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Wealth, Health, and Child Development: Evidence from Administrative Data on Swedish Lottery Players

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WEALTH, HEALTH, AND CHILD DEVELOPMENT: EVIDENCE FROM ADMINISTRATIVE DATA ON SWEDISH LOTTERY PLAYERS*

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We use administrative data on Swedish lottery players to estimate the causal impact of substantial wealth shocks on players' own health and their children's health and developmental outcomes. Our estimation sample is large, virtually free of attrition, and allows us to control for the factors conditional on which the prizes were randomly assigned. In adults, we find no evidence that wealth impacts mortality or health care utilization, with the possible exception of a small reduction in the consumption of mental health drugs. Our estimates allow us to rule out effects on 10-year mortality one sixth as large as the cross-sectional wealth-mortality gradient. In our intergenerational analyses, we find that wealth increases children's health care utilization in the years following the lottery and may also reduce obesity risk. The effects on most other child outcomes, which include drug consumption, scholastic performance, and skills, can usually be bounded to a tight interval around zero. Overall, our findings suggest that in affluent countries with extensive social safety nets, causal effects of wealth are not a major source of the wealth-mortality gradients, nor of the observed relationships between child-developmental outcomes and household income. JEL Codes: I10, I14, J24.

I. Introduction

At every stage in the life cycle, health is robustly associated with various markers for socioeconomic status (SES) such as income, educational attainment, and occupational prestige (Smith 1999; Currie 2009; Cutler, Lleras-Muney, and Vogl 2012). These relationships manifest themselves early. For example, children from low-income households weigh less at birth, are more likely to be born prematurely, and are increasingly at greater risk for chronic health conditions as they age (Brooks-Gunn and Duncan 1997; Newacheck and Halfon 1998; Currie 2009). Childhood health is in turn positively related to a number of later outcomes, including skills, scholastic achievement, and adult economic status (Currie 2009; Smith 2009). In adults, it is also a well-established fact that individuals with higher incomes enjoy better health outcomes (Smith 1999; Deaton 2002). Descriptive research has uncovered these positive relationships in many different countries and time periods, and in many different subpopulations (Smith 1999; Deaton 2002; Cutler, Lleras-Muney, and Vogl 2012).

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Although the existence of these gradients for adult health and child outcomes is not controversial, credibly elucidating their underlying causal pathways has proven challenging, as concerns about reverse causation and omitted variable bias often loom large (Mayer 1998; Deaton 2002; Currie 2009; Chandra and Vogl 2010; Baker and Stabile 2012; Cutler, Lleras-Muney, and Vogl 2012). One review article on the causes and consequences of early childhood health notes that "[t]he number of studies associating poor child outcomes with low SES far exceeds the number that make substantive progress on this difficult question of causality" (Baker and Stabile 2012, p. 8). Writing about the adult health gradient, Deaton (2002) concludes that "there is no general agreement about [its] causes ... [and] what apparent agreement there is is sometimes better supported by repeated assertion than by solid evidence" (p. 15).

In this paper, we use the randomized assignment of lottery prizes in three samples of Swedish lottery players to estimate the causal effect of wealth on players' health and their children's health and development. The prizes vary in magnitude, but 75% of the combined prize pool is accounted for by prizes whose value is in the range 1 to 40 Swedish median annual disposable incomes (approximately $20,000 to $800,000). The estimates we report are therefore useful for testing and refining hypotheses about the sources of the relationship between permanent income and the outcomes we consider.

Our study has three key methodological features that enable us to make stronger inferences about the causal impact of wealth than previous lottery studies evaluating the effect of wealth on health (Lindahl 2005; Gardner and Oswald 2007; van Kippersluis and Galama 2013; Apouey and Clark 2015) and health expenditure (Cheng, Costa-i-Font, and Powdthavee 2015). First, we observe the factors conditional on which the lottery wealth is randomly assigned, allowing us to leverage only the portion of lottery-induced variation in wealth that is exogenous. Second, the size of the prize pool is almost 1 billion dollars – two orders of magnitude larger than in any previous study of lottery players' health. Third, Sweden's high-quality administrative data allow us to observe a rich set of outcomes, some of which are realized over 20 years after the event, in a virtually attrition-free sample. Additionally, our data also allow us to address many (but not all) concerns that are often voiced about the external validity of studies of lottery players.

We conduct two sets of analyses. In our adult analyses, our primary outcomes of interest are total and cause-specific mortalities, but we also report estimates of the impact of wealth on an array of hospitalization and drug-prescription variables. Several of the specific outcomes considered in our adult analyses are included because of their hypothesized relationships to health behaviors and stress, the two primary mechanisms through which epidemiologists have proposed that low income can adversely impact cardiovascular health, mental health, and the risk of autoimmune disease (Williams 1990; Brunner 1997; Adler and Newman 2002; Stansfeld et al. 2002; Marmot and Wilkinson 2006). In our intergenerational analyses, we study how wealth impacts a number of infant and child health characteristics that have featured in earlier work (Currie 2009; Baker and Stabile 2012). Given the known associations between early health and subsequent psychological development, we also examine children's scholastic achievement and cognitive and non-cognitive skills. Throughout, we try to facilitate the interpretation of our findings by reporting the causal estimates from the adult sample alongside cross-sectional wealth gradients, and the causal estimates from the intergenerational sample alongside the estimated gradient with respect to

a 10-year total of household disposable income.

In our adult analyses, we find that the effect of wealth on mortality and health care utilization can be bounded to a tight interval around zero. For example, our estimates allow us to rule out a causal effect of wealth on 10-year adult mortality one sixth as large as the cross-sectional gradient between mortality and wealth. We continue to find effects that can be bounded away from the gradient when we stratify the sample by age, income, sex, health, and education. In our intergenerational analyses, the estimated effect of wealth on scholastic achievement, cognitive and non-cognitive skills is always precise enough to bound the parameter to a tight interval around zero, and we can reject effect sizes much smaller than the household-income gradient. The estimated effects on the child health outcomes – with all-cause hospitalizations as the exception – are not statistically distinguishable from the household-income gradients, but often estimated with enough precision to allow us to reject effects of the magnitude reported in most previous research finding positive effects (Duncan, Morris, and Rodrigues 2011; Cooper and Stewart 2013).

There are some exceptions to the overall pattern of null results. In the adult analyses, we find suggestive evidence that wealth causes a small reduction in the consumption of anxiolytics ("anti-anxiety" medication) and hypnotics and sedatives ("sleeping pills"). In the intergenerational analyses, we find that wealth increases children's hospitalization risk in the two and five years after the lottery, and reduces obesity risk around the age of 18. Yet taken in their entirety, the findings of this paper provide little reason to interpret the wealth-health gradients and wealth-child development gradients in our Swedish sample as primarily arising due to causal effects of wealth. Rather, our findings suggest that, as many authors have cautioned (Mayer 1998; Smith 1999; Deaton 2002; Chandra and Vogl 2010; Cutler, Lleras-Muney, and Vogl 2012), causal interpretations of the gradients observed in developed countries should be viewed skeptically.[1]

The paper is structured as follows. Section II briefly reviews the register data and describes our pooled lottery data. Section III describes our identification strategy, provides evidence of the (conditional) random assignment of wealth in our data, and discusses the appropriateness of generalizing from our Swedish sample of lottery players to the Swedish population. In Sections IV and V, we report the results from the adult and intergenerational analyses. Section VI concludes with a discussion that places our findings in the context of the wider literature. Throughout the manuscript, referenced tables and figures whose names are prefaced by the letter "A" are available in the Online Appendix (hereafter, "OA").

II. Data

To set the stage, Table I gives a summary overview of the registers from which we derive our main outcome variables (Panel A) and the wealth and household-income variables we use to estimate cross-sectional gradients often used to benchmark our lottery-based estimates (Panel B). Additional details on

[1] For quasi-experimental evidence on adults, see Adams et al. (2003), Meer, Miller, and Rosen (2003), Frijters, Haisken-DeNew, and Shields (2005), Snyder and Evans (2006), Adda, Banks, and von Gaudecker (2009), Erixson (2014), and Schwandt (2014). For evidence on child outcomes, see Salkind and Haskins (1982), Sacerdote (2007), Akee et al. (2010), Duncan, Morris, and Rodrigues (2011), Milligan and Stabile (2011), Dahl and Lochner (2012), and Bleakley and Ferrie (2015).

the net wealth measure, which is constructed by summing the year-end value of an individual's financial assets, real assets, other assets and subsequently subtracting debt, is available in OA VI.D. Panel C defines three sets of characteristics – birth demographics, other demographics, and health characteristics – that will play a key role in many of our analyses. The birth demographics are a third-order age polynomial, an indicator for female, and an indicator for being born in a Nordic country. The other demographics are income, and indicator variables for college completion, marital status, and retirement status. Finally, the health characteristics are (a proxy for) the Charlson co-morbidity index[2] (Charlson et al. 1987) and indicator variables for having been hospitalized in the past five years (i) at all, (ii) for more than one week, (iii) for circulatory disease, (iv) for respiratory disease, or (v) for cancer. Throughout the paper, we refer to all these characteristics collectively as our set of "baseline" controls. All variables measured in monetary units (e.g. lottery prizes, wealth or household income) are measued in year-2010 prices.

[TABLE I HERE]

Our analyses are based on a pooled sample of lottery players who, along with their children, were merged to administrative records, using information about players personal identification numbers (PINs). Our basic identification strategy is to use the data and knowledge about the institutional details of each of the three lotteries that comprise the pooled sample to define cells within which lottery prizes are randomly assigned. In our analyses, we then control for the cell fixed effects in regressions of health and child outcomes on the size of the lottery prize won. Because the construction of the cells varies by lottery, we discuss each separately. For expositional clarity, we begin by describing the construction of the cells used in the adult analyses; the construction of the intergenerational cells is a straightforward extension described in Section III.[3]

II.A. Prize-linked Savings Accounts

Prize-linked savings accounts (PLS) are savings accounts that incorporate a lottery element instead of paying interest (Kearney et al. 2011). PLS accounts have existed in Sweden since 1949 and were originally subsidized by the government. The subsidies ceased in 1985, at which point the government authorized banks to offer PLS products under new names. Two systems were put into place. The savings banks ("Sparbankerna") started offering their clients a PLS product known as the Million Account ("Miljonkontot"), whereas the remaining banks joined forces and offered a PLS product known as the Winner Account ("Vinnarkontot"). Each system had over 2 million accounts, implying that one in two Swedes held a PLS account.

Our analyses are based on two sources of information about the Winner Account system that were retrieved from the National Archives: a set of microfiche images with account data and prize lists printed on paper (see PLS Figures II-IV in the OA). One separate microfiche volume exists for each monthly PLS draw that took place between December 1986 and December 1994 (the "fiche period"). Each volume

[2] See OA VI.E. for details on the constuction of the Charlson index.

[3] See OA III (PLS), IV (Triss) and V (Kombi) for a detailed description of how the data from the lotteries were processed and quality controlled.

contains one row of data for each account in existence at the time, with information about the account number, the account owner's PIN, and the number of tickets purchased. The prize lists, which are available for each draw until 2003, contain information about the account numbers of winning accounts and the prizes won (type of prize and prize amount). The prize lists do not contain the account owner's PIN, so the fiches are needed to identify the unique mapping from account number to PIN. After the fiche period, we can identify the PIN of winners as long as the winning account was active during the fiche period.

Two research assistants working independently manually entered each prize list. We relied on Optical Character Recognition (OCR) technology to digitize the microfiche cards, which contain almost 200 million rows of data. We also supplemented the OCR-digitized data with manually gathered data for all accounts that won 100,000 SEK or more during the fiche period. In the OA, we provide a detailed account of how we processed the digitized data to construct a monthly panel for the years 1986 to 1994 with information about accounts, their balance, and the PIN of the account holder. Our quality checks, which rely in part on the manually collected data, showed our algorithm was very effective at correctly mapping prize-winning accounts to a PIN and determining their account balances (OA III.C-III.E).

PLS players could win two types of prizes: fixed prizes and odds prizes. To select the winners, each account was first assigned one unique integer-valued lottery ticket per 100 SEK in balance. Each prize was then awarded by randomly drawing a winning ticket. Fixed prizes varied between 1,000 and 2 million SEK net of taxes and (conditional on winning) did not depend on the account balance. The odds prizes were prizes that instead paid a multiple of 1, 10, or 100 of the account balance to the winner, with the prize amount capped at 1 million SEK. Conditional on winning an odds prize, an account with a larger balance hence won a larger prize (except when the cap was binding).

To construct the cells used in our adult analyses, we use different approaches depending on the type of prize won. For fixed-prize winners, our identification strategy exploits the fact that in the population of players who won exactly n fixed prizes in a particular draw, the total sum of fixed prizes won is independent of the account balance (see OA III.I for a formal treatment). For each draw, we therefore assign winners to the same cell if they won an identical number of fixed prizes in that draw and define the treatment variable as the sum of fixed prizes won. Several previous papers have used this identification strategy (Imbens, Rubin, and Sacerdote 2001; Hankins and Hoekstra 2011; Hankins, Hoekstra, and Skiba 2011). Because it does not require information about the number of tickets owned, we can use it for fixed prizes won both during and after the fiche period.

For odds-prize winners, matching on number of prizes won and draw is insufficient because winners of larger odds prizes, on average, have larger account balances (which may be correlated with unobservable determinants of health). We therefore match individuals who won exactly one odds-prize to controls who won exactly one prize (odds or fixed) in the same draw and whose account had a near-identical balance to that of the winning account.[4] A fixed-prize winner who is successfully matched to an odds-prize winner is moved from the original fixed-prize cell to the cell of the odds-prize winner. After the fiche period, we do not observe account balances; therefore we restrict attention to odds prizes won during the fiche period (1986-1994).

[4] Details on the exact matching procedure are available in OA III.F.

Our final sample is restricted to prize-winning accounts only, because we find some indications that in the full panel, non-winning accounts in a given draw are not missing at random.[5] For the prize-winning accounts, we were able to reliably match 98.7% of the winning accounts from the fiche period to a PIN, implying a negligibly small rate of attrition. In practice, little variation in lottery prizes is lost by comparing winners of large prizes to winners of small prizes (typically 1,000 SEK in the PLS data) instead of comparing winners of large prizes to non-winners. Because the majority of PLS prizes are small, the small-prize winners can still be used to accurately estimate the counterfactual trajectories of large winners. OA III.F contains an illustration, based on hypothetical data, of the procedure used to generate the PLS cells.

II.B. The Kombi Lottery

Kombi is a monthly subscription ticket lottery whose proceeds are given to the Swedish Social Democratic Party and its youth movement. Participants are therefore unrepresentative of the Swedish population in terms of political ideology. Subscribers are billed monthly for their tickets. Ticket owners automatically participate in regular prize draws in which they can win cash prizes or merchandise.

Kombi provided us with an electronic data set with information about the monthly ticket balance of all Kombi participants since January 1998.[6] They also provided us with a list of all individuals who won 1 million SEK (net of taxes) or more, along with information about the month and year of the win. Our empirical strategy is to compare each winner of a large prize with "matched controls" who did not win a large prize but owned an identical number of tickets at the time of the draw. We matched each winner of a large prize to (up to) 100 controls. To improve the precision of our estimates, we choose controls similar in age and sex whenever possible. In those cases in which we had fewer than 100 controls, we included all of them. Our final estimation sample includes the winners of 462 large prizes matched to 46,024 controls (comprising 40,366 unique individuals).

II.C. The Triss Lottery

Triss is a scratch-ticket lottery run since 1986 by Svenska Spel, the Swedish government-owned gambling operator. Triss lottery tickets can be bought in virtually any Swedish store. Our sample contains winners of two types of Triss prizes: Triss-Lumpsum and Triss-Monthly.

Winners of the Triss-Lumpsum and Triss-Monthly prize are eligible to participate in a morning TV show broadcast on national television ("TV4 Morgon"). At the show, Triss-Lumpsum winners draw a prize from a stack of tickets. This stack of tickets is determined by a prize plan that is subject to occasional revisions. Triss-Lumpsum prizes vary in size from 50,000 to 5 million SEK (net of taxes). Triss-Monthly winners participate in the same TV show, but draw one ticket that determines the size of a monthly installment and a second that determines its duration. The tickets are drawn independently. The durations range from 10 to 50 years, and the monthly installments range from 10,000 to 50,000 SEK.

[5] See the discussion in OA III.E.

[6] Approximately 1% of the participants are excluded from the panel because they did not provide a valid PIN upon enrollment. This missingness is not endogenous to the lottery and hence not a source of bias.

To make the monthly installments in Triss-Monthly comparable to the lump-sum prizes in the other lotteries, we convert them to present value using a 2% annual discount rate.

Svenska Spel supplied us with a spreadsheet with information on all participants in Triss-Lumpsum and Triss-Monthly prize draws in the period between 1994 and 2010. The Triss-Monthly prize was not introduced until 1997. Around 25 Triss-Lumpsum prizes and five Triss-Monthly prizes are awarded each month. With the help of Statistics Sweden, we were able to use the information in the spreadsheet (name, age, region of residence, and often also the names of close relatives), to reliably identify the PINs of 98.7% of the winners of Triss prizes. The spreadsheet also notes instances in which the participant shared ownership of the ticket. Our main analyses are based exclusively on the 90% of winners who did not indicate prior to the TV show that they shared ownership of the lottery tickets. However, all of our main results are substantively identical with shared prizes included (see OA IV.D).

Our empirical strategy makes use of the fact that, conditional on making it to the show, prizes are drawn randomly conditional on the prize plan. We assign players to the same cell if they won the same type of lottery prize (Triss-Lumpsum or Triss-Monthly) under the same prize plan and in the same year.

III. Identification Strategy

In our adult analyses, each observation corresponds to a prize won by a player aged 18 or above at the time of the lottery. Normalizing the year of the lottery to $t = 0$, our main estimating equation is given by,

$$Y_{i,t} = \alpha_t P_{i,0} + \mathbf{X}_i \boldsymbol{\beta}_\mathbf{t} + \mathbf{Z}_{i,-1} \boldsymbol{\gamma}_\mathbf{t} + \epsilon_{i,t}, \tag{1}$$

where $Y_{i,t}$ is the (possible time-varying) post-lottery outcome of interest, $P_{i,0}$ is the prize amount won in million SEK, and X_i is a vector of cell fixed effects. The key identifying assumption is that $P_{i,0}$ is independent of potential outcomes conditional on X_i. We include the vector of baseline controls measured at year-end in $t = -1$, $Z_{i,-1}$, in order to improve the precision of our estimates. Unless otherwise noted, we estimate Equation (1) using ordinary least squares (OLS).

Our intergenerational analyses are based on a version of Equation (1) in which the unit of analysis is the child of a player. In these analyses, we distinguish between pre- and post-lottery children. Players' children who were conceived but not yet aged 18 at the time of the lottery are defined as pre-lottery children. We refer to children conceived after the lottery as post-lottery children. If the impact of wealth on fertility is heterogeneous, it could invalidate any experimental comparisons of the post-lottery children of winners who won small prizes to post-lottery of winners who won large prizes. Though wealth effects on the composition of births are interesting, we restrict the estimation sample to pre-lottery children except when studying infant health outcomes (which by definition are realized before the lottery in virtually all pre-lottery children). We discuss and evaluate possible selection effects in the infant health analyses in Section III.B below.

The cells used in all of our intergenerational analyses are generated following a procedure analogous to that used for the adult sample, with two exceptions. First, when generating the cells, we condition on the lottery-playing parent's number of pre-lottery children, thus ensuring the amount won per child

is the same within a cell regardless of whether $P_{i,0}$ is defined as the prize won by the winning parent or the prize won per pre-lottery child. In our primary specification, $P_{i,0}$ is defined as in the adult analyses, but we also report a robustness check where the treatment variable is defined as the lottery prize per child. The second difference is that we drop all odds-prize cells in the intergenerational analyses.[7] In the intergenerational analyses, we control for the child's parent's baseline characteristics (except retirement status, due to the low fraction of parents who are retired) and for the child's birth demographics. Table II summarizes our identification strategy in the adult and intergenerational analyses.

[TABLE II HERE]

III.A. Inference

We took a number of steps to ensure the standard errors we report convey the precision of our estimates as accurately as possible. Throughout the paper, we adjust the analytical standard errors for two sources of non-independence. First, players who win more than one prize will typically appear more than once in the sample (as will the children of such players). Second, in the intergenerational analyses, siblings' outcomes are clearly not independent. We therefore reported clustered standard errors (Liang and Zeger 1986) throughout the manuscript. We cluster at the level of the player in our adult analyses and at the household level in the intergenerational analyses (using an iterative process that always assigns half-siblings to the same cluster).

The analytical standard errors rely on an asymptotic approximation that may introduce substantial biases in finite samples. Though our sample size is large, some of the variables are heavily skewed, so standard rules of thumb about the appropriate sample sizes may not apply. To quantify the amount of finite-sample bias, we conducted Monte Carlo simulations in our adult and intergenerational estimation samples. In the simulations, we exploit the fact that the prizes are randomly assigned within cells to obtain the approximate finite-sample distribution of our test statistics under the null hypothesis that the effect of wealth is zero. Procedurally, we generated 1,000 data sets in which the prizes won by the players (and hence also their children) were permuted within each cell. For each outcome and each permuted sample, we then estimated Equation (1).

In the simulated data, prize amount is (conditionally) independent of the outcome by construction, so if the p-values obtained from analytical standard errors follow a uniform distribution, we interpret this finding as evidence that they are reliable. By this criterion, the analytical standard errors we report in our main analyses are generally reliable. In all our major analyses, we nevertheless supplement analytical standard errors with resampling-based p-values (constructed from the resampling distribution generated in the Monte Carlo simulations). In some analyses of either skewed variables (e.g., prescription-drug consumption) or rare binary variables (e.g., short-run cause-specific hospitalizations), we occasionally observe non-trivial differences between the analytical and resampling-based p-values. In such cases, we rely on the resampling-based p-values, which are usually more conservative.

[7] Because the odds prizes are randomly assigned conditional on account balances, partitioning the odds-prize cells by the number of pre-lottery children would leave little useful identifying variation.

III.B. Random Assignment

If the identifying assumptions of Table II are correct, no covariates determined before the lottery should have predictive power for the lottery outcome once we condition on the cell fixed effects. Normalizing the time of the lottery to 0, we test for (conditional) random assignment by running regressions of the following form:

$$P_{i,0} = \mathbf{X}_{i,0}\boldsymbol{\beta} + \mathbf{Z}_{-1}\boldsymbol{\gamma} + \epsilon_{i,0}, \tag{2}$$

where $P_{i,0}$ is prize money at the time of the event, $X_{i,0}$ is the matrix of cell fixed effects, and Z_{-1} is the full set of baseline controls (see Table I) measured at $t = -1$. To test for random assignment, in Table III, we report omnibus p-values for joint significance of the demographic characteristics defined in Table I, the health characteristics, and their union. We run these randomization tests in the pooled adult sample, in the four lottery samples, and for parents of pre-lottery children or post-lottery children. In the pooled sample, we also estimate Equation (2) without cell fixed effects. Overall, the results in Table III are consistent with our null hypothesis that wealth is randomly assigned if and only if we condition on the cell fixed effects.[8]

[TABLE III HERE]

Because the hypothesis of conditional random assignment of wealth is the least credible in the potentially selected sample of post-lottery children, we also tested whether wealth shocks have an impact on fertility (a fundamental question in its own right – see Becker and Tomes [1976]). As is common in studies of infant health, we restricted our analyses to post-lottery children of female players. As shown in Table AIII, we find no evidence that the lottery wealth impacts fertility of women below the age of 50 in our sample.

III.C. External Validity

In this section, we address a number of questions about the appropriateness of generalizing from our Swedish sample of lottery players to the Swedish population.

How Representative Are Players? Table IV compares our pooled adult sample to representative samples of Swedes. Column (1) reports unweighted summary statistics for the pooled lottery sample; column (2) reports summary statistics with observations weighted by prize won. All characteristics are measured the year before the lottery. The average difference between player's age in the year of a win and that of a randomly drawn Swedish adult is 10 years. In columns (3) and (4), we therefore report descriptive statistics for representative samples of Swedes reweighted to match the age- and sex distributions in columns (1) and (2). We find that after adjusting for age differences, players are similar to the representative sample in terms of their baseline characteristics, which include health. The OA contains additional analyses of the representativeness of players stratified by lottery (Table AIV), players

[8] As an alternative randomization check, Table AI shows that within cells, pre-lottery baseline characteristics are balanced across individuals who won above- and below-median prizes. Table AII reports estimates of how wealth impacts the likelihood of being missing from several of the key registers listed in Table I. We find no evidence of such effects.

with pre- or post-lottery children (Table AV), and the representativeness of players' children themselves (Table AVI). Figure AI shows the age distribution at the time of the win of the members of the adult sample (mean age = 58.7) and their pre-lottery children (mean age = 9.8).

[TABLE IV HERE]

How Large Are the Wealth Shocks? Table V reports summary information about the distribution of lottery prizes in the pooled adult sample, its four lottery subsamples, and the pooled intergenerational samples. For each sample, the table shows the number of prizes awarded in five mutually exclusive prize-range categories. For the adult sample, we also report the average prize in each category. Although 91% of the winners in the adult sample are small-prize winners from PLS, these small prizes only account for 7% of the total value of the prizes disbursed to winners. Dropping these prizes altogether reduces the amount of identifying variation by 10%.[9] The estimates we report therefore assign relatively little weight to the marginal effects of small lottery prizes, even though small prizes account for a large fraction of the total number of prizes won. By contrast, prizes greater than 500,000 SEK account for over 70% of the total prize pool. To convey a sense of the magnitudes of the large prizes that account for most of our identifying variation, the $10^{\mathbf{th}}$, $50^{\mathbf{th}}$ (median) and $90^{\mathbf{th}}$ percentiles of the distribution of adult Swedes' annual disposable income in 1999 were 65,000 ($9,100), 144,000 ($20,100) and 247,000 SEK ($34,500).[10] Thus, a prize of 1M SEK, the typical prize won by large-prize winners in both PLS and Kombi, is approximately equal to 7 median annual disposable incomes.

[TABLE V HERE]

Is Lottery Wealth Different? A general concern often voiced about studies of lottery winners is that people may react differently to lottery wealth than other types of wealth shocks (e.g., changes in taxes, welfare systems, or asset-price fluctuations). This argument can take many specific forms, one of them being that lottery prizes are usually paid in lump sums, whereas many policy changes involve changes to income flows. Throughout the paper, we therefore test for heterogeneity by lottery and by type of prize (monthly installments vs. lump sum). In interpreting these estimates, recall that the Triss-Monthly prizes supplement monthly incomes by 10,000 to 50,000 SEK ($1,400-$7,000). Such figures are far too large to realistically replicate the features of most income-support programs. Rather, they allow us to evaluate whether our conclusions about the effects of substantial shocks to permanent income are robust to the mode of payment. The informativeness of the estimates from the Triss-Monthly sample also varies across outcomes depending on the effective sample size.

According to a folk wisdom, lottery winners spend lottery wealth more frivolously than other types of wealth. In a companion paper on labor supply (Cesarini et al. 2015), we show that the earnings response to the lottery wealth shock is immediate, modest in size, seemingly permanent, and similar across lotteries. Figure I shows the trajectory of net wealth in the years following the lottery win. Plotted are coefficient

[9] Because all analyses include cell-fixed effects, none of our identifying variation comes from between-cell comparisons. The total amount of variation is therefore appropriately defined as the total sum of squares of the cell-demeaned prizes.

[10] For net wealth, the analogous figures are -121,000 ($-16,900), 98,000 ($13,700) and 1,264,000 SEK ($176,700).

estimates for $t = -1, .., 10$ obtained from our main estimating equation with the outcome variable defined as (household-level) year-end net wealth. In the year of the lottery, measured net wealth increases by over 60% of the prize amount won. The net-wealth trajectories are similar across lotteries, and suggest winners spread out the consumption of lottery wealth fairly evenly over time. The indications are thus that winners of large prizes in all lotteries enjoy a modest but sustained increase in consumption and leisure for an extended period of time.

[FIGURE I HERE]

IV. Adult Health

We use information from the *Cause of Death Register* to study both overall mortality and cause-specific mortalities, and information from additional registers to study in-patient hospitalizations and consumption of prescription drugs. We examine deaths and health care utilization events classified into two cause categories: common causes and hypotheses-based causes. The common causes are cancer, respiratory disease, cardiovascular disease, and other. The hypotheses-based causes, which we sought to harmonize across registers, include diabetes, ischemic heart disease, hypertension, cerebrovascular disease, alcohol consumption, injury, and smoking.[11]

We chose these categories to test some of the hypotheses about the causal pathways from income to health that have been proposed in economics and epidemiology. Epidemiologists argue that the stress induced by low income has deleterious health effects, either through relatively proximal biological mechanisms that divert resources away from the maintenance of long-term health (the "fight or flight" response) or through behavioral responses such as smoking, excessive drinking, or unhealthy dieting (Williams 1990; Adler and Newman 2002). These biological mechanisms, in turn, increase the risk of bad health in the categories covered by our hypotheses-based classification. In the framework that economists use to study the wealth-health relationship (Grossman 1972), health is a stock whose malleability may vary over the life cycle (Cutler, Lleras-Muney, and Vogl 2012). Plausible channels through which wealth could impact health include changes to lifestyle factors, such as consumption of cigarettes, alcohol, or an unhealthy diet, and health investments with a substantial time cost, such as exercise or access to medical services that require multiple time-consuming interactions with the health care system before being offered.

IV.A. Total and Cause-specific Mortality

We begin with mortality because it is the most objective health measure available in our data. In our main analyses of mortality, the dependent variable is an indicator variable that takes the value 1 if the individual was deceased $t = 1, ..., 10$ years after the lottery. For each of these 10 survival horizons, we estimate a separate linear probability model. In all lottery regressions, we control for the full set of baseline characteristics measured at $t = -1$ and scale the treatment variable so that a coefficient of 1.00 means 1M SEK decreases the survival probability over the relevant time horizon by 1 percentage point.

[11] Table AVII describes the mapping from the International Classification of Diseases (ICD) diagnoses codes to the cause-specific death and hospitalization events.

Given that wealth-mortality gradients are sometimes given causal interpretations, we compare the lottery-based estimates to the cross-sectional gradients estimated from non-experimental variation in a Swedish representative sample drawn in 2000 and a US sample. The US analyses are based on all adult members of the Health and Retirement Study's AHEAD cohort who were alive in 1993. To maximize comparability to the lottery estimates, we re-weight both cross-sectional samples to match the age and sex distributions of the pooled lottery sample. We estimate Swedish cross-sectional gradients from regressions of the form

$$Y_{i,t} = \alpha_t W_{i,1999} + \mathbf{Z}_{i,1999}\boldsymbol{\gamma} + \epsilon_i, \tag{3}$$

where $Y_{i,t}$ is an indicator variable equal to 1 if individual i is deceased in year t, $W_{i,1999}$ is net wealth by December 31, 1999, and $Z_{i,1999}$ is a set of controls. We estimate a separate regression for $t = 2001, ..., 2010$. The US gradients are estimated using an analogous specification, except that covariates are measured in 1992, and mortality observed for $t = 1994, ..., 2003$. We winsorize net wealth in both samples at the 1st and 99th percentiles and convert the winsorized variable to year-2010 SEK.

Figure II graphically illustrates the estimated coefficients in (i) our pooled lottery sample, (ii) the weighted Swedish representative sample controlling for just the birth demographics, (iii) the weighted Swedish sample controlling for the baseline characteristics, and (iv) the weighted US sample with controls for birth demographics. The estimates for $t = 2$, 5, and 10 are reported in table format in Table AVIII, which also shows the fraction of individuals deceased at $t = 2, 5$, and 10 in the lottery sample and the two representative samples.

[FIGURE II HERE]

The wealth-mortality gradients in Sweden and the United States are of similar magnitude and exhibit similar trajectories over time.[12] In Sweden, an additional 1M is associated with approximately a 2.8 percentage point decrease in 10-year mortality risk, and the point estimate only falls to 2.1 if we control for the full set of baseline characteristics (Table AVIII).

In sharp contrast to the cross-sectional gradients, the lottery-based estimates are close to zero and never statistically distinguishable from zero in the pooled sample. For all survival horizons greater than two years, the lottery-based estimates are statistically distinguishable from the gradients. For 10-year mortality, the 95% confidence interval allows us to rule out causal effects one sixth of the gradient. We find no evidence of a positive gradual accumulation of effects. If anything, the temporal pattern appears to be the opposite: positive effects that fade to zero and may even be negative over longer horizons. The estimates and their standard errors are substantively identical if we use the Probit estimator (Table AVIII), and our conclusions are robust to restricting the sample to lottery players who can be followed for at least 10 years after the lottery (thus holding the composition of the lottery sample fixed; see Figure AII).

We repeated the above analyses for all the common and hypotheses-based cause-specific mortalities

[12] For a comparison of wealth gradients across 16 countries, including Sweden and the United States, see Semyonov, Lewin-Epstein, and Maskileyson (2013).

at $t = 5$ and 10 (Figures AIII-AIV and Table AIX).[13] We find no evidence that lottery wealth affects the probability of death due to any of these causes. Compared with the respective gradients, the lottery-based estimates almost always imply a smaller protective effect (or even a harmful effect) of wealth. Our lottery-based estimates are statistically distinguishable, at the 5% level, from several of the cause-specific 10-year mortality gradients: alcohol, circulatory disease, diabetes, ischemic heart disease, other, and respiratory disease.

To investigate if the small effects on overall mortality masks any heterogeneous effects, we conducted additional analyses in a number of subpopulations. Health is a stock whose correlation with income varies over the life cycle, and so may the mix of causal forces that give rise to the correlation at different ages (Smith 2007; Cutler, Lleras-Muney, and Vogl 2012). We therefore reran our main analyses of overall mortality at $t = 2$, 5 and 10 in three subsamples defined by age at the time of the lottery: early (ages 18-44), middle (45-69), and late adulthood (70+). We also test for heterogeneity by sex, health status (hospitalized or not during the last five years), college completion, and income (individual disposable income above vs. below the median in the individual's age category). In each heterogeneity analysis, we estimated an extended version of Equation (1) in which all coefficients are allowed to vary flexibly by subsample. We then conduct a conventional F-test of the null hypothesis that the effect of wealth is the same across all subgroups.

As shown in Tables AX-AXI, we find no strong evidence of heterogeneous effects, but we observe nominally significant effects of wealth on mortality in some of the subsamples; for example, we find signs that wealth increases 10-year mortality in players above 70 years of age, in female players, and in players with below-median income, and suggestive evidence that wealth is protective in individuals with college degrees. Given the large number of hypotheses tested, we interpret these results cautiously. The most important conclusion from our heterogeneity analysis is that in each of the 11 subsamples, some of which include fewer than 15% of the members of the pooled sample, the estimated effect of wealth on 10-year mortality is precise enough to rule out even the more conservatively estimated Swedish gradient of -2.1. In fact, we can reject causal effects one third as large as this gradient in seven of our 11 subsamples, including in several populations (e.g., low-income households) sometimes identified as vulnerable in the literature (Figure AV).

We also investigated whether the effect of wealth on overall mortality varied by lottery (Table AXII). Because most players whom we can follow for 10 years are from the PLS lottery, the 10-year mortality estimates in remaining lotteries are too imprecise to convey any valuable information about heterogeneity. For two- and five-year mortality, the estimated effects are similar across the lotteries and estimated with reasonable precision. The cross-sectional gradient for five-year mortality is -1.57 controlling for the demographics, a magnitude we can reject at the 5% level in all lotteries except Kombi (95% CI -1.85 to 2.50).

To better understand what sort of nonlinear effects are consistent with our results, we re-estimated our main mortality regressions, dropping small (<10K), large (>2M), or very large (>4M) prizes altogether.

[13] We abstain from reporting results for $t = 2$ because the fraction of individuals deceased from several of our specific causes is too low over this time horizon to generate informative estimates.

These sample restrictions appear to have little systematic impact on our estimates, suggesting that none of our results are driven by extreme prizes (Table AXIII). We also estimated two piecewise linear models, the first with a single knot at 1M and the second with knots at 100K and 1M. If lottery wealth has positive and diminishing marginal health benefits (Adler and Newman 2002), we expect negative coefficients that are further away from zero at lower prizes. Our point estimates suggest the opposite pattern – increases in mortality risk that are greatest at lower levels of wealth. Figure AVI illustrates the spline estimates. Though we can never statistically reject constant marginal effects, the upper panel shows we can rule out even modest positive diminishing marginal effects of wealth. For example, according to the first model, the effect of a 1M wealth shock, relative to a counterfactual of winning zero, on 10-year survival probability is at most 0.2 percentage points. The lower panel shows the marginal effect of wealth below 100K is estimated with too little precision to convey useful information.

We supplement our main results with estimates from duration models, which make stronger parametric assumptions about the relationship between wealth and mortality, but also accommodate the right censoring of the data and thus make more efficient use of the full data set (which includes some players observed up to 24 years after the lottery event). We estimate an exponential proportional hazard model in which, again normalizing the time of the lottery to $t = 0$, the hazard of death individual i faces at t is assumed to be given by,

$$h_i\left(t|P_{i,0}, \mathbf{X}_i, \mathbf{Z}_{i,-1}\right) = \exp(\sum_{j=1}^{3} A_{it}^{j}\gamma_j)\lambda_0 \exp\left(\alpha P_{i,0} + \mathbf{X}_{i,0}\,\boldsymbol{\beta} + \mathbf{Z}_{i,-1}\boldsymbol{\gamma}\right), \tag{4}$$

where $P_{i,0}$ is the lottery prize won at event time $t = 0$, A_{it} is the age (in years) of individual i at time t, $X_{i,0}$ is the vector of cell fixed effects, $Z_{i,-1}$ is the vector of predetermined covariates except for age, and λ_0 is the baseline hazard. Because we do not wish to impose the implausible restriction that individuals face a constant hazard of death over the life cycle, we allow the hazard to vary flexibly with age. The key assumption in Equation (4) is that all of the exponentiated covariates in the equation above proportionally affect this age-varying baseline hazard. In Table VI, we report estimates of Equation (4) obtained from the full adult sample, and the subsamples used in the heterogeneity analyses above. The first column of Table VI shows the estimated effect of wealth in the full sample. The estimates are all shown as hazard ratios, so the estimate in column (1) of 1.015 (95% CI 0.964-1.066) means the mortality risk increases by 1.5% for each million SEK won. The next two columns contains estimates from the reweighted Swedish 2000 representative sample. The hazard ratio is 0.874 with the baseline set of covariates and 0.828 with the narrower set of controls. In the cross section, 1M SEK of net wealth is thus associated with a 17.2% or 12.6% lower mortality risk. The results from the heterogeneity analyses are qualitatively similar to the OLS findings. Hazard ratios hover around 1.00 and are estimated with enough precision to reject the gradient in all subsamples except college-educated winners and winners aged 18-44.

As an alternative benchmark for these estimates, an extra year of schooling is believed to reduce mortality rates by about 8% across the entire life cycle (Deaton 2002, p. 21). Our estimates allow us to reject that 100,000 SEK – roughly the annual US per-pupil spending in high school – reduces the mortality rate by more than 0.4%. We also sought to evaluate whether the effects are small or large from

a welfare perspective, by calculating the cost per life year saved at the bounds of our confidence intervals. Even if we take the lower bound of our 95% CI for the hazard, the estimated hazard translates into an average prolonged life of four months per 1M SEK in our sample. Our estimates therefore allow us to reject costs smaller than 3M SEK per year of life saved, roughly three times larger than a recent Swedish estimate of the value of a quality-adjusted life year of 1.2M SEK (Hultkrantz and Svensson 2012, p. 309).

[TABLE VI HERE]

IV.B. Health Care Utilization

We examine two types of health care utilization: hospitalizations requiring in-patient care and consumption of prescription drugs. The hospitalization analyses are based on data on in-patient care from the *National Patient Register*. From 1987 and onward, this register contains information about each patient's arrival and discharge dates, and diagnoses codes (in ICD format). Our analyses of drug prescriptions are based on data from the *Prescribed Drug Register*, which contains information about all over-the-counter sales of prescribed medical drugs between 2006 and 2010. During this period, we observe on which day a prescription was purchased, the Anatomical Therapeutic Chemical Classification System (ATC) code of the drug, and the number of defined daily doses (DDDs) purchased. A DDD is an estimate of the maintenance dose per day of a drug when it is used for its main indication.

Unlike the well-studied wealth-mortality gradients, health-care-utilization gradients with respect to income are on average quite small, but vary both in their sign and their magnitude across countries (Majo and van Soest 2012). Given Sweden's universal coverage, extrapolations of the findings reported here to other settings are clearly fraught with numerous difficulties. To facilitate the exposition, we seek to mimic the mortality analyses as closely as possible, but relegate most of the findings to the OA. In our analyses of cause-specific hospitalizations, we consider the same common and hypotheses-based causes as in the mortality analyses. In our hospitalization analyses, the main outcome variables are a set of binary outcome variables equal to 1 if in at least one of the two, five, and 10 years following the lottery, the individual was hospitalized for at least one night. We construct one variable for each of the specific causes, and also an omnibus ("all-cause") variable that includes hospitalizations due to all causes except pregnancy. We also construct an alternative all-cause hospitalization variable for hospitalizations whose duration exceeds at least one week.

In our drug prescription analyses, the nature of the data required us to make some minor changes to the categories,[14] the most important of which is that we include mental health as one of the hypotheses-based causes.[15] We estimate the impact of wealth on the intensive- and extensive-margin drug consumption 2006-2010. Our primary outcome is the sum of DDDs prescribed between 2006 and 2010 in all drug

[14] First, we merge ischemic heart disease and hypertension into a single category ("Heart") because many drugs are prescribed to treat both ischemic heart disease and hypertension. Second, we make no attempt to identify drugs used to treat diseases often caused by alcohol and tobacco consumption; the structure of the drug-prescription data makes the identification of such drugs difficult. Table AVII shows how we use a drug's ATC to assign it to one of the categories.

[15] We did not include mental health in the other analyses because inferring mental health status from hospitalization records and death certificates is difficult (and outcomes such as suicide are too rare to allow well-powered analyses). Our decision to study mental health drugs was made prior to accessing the data and was in fact one of the major motivations we gave when requesting access to the data on drug prescription.

categories except contraceptives. We also construct a health index that aggregates the information available about a person's health in the registries to predict five-year mortality risk. The index, which ranges from 0 to 100, is defined so higher values denote worse health (see OA VI.C for details). In all health-care-utilization analyses, we restrict the estimation sample to individuals who were alive for the entire period over which a variable is defined. For example, the drug prescription analyses are restricted to a sample of individuals who won before 2006 (the first year for which we have data) and were alive in 2010 (the last year with data).

To give a broadbrush summary of the overall pattern of results, Table VII reports results for eight key outcomes: all-cause hospitalizations and the health index at $t = 2, 5, 10$, total drug consumption, and intensive-margin consumption of mental health drugs. As a benchmark, the table also reports the wealth gradient estimated in a representative sample reweighted so its sex- and age distribution matches the lottery sample.[16] The causal estimates on the three all-cause hospitalization variables are not statistically distinguishable from zero. The estimated marginal effects of 1M SEK on the probability of hospitalization within five and 10 years are 0.39 (95% CI -0.82 to 1.60) and -0.03 percentage points (95% CI -1.54 to 1.48), respectively. Given baseline probabilities of 38.3% and 51.2%, these estimates allow us to rule out effects on relative risk that are fairly small. For $t = 2$ and $t = 5$ hospitalizations, the causal estimates are statistically distinguishable from the gradients at the 10% level.

The estimated effect of 1M on the health index, whose value ranges from 0 to 100, is -0.08 (95% CI -0.36 to 0.20) at $t = 2$, 0.30 (95% CI -0.23 to 0.82) at $t = 5$ and 0.45 (95% CI -0.24 to 1.15) at $t = 10$. These estimates, by contrast, are clearly statistically distinguishable from the gradients.

[TABLE VII HERE]

Our estimated effects on total and mental health drug consumption are also small. The 95% confidence interval for the estimated impact of 1M SEK on total drug consumption is -0.04 to 0.01 SD units. For mental health, our estimates suggest wealth significantly reduces intensive-margin consumption, but the coefficient estimate (-32.50) is small. In standard-deviation units, 32.5 DDDs corresponds to an effect of 0.03, so the estimated effect is not an exception to the overall pattern of small effects of wealth. Moreover, mental health was one of several categories of prescription drugs considered, and the result does not survive a multiple-hypotheses correction that takes into account all the drug-prescription variables analyzed (adjusted p-value = 0.21).[17] We therefore interpret the finding cautiously. In post hoc analyses reported in Table AXVI, we found that the effect, if real, is explained mostly by reductions in the consumption of anxiolytics (used to treat anxiety) and hypnotics and sedatives (used to treat insomnia). The mental health result shows up robustly if a count model is used in lieu of our baseline OLS specification, but

[16] To maximize comparability, we also limit the representative sample to individuals who were alive for the entire period over which the dependent variable is defined, and then reweight it to match the sex and age distribution in our lottery estimation sample.

[17] To implement the multiple-hypotheses correction, we simulate the lottery 10,000 times, each time randomly permuting the prize within each cell. In each simulated data set, we then run 17 separate outcome regressions, one for each of outcome listed in Tables AXIV and AXV. In each simulated data set, we compute the minimum of the 17 p-values from the null that the effect of wealth is zero. The resampling-based p-value of 0.018 is lower than the minimum of the 17 p-values only 21% of the time.

the estimated effect is not statistically significant if we winsorize the mental health variable at the 99th percentile (Table AXVII).

For four of the outcomes in Table VII – five-year hospitalization, five-year health index, total drug consumption, and mental health drug consumption – we also undertook a series of additional heterogeneity and non-linearity analyses analogous to those conducted for overall mortality. The estimated effect on mental health is negative in 10 out of 11 subsamples considered in the heterogeneity analyses (Tables AXIX-AXX; the exception is individuals above 70) and in all four lotteries (Table AXXI), and appears to be driven primarily by large-prize winners (Table AXXII).

Table AXVIII reports lottery-based estimates and wealth gradients for the full set of cause-specific hospitalization variables we considered. For all but two of the 26 outcomes, the gradients are negative, implying higher net wealth is associated with lower hospitalization risk. The exceptions are 10-year hospitalization risk for cancer and cerebrovascular disease. The causal estimates also exceed the gradients for all but two outcomes, implying that the evidence in its entirety suggests that the causal benefits of wealth on health are of smaller magnitude than the gradient. However, the difference is only statistically significant at the 5% level for six outcomes: all-cause hospitalizations lasting at least a week ($t = 5$), alcohol ($t = 5$ and $t = 10$), diabetes ($t = 5$ and $t = 10$), and ischemic heart disease ($t = 5$). An analogous analysis of the drug-prescription variables yields similar conclusions. For example, the estimated causal effects on the eight extensive-margin drug variables are always greater than the gradients, though the difference is only significant for cerebrovascular disease and diabetes (Table AXIV).

V. Intergenerational Analyses

To minimize concerns about undisclosed multiple-hypothesis testing, our intergenerational analyses were pre-specified in an analysis plan posted in the public domain before running any regressions of child outcomes on the treatment variable (Cesarini et al. 2014).[18] The plan defines our set of child health and child development outcomes and specifies all major aspects of the analyses, including the main estimating equation, the construction of the intergenerational cells, sample-selection criteria, and the heterogeneity analyses. The plan also contains some basic descriptive analyses of how the outcomes vary by household income.

Overall, we sought to examine outcomes resembling those that have featured prominently in earlier research on child health and development (Brooks-Gunn and Duncan 1997; Newacheck and Halfon 1998; Currie 2009). This line of research has shown that on average, children from households with lower incomes weigh less at birth, are more likely to suffer health insults due to accidents or injury, and are at greater risk for chronic conditions such as asthma, attention deficit hyperactivity disorder (ADHD), and overweight. We therefore consider several health outcomes in these domains, for example, hospitalizations due to respiratory disease, consumption of ADHD prescription drugs, and obesity. Many markers of childhood health are also predictive of subsequent cognitive and emotional development (Currie 2009), so we also examine how wealth impacts childrens' cognitive and non-cognitive skills and their scholastic

[18] The analysis plan was posted and archived on July 18, 2014, at https://www.socialscienceregistry.org/trials/442.

performance. Table AXXIII contains detailed information about the definition of our outcome variables and sample selection criteria.

Throughout this section, we eschew comparisons to the cross-sectional gradient with respect to net wealth, because net wealth measured early in the life cycle is a poor proxy for permanent income.[19] Instead, we benchmark our estimates against the gradient with respect to a 10-year sum of household income. We define household income as the sum of the disposable incomes of the child's two biological parents in the first 10 years of the child's life. We estimate these gradients in a large representative sample, controlling for parents' birth demographics, and imposing the same cohort and age restrictions used in the lottery analyses.[20] The OA contains additional methodological details (IX.D) and a comparison of the Swedish and US gradients for a selected set of development and health outcomes (Figure AVII; OA IX.E).

V.A. Child Health

Table VIII summarizes the results for our 18 pre-specified child health outcomes. For each outcome, we report lottery-based estimates alongside household-income gradients.

[TABLE VIII HERE]

We begin with the analyses of infant health, which are based on variables derived from the *Medical Birth Register*. Columns (1)-(3) of Table VIII display the results for our three pre-specified (and commonly studied) outcomes – birth weight (in grams), preterm birth (gestation length < 37 weeks), and low birth weight (birth weight < 2500 grams). Whereas all remaining analyses are based on pre-lottery children, the infant health analyses are restricted to the smaller sample of post-lottery children born to female players, leading to less precise estimates. None of the three causal estimates are robustly distinguishable from either zero or the household-income gradients.

We use data from the *Swedish Conscript Register* to construct three measures of body weight: BMI measured on a continuous scale, and indicator variables for having a BMI above 25 and 30, the standard cutoffs used to define "overweight" and "obesity." Conscription was mandatory for all Swedish men until 2010, so these analyses are limited to male pre-lottery children who reached conscription age (18-19) no later than 2010. The results from these analyses are shown in columns (4)-(6). For BMI, the point estimate is -0.11 with a 95% CI that ranges from -0.55 to 0.33 (-0.18 to 0.10 in SD units). We estimate a statistically significant effect of wealth on obesity risk: according to our point estimate, 1M SEK reduces the probability of being obese around age 18 by 2.1 percentage points, roughly twice the size of the gradient. The obesity result survives an adjustment for multiple-hypothesis testing[21] (adjusted p-value =

[19] In our representative sample drawn in 2000, the R^2 from the regression of 10-year disposable income (measured 2001-2010) on net wealth measured in 1999 varies substantially by age. The R^2 is 1% in individuals aged 15-24, 5% for individuals aged 25-34, and rises monotonically to 25% for individuals aged 65-74.

[20] Based on exploratory analyses described in the analysis plan, we decided to restrict the estimation samples for some outcomes. For example, ADHD medication is not prescribed to children below the age of 6, so the ADHD-analyses are restricted to person-year observations in which the child was at least 6 years old.

[21] We use a procedure analogous to the one described in footnote 17 to adjust for the multiple hypotheses tested in the child health analyses. The adjustment is restricted to the 15 out of 18 child health outcomes studied in pre-lottery children. We

0.04), but because the estimated effect is outside the range we consider plausible and is not statistically significant in several of our sensitivity analyses, we interpret the result with some caution.[22]

We also used data from the *Prescribed Drug Register* to study prescription-drug consumption in four categories. The first three categories – Asthma & Allergy, Mental Health, ADHD – have featured prominently in the US literature on child development (Currie 2009). The fourth category – Total – includes all drug consumption except contraceptives and drugs included in the first three categories. The results from the prescription-drug analyses are shown in columns (7)-(10). We find no evidence that wealth impacts the total number of DDDs consumed in any of the four categories. The estimates consistently have good precision in the sense that we can bound the effect of 1M SEK to within ±0.03 SD units of the point estimate. The estimates are not statistically distinguishable from the household-income gradients.

Our final set of child health outcomes are hospitalization variables derived from the *National Patient Register*. We consider in-patient hospitalizations due to respiratory disease, external causes (accidents, injuries, and poisoning), and an omnibus ("all-cause") category covering all hospitalizations except those due to pregnancy. For each category, we define indicator variables equal to 1 if a child was hospitalized due to that cause within two or five years of the lottery event. For the "all-cause" category, we also define a second set of indicators for hospitalizations lasting at least a week. We focus on respiratory disease and external causes because these are the most common chronic physical health conditions afflicting children in developed countries (Currie 2009).

The results of the hospitalization analyses are reported in columns (11)-(18). According to our lottery estimates, a 1M SEK positive wealth shock increases the probability of all-cause hospitalization within two years by 2.1 percentage points and within five years by 3.4 percentage points. The estimated effects on our two- and five-year all-cause hospitalization variables are statistically distinguishable both from zero and from the gradients, which are negative. The five-year effect surives an adjustment for multiple-hypothesis testing (following the same procedure as for obesity; adjusted p-value = 0.01). Expressed as relative risks, our estimates imply that 1M SEK increases both two- and five-year hospitalization risk by 19%. For hospitalizations due to both respiratory disease and external causes, the effects of wealth are of similar magnitude but less precisely estimated.

V.B. Child Development

Table IX reports results for our six development variables. Our first two outcomes, cognitive and non-cognitive skills, are obtained from the *Swedish Conscript Register*, and hence available only for male children. Our remaining four variables measure scholastic achievement in ninth grade, the last year of

exclude the infant health characteristics from the multiple-hypotheses adjustment for practical reasons; because of Statistics Sweden's privacy rules, the characteristics were supplied to us in another data set with exactly the same subjects, but a different set of masking identifiers.

[22] Given that 2.9% of the conscripts in our sample are obese, we consider a point estimate of -2.1 implausibly large. Those who share this assessment may nevertheless find our result informative. To illustrate, consider a Bayesian whose prior about the true effect size is uniform on the interval from -1.00 to 1.00 percentage points. Upon seeing our results, the mean of her posterior is -0.66 and the probability assigned to the event that the sign of wealth is negative should change from 50% to 94%.

compulsory schooling. Our primary outcome, obtained from the *Ninth Grade Register*, is the child's overall GPA. From 2003, we also have information from the *National Tests Register* about performance on mandatory tests in Swedish, English, and Mathematics. The six outcomes in Table IX have been normalized in a representative sample so that the effect-size estimates can be interpreted in population-standard-deviation units.

Our measure of cognitive skills is the recruit's score on a cognitive test similar to the Armed Forces Qualification Test. In addition to taking the test, recruits also meet with a military psychologist who assesses their ability to deal with the psychological requirements of military service. The psychologist's assessment is our measure of non-cognitive skill. Previous work has shown it to be a reliable predictor of labor market outcomes even conditional on cognitive skills (Lindqvist and Vestman 2011). The estimated effect on cognitive skills is -0.11 SD units and borderline significant (95% CI -0.21 to -0.02). The estimated effect on non-cognitive skills is -0.03 SD units (95% CI -0.19 to 0.12). For both outcomes, our estimates are clearly bounded away from the household-income gradients, which are 0.22 for cognitive skills and 0.16 for non-cognitive skills.

The estimated effects on scholastic achievement, reported in columns (3)-(6), are also all negative, ranging from -0.02 SD units for GPA to -0.08 SD units for English. For GPA, our 95% CI is -0.08 to 0.03 SD units. By way of comparison, the estimated household-income gradient is 0.26. We can thus reject causal effects far smaller than the gradient. The same conclusion holds for test scores in Swedish (95% CI -0.11 to 0.04 SD units), English (95% CI -0.17 to 0.00 SD units), and Mathematics (95% CI -0.13 to 0.07 SD units).

[TABLE IX HERE]

V.C. Robustness and Heterogeneity

We conducted a number of follow-up analyses of the findings in Tables VIII and IX. To simplify the exposition, we restrict these analyses to 15 variables, nine key health outcomes and the six developmental outcomes. We relegate most results to the OA. Our discussion here is selective and seeks to accomplish two primary aims, the first of which is to evaluate the robustness and heterogeneity of the two effects that survived multiple-hypothesis correction: obesity and five-year all-cause hospitalizations (hereafter, "hospitalization").

Table AXXIV shows that the effect-size estimates for obesity and hospitalization do not change appreciably if the treatment variable is instead defined as the prize per child, although the obesity effect is no longer statistically significant. In our nonlinearity analyses (Table AXXV), we find that the estimated effect on hospitalization is of similar magnitude when we drop small (<10K), large (>2M) and very large prizes (>4M) prizes, whereas the obesity coefficient falls by 30%-40% if the very prizes are dropped. In our main heterogeneity analyses, we stratified the sample by: (i) household-income (bottom quartile vs top three quartiles), (ii) child age at the time of the lottery (above or below 9), (iii) winning parent (father or mother) and (iv) child's sex (male or female). In all eight subsamples, wealth is estimated to increase hospitalization risk, and significantly so in six cases (Tables AXXVII-AXXVIII). The estimated

effects on obesity are also directionally consistent across all the heterogeneity analyses, and statistically significant in two subsamples: low-income households and households where the mother won. Overall, these analyses suggest that the hospitalization effect shows up robustly across specifications, whereas the obesity findings are less definitive.

The second aim is to systematically compare the causal effect estimates obtained from each of our eight subsamples to the household-income gradient. For the six developmental outcomes, we conducted a total of 44 heterogeneity analyses: eight for each of the four scholastic achievement variables, and six for each of the two skills variables (only available in males, making sex-stratified analyses impossible). Of the 44 estimated treatment effects, all but two are statistically distinguishable from the population gradients at the 5% level,[23] and seven are statistically distinguishable from zero at the 5% level, always with estimates implying adverse effects on developmental outcomes. The difference between the causal estimates and the household-income gradients is especially striking for GPA, where our 95% CIs allow us to reject positive effects one third as large as the household-income gradients in all eight subsamples. For health, the overall pattern of results is very different: our lottery-based estimates are typically not distinguishable from the household-income gradients. The only major exception is our hospitalization variable, for which we can reject the household-income gradient in all eight subsamples.[24]

V.D. Comparison to Previous Causal Estimates

As a complementary benchmark for our estimates, we compared them to the range of effect sizes reported in meta analyses of the causal impact of income on child outcomes (Duncan, Morris, and Rodrigues 2011; Cooper and Stewart 2013). These studies measure effects in units of SDs of the outcome per $1,000 of annual income. Using this scale, Cooper and Stewart (2013) reported effects in the range 0.04-0.22 for studies finding effects on skills and scholastic achievement (of the 21 studies that satisfied the inclusion criteria in their study and examined cognitive/scholastic outcomes, 16 reported positive effects and five reported null results). The meta analysis of Duncan, Morris, and Rodrigues (2011) was restricted to welfare-to-work experiments and reported an effect-size range of 0.04-0.05 for cognitive skills.[25] It is noteworthy that even in the studies reporting positive effects at the lower end of these effect-size ranges, the causal estimates exceed the income gradients.[26]

There is no unassailable way of making our estimates comparable to those in previous studies. We proceed by measuring the shock to annual income as the annual payout that each lottery prize would generate if it were annuitized over a 20-year period at an actuarially fair price and a real return of 2.3%.[27]

[23] Both exceptions are observed in the sample of children treated after the age of 9. In this population, the upper bound of our 95% CI is 0.26 for mathematics (gradient 0.21) and 0.22 for Swedish (gradient 0.20).

[24] The fact that our estimated effects are robustly bounded away from the household-income gradients for the developmental outcomes but not the health outcomes is primarily due to the gradients being smaller for the health outcomes.

[25] The original effect-size ranges cited in this paragraph were measured in year-2000 or year-2001 prices. To make them comparable to our estimates, the coefficients have been converted to year-2010 prices.

[26] Two prominent examples are Milligan and Stabile (2011) and Dahl and Lochner (2012).

[27] This is the historical return on long-term Swedish government bonds calculated by Waldenström (2014). Using the 2010 SEK/USD exchange rate, 1M year-2010 SEK will generate an annual income stream of $8,799. Hence, the original coefficient estimates must be divided by a factor of $8.799 \times \sigma_Y$, where σ_Y is the standard deviation of the dependent variable, to generate what we refer to as our rescaled estimates.

For point of reference, 1M SEK thus annuitized corresponds to an annual income increase of $8,800. A sustained increase in net annual income of that magnitude is large enough to move most households 2-3 deciles up the distribution of permanent income. Because the households in our sample enjoy an increase in income that lasts longer than the income supplements studied in previous quasi-experimental or experimental studies, our rescaled estimates will, all else equal, overstate the benefits of wealth in Sweden if the effects of income are positive and cumulative.

Figure III plots our estimates, rescaled as described above, of the causal effects on GPA from our pooled sample and four subsamples. In all eight subsamples, four of which are included in Figure III, we can rule out wealth effects on GPA smaller than 0.01 SDs, one quarter as large as the effects at the bottom end of the range reported in earlier studies with positive findings. To illustrate the point that the effect sizes in most published quasi-experimental studies exceed the gradient, the household-income gradient is depicted on the far left of the graph.[28] Replacing GPA in Figure III by any of the other five developmental outcomes gives substantively identical results.

[FIGURE III HERE]

Cooper and Stewart's meta analysis also covers physical and mental health outcomes. For these, effect sizes in studies with positive findings always exceed 0.03 SD units (but are usually much larger). There are several reasons why this lower bound may be a less informative benchmark for our health estimates than the corresponding lower bound for our developmental outcomes. The studies of health are fewer and tend to focus on outcomes whose comparability with our outcomes is more limited. Also, the tendency for health gradients to be smaller in Sweden (Figure AVIII), perhaps due to features of the Swedish health care system, may exacerbate concerns about external validity. With these caveats in mind, it is nevertheless interesting to note that our rescaled effect-size estimates for the health outcomes studied in pre-lottery children consistently allow us to rule out effects far below 0.03 SD units.[29] Figure AVIII provides a graphical illustration for the nine key health outcomes considered in our follow-up analyses. For total drug consumption, we can for example rule out effects with an absolute value above 0.004 SD units.

Considered in their entirety, our results clearly show that the marginal effects implied by our estimates are substantially smaller than previously reported causal estimates. While heterogeneity in the outcome measures considered could explain why we consistently find smaller effects on health, this explanation is not credible for the developmental outcomes, where we use variables similar to those studied in most earlier work. There are several plausible reasons, none of them mutually exlusive, that may help explain why we systematically find smaller effects.

A first possible reason is population heterogeneity; most studies have focused on children from disadvantaged backgrounds, often in countries that differ in important ways from Sweden. Yet, our estimates allow us to reject positive effects greater than 0.03 SD units for outcomes such as drug consumption

[28] To generate household-income gradients comparable to our rescaled estimates, we calculate the yearly average of 10-year household income and convert it to $1,000.

[29] For birth weight our rescaled estimates are less precise. One study reports significant positive effects that are contained within our confidence intervals (Hoynes, Miller, and Simon 2015).

and school achievement even when we restrict our subsample to low-income households. For the two skills variables, we can rule out positive effects altogether in our low-income sample. Our estimates are also smaller than the positive effects found for some child health and development outcomes in quasi-experimental studies in Canada and Norway, two countries that share many institutional features with Sweden (Milligan and Stabile 2011; Løken, Mogstad, and Wiswall 2012).

Another potential source of the difference is that most policy changes or welfare-to-work experiments that have been exploited in earlier studies of income effects involve change to both incomes and prices (e.g., through changes to taxes or child care subsidies). Some authors have argued that these studies cannot credibly separate income effects from other changes and may be picking up difficult-to-model substitution effects (Currie 2009; Mayer 2010; Heckman and Mosso 2014).[30] Our estimates, by contrast, can be interpreted quite unambiguously as income effects. Finally, most earlier studies have evaluated the consequences of more modestly sized, usually monthly, income supplements. One of our lotteries (Triss-Monthly) pays prizes in monthly supplements, but the supplements are much larger than those considered in previous studies of income-support programs. For some child outcomes, such as GPA, we continue to be able to rule out effects of $1,000 in annual income larger than 0.05 SD units even when we restrict the sample to Triss-Monthly. The lower effects we report could reflect diminishing marginal effects of income supplements.

V.E. Parental Behavior

Even absent any direct evidence of an impact of wealth on most of our child outcomes, knowing if the wealth shocks have any discernible impact on parental behaviors is valuable. No variables in administrative records can be unambiguously interpreted as measures of parental investments or parenting quality, but our analysis plan (Cesarini et al. 2014) specified five outcomes that may be of some relevance for testing theories of how wealth impacts children's outcomes. These five variables are asset transfers, local school quality, parental mental health, maternal smoking, and duration of parental leave.[31] A summary of the results is that none of the causal estimates are statistically distinguishable from zero (see Table AXXIX for the full set of results). For three of the outcomes – duration of maternal leave, paternal leave, and school quality – we consider the estimates precise enough to conclude that the behavioral responses on these margins must be very small. For example, we can reject positive effects of 1M SEK on maternity leave larger than 13 days, a small effect given that the average mother claims 386 days of maternity leave benefits.

[30] The challenge is to credibly rule out the possibility that the policy changes used in quasi-experimental studies only impact wealth. See for example the concerns voiced by Currie (2009, p. 96) about the welfare-to-work experiments and by Heckman and Mosso (2014, p. 717) about the studies of Duncan, Morris, and Rodrigues (2011) and Milligan and Stabile (2011).

[31] Psychologists have argued that by reducing stress, more income can improve parenting and maternal health behaviors (Elder 1974; Conger et al. 1994). In the framework economists use to analyze child development, wealth impacts children through parental investments in goods and services.

VI. Discussion

Observational studies consistently find strong gradients between markers for SES and health and child outcomes. But the causal processes that underlie these relationships remain poorly understood (Smith 1999; Deaton 2002; Currie 2009; Mayer 2010; Baker and Stabile 2012; Cutler, Lleras-Muney, and Vogl 2012). We contribute to this literature by providing credible and statistically precise estimates of the causal impact of substantial wealth shocks on a rich set of outcomes available in administrative registers. For most outcomes, we report estimates that are not statistically distinguishable from zero but often precise enough to bound the parameter being estimated to a tight range around the point estimate. Three possible exceptions to our overall finding of zero effects are that wealth appears to improve adults' mental health, increase hospitalization of children, and perhaps reduce children's obesity risk.

There are limitations to what can be learned about health and child achievement gradients by studying the randomized assignment of large lottery prizes to Swedish lottery players. Sweden has a publicly funded and universal health care system, and an educational system under which schools are prohibited from charging tuition and required to follow a national curriculum. Consequently, caution is warranted in extrapolating our results to other settings, especially to developing countries, where we have sound theoretical reasons to expect larger effects and evidence consistent with this expectation (Case 2004). Nor should one infer from our findings that large, positive, wealth shocks will necessarily have small impacts on health care demand also in developed countries without universal health care. However, health and child achievement gradients show up reliably across developed countries with quite different institutional arrangements.[32] Many theories of the gradients therefore invoke causal mechanisms that plausibly operate in Sweden and across a wide range of other cultural and policy environments.

We view our intergenerational estimates as useful primarily for testing hypotheses about the causes of the graded association between permanent income and child outcomes. Because our wealth shocks are large even from a life-cycle point of view, and children in our sample come from a heterogeneous collection of backgrounds, the setting we consider is especially suitable for testing predictions about the effects of large windfall gains on children from households with incomes spanning the entire income distribution.

Our results suggest that in a model of child development parameterized to match conditions in Sweden, the effect of permanent income on children's outcomes is small. With the exception of obesity risk, we estimate precise zero or negative effects in subpopulations for which theories of child development predict larger benefits of wealth. For example, though the mechanism differs, investment models (Becker and Tomes 1979) and parental stress models (Bradley and Corwyn 2002) predict larger positive effects of wealth shocks in families with low incomes. The small impact of wealth on proxies for parenting behavior may explain why the shocks to permanent income appear to have few discernible intergenerational impacts. Our conclusions about the impact of wealth are consistent with the findings from a rigorous study of Korean-born adoptees who were assigned to US families using a plausibly random mechanism (Sacerdote 2007), and the structural literature on child development (Heckman and Mosso 2014). Our findings are also strikingly similar to those reported by Bleakley and Ferrie (2015) in a study exploiting

[32] For example, neither within- nor cross-country analyses find a strong relationship between the strength of the health gradient and health care institutions (Cutler, Lleras-Muney, and Vogl 2012).

the randomized assignment of land in a lottery held in early $19^{\mathbf{th}}$-century Georgia. Winners were assigned a piece of land whose value was roughly equal to the median wealth at the time. Bleakley and Ferrie find no evidence that these substantial wealth shocks impacted the human-capital outcomes of winners' children or grandchildren.

In our adult analyses, our estimates allow us to rule out all but very modest effects of wealth on overall long-run mortality, cause-specific mortality, and an array of health-care-utilization variables. Three previous studies of lottery players' health report statistically significant positive effects on mental health (Lindahl 2005; Gardner and Oswald 2007; Apouey and Clark 2015). The findings of these previous studies are qualitatively but not quantitatively similar to our mental health results. Indeed, one interpretation of our results is that previous studies have lacked statistical power to detect effect sizes that our study suggests are plausible. If so, the effect sizes we estimate are useful for evaluating the credibility of statistically significant findings in earlier studies on smaller samples. To illustrate, consider the pioneering study of Lindahl (2005). Converting Lindahl's estimates to make them comparable to ours, the estimated effect of 1M SEK on an index of mental health is 0.42 in SD units (SE = 0.19). In our sample, we can reject effects on our mental health variable greater than 0.06 SD units. If we assume 0.06 is a realistic effect size, then Lindahl's statistical power to detect such an effect at the 95% level was 6.2%. Conditional on observing an effect that is significant at the 5% level, a study with such low power has a 19% chance of incorrectly signing the coefficient, and will overestimate the effect size by a factor of 7.4.[33]

Differences in the definition of the outcome variables could explain why previous studies have found order-of-magnitude stronger effects on mental health, but not why our mortality results differ from those reported by Lindahl. He estimates that 100K reduces five-year mortality by 1.30 percentage points (95 % CI -2.22 to -0.41 percentage points) and 10-year mortality by 1.89 percentage points (95 % CI -3.30 to -0.49 percentage points). In our pooled sample, the analogous estimates are 0.00 for five-year mortality (95 % CI -0.06 to 0.06 percentage points) and 0.07 for 10-year mortality (95% CI -0.03 to 0.16).[34] In each of the 11 subsamples considered in our heterogeneity analyses, our study had 99% power to detect effect sizes one quarter the size of Lindahl's. In this specific case, the discrepancy in findings is not plausibly attributable to treatment-effect heterogeneity.

Our findings of small effects are consistent with the conclusions of a number of quasi-experimental papers using natural experiments other than lotteries (Meer, Miller, and Rosen 2003; Frijters, Haisken-DeNew, and Shields 2005; Snyder and Evans 2006; Erixson 2014; Schwandt 2014). Economists, placing the most weight on this evidence, have often concluded that omitted variables and reverse causation from health to wealth primarily explain the gradients between income and health (Smith 1999; Deaton 2003; Chandra and Vogl 2010), whereas epidemiologists frequently point to the substantial health-wealth correlations – and their apparent robustness to the inclusion of a large set of controls – in support of

[33] These calculations are based on Gelman and Carlin (2014). The implied effect reported by Gardner and Oswald (2007) is an order of magnitude larger than Lindahl's estimate of 0.42 SD units, so similar analyses of their results would yield even more dramatic conclusions.

[34] Lindahl's estimates are reported in units of 130K year-1998 SEK. Inflation was 18% between 1998 and 2010, so the estimates in his Table 4 need to be multiplied by a factor of 100,000/(1.18*130,000) to be expressed in units of 100K year-2010 SEK. Our coefficient estimates are comparable to these transformed estimates if we divide them by 10.

their positions (Marmot 1994). Our estimates reinforce the economists' skepticism.[35]

We find little support for what we take to be the predictions from the epidemiological literature about the impacts of wealth. Our mental health findings are consistent with income conveying some psychosocial benefits (Adler and Newman 2002; Marmot and Wilkinson 2006), but we find no evidence that these benefits translate into improved autoimmune or cardiovascular health. Nor do we find positive effects on health outcomes in individuals with low socioeconomic status. In our mortality analyses, we find no evidence for a gradual accumulation of positive effects or that wealth confers large benefits to members of groups that the epidemiological literature has traditionally identified as vulnerable.

The identification of the causal processes that produce the relationships between SES and health over the life cycle is fraught with methodological hazards. One such hazard is that treating SES as a unified concept may obfuscate the heterogenous effects of its various dimensions over the life-cycle (Deaton 2002; Cutler, Lleras-Muney, and Vogl 2012). Though no silver bullet can answer these pressing research questions, studies of lottery players are one attractive research strategy for understanding how economic resources, one important dimension of SES, impact health and child outcomes (Mayer 1998; Smith 1999). We find that overall, the effects of substantial, positive, wealth shocks are small, both in the aggregate and in subsamples. Our results are not incompatible with the existence of substantial causal pathways from some dimensions of SES to health, but may help narrow the set of hypotheses about the causes of the gradients that should be considered plausible.

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Supplementary Material

An Online Appendix for this article can be found at QJE online (qje.oxfordjournals.org).

References

Adams, Peter, Michael D. Hurd, Daniel McFadden, Angela Merrill, and Tiago Ribeiro, "Healthy, Wealthy, and Wise? Tests for Direct Causal Paths Between Health and Socioeconomic Status," *Journal of Econometrics*, 112 (2003), 3–56.

Adda, Jérôme, James Banks, and Hans-Martin von Gaudecker, "The Impact of Income Shocks on Health: Evidence from Cohort Data," *Journal of the European Economic Association*, 7 (2009), 1361–1399.

[35] Our findings are not at odds with a US literature that shows a within-month mortality spike in connection with receipt of government transfer payments (Dobkin and Puller 2007; Evans and Moore 2011; Evans and Moore 2012). Similar spikes have been documented in Swedish data (Andersson, Lundborg, and Vikström 2015). These studies examine the impact of a modestly sized and anticipated liquidity shocks; our study examines the effects of positive shocks to permanent income.

Adler, Nancy E., and Katherine Newman, "Socioeconomic Disparities in Health: Pathways and Policies," *Health Affairs*, 21 (2002), 60–76.

Akee, Randall K. Q., William E. Copeland, Gordon Keeler, Adrian Angold, and E. Jane Costello, "Parents' Incomes and Children's Outcomes: A Quasi-Experiment Using Transfer Payments from Casino Profits," *American Economic Journal: Applied Economics*, 2 (2010), 86–115.

Andersson, Elvira, Petter Lundborg, and Johan Vikström, "Income Receipt and Mortality – Evidence from Swedish Public Sector Employees," *Journal of Public Economics*, 131 (2015), 21–32.

Apouey, Benedicte, and Andrew E. Clark, "Winning Big but Feeling no Better? The Effect of Lottery Prizes on Physical and Mental Health," *Health Economics*, 24 (2015), 516–538.

Baker, Michael, and Mark Stabile, "Determinants of Health in Childhood," in *The Oxford Handbook of Health Economics*, Sherry Glied, and Peter C. Smith, eds., (New York, NY: Oxford University Press, 2012).

Becker, Gary S., and Nigel Tomes, "Child Endowments and the Quantity and Quality of Children," *Journal of Political Economy*, 84 (1976), S143-S162.

Becker, Gary S., and Nigel Tomes, "An Equilibrium Theory of the Distribution of Income and Intergenerational Mobility," *Journal of Political Economy*, 87 (1979), 1153–1189.

Bleakley, Hoyt, and Joseph Ferrie, "Shocking Behavior: Random Wealth in Antebellum Georgia and Human Capital Across Generations," *Quarterly Journal of Economics*, [conditionally accepted].

Bradley, Robert H., and Robert F. Corwyn, "Socioeconomic Status and Child Development," *Annual Review of Psychology*, 53 (2002), 371–399.

Brooks-Gunn, Jeanne, and Greg J. Duncan, "The Effects of Poverty on Children," *The Future of Children*, 7 (1997), 55–71.

Brunner, Eric, "Stress and the Biology of Inequality," *British Medical Journal*, 314 (1997), 1472-1476.

Case, Anne, "Does Money Protect Health Status? Evidence from South African Pensions," in *Perspectives on the Economics of Aging*, David A. Wise, ed., (Chicago, IL: University of Chicago Press, 2004).

Cesarini, David, Erik Lindqvist, Matthew Notowidigdo, and Robert Östling, "The Effect of Wealth on Individual and Household Labor Supply: Evidence from Swedish Lotteries," Mimeo, IFN, 2015.

Cesarini, David, Erik Lindqvist, Robert Östling, and Björn Wallace, "Pre-Analysis Plan for Wealth, Health and Child Development: Evidence From Administrative Data on Swedish Lottery Players," AEA RCT Registry ID AEARCTR-0000442, 2014.

Chandra, Amitabh, and Tom S. Vogl, "Rising up with Shoe Leather? A Comment on Fair Society, Healthy Lives (The Marmot Review)," *Social Science & Medicine*, 71 (2010), 1227–1230.

Charlson, M. E., Peter Pompei, K. L. Ales, and C. R. MacKenzie, "A New Method of Classifying Prognostic Comorbidity in Longitudinal Studies: Development and Validation," *Journal of Chronic Diseases*, 40 (1987), 373–383.

Cheng, Terence C., Joan Costa-i-Font, and Nattavudh Powdthavee, "Do You Have to Win It to Fix It? A Longitudinal Study of Lottery Winners and Their Health Care Demand," CEP Discussion Paper No. 339, 2015.

Conger, Rand D., Xiaojia Ge, Glen H. Elder Jr., Frederick O. Lorenz, and Ronald L. Simons, "Economic Stress, Coercive Family Process, and Developmental Problems of Adolescents," *Child Development*, 65 (1994), 541–561.

Cooper, Kerris, and Kitty Stewart, "Does Money Affect Children's Outcomes? A Systematic Review," Discussion paper, Joseph Rowntree Foundation, 2013.

Currie, Janet, "Healthy, Wealthy, and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development," *Journal of Economic Literature*, 47 (2009), 87–122.

Cutler, David M., Adriana Lleras-Muney, and Tom Vogl, "Socioeconomic Status and Health: Dimensions and Mechanisms," in *The Oxford Handbook of Health Economics*, Sherry Glied, and Peter C. Smith, eds., (New York, NY: Oxford University Press, 2012).

Dahl, Gordon B., and Lance Lochner, "The Impact of Family Income on Child Achievement: Evidence from the Earned Income Tax Credit," *American Economic Review*, 102 (2012), 1927–1956.

Deaton, Angus, "Policy Implications of the Gradient of Health and Wealth," *Health Affairs*, 21 (2002), 13-30.

— —, "Health, Inequality, and Economic Development," *Journal of Economic Literature*, 41 (2003), 113-158.

Dobkin, Carlos, and Steven L. Puller, "The Effects of Government Transfers on Monthly Cycles in Drug Abuse, Hospitalization and Mortality," *Journal of Public Economics*, 91 (2007), 2137–2157.

Duncan, Greg J., Pamela A. Morris, and Chris Rodrigues, "Does Money Really Matter? Estimating Impacts of Family Income on Young Children's Achievement with Data from Random-Assignment Experiments," *Developmental Psychology*, 47 (2011), 1263–1279.

Elder, Glen H., *Children of the Great Depression: Social Change in Life Experience*, (Chicago, IL: The University of Chicago Press, 1974).

Erixson, Oscar, "Health Responses to a Wealth Shock: Evidence from a Swedish Tax Reform," IFN Working Paper No. 1011, 2014.

Evans, William N., and Timothy J. Moore, "The Short-Term Mortality Consequences of Income Receipt," *Journal of Public Economics*, 95 (2011), 1410–1424.

— —, "Liquidity, Economic Activity and Mortality," *Review of Economics and Statistics*, 94 (2012), 400-418.

Frijters, Paul, John P. Haisken-DeNew, and Michael A. Shields, "The Causal Effect of Income on Health: Evidence from German Reunification," *Journal of Health Economics*, 24 (2005), 997–1017.

Gardner, Jonathan, and Andrew J. Oswald, "Money and Mental Wellbeing: A Longitudinal Study of Medium-Sized Lottery Wins," *Journal of Health Economics*, 26 (2007), 49–60.

Gelman, Andrew, and John Carlin, "Beyond Power Calculations: Assessing Type S (Sign) and Type M (Magnitude) Errors," *Perspectives on Psychological Science*, 9 (2014), 641–651.

Grossman, Michael, "On the Concept of Health Capital and the Demand for Health," *Journal of Political Economy*, 80 (1972), 223-255.

Hankins, Scott, and Mark Hoekstra, "Lucky in Life, Unlucky in Love? The Effect of Random Income Shocks on Marriage and Divorce," *Journal of Human Resources*, 46 (2011), 403–426.

Hankins, Scott, Mark Hoekstra, and Paige Marta Skiba, "The Ticket to Easy Street? The Financial Consequences of Winning the Lottery," *Review of Economics and Statistics*, 93 (2011), 961–969.

Heckman, James J., and Stefano Mosso, "The Economics of Human Development and Social Mobility," *Annual Review of Economics*, 6 (2014), 689–733.

Hoynes, Hilary, Doug Miller, and David Simon, "Income, the Earned Income Tax Credit, and Infant Health," *American Economic Journal: Economic Policy*, 7 (2015), 172–211.

Hultkrantz, Lars, and Mikael Svensson, "The Value of a Statistical Life in Sweden: A Review of the Empirical Literature," *Health Policy*, 108 (2012), 302–310.

Imbens, Guido W., Donald B. Rubin, and Bruce I. Sacerdote, "Estimating the Effect of Unearned Income on Labor Earnings, Savings, and Consumption: Evidence from a Survey of Lottery Players," *American Economic Review*, 91 (2001), 778–794.

Kearney, Melissa S., Peter Tufano, Jonathan Guryan, and Erik Hurst, "Making Savers Winners: An Overview of Prize-Linked Saving Products," in *Financial Literacy: Implications for Retirement Security and the Financial Marketplace*, Olivia S. Mitchell, and Annamaria Lusardi, eds., (New York, NY: Oxford University Press, 2011).

Liang, Kung-Yee, and Scott L. Zeger, "Longitudinal Data Analysis Using Generalized Linear Models," *Biometrika*, 73 (1986), 13-22.

Lindahl, Mikael, "Estimating the Effect of Income on Health and Mortality Using Lottery Prizes as an Exogenous Source of Variation in Income," *Journal of Human Resources*, 40 (2005), 144–168.

Lindqvist, Erik, and Roine Vestman, "The Labor Market Returns to Cognitive and Noncognitive Ability: Evidence from the Swedish Enlistment," *American Economic Journal: Applied Economics*, 3 (2011), 101–128.

Løken, Katrine V., Magne Mogstad, and Matthew Wiswall, "What Linear Estimators Miss: The Effects of Family Income on Child Outcomes," *American Economic Journal: Applied Economics*, 4 (2012), 1–35.

Majo, Maria Cristina, and Arthur van Soest, "Income and Health Care Utilization Among the 50+ in Europe and the US," *Applied Econometrics*, 4 (2012), 3–22.

Marmot, Michael, "Social Differentials in Health Within and Between Populations," *Daedalus*, 123 (1994), 197–216.

Marmot, Michael, and Richard G. Wilkinson, eds., *Social Determinants of Health*, 2nd ed. (New

York, NY: Oxford University Press, 2006).

Mayer, Susan E., *What Money Can't Buy: Family Income and Children's Life Chances* (Cambridge, MA: Harvard University Press, 1998).

— —, "Revisiting an Old Question: How Much Does Parental Income Affect Child Outcomes?," *Focus*, 27 (2010), 21-26.

Meer, Jonathan, Douglas L. Miller, and Harvey S. Rosen, "Exploring the Health-Wealth Nexus," *Journal of Health Economics*, 22 (2003), 713-730.

Milligan, Kevin, and Mark Stabile, "Do Child Tax Benefits Affect the Well-Being of Children? Evidence from Canadian Child Benefit Expansions," *American Economic Journal: Economic Policy*, 3 (2011), 175–205.

Newacheck, Paul W, and Neal Halfon, "Prevalence and Impact of Disabling Chronic Conditions in Childhood," *American Journal of Public Health*, 88 (1998), 610–617.

Sacerdote, Bruce, "How Large are the Effects from Changes in Family Environment? A Study of Korean American Adoptees," *Quarterly Journal of Economics*, 122 (2007), 119–157.

Salkind, Neil J., and Ron Haskins, "Negative Income Tax: The Impact on Children from Low-Income Families," *Journal of Family Issues*, 3 (1982), 165–180.

Schwandt, Hannes, "Wealth Shocks and Health Outcomes: Evidence from Stock Market Fluctuations," CEP Discussion Paper No. 1281, 2014.

Semyonov, Moshe, Noah Lewin-Epstein, and Dina Maskileyson, "Where Wealth Matters More for Health: The Wealth-Health Gradient in 16 Countries," *Social Science & Medicine*, 81 (2013), 10–17.

Smith, James P., "Healthy Bodies and Thick Wallets: The Dual Relation between Health and Economic Status," *Journal of Economic Perspectives*, 13 (1999), 145-166.

— —, "The Impact of Socioeconomic Status on Health over the Life-Course," *Journal of Human Resources*, 42 (2007), 739–764.

— —, "The Impact of Childhood Health on Adult Labor Market Outcomes," *Review of Economics and Statistics*, 91 (2009), 478–489.

Snyder, Stephen E., and William N. Evans, "The Effect of Income on Mortality: Evidence from the Social Security Notch," *Review of Economics and Statistics*, 88 (2006), 482–495.

Stansfeld, Stephen A., Rebecca Fuhrer, Martin J. Shipley, and Michael G. Marmot, "Psychological Distress as a Risk Factor for Coronary Heart Disease in the Whitehall II Study," *International Journal of Epidemiology*, 31 (2002), 248–255.

Van Kippersluis, Hans, and Titus J. Galama, "Why the Rich Drink More, but Smoke Less: The Impact of Wealth on Health Behavior," RAND Working Paper Series WR-988, 2013.

Waldenström, Daniel, "Swedish Stock and Bond Returns, 1856-2012," IFN Working Paper No. 1027, 2014.

Williams, David R., "Socioeconomic Differentials in Health: A Review and Redirection," *Social Psychology Quarterly*, 53 (1990), 81–99.

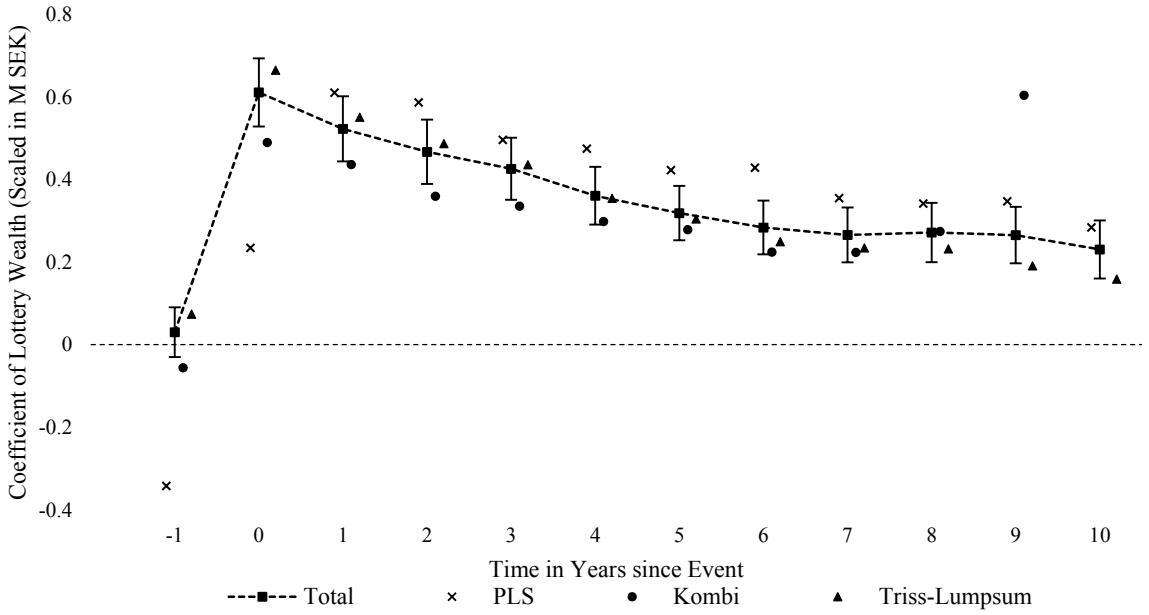

FIGURE I

The Effect of Lottery Wealth on Net Wealth According to Administrative Registers

See OA VI.D for details on the net wealth variable. Plotted in the figure are regression coefficients from Equation (1) with the dependent variable defined as household net wealth at t = -1,. . . ,10. Standard errors are clustered by individual, and the error bars give 95% confidence intervals. The seemingly anomalous lottery-specific estimates (e.g. PLS at t = 0 and Kombi at t = 9) are all imprecisely estimated. For example, the t = 9 Kombi estimate is based on draws from a single year of data; the Kombi panel begins in 1998 and our last year with wealth data is 2007. Household wealth is defined as the wealth of the winner plus, if applicable, the wealth of the spouse or cohabitating partner. Coefficients can be interpreted as the effect of a one-SEK increase in lottery prize on measured net wealth. Only the lump-sum lotteries are included in the Total.

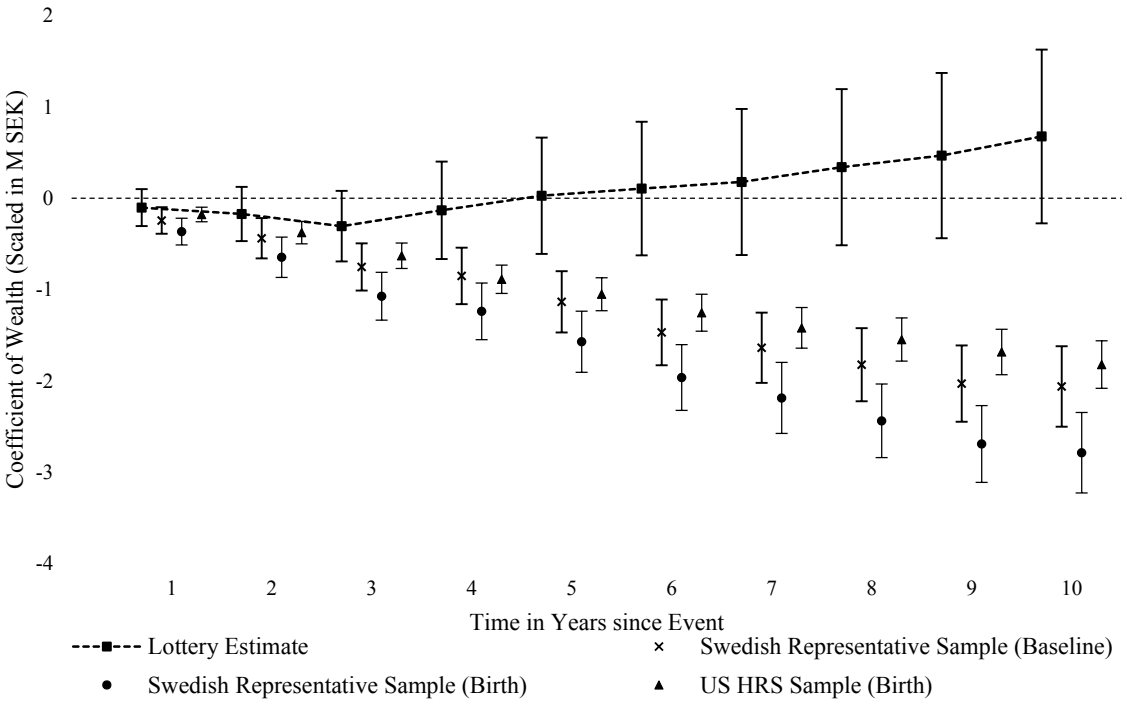

FIGURE II
Wealth and Mortality

This figure contrasts our lottery-based estimates of the effect of wealth on mortality to gradients estimated in Swedish and US population samples. The population samples have been re-weighted to match the sex and age distribution of our sample of lottery winners. Gradients are separately estimated with controls for birth demographics for Sweden and the US, as well as with the full set of baseline controls for Sweden. Standard errors are clustered by individual, and the error bars give 95% confidence intervals of the coefficient.

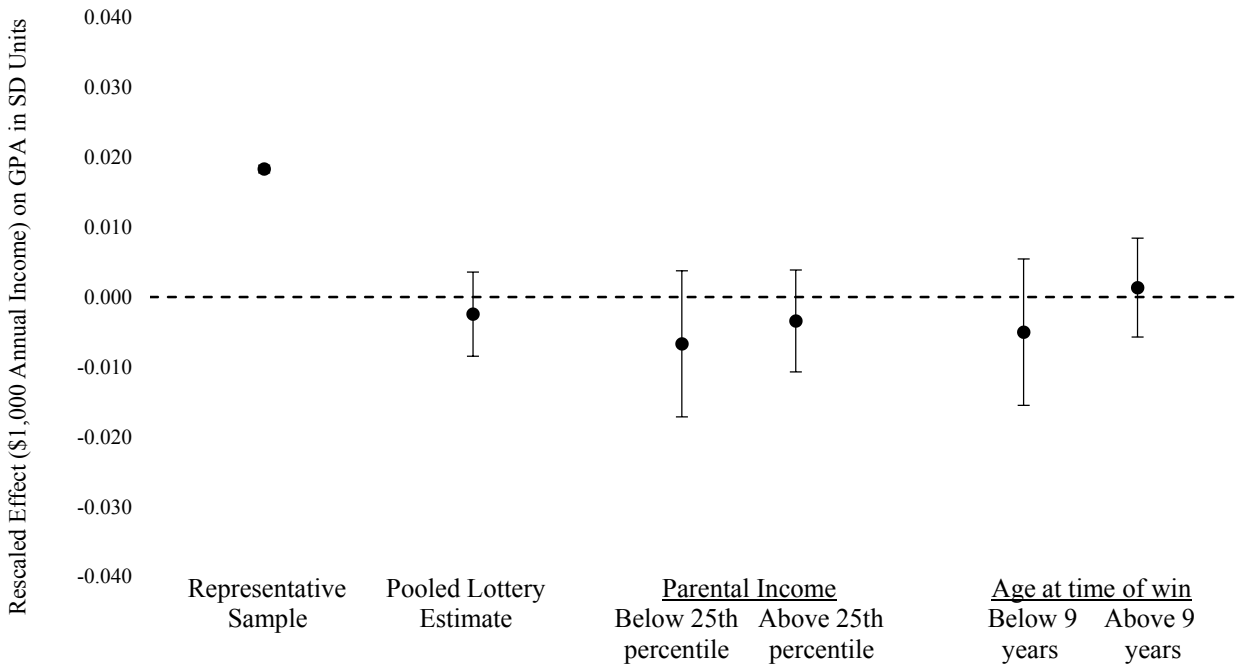

FIGURE III
Household Income and Child Grade Point Average

This figure compares our lottery-based estimates, rescaled for comparability, of the causal impact of wealth on child GPA in different subsamples to the household-income gradient estimated in a large representative sample of children. All coefficients are in standardized GPA units. Gradients include controls for birth demographics and the lottery estimates additionally include demographic controls for the winning parent. Standard errors are clustered using an iterative algorithm that assigns siblings and half-siblings to the same cluster.

TABLE I

OVERVIEW OF MAIN REGISTERS AND CONTROL VARIABLES

Panel A: Registers Used to Generate Main Outcome Variables

Register Name (Period)	Unit of Observation	Data Description	Outcome Measured
Cause of Death (1952-)*	Death Event	Date & Cause of Death (ICD)	Up to 24 Post-Lottery Years
Conscription (1969-)**	~18-Year Old Male	Cog. & Noncog. Skills, BMI	At Age ~18
Medical Birth (1973-)**	Infant	Birth Weight, Gestation	At Birth
Ninth Grade (1988-)**	Ninth Grade Student	GPA	At Age ~15
National Tests (2003-)**	Ninth Grade Student	Test Scores in English, Mathematics, Swedish	At Age ~15
Patient (1987-)	In-Patient Hospitalization	Entry & Discharge Dates, Diagnoses (ICD)	Up to 10 Post-Lottery Years
Prescribed Drug (2006-)	Drug Purchase	Date, Type (ATC), Quantity (DDD)	2006-2010

Panel B: Wealth- and Income Gradients

Register Name (Period)	Income/Wealth Variable
Wealth (1999-2007)*	Net Wealth: Year-end Sum of Real and Financial Assets Minus Debt.
Income & Taxation (1978-)**	Household Income: Sum of Biological Parents' Disposable Income in Child's First Ten Years of Life.

Panel C: Definition of Baseline Controls

Birth Demographics	Other Demographics	Health Characteristics
Age	1 if Married	1 if Hospitalized
Age^2	1 if College-Graduate	1 if Hospitalized $\geq$ 7 days
Age^3	# Children	1 if Hospitalized for Cancer
1 if Female	Labor Income	1 if Hospitalized for Respiratory
1 if Born in Nordic Country	1 if Retired*	1 if Hospitalized for Circulatory
		Charlson Index

Notes. Panel A provides background information about the registers from which several of our key variables are derived. Panel B defines the wealth and income variables we use to estimate gradients against which we sometimes benchmark our causal estimates. Panel C defines a set of variables that will be used throughout the paper. We refer to the union of birth and other demographics as "demographic characteristics" and to the union of demographic and health characteristics as the "baseline characteristics" or "baseline controls". Annual labor income is measured in SEK. All hospitalization variables are binary and defined over the preceding five years. Additional details on the construction of the Charlson index are available in OA Section VI.E. BMI: Body Mass Index. ATC: Anatomical Therapeutic Chemical Classification. ICD: International Classification of Diseases. DDD: Defined Daily Dose. * used in adult analyses only, ** intergenerational analyses only.

TABLE II

OVERVIEW OF IDENTIFICATION STRATEGY

Lottery	Period	Type	Cell Construction		Number of Cells		
			Adult Analyses	Intergenerational Analyses	Adult	Pre-lottery	Post-lottery
PLS Fixed Prizes	1986-2003	Lumpsum	Draw × # Fixed Prizes	Draw × # Fixed Prizes × # Children	228	487	400
PLS Odds Prizes	1986-1994	Lumpsum	Draw × # Prizes × # Tickets	Excluded	1881	0	0
Kombi	1998-2010	Lumpsum	Draw × # Tickets	Draw × # Tickets × # Children	262	51	6
Triss-Lumpsum	1994-2010	Lumpsum	Year × Prize Plan	Year × Prize Plan × # Children	18	67	51
Triss-Monthly	1997-2010	Monthly Installments	Year × Prize Plan	Year × Prize Plan × # Children	19	37	12

Notes. This table provides a summary overview of the identification strategies used in the adult and intergenerational analyses. Our identification strategy uses the available data and detailed knowledge about the lotteries to define subsamples/cells within which wealth is randomly assigned. "Cell Construction" describes the characteristics two players must share in order to be assigned to the same cell. Pre-lottery children are children conceived before the lottery and post-lottery children are children conceived after the lottery. Post-lottery children are only included in our analyses of infant health outcomes, whereas pre-lottery children are included in all other intergenerational analyses.

TABLE III

TESTING FOR THE CONDITIONAL RANDOM ASSIGNMENT OF LOTTERY PRIZES

	Pooled Sample		Individual Lottery Samples				Parents with…	
			PLS	Kombi	Triss-Lumpsum	Triss-Monthly	Pre-lottery Child(ren)	Post-lottery Child(ren)
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Fixed Effects	None	Cells	Cells	Cells	Cells	Cells	Cells	Cells
N	439,234	439,234	387,813	46,486	4,250	685	68,584	34,187
R^2	0.001	0.477	0.054	0.002	0.009	0.107	0.572	0.525
Lagged Demographic Characteristics								
F-statistic	30.84	0.46	0.29	1.01	1.65	0.56	1.04	0.51
p (analytical)	[<0.001]	[0.926]	[0.989]	[0.431]	[0.078]	[0.845]	[0.405]	[0.897]
p (resampling)	[0.001]	[0.943]	[0.990]	[0.958]	[0.466]	[0.888]	[0.805]	[0.973]
Lagged Health Characteristics								
F-statistic	1.18	1.66	0.86	1.26	1.40	1.37	1.00	1.74
p (analytical)	[0.309]	[0.114]	[0.536]	[0.269]	[0.199]	[0.213]	[0.427]	[0.095]
p (resampling)	[0.322]	[0.163]	[0.596]	[0.336]	[0.391]	[0.342]	[0.667]	[0.353]
Lagged Baseline Characteristics								
F-statistic	24.63	0.97	0.51	1.05	1.43	0.91	1.05	1.05
p (analytical)	[<0.001]	[0.495]	[0.956]	[0.404]	[0.108]	[0.565]	[0.402]	[0.403]
p (resampling)	[0.001]	[0.573]	[0.965]	[0.915]	[0.559]	[0.705]	[0.865]	[0.820]

Notes. This table reports results from our randomization tests. Each column corresponds to a regression where the dependent variable is lottery prize and we control for baseline characteristics measured the year before the lottery event in all specifications. Under the null hypothesis of conditional random assignment, variables determined before the lottery should not have any predictive power conditional on the cell fixed effects. Column (1) shows the specification without cell fixed effects and column (2) the specification that controls for cell fixed effects. Columns (3)-(6) report the results separately by lottery and columns (7) and (8) display the results when we restrict the sample to players with pre- or post-lottery children, respectively. The reported p-values are from tests of the joint significance of the demographic characteristics, the health characteristics and the baseline characteristics. The non-parametric p-values are obtained from the resampling distribution of the F-statistic in 1,000 Monte Carlo simulation draws in which prizes are randomly permuted within each cell.

TABLE IV

SIMILARITY OF POOLED LOTTERY SAMPLE TO GENERAL POPULATION

	Pooled Lottery Sample		Sex- and Age-Reweighted Representative Sample	
	Unweighted	Prize-weighted	Weighted to Match (1)	Weighted to Match (2)
	(1)	(2)	(3)	(4)
Birth Year	1935.9	1943.3	1935.9	1943.3
Female	51.0%	49.2%	51.0%	49.2%
Nordic Born	97.2%	95.8%	94.6%	92.9%
# Children	1.63	1.69	1.78	1.74
College-Graduate	18.0%	18.3%	16.0%	21.2%
Married	57.8%	55.5%	54.6%	54.1%
Retired	38.9%	27.8%	34.8%	26.3%
Labor Income/1000	127.5	158.3	112.2	145.3
Hospitalized	31.9%	27.4%	34.4%	29.4%
Hospitalized ≥ 7 days	15.8%	12.0%	18.3%	13.5%
Hospitalized for Cancer	3.9%	3.5%	5.0%	3.9%
Hospitalized for Respiratory	3.5%	3.3%	4.5%	3.5%
Hospitalized for Circulatory	10.2%	7.5%	11.8%	8.8%
N	439,234	439,234	91,036	90,992

Notes. This table compares the baseline characteristics of individuals in the pooled lottery sample to those of the general population. All player characteristics are measured the year before the lottery event. Column (1) reports unweighted summary statistics for the pooled lottery sample and column (2) summary statistics with observations weighted by prize amount won. Columns (3) and (4) shows descriptive statistics for a representative sample of Swedes aged 18 and above which has been reweighted to match the age and sex distribution of (1) and (2), respectively. PLS winners were matched to a representative sample from 1990 and the other lotteries to a representative sample from 2000. The number of observations refers to the total number of lottery prizes in columns (1) and (2) and to individuals in columns (3) and (4).

TABLE V

DISTRIBUTION OF PRIZES AWARDED

	Adult Sample	Mean Prize	Adult Lottery Samples by Lottery				Parents with Pre-Lottery Child(ren)	Parents with Post-Lottery Child(ren)
			PLS	Kombi	Triss-Lumpsum	Triss-Monthly		
<10K	404,165	1.2K	358,141	46,024	0	0	61,944	30,796
10K to 100K	27,109	17K	25,926	0	1,183	0	5,053	2,827
100K to 500K	5,293	173K	2,650	0	2,643	0	1,147	450
500K to 1M	527	671K	324	0	203	0	68	32
$\geq$ 1M	2,140	2.08M	772	462	221	685	372	82
Total	439,234	15K	387,813	46,486	4,250	685	68,584	34,187

Notes. This table shows the number of prizes assigned to individuals in our final estimation sample. Matched controls from the Kombi sample are included in the category "<10K". For Triss-Monthly, we define the prize size as the net present value of the monthly installments assuming an annual discount rate of 2%. Mean prize is the average value of prizes in the category in the adult sample.

TABLE VI

PROPORTIONAL HAZARD MODEL ESTIMATES OF THE EFFECT OF WEALTH ON MORTALITY

	Pooled Lottery	Representative Sample		Sex		College-Graduate	
		Baseline	Birth	Female	Male	Yes	No
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Effect/Gradient	1.015	0.874	0.828	1.058	0.973	0.960	1.020
SE	(0.026)	(0.015)	(0.015)	(0.041)	(0.035)	(0.086)	(0.027)
p	[0.564]	[<0.001]	[<0.001]	[0.147]	[0.437]	[0.647]	[0.461]
Number at Risk	439,234	39,019	39,019	224,083	215,151	78,869	360,365
Number of Deaths	139,049	5,138	5,138	67,003	72,046	12,521	126,528
Heterogeneity *p*	[<0.001]			[<0.001]	[0.009]	[0.327]	[<0.001]
		Age		Hospitalized		Labor Income > Median	
	18-44	45-69	70+	No	Yes	Below	Above
	(8)	(9)	(10)	(11)	(12)	(13)	(14)
Effect	0.920	0.963	1.054	1.016	1.026	1.018	1.015
SE	(0.078)	(0.041)	(0.036)	(0.040)	(0.036)	(0.028)	(0.066)
p	[0.322]	[0.370]	[0.127]	[0.691]	[0.467]	[0.506]	[0.818]
Number at Risk	88,738	220,271	130,225	299,037	140,197	219,655	219,579
Number of Deaths	2,231	44,928	91,890	71,084	67,965	116,365	22,684
Heterogeneity *p*	[0.560]	[0.040]	[<0.001]	[<0.001]	[<0.001]	[<0.001]	[0.038]

Notes. This table shows the estimated hazard ratios from survival models with right censoring. All regressions except (3) includes the full set of baseline controls. "Hospitalized" refers to whether the winner was hospitalized during the five years preceding the lottery event. Coefficients are expressed as hazard ratios, so an estimate of 1.1 would imply that 1M SEK increases the hazard by 10%. Standard errors are clustered by individual. Heterogeneity *p* refers to the *p*-value from test of null hypothesis that the hazard ratio is equal to the hazard ratio in column (2).

TABLE VII

EFFECT OF WEALTH ON HEALTH CARE UTILIZATION

	Hospitalization			Health Index			Drug Consumption (2006-2010)	
	$t = 2$	$t = 5$	$t = 10$	$t = 2$	$t = 5$	$t = 10$	Total	Mental Health
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Effect (M SEK)	0.304	0.393	-0.027	-0.080	0.297	0.453	-97.56	-32.50
SE	(0.505)	(0.617)	(0.770)	(0.143)	(0.268)	(0.355)	(84.62)	(10.33)
p (analytical)	[0.547]	[0.524]	[0.972]	[0.577]	[0.267]	[0.202]	[0.249]	[0.002]
p (resampling)	[0.525]	[0.555]	[0.977]	[0.488]	[0.220]	[0.210]	[0.268]	[0.018]
Mean/SD	25.6%	38.3%	51.2%	14.5/24.2	21.0/31.5	31.1/38.5	4,375/6,168	311/945
N	415,215	378,099	296,904	431,064	418,002	367,863	279,784	279,784
Gradient (M SEK)	-0.716	-0.764	-0.288	-0.414	-1.133	-1.410	-194.15	-33.06
SE	(0.280)	(0.316)	(0.334)	(0.092)	(0.128)	(0.157)	(44.112)	(6.216)
Mean/SD	24.77%	37.66%	50.86%	14.6/24.2	21.3/31.7	32.8/39.1	4,659/6,500	393/1,130
Heterogeneity p	[0.077]	[0.095]	[0.756]	[0.049]	[<0.001]	[<0.001]	[0.311]	[0.963]

Notes. This table reports estimates of the causal impact of wealth on some key health care utilization variables. For the binary hospitalization variables, wealth is scaled so a coefficient of 1.00 implies that 1M SEK increases the probability of hospitalization by one percentage point. For remaining outcomes, wealth is scaled in million SEK. Wealth gradients are estimated in a representative sample of adults that has been reweighted to match the sex- and age-distribution of the lottery sample. All regressions include controls for the full set of baseline characteristics. Higher values for the health index denotes worse health. Standard errors are clustered by individual. Resampling-based p-values are obtained from 1,000 Monte Carlo simulation draws in which the prizes are permuted randomly within each cell. The heterogeneity p-value is from a two-sided t-test of the null hypothesis that the gradient and causal parameter are equal.

TABLE VIII

WEALTH AND CHILD HEALTH OUTCOMES

	Infant Health			Body Mass			Drug Consumption			
	Birth Weight (grams)	Low Birth Weight	Premature Birth	BMI	Over-weight	Obese	Total	Mental Health	Allergy & Asthma	ADHD
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Effect (M SEK)	16.265	-1.140	-1.461	-0.113	-0.919	-2.141	8.895	-3.710	5.378	-11.247
SE	(42.802)	(1.162)	(0.741)	(0.225)	(2.324)	(0.858)	(27.656)	(12.463)	(12.930)	(7.645)
p (analytical)	[0.704]	[0.327]	[0.049]	[0.614]	[0.692]	[0.013]	[0.748]	[0.766]	[0.677]	[0.141]
p (resampling)	[0.815]	[0.723]	[0.128]	[0.585]	[0.695]	[0.002]	[0.577]	[0.909]	[0.599]	[0.502]
Mean/SD	3,547/583	4.00%	5.91%	22.4/3.2	15.87%	2.91%	468/1,540	106/576	91.5/357	13.4/156
N	24,977	24,977	25,026	32,646	32,646	32,646	112,223	105,197	21,298	20,656
Gradient (M SEK)	23.537	-0.196	-0.192	-0.174	-2.486	-0.984	7.276	1.525	3.974	-4.101
SE	(1.808)	(0.057)	(0.068)	(0.024)	(0.275)	(0.119)	(2.921)	(1.807)	(1.032)	(0.539)
Heterogeneity *p*	[0.865]	[0.418]	[0.088]	[0.787]	[0.503]	[0.182]	[0.954]	[0.678]	[0.914]	[0.351]

	Hospitalizations							
	All Causes		All Causes ≥ 7 days		Respiratory		External	
	t = 2	*t* = 5	*t* = 2	*t* = 5	*t* = 2	*t* = 5	*t* = 2	*t* = 5
	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)
Effect (M SEK)	2.119	3.443	0.400	0.227	0.864	0.949	0.320	1.017
SE	(0.747)	(0.913)	(0.291)	(0.320)	(0.519)	(0.776)	(0.381)	(0.768)
p (analytical)	[0.005]	[0.0002]	[0.170]	[0.478]	[0.096]	[0.222]	[0.400]	[0.185]
p (resampling)	[0.002]	[<0.001]	[0.142]	[0.440]	[0.018]	[0.124]	[0.388]	[0.178]
Proportion	10.89%	17.99%	1.70%	2.78%	2.76%	4.78%	2.66%	4.93%
N	114,160	111,064	114,160	111,064	100,327	75,382	100,327	75,382
Gradient (M SEK)	-0.097	-0.288	-0.009	-0.012	-0.071	-0.147	-0.037	-0.102
SE	(0.070)	(0.107)	(0.026)	(0.045)	(0.024)	(0.038)	(0.041)	(0.070)
Heterogeneity *p*	[0.003]	[<0.001]	[0.162]	[0.459]	[0.072]	[0.159]	[0.351]	[0.147]

Notes. This table summarizes the results from our analyses of the pre-specified child health outcomes. In the regressions with binary dependent variables, wealth is scaled so a coefficient of 1.00 implies that 1M SEK increases the probability by one percentage point. For remaining outcomes, wealth is scaled in million SEK. Household-income gradients are obtained by regressing each outcome on a 10-year sum of household disposable income in a representative sample selected using the same cohort and age restrictions used in the lottery analyses (Table AXXIII). Except in the infant health analyses, we control for the child's birth characteristics. In the lottery regressions, we also control for the winning parent's demographic characteristics. Standard errors are clustered using an iterative algorithm that assigns siblings and half-siblings to the same cluster. Resampling-based *p*-values are obtained from 1,000 Monte Carlo simulation draws in which the prizes are permuted randomly within each cell. The heterogeneity *p*-value is from a two-sided *t*-test of the null hypothesis that the gradient and causal parameter are equal. BMI: Body Mass Index. ADHD: Attention Deficit Hyperactivity Disorder.

TABLE IX

WEALTH AND CHILD DEVELOPMENT

	Skills		GPA	Ninth Grade National Test Scores		
	Cognitive	Noncognitive		Swedish	English	Math
	(1)	(2)	(3)	(4)	(5)	(6)
Effect (M SEK)	-0.113	-0.031	-0.022	-0.034	-0.081	-0.029
SE	(0.048)	(0.077)	(0.027)	(0.039)	(0.043)	(0.052)
p (analytical)	[0.019]	[0.686]	[0.424]	[0.385]	[0.060]	[0.579]
p (resampling)	[0.074]	[0.573]	[0.679]	[0.478]	[0.172]	[0.611]
Mean/SD	0.17/0.98	0.14/0.98	0.27/0.94	0.16/1.00	0.11/0.99	0.2/1.01
N	36,435	31,550	74,187	25,079	25,286	23,990
Gradient (M SEK)	0.221	0.163	0.256	0.199	0.204	0.213
SE	(0.007)	(0.008)	(0.004)	(0.005)	(0.005)	(0.005)
Heterogeneity *p*	[<0.001]	[0.012]	[<0.001]	[<0.001]	[<0.001]	[<0.001]

Notes. This table summarizes the findings from our analyses of the pre-specified child development outcomes. Wealth is scaled in million SEK and all variables are standardized so that coefficient estimates are in population standard-deviation units. Gradients are obtained by regressing each outcome on the 10-year sum of annual household disposable income in a representative sample selected using the same cohort and age restrictions as in the lottery analyses (see Table AXXIII). We control for the child's birth characteristics. In all lottery regressions, we also control for the winning parent's demographic characteristics. Standard errors are clustered using an iterative algorithm that assigns siblings and half-siblings to the same cluster. Resampling-based *p*-values are obtained from 1,000 Monte Carlo simulation draws in which the prizes are permuted randomly within each cell. The heterogeneity *p*-value is from a two-sided *t*-test of the null hypothesis that the gradient and causal parameter are equal.