pu(y+I-c) \tag{5}$$

$$EU_{INS} = (1-p)u(y) + pu(y-c+B) \tag{6}$$

Equation 6 can be rewritten as follows:

$$EU_{INS} = (1-p)\,u\left((y+I)-I\right) + pu\left((y+I)-I-c+B\right) \tag{7}$$

The expected utility of the insurance group compared to the cash transfer group is therefore:

$$EU_{INS} - EU_{UCT} = (EU_{Insurance} - EU_{NoInsurance}) \text{ estimated at } y+I \tag{8}$$

Thus, this design allows us to estimate the expected utility of having health insurance, controlling for the income effect of having received the insurance free of charge. For risk-averse individuals, if p is large enough, we have $EU_{INS} - EU_{UCT} \geq 0$. In contrast, if p is very low, or perceived to be very low, then $EU_{UCT} \approx u(y+I) \geq u(y) \approx EU_{INS}$, and expected utility from cash is greater than from free insurance. Thus, theoretical predictions as to the value of insurance are ambiguous.

One concern with this experimental design is that it evaluates the impact of insurance at $y+I$, not y. Because the utility of receiving insurance relative to cash is decreasing in y [12], it is conceivable that I is large enough to make the effect of insurance very small. However, this is unlikely to be a concern in our case, since the market value of health insurance is at

study reporterd a willingness-to-pay of USD 12.41 PPP per month for inpatient insurance, USD 6.82 PPP for outpatient insurance with copay, and USD 8.13 PPP for outpatient insurance without copay. Because the actual monthly premium for the policy we provided was USD 27.35 PPP, this evidence suggests that our results provide *lower* bounds on the effects of insurance.

[12] Set $p = 1$ and notice that, by Jensen's inequality, $\frac{\partial(EU_{Insurance} - EU_{NoInsurance})}{\partial y} = \frac{\partial(u(y-I-c+B) - u(y-c))}{\partial y} < 0$

an average of USD 338 PPP, which corresponds to only 8 percent of our sample households' average yearly income.