The Fetal Origins Hypothesis in Finance: Prenatal Environment and Financial Risk Taking

This draft: April 30, 2014

Abstract

We find that differences in an individual's prenatal environment explain heterogeneity in financial risk taking propensities much later in life. An exogenous increase in exposure to prenatal testosterone, the most potent steroid hormone in humans, is related to elevated risk taking in financial market in adulthood. We also examine birth weight, the most widely used summary measure of the early life environment. We find that those with lower birth weight are less likely to hold risky assets, but, conditional on holding risky assets, prefer more volatile equity portfolios than those with higher birth weight. This study is one of the first attempts to incorporate into finance research the fetal origins hypothesis and the notion that prenatal environment programs a fetus in the womb to have persistent behavioral characteristics. Our results contribute to the understanding of how environmental conditions and circumstances shape individuals' behavior in financial markets.

I Introduction

A large literature in economics shows the importance of the early life environment for economic outcomes much later in life. In fact, several "fetal origins" studies have shown that conditions and circumstances even before birth are of first-order importance when it comes to explaining the observed heterogeneity in individuals' life trajectories, in particular their long-term human and health capital. In their recent review article, Almond and Currie (2011b) go as far as asking: "[W]hat if the nine months *in utero* are one of the most critical periods in a person's life [...]?"

In financial economics research, specifically related to individual investor behavior, the importance of the early life environment has received relatively limited attention. Some studies, which focus on the *postnatal* environment, have recently attempted to fill this void. For example, the evidence reported by Malmendier and Nagel (2011) suggests that "Depression Babies" develop more aversion to financial risk taking later in life. Cronqvist, Siegel, and Yu (2014) show that individuals who grew up during the Depression era, or in relatively less wealthy families, develop a more value-oriented investment style later in life. Chetty et al. (2011) report that the pre-school (kindergarten) environment explains some asset allocation decisions among adults, such as contributing to a 401(k) retirement savings plan and owning a home.[1]

In this study, we extend these efforts by examining whether differences in the *prenatal*, i.e., pre-birth, environment explains heterogeneity in individuals' financial risk taking propensities much later in life. First, we examine the long-term effects of differential prenatal exposures to testosterone. We focus on testosterone as it is the most potent steroid hormone in humans, and perhaps more importantly, we can hypothesize the direction of an effect of pre-birth testosterone exposure on financial risk taking. Existing research on the effect of prenatal testosterone on risk taking has generally relied on the 2D:4D finger ratio, i.e., the ratio of the index and ring finger lengths, a noisy biomarker of pre-birth testosterone exposure, and has provided inconclusive evidence (e.g., Apicella et al. (2008) and Sapienza, Zingales, and Maestripieri (2009)). Our

[1]Several recent studies have also found that experiences in adulthood are important for an individual's investment behavior later in life (e.g., Malmendier and Nagel (2014) and Knüpfer et al. (2014)).

empirical identification strategy instead relies on a natural experiment that occurs in some twins pregnancies. More specifically, the "Twin Testosterone Transfer" (TTT) hypothesis predicts that, in the case of opposite sex twins, the higher level of prenatal testosterone in the amniotic fluid of a male fetus increases the pre-birth testosterone exposure of the female fetus that shares the womb with the male fetus.

Second, we study the long-term effects of differences in birth weights. While the limitations of birth weight as a summary measure of endowments at birth is increasingly well-recognized (e.g., Almond et al. (2005)), little progress has been made towards identifying a superior measure. We use a sample of identical twins to control for confounding factors, i.e., our identification strategy allows us to control for unobserved characteristics of the mother as well as the genetic make-up of the twins. It therefore ensures that the birth weight differences are driven by environmental factors (e.g., nutritional intake within the uterus) rather than by genetic factors.

The data we use for this study come from the Swedish Twin Registry (STR), the world's largest twin registry with very detailed information on over 50,000 different same- and opposite-sex twin pairs from birth cohorts dating back to the 19th century, and constitute a combination of register and survey data. These data have been matched with detailed financial data from the Swedish Tax Authority and other individual data (e.g., family structure and education data) from Statistics Sweden, and allow us to measure financial risk taking in several alternative ways, e.g., the proportion of an individual's financial assets allocated to risky equities.

Our empirical evidence is consistent with the fetal origins hypothesis and suggests that the prenatal environment is important for an individual's financial risk taking propensity several decades later in life. First, we find that a female with a male co-twin, i.e., an individual in the treatment group, takes significantly more risk later in life compared to a female with a female co-twin, i.e., an individual in the control group. The economic magnitude of the effect is sizable: A treated female allocates about 1.24 percentage points more of the financial assets to equities compared to a female in the control group (about 3% compared to the mean allocation). These treatment effects are significant also relative to the gender gap in financial risk taking (e.g.,

Croson and Gneezy (2009) and Sundén and Surette (1998)). More specifically, we find that a significant proportion, about 39%, of the the gender gap is explained by pre-birth exposure to the testosterone hormone, suggesting that biological factors explain a sizable proportion of the gender gap in financial risk taking. We verify that intra-twin pair social interactions in adulthood cannot easily explain our results and that the effect of a male co-twin is not merely reflecting a more general male sibling effect.

Second, controlling for twin pair fixed effects, we find that those with lower birth weight, i.e., with more adverse prenatal conditions in a general sense, are less likely to hold risky assets, but, conditional on holding risky assets, prefer more volatile equity portfolios than those with higher birth weight. A one standard deviation decreases in *Birth Weight (ln)* increases the *Portfolio Volatility* by about 0.70 percentage points, or about 5% of the mean portfolio volatility (15.26%) in the entire sample. That is, those with lower birth weight select more volatile portfolios relative to those with higher birth weight. The effect of birth weight does not appear to operate through individual characteristics that also vary with birth weight (e.g., health and education), but rather through a direct effect of birth weight on financial behavior, suggesting that, e.g., nutritional intake in utero programs a fetus to specific behaviors later in life.

Our paper contributes to several pre-existing literatures in finance and economics research. First, this is one of the first attempt to incorporate the fetal origins hypothesis into financial economics. This hypothesis has been very useful for economists' understanding of long-term effects of the early environment on health and human capital (e.g., Almond and Currie (2011b) and Currie (2011)), and we show that it is useful also for understanding individual investors' financial risk taking propensities later in life. Second, with a growing literature in finance having established the importance of genetics in explaining cross-sectional heterogeneity in financial risk taking (e.g., Cesarini et al. (2009), Barnea, Cronqvist, and Siegel (2010), and Cesarini et al. (2010)), the focus is shifting to a search for the environmental circumstances and life experiences that explain outcomes of interest to financial economists. Our research is one of the first attempts in finance to show that the early life environment, even pre-birth experiences in the

womb, explains differences in risk taking in financial markets. Finally, our paper contributed to a literature in the intersection of finance and neuroscience which seeks to establish causal effects of prenatal testosterone exposure, but which has provided inconclusive evidence (e.g., Apicella et al. (2008) and Coates et al. (2009), and Sapienza, Zingales, and Maestripieri (2009)). Using an different identification strategy and a large-scale field data on individuals' asset allocations, our research has the potential to clarify the role prenatal testosterone exposure plays for risk taking behavior later in life.

The paper is organized as follows. Section II reviews related research. Section III describes our data. Sections IV and V report our results and robustness checks. Section VI concludes.

II Related Research

In this section, we review the scientific evidence on which we base our hypothesis that different prenatal environments might explain heterogeneity in risk taking propensities much later in life. We first introduce the fetal origins hypothesis which originates from medical research, and review the empirical evidence in applied economics research related to fetal programming and health capital as well as human capital later (sometimes several decades later) in an individual's life. We also explain our identification strategy, in particular the "Twin Testosterone Transfer" (TTT) hypothesis. Finally, we review the pre-existing empirical evidence related to at-birth endowments, proxied by birth weight, and long-term economic outcomes.

A Fetal Origins Hypothesis

The fetal origins hypothesis was pioneered in medical research by Barker (1990); Barker and Robinson (1992) who argued that the intrauterine environment may program a fetus to have particular characteristics which may affect the individual in adulthood. According to this hypothesis, the effects of prenatal conditions and circumstances may be very persistent. More specifically, Barker argued that individuals who are starved or otherwise experience poor nutrition *in utero* are significantly more likely to become overweight as adults, possibly because of

compensating programming taking place *in utero*, and that these individuals are actually more likely to suffer from diseases associated with obesity, including diabetes and cardiovascular-related diseases (e.g., Barker (1995)). This mechanism is called "fetal programming" and is just started to be researched and understood in depth. One possibility is that the epigenome, which may be thought of as a set of switches that cause parts of the genome to be expressed or not, is affected in a significant way by the pre-birth environment (e.g., Petronis (2010)).[2] Pre-existing scientific evidence related to the fetal origins hypothesis constitutes the basis for the empirical analysis pursued in this paper, i.e., financial risk taking later in life may in part be the outcome of fetal programming.

Over the past decade, the fetal origins hypothesis has made its way from medical research into economic research. Currie and Hyson (1999) was first in economics research to conclude that the fetal origin effects were not confined only to long-term health capital, but they extend also to human capital measures, e.g., IQ and educational attainment. Studies in applied economics have used exogenous variation in factors such as nutrition, diseases, and pollution to identify causal treatment effects of the prenatal environment.

To provide only a few examples from applied economics research, the long-term effects of poor nutrition *in utero* have been studied using data from the *Hongerwinter* of 1944-45 when the Nazi Germany effectively stopped all food supplies to the Netherlands, and adult rations dropped as low as 580 kilocalories per day, and significant effects on disease rates later in life have been reported (e.g., Stein et al. (1975) and Ravelli et al. (1976)). Other studies of the long-term effects on health as well as human capital of prenatal nutrition include studies of the *Phylloxera* insect which asymmetrically affected available income and food resources at different vineyards in France in the late 19th century (e.g., Banerjee et al. (2010)), and studies of fasting during the Ramadan among pregnant mothers (e.g., Almond and Mazumder (2011)). Turning to studies of the prenatal effect of diseases, Almond et al. (2005) and Almond (2006) studied children to mothers who were pregnant during the influenza epidemic of 1918 in the U.S. and

[2]See, e.g., Lombardo et al. (2012a) and Lombardo et al. (2012b) for recent scientific papers related to fetal programming.

found that they experienced reduced educational attainment, lower income and socioeconomic status, as well as accelerated disability rates as adults (some of these differences remaining observable in the "treated" individuals even when they were in their 80s). Others have studied the long-term treatment effects on cognitive ability of heterogeneity in pre-birth exposure to pollution such as exposure to Chernobyl fallout in Sweden (e.g., Almond et al. (2009)) and the effects on educational attainment of particulate matter (PM) in the air, which varies exogenously with the business cycle (e.g., Sanders (2012)).

The overall conclusion from this literature is the importance of the prenatal environment for long-term health and human capital.[3]

B Twin Testosterone Transfer Hypothesis

Because of our focus in this paper on financial risk taking, we examine the long-term effects of heterogeneous prenatal exposure to testosterone, the most potent steroid hormone in humans and one which has consistently been found to be related to risk taking among adults. In the mother's womb, a human fetus endogenously generates testosterone and the exposure to this hormone has been shown to cause permanent changes in the brain's development, the so-called organizational effects of testosterone. Studies show that there is indeed significant between- as well as within-gender differences in pre-birth testosterone exposure levels. As an example of differences in magnitudes, Baron-Cohen et al. (2005) report significant cross-sectional variation in prenatal testosterone among both male fetuses (N=41; prenatal T range in nmol/l is 0.125-1.800 with a mean of 0.943 and a standard deviation of 0.365) and female fetuses (N=30; prenatal T range in nmol/l is 0.150-0.800 with a mean of 0.358 and a standard deviation of 0.161). As a result, *in utero* testosterone exposure is a promising approach to study the effects of very different pre-birth environments on financial risk taking propensities later in life.

Any study of prenatal testosterone is associated with several empirical challenges. First, the direct measurement of prenatal testosterone in the amniotic fluid in pregnant mothers

[3]We refer to Almond and Currie (2011a) and Almond and Currie (2011b) for additional references and a more complete and in-depth review of the fetal origins hypothesis.

(via amniocentesis) is invasive and has therefore been restricted to small and potentially non-representative samples (e.g., van de Beek et al. (2004) and Baron-Cohen et al. (2004)). Second, while exogenous manipulation of testosterone is increasingly used in research in the intersection of economics, finance, and neuroscience to cause treatment effects (e.g., Zak et al. (2009) and Eisenegger et al. (2009)), such manipulation during pregnancy is precluded in research on human fetuses. Finally, exogenous prenatal testosterone manipulation would be impractical for our study as it would take 50+ years to conduct the treatment and then observe financial risk taking later in life. Existing research on the effect of prenatal testosterone on risk taking has therefore generally relied on the 2D:4D finger ratio, i.e., the ratio of the index and ring finger lengths, a noisy biomarker of prenatal testosterone exposure, but has provided inconclusive evidence. Apicella et al. (2008) and Sapienza et al. (2009) find no statistically significant relation between 2D:4D ratio and financial risk taking. Coates et al. (2009) find that the 2D:4D ratio is related to the profitability of 44 professional traders at the London Stock Exchange, even though it is possible that this result reflects a cognitive ability effect, as opposed to a risk taking effect (e.g., Coates and Herbert (2008)).

The identification strategy in this study relies on an experiment that occurs naturally in some twin births, and is referred to as the "Twin Testosterone Transfer" (TTT) hypothesis. Testosterone transfer from male fetuses to neighboring fetuses via diffusion across fetal membranes was first confirmed in animals (e.g., vom Saal et al. (1980) and Hauser and Gandelman (1983)).[4] Several studies of humans have reported evidence consistent with the TTT hypothesis, both with respect to elevated testosterone levels as well as the masculinization of anatomical, physiological, and behavioral traits caused by the presence of a male fetus in the womb (e.g., Slutske et al. (2011)).[5] We refer to Tapp et al. (2011) for additional references related to the TTT hypothesis.

[4]Consistent with the TTT, researchers have documented that the intra-uterine position (IUP) is important (e.g., Ryan and Vandenbergh (2002)). That is, for animals for which multiple births are common (e.g., mice), female fetuses developing in-between two males in the womb show significantly more masculinized traits later in life.

[5]Some studies have reported a relation between prenatal testosterone and cognitive skills (e.g., Finegan et al. (1992)).

C Birth Weight

A large literature in economics documents that birth weight is predictive of long-term outcomes for adults. More specifically, differences in birth weight are related to differences in health capital as well as human capital much later in life. Birth weight is the most widely available and used proxy summary measure of the prenatal environment. Some researchers have emphasized that birth weight does not fully capture fetal origins effects, particularly because shocks in the first trimester of the pregnancy have been found to be extra critical while the fetus gains most of its weight in the third trimester (e.g., Almond et al. (2005)). As a result, birth weight may not be a sensitive measure of circumstances during the most critical period of the development of a human fetus. Nonetheless, birth weight remains an important measure in economic research on the effects of the prenatal environment because little substantial progress has been made towards identifying an alternative, superior summary measure.

Several studies have used cross-sectional data to show that low birth weight is related to long-term economic outcomes such as educational attainment, employment, and earnings (e.g., Currie and Hyson (1999)). One empirical challenge for these studies is that it is possible that there are no underlying causal relationship, as low birth weight may be correlated with many difficult-to-measure omitted socioeconomic and genetic variables. That is, many variables may be correlated with both negative birth outcomes and lower future performance. As a result, more recent studies have used within-sibling or within-twin variation to identify the effects of birth weight and confirmed the previous results, even though the economic magnitude of some of the documented effects is reduced (e.g., Behrman and Rosenzweig (2004) and Almond et al. (2005)).[6]

Birth weight may be directly or indirectly related to financial risk taking later in life. First, fetal programming may directly affect risk preferences. On the one hand, those with higher birth weight, i.e., better endowments at birth in a general sense, may be expected to take more risk. On the other hand, from an evolutionary perspective where maximizing the propagation of an individual's genes is of importance (e.g., Robson (2001a,b)), individuals with lower birth weight

[6] We also refer to Currie (2009), Almond and Currie (2011a), and Currie (2011) for a more detailed review of empirical evidence related to birth weight and health and human capital later in life.

may have been programmed to take more risk to compensate for lagging behind at birth (e.g., Hack et al. (2002)). Increased risk taking may be compensatory behavior through which those with a poor start (in the form of a lower birth weight) attempt to mitigate the effects by taking more risk (e.g., Metcalfe and Monaghan (2001)). Second, there may be an indirect effect on risk preferences because birth weight has been found to be related to socioeconomic outcomes, including education, IQ, and earnings (e.g., Behrman and Rosenzweig (2004) and Black et al. (2007)), which may correlate with individuals' financial risk taking propensities.

III Data

A Data Sources and Summary Statistics

Our data come from the Swedish Twin Registry (STR), the world's largest twin registry with very detailed information on over 50,000 different same- and opposite-sex twin pairs from birth cohorts dating back to the 19th century, and constitute a combination of registry and survey data. These data have been matched with detailed financial data from the Swedish Tax Authority and other individual data (e.g., family structure and education data) from Statistics Sweden for the period 1999-2007. For each individual, our data set contains the number of securities owned at the end of the year, and security-level data have been collected from Bloomberg, Datastream, Morningstar, SIX Telekurs, Standard & Poor's, and the Swedish Investment Fund Association. We select twins that in a given year are at least 18 years old and that have positive disposable income and net-worth.

For our tests of the twin testosterone hypothesis, we select all fraternal twins. For the majority of our analysis of the twin testosterone hypothesis we will compare fraternal female twins with male co-twins (i.e., those of opposite sex twin pairs) to fraternal female twins with female co-twins (i.e., those of same sex twin pairs), but to measure gender differences in risk taking,we also includes fraternal male twins. Our final sample of fraternal twins contains 9,410 female twins of opposite sex pairs, 9,093 female twins of same sex pairs, and 15,957 male fraternal

twins. For each of these twins, we have up to nine years of data. For this sample, Table 1 Panel A reports summary statistics of selected socioeconomic characteristics, pooled across all years, but separately for women and men and, in the case of women, for those with male and female co-twins. The average age of the female twins is 57 during our sample period, while the average age of men is 56, suggesting that on average the twins in our data set were born in the 1940s. Female twins with female co-twins and female twins with male co-twins differ with respect to the number of siblings they have (excluding their co-twin) and in their birth order. Same-sex female twins are slightly more likely to be first-borns than opposite-sex or same-sex male twins. In our empirical analysis, we always control for age differences as well as differences in family structure between females twins with same sex and opposite sex co-twins.

We provide a detailed definition of all variables in Appendix Table A.1. We note that several economic outcomes, for example, business ownership, disposable income, and net worth, exhibit a clear gender difference, while the difference between the treatment and the control group of female twins is typically small. Nevertheless, we do note that values for female of opposite-sex pairs different slightly from those of female of same-sex pair, tilted towards the corresponding values for men.

For our analysis of the effects of birth weight, we focus on identical twins in order to attribute within-pair differences to environmental as opposed to genetic differences. In addition, we only include those twin-years for which we have non-missing observations for both twins of a given twin pair. Our final sample contains 2,466 identical twins with a total of 17,510 twin-year observations between 1999 and 2007. Panel B again reports socioeconomic characteristics separately for the lowest and the highest birth quartiles, and for the entire sample. We note that for some socioeconomic variables, such age, birth order, number of siblings and years of education there are some clear differences between the lowest and the highest quartiles. In our empirical analysis, we include twin pair fixed effects to account for common environmental and genetic factors.

B Measuring Financial Risk Taking

To measure the tendency of individuals to take financial risk, we use several standard proxies that have been widely used in the extant literature on financial risk taking. Our first measure is the share of risky (equity) assets (*Risky Share*) out of all financial assets (see, e.g., Merton (1969) and Samuelson (1969)). Our second measure is the volatility of portfolio of risky financial assets. That is, conditional on stock market participation and using twelve monthly return observations, we calculate the annualized, value-weighted portfolio return volatility (*Portfolio Volatility*) for each twin and year. We also calculate the fraction of risky assets held in direct equities as opposed to likely well-diversified mutual funds (*Proportion Stocks*).

Table 2 Panel A reports summary statistics for our sample of fraternal twins used in the twin testosterone analysis. Across all three risk proxies, men take more risk than women, while at the same time female twins with male co-twins take more risk than female twins with female co-twins. Table 2 Panel A again documents a substantial gender gap, but also a difference between the treatment and control groups.

Table 2 Panel B reports summary statistics for the sample of identical twins. Compared to twins in the lowest birth quartiles, twins in the highest quartile hold more risky assets, invest (slightly) less in individual stocks, and experience lower volatility in their overall financial portfolio.

IV Effects of Prenatal Environment on Financial Risk Taking

A Identification and Empirical Approach

Our identification strategy is based on the "Twin Testosterone Transfer" (TTT) hypothesis: A female who shares the womb with a male co-twin (F_M) is exposed to a higher level of prenatal testosterone than a female who shares the womb with a female co-twin (F_F). As a result, we examine whether an F_M, i.e., an individual in the treatment group, takes more financial risk later in life compared to an F_F, i.e., an individual in the control group.

Using data on female fraternal twins, we use the following panel data model specification to estimate the treatment effect (I^{F_M}) of exogenously increased prenatal testosterone exposure on financial risk taking later in life:

$$y_{ijt} = \beta_0 + \beta_1 I_j^{F_M} + \beta_2 Age_{jt} + \beta_3 Family_j + \epsilon_{ijt}, \tag{1}$$

where y_{ijt} is a measure of financial risk taking of twin i of pair j in year t. I^{F_M} is one for a female with a male co-twin, and zero otherwise. We control for an individual's age (Age) by using age fixed effects for individuals below 35 years, between 35 and 49 years, between 50 and 65, and above 65 years. We also control for the family structure ($Family$) by including both the number of non-twin siblings and the birth order of the twin because if a same-sex female twin pair is born first some parents (at least older cohorts) may have a preference for more children to increase the probability of having a male child.

It is important to emphasize several aspects of this empirical approach. First, the comparison of F_M versus F_F twins is particularly appropriate from an econometric identification perspective because the gender of fraternal twins is determined exogenously relative to parental as well as the twin's own (genetic) characteristics. Second, our study focuses entirely on organizational, i.e., *prenatal*, effects of testosterone, which are predetermined with respect to financial risk taking later in life, as opposed to the effects of circulating testosterone in adults the level of which responds endogenously to environmental conditions.[7] Third, this study is not about treatment effects specific to a sample of twins. Every human fetus endogenously generates a different level of *in utero* testosterone. Using data on twins is simply our empirical approach to create exogenous cross-sectional differences in treatments to prenatal testosterone. That is, our approach is used to confront the challenges of causality, i.e., to examine whether the prenatal environment causes

[7]Men generally have higher levels of circulating testosterone than women during puberty and in adulthood. Circulating testosterone can be measured in saliva or blood and exogenously manipulated in experiments, and some studies have examined the effects of circulating testosterone on financial risk preferences, but the empirical evidence is so far inconclusive. More specifically, higher circulating testosterone has been found to increase risk taking in investment games in the lab in men (e.g., Apicella et al. (2008)) or only in women and not in men (e.g., Sapienza, Zingales, and Maestripieri (2009)).

differences among individuals later in life. Finally, because a female fetus on average generates significantly less *in utero* testosterone compared to males, we expect the strongest treatment effect for females who share the womb with a male co-twin. For a male who shared the womb with another male the average effect is more ambiguous as the male co-twin may generate either more or less testosterone.

Because all our measures of financial risk taking (y_{ijt}) have non-negative values, we estimate Equation (1) using a standard Tobit model with zero as the lower bound. All reported standard errors are double-clustered at the level of the individual as well as the year.

B Effects of Prenatal Testosterone Exposure

Table 3 reports our main results and shows that differences in the prenatal environment, specifically related to testosterone exposure, explains heterogeneity in financial risk taking propensity much later in life. For each of four measures of financial risk taking previously introduced, females who shared the womb with a male co-twin (F_M) take more financial risk than females who shared the womb with a female co-twin (F_F).

For the *Risky Share*, the estimated treatment effect, i.e., the point estimate on *Male Co-Twin* (F_M) is statistically significant at the 5% level. The economic magnitude of the effect is also sizable. An F_M twin allocates about 1.24 percentage points more of the financial assets to equities compared to an F_F twin. This corresponds to an increase of about 3% compared to the mean allocation (41.6%) of the control group of F_F twins.

Similarly, a treated female's portfolio exhibits a 3% higher volatility (*Portfolio Volatility*) and a 14% higher allocation to individual stocks relative to mutual funds (*Proportion Stocks*) in comparison with the control group (coefficients statistically significant, respectively, at the 1% and 5% level).

Finally, *Risky Share* decreases monotonically with age, consistent with a positive association between risk aversion and age (see, e.g, Barsky et al. (1997)), while *Proportion Stocks* increases until age 65, possibly reflecting increasing familiarity with individual stocks over the course of

the working life. While *Portfolio Volatility* is lower for those of retirement age (i.e., 66 years or older), no monotonic association with age exists until age 65. We also note that neither the number of siblings nor the the twin's birth order is significantly related to any of the measures of financial risk taking.

Our empirical evidence is overall consistent with the fetal origins hypothesis, and more specifically the TTT hypothesis, and suggests that the prenatal environment is important for an individual's financial risk taking propensity several decades later in life.

The twin testosterone hypothesis builds on the general understanding that the development of the male phenotype is dependent on the exposure to prenatal testosterone, while the female phenotype typically develops in the absence of testosterone exposure. Therefore, the increased level of testosterone due to a male co-twin is expected to lead to a (slight) masculinization of the female fetus. In animal studies, this masculinization has been shown to have anatomic as well as physiological consequences in addition to behavioral effects (see, e.g., vom Saal et al. (1980)). In untabulated results, we therefore apply our empirical model to a subset of slightly older female twins for whom we have data on their birth weight as well as adult height and weight. Differently from Glinianaia et al. (1998) who reports a significantly larger birth weight for females with male co-twins, we do not find statistically significant treatment effects with respect to either birth weight or height or weight later in life. But we do find a statistically significant increase in the Body Mass Index (BMI), which corresponds to in increase of about 1% relative to the mean BMI of the control group.

C Explaining the Gender Gap in Financial Risk Taking

A large scientific literature shows the importance of prenatal exposure to the testosterone hormone for the development of male characteristics in humans. As a result, it may be useful to compare the economic magnitude of the estimated treatment effect to the overall difference in risk taking between men and women, i.e., the "gender gap." More specifically, we ask to what extent the gender gap in financial risk taking may be explained by differences in prenatal testosterone

exposure. In Table 4, we therefore add fraternal male twins to our sample and re-estimate Equation (1), also adding a *Male* indicator variable.

First, we confirm the existence of an economically and statistically significant gender gap also in our sample with respect to all four risk-taking measures. For example, the estimated coefficient on the *Male* indicator is 3.30 percentage points, corresponding to men's *Risky Share* being about 8% higher than that of a female. This result is consistent with previous studies that have documented a significant gender gap in financial risk taking (e.g., Croson and Gneezy (2009) and Sundén and Surette (1998)).

Second, and most important given the goals of this paper, we compare the economic magnitude of the estimated treatment effect of prenatal testosterone exposure to the estimated gender gap. For *Risky Share*, we find that the treatment effect is about 39% (= 1.273/3.299) of the gender gap, i.e., a female who shared the womb with a male on average has a 39% smaller gender gap in financial risk taking compared to a female in the control group. For the other two measures, we find smaller but comparable effects: 10% for *Portfolio Volatility*, 11% for *Proportion Stocks*.

To the best of our knowledge, no data exist on the magnitude of the increase in prenatal testosterone exposure due to a male co-twin for humans. But animal studies conducted on mice suggest that the increase in blood testosterone levels in female fetuses due to testosterone transfer from male fetuses corresponds to about 10% of the difference in testosterone levels between male and female fetuses (vom Saal et al. (1980)). Assuming that these studies have some relevance for humans and that the relationship between testosterone levels and risk taking is approximately linear, the size of the treatment effect we document relative to the overall gender gap would appear plausible.[8]

Our results suggest that differential pre-birth exposure to the testosterone hormone can explain a significant proportion of the gender gap in financial risk taking.[9] This evidence is

[8] In untabulated results, we extend the gender gap analysis to *BMI* and find that the treatment effect of a male co-twin corresponds to about 12% of the gender gap in *BMI*.

[9] While gender differences in general reflect biological as well as social factors, Swedish data may be relatively less affected by gender identity effects, given the strong emphasis on gender equality in Sweden (e.g., Guiso, Monte, Sapienza, and Zingales (2008)).

consistent with a masculinazing effect of prenatal testosterone exposure also in a financial domain.

D Social Interaction Effects

We examine the possibility that our results are attributable to intra-twin pair social interactions. A co-twin may be a particularly low-cost source of casual financial advice. For example, female twins may seek financial advice from their male co-twins because of a cultural norm that "men make the financial decisions." Because men on average take more financial risk compared to women, male twins may provide advice to their female co-twins that involve more risk taking compared to any advice provided by female co-twins. If social interaction between twins in adulthood is the cause of the reported treatment effect, we expect the effects to be weaker among subsets of twins who as adults do not interact with each other a lot or at all.

In Table 5, we re-estimate Equation (1) across all three measures of risk taking for several different subsets of twins that are less likely to interact around the time we observe their financial portfolios. In Panel A, we consider only the subsample of those who are below the median in the intra-twin pair communication and contact frequency distribution, which is based on surveys conducted by the Swedish Twin Registry. In Panel B, we use travel time as a measure of geographic distance between the twins, although it may be a less direct measures of lack of communication in today's society.[10] Finally, in Panel C, we examine twin pairs whose portfolios overlap by less than 50% because twins within a pair may choose the same securities, e.g., after discussing individual stocks, and as a result their financial risk taking will also be similar. With the exception of *Proportion Stocks*, we find that the point estimates of the treatment effects are very similar or larger than those reported previously and that the statistical significance is also comparable, accounting for a smaller sample than the previous one.

The evidence in Table 5 shows that intra-twin pair social interactions in adulthood can not easily explain our results. That is, we observe elevated financial risk taking propensities even among treated females who do not interact with their male co-twins a lot or at all.

[10]The results are similar if we use different regions or cities to measure geographic distance (untabulated).

E Effects of Male Sibling

We also examine the possibility that our results are attributable to a more general male sibling effect. For example, a female with a male co-twin may be exposed to relatively more aggressive or risk taking male behaviors within the family and when growing up, increasing the probability that the individual chooses to adopt such behaviors by way of imitation, which later in life may reflect themselves as increased financial risk taking. Differently from an effect of the fetal origins hypothesis, such an effect should not be limited to male co-twins, but may occur with male siblings in general.

In Table 6 Panel A, we re-estimate Equation (1), also adding an indicator variable for the presence of, respectively, male and female non-twin siblings in the family. First, we find in the first column that controlling for the presence of non-twin male siblings does not reduce the previously reported economic magnitude or statistical significance of the treatment effects. For example, the point estimate is 1.25 (compared to 1.24 in Table 3), and remains statistically significant at the 5%-level. We also find that the difference between "Non-Twin Male Sibling" and "Non-Twin Female Sibling", i.e., the effect of the presence of a male non-twin sibling relative to the effect of the presence of a female non-twin sibling, is not statistically significant for any of the financial risk taking measures (this difference is actually negative for *Portfolio Volatility* and *Proportion Stocks*). Last, we also compare the male co-twin treatment effect to the male sibling effect. With the exception of *Risky Share*, we find that the male co-twin treatment effect is substantially larger than the male sibling effect, and, in the case of *Portfolio Volatility* and *Proportion Stocks*, this difference is also statistically significant.

In Table 6 Panel B, we examine the financial risk taking propensity of the non-twin female siblings in our sample. We do not find that the presence of any male (twin or non-twin) sibling significantly affects the financial risk taking propensity. We also find that the difference between "Any Male Sibling" and "Any Female Sibling", i.e., the effect of the presence of a any male sibling relative to the effect of the presence of any female sibling, is always negative but not statistically significant.

In conclusion, the evidence in Table 6 suggests that the effect of a male co-twin is not merely reflecting a more general male sibling effect.

V Effects of Birth Weight on Financial Risk Taking

A Identification and Empirical Approach

In economic research, birth weight is the most widely available and used summary measure of the prenatal environment. In this section, we analyze whether birth weight is predictive of financial risk taking. Using data on identical twins, we use the following model specification to estimate the effect of birth weight on financial risk taking later in life:

$$y_{ijt} = \delta_0 + \delta_1 BW_{ij} + a_j + c_j + \omega_{ijt}, \tag{2}$$

where y_{ijt} is a measure of financial risk taking of twin i of pair j in year t. BW_{ij} is an individual's birth weight. a_j and c_j are, respectively, unobservable genetic endowments and environmental effects common to each twin pair, such as, e.g., the mother's health during the pregnancy or the parents' socioeconomic status. Because birth weight may be correlated with these genetic endowments and common environmental effects, we include twin pair fixed effects in order to isolate the individual-specific effects of the prenatal environment, such as better or worse nutritional intake of one twin relative to the other twin in the same pair. That is, by simultaneously accounting for a_j and c_j, twin pair fixed effects result in an unbiased estimate of δ_1 (e.g., Behrman and Rosenzweig (2004) and Black et al. (2007)). To understand any bias attributable to unobservable genetic and common environmental variation, we also report estimates without twin pair fixed effects.

We estimate Equation (2) using standard ordinary least squares. All reported standard errors are double-clustered at the level of the individual as well as the year.

B Effects of Birth Weight

The effect of birth weight on risk taking is unclear *ex ante*. On the one hand, the existing literature shows that higher birth weight leads to higher education, higher earnings, and better health (e.g., Behrman and Rosenzweig (2004) and Black et al. (2007)). Higher economic resources and human capital could allow those with higher birth weight to take more risk in financial markets. On the other hand, Hack et al. (2002) hypothesize that lower birth weight might lead to more risk taking later in life. Individuals might engage in compensatory behaviors and take more risk to mitigate the effects of a poor start (in term of lower birth weight).[11]

In Table 7, we report the effect of birth weight, measured using the natural logarithm (*Birth Weight (ln)*), on our measures of financial risk taking. In columns 1, 3, and 5, we report results without twin pair fixed effect, while we also include twin pair fixed effects in columns 2, 4 and 6.[12] The inclusion of twin pair fixed effects significantly increases *R-squared*. This result is not surprising and reflects the significant commonality between identical twins. The relative importance of genetic and common environmental effects is consistent with the recent studies by, e.g., Barnea et al. (2010) and Cesarini et al. (2010). We find that birth weight has a positive effect on *Risky Share*. The estimated effect is larger, but the statistical significance is somewhat weaker, in the second column where we also include twin pair fixed effects. The economic magnitude of the estimated effect is sizable: in the fixed effects model, a one standard deviation increase in *Birth Weight (ln)* increases the *Risky Share* by about 1.46 percentage points, or about 3.3% of the mean allocation (45.0%) in the entire sample. We also find that birth weight has a negative effect on *Portfolio Volatility* and *Proportion Stocks*. Both effects are highly statistically significant after we account for twin pair fixed effects (p-value = 0.000 and p-value = 0.011). The estimates in column 4 imply that a one standard deviation increase in *Birth Weight (ln)* decreases the *Portfolio Volatility* by about 4.6% relative to the sample mean (15.3%). From estimates in Column 6, we find that an analogous change in birth weight generates an even larger effect and

[11] Metcalfe and Monaghan (2001) provides an overview on compensatory behaviors in response to low birth weight.

[12] In the model specification without twin pair fixed effects, we also control for gender as we use both male and female pairs of identical twins.

reduces the *Proportion Stocks* by about 10.4% of the mean direct equity holdings (28.6%) in the entire sample.

The evidence in Table 7 reveals that lower birth weight individuals are less likely to invest in risky (equity) assets, but that, conditional on holding risky assets, lower birth weight individuals choose more volatile portfolio that contain a higher portion of individual stocks relative to mutual funds. These findings are consistent with the interpretation that adverse prenatal conditions, experienced by an individual in the womb and orthogonal to genetic endowments, prevent or limit investments in risky assets later in life. Conditional on holding any risky assets, though, those with a lower birth weight select significantly more volatile assets, consistent with compensatory behaviors in response to unfavorable starting conditions.

The birth weight results raise the question whether the previously reported results related to prenatal testosterone exposure may be explained by differences in birth weight. We have already commented on how in our sample the difference in birth weight between female twins with a male co-twin and those with a female co-twin is positive, but not statistically significant. To formally test for this possibility, we have re-estimated Equation (1), adding *Birth Weight (ln)* to the model. In untabulated results, we find that our estimates of the effect of a *Male Co-Twin* do not change after we control for birth weight. That is, the effect of prenatal testosterone exposure is orthogonal to general prenatal conditions and circumstances as captured by birth weight.

C Direct and Indirect Effects

Birth weight may be directly or indirectly related to financial risk taking later in life. First, fetal programming may directly affect an individual's risk preferences. Second, parents and the general environment may reinforce differences between individuals by providing more economic resources to the stronger child (e.g., Hsin (2012)). In addition, birth weight might affect risk taking later in life through its effect on other economic outcomes, such as education and earnings (e.g., Behrman and Rosenzweig (2004) and Black et al. (2007)) that have been shown to correlate with investment decisions. We shed some light on the source of the birth weight effects.

In a first step, we examine the effects of birth weight on several potential determinants of risk taking established in previous studies: *Net Worth* (e.g., Brunnermier and Nagel (2008)), *Labor Income Volatility* and *Business Owner* (e.g. Heaton and Lucas (2000)), cognitive abilities proxied for by *Years of Education* (e.g., Grinblatt et al. (2011)), and *Poor Health* (e.g., Rosen and Wu (2004)). Table 8 shows that birth weight is significantly related to several of these individual characteristics. In particular, higher birth weight is predictive of higher net worth, less volatile labor income, an increase in years of education (confirming the results in Black et al. (2007)), and a reduction in the risk of experiencing poor health as an adult.

In a second step, we re-estimate Equation (2), including the determinants of risk taking through which birth weight could operate. We also include several additional socioeconomic characteristics commonly used in the empirical portfolio choice literature.[13] Table 9 reveals that the effects of birth weight on our measures of financial risk-taking remain largely unchanged. For *Risky Share* and controlling for twin pair fixed effects, the effect of *Birth Weight (ln)* is 5.92 (compared to 5.96 in Table 7), and remains statistically significant at the 10%-level (p-value = 0.078). For *Portfolio Volatility* and *Proportion Stocks* the absolute size of the birth weight effect even increases. That is, the inclusion of possible channels through which birth weight could affect financial decisions indirectly, does not seem to significantly alter the direct effect of birth weight on financial risk taking.

In conclusion, the evidence in Tables 8 and 9 suggests that the effects of birth weight on financial risk taking are not easily explained by known factors that affect financial risk taking such as education or net worth. The prenatal environmental causes of lower birth weight seem to have persistent and direct effects on financial decisions much later in life.

[13]Specifically, we include, but do not explicitly report: *Single*, *Divorced*, *Number of Children*, *Retired*, *Disposable Income (ln)*.

VI Conclusion

A large and growing literature in economics has recently documented the importance of the prenatal, i.e., pre-birth, environment for economic outcomes much later in life (e.g., Almond and Currie (2011b) and Currie (2011)). This scientific evidence has even made its way into mainstream media, for example Paul's (2011) book "Origins: How the Nine Months Before Birth Shape the Rest of Our Lives" and an article in Time magazine summarizing the evidence: The "quality of nutrition [we] received in the womb; the pollutants, drugs and infections [we] were exposed to during gestation [...] shape our susceptibility to disease, our appetite and metabolism, our intelligence and temperament." In this paper we have asked whether the prenatal environment also affects outcomes in the domain of investment decisions. More specifically, do differences in the prenatal environment explain heterogeneity in individuals' financial risk taking propensities later in life?

We find that differences in an individual's prenatal environment explain heterogeneity in financial risk taking propensities much later in life. An exogenous increase in exposure to prenatal testosterone is related to elevated risk taking in financial market in adulthood. We also examine birth weight, the most widely used summary measure of the prenatal environment. Even after controlling for identical twin pair fixed effects, we find that those with lower birth weight, i.e., those that experience more adverse prenatal conditions in a general sense, are less likely to invest in risky financial assets, and conditional on holding risky financial assets prefer more volatile equity portfolios with a relatively larger allocation to individual stocks rather than mutual funds.

This study is one of the first attempts to incorporate into finance research the fetal origins hypothesis, a growing literature in economic research which has documented that the prenatal environment programs a fetus in the womb to have persistent behavioral characteristics. Our results contributes to our understanding of how (prenatal) environmental conditions can shape individuals' behavior in financial markets.

Future research may focus on how different prenatal environmental factors, other than testosterone exposure or birth weight, affect financial decisions. Several economists have also

emphasized the importance of the *postnatal* early life environment for outcomes much later in life (e.g., Garces, Thomas, and Currie (2002) and Cunha and Heckman (2010)), which provides another direction for related research.

References

Almond, D., 2006. Is the 1918 influenza pandemic over? Long-term effects of in utero influenza exposure in the post-1940 U.S. population. Journal of Political Economy 114 (4), 672–712.

Almond, D., Chay, K. Y., Lee, D. S., 2005. The costs of low birth weight. Quarterly Journal of Economics 120 (3), 1031–1083.

Almond, D., Currie, J., 2011a. Human capital development before age five. Handbook of Labor Economics 4, 1315–1486.

Almond, D., Currie, J., 2011b. Killing me softly: The fetal origins hypothesis. Journal of Economic Perspectives, 153–172.

Almond, D., Edlund, L., Palme, M., 2009. Chernobyl's subclinical legacy: Prenatal exposure to radioactive fallout and school outcomes in Sweden. Quarterly Journal of Economics 124 (4), 1729–1772.

Almond, D., Mazumder, B., 2011. Health capital and the prenatal environment: The effect of Ramadan observance during pregnancy. American Economic Journal: Applied Economics 3 (4), 56.

Apicella, C. L., Dreber, A., Campbell, B., Gray, P. B., Hoffman, M., Little, A. C., 2008. Testosterone and financial risk preferences. Evolution and Human Behavior 29 (6), 384–390.

Banerjee, A., Duflo, E., Postel-Vinay, G., Watts, T., 2010. Long-run health impacts of income shocks: Wine and Phylloxera in nineteenth-century France. Review of Economics and Statistics 92 (4), 714–728.

Barker, D. J., 1990. The fetal and infant origins of adult disease. British Medical Journal 301, 1111.

Barker, D. J., 1995. Fetal origins of coronary heart disease. British Medical Journal 311, 171–174.

Barker, D. J. P., Robinson, R. J., 1992. Fetal and infant origins of adult disease. British Medical Journal, London.

Barnea, A., Cronqvist, H., Siegel, S., 2010. Nature or nurture: What determines investor behavior? Journal of Financial Economics 98, 583–604.

Baron-Cohen, S., Knickmeyer, R. C., Belmonte, M. K., 2005. Sex differences in the brain: implications for explaining autism. Science 310 (5749), 819–823.

Baron-Cohen, S., Lutchmaya, S., Knickmeyer, R., 2004. Prenatal testosterone in mind: Amniotic fluid studies. MIT Press.

Barsky, R. B., Juster, F. T., Kimball, M. S., Shapiro, M. D., 1997. Preference parameters and behavioral heterogeneity: An experimental approach in the health and retirement study. Quarterly Journal of Economics 112, 537–579.

Behrman, J. R., Rosenzweig, M. R., 2004. Returns to birthweight. Review of Economics and Statistics 86 (2), 586–601.

Black, S. E., Devereux, P. J., Salvanes, K. G., 2007. From the cradle to the labor market? The effect of birth weight on adult outcomes. Quarterly Journal of Economics 122 (1), 409–439.

Brunnermier, M. K., Nagel, S., 2008. Do wealth fluctuations generate time-varying risk aversion? Micro-evidence on individuals' asset allocation. American Economic Review 98, 713–736.

Cesarini, D., Dawes, C. T., Johannesson, M., Lichtenstein, P., Wallace, B., 2009. Genetic variation in preferences for giving and risk taking. Quarterly Journal of Economics 124, 809–842.

Cesarini, D., Johannesson, M., Lichtenstein, P., Sandewall, O., Wallace, B., 2010. Genetic variation in financial decision making. Journal of Finance 65, 1725–1754.

Chetty, R., Friedman, J. N., Hilger, N., Saez, E., Schanzenbach, D. W., Yagan, D., 2011. How does your kindergarten classroom affect your earnings? Evidence from Project STAR. Quarterly Journal of Economics 126 (4), 1593–1660.

Coates, J. M., Gurnell, M., Rustichini, A., 2009. Second-to-fourth digit ratio predicts success among high-frequency financial traders. Proceedings of the National Academy of Sciences 106 (2), 623–628.

Coates, J. M., Herbert, J., 2008. Endogenous steroids and financial risk taking on a London trading floor. Proceedings of the National Academy of Sciences 105 (16), 6167–6172.

Cronqvist, H., Siegel, S., Yu, F., 2014. Value versus growth investing: Why do different investors have different styles? Working Paper, China Europe International Business School and University of Washington.

Croson, R., Gneezy, U., 2009. Gender differences in preferences. Journal of Economic Literature 47 (2), 448–474.

Cunha, F., Heckman, J., 2010. Investing in our young people. In: Reynolds, A. (Ed.), Cost Effective Early Childhood Programs in the First Decade: A Human Capital Integration. Cambridge University Press.

Currie, J., 2009. Healthy, wealthy, and wise: Socioeconomic status, poor health in childhood, and human capital development. Journal of Economic Literature 47 (1), 87–122.

Currie, J., 2011. Inequality at birth: Some causes and consequences. American Economic Review 101 (3), 2–22.

Currie, J., Hyson, R., 1999. Is the impact of health shocks cushioned by socioeconomic status? The case of low birthweight. American Economic Review 89 (2), 245–250.

Eisenegger, C., Naef, M., Snozzi, R., Heinrichs, M., Fehr, E., 2009. Prejudice and truth about the effect of testosterone on human bargaining behaviour. Nature 463 (7279), 356–359.

Finegan, J.-A. K., Niccols, G. A., Sitarenios, G., 1992. Relations between prenatal testosterone levels and cognitive abilities at 4 years. Developmental Psychology 28, 1075–1089.

Garces, E., Thomas, D., Currie, J., 2002. Longer-term effects of Head Start. American Economic Review 92 (4), 999–1012.

Glinianaia, S. V., Magnus, P., Harris, J. R., Tambs, K., 1998. Is there a consequence for fetal growth of having an unlike-sexed cohabitant in utero? International Journal of Epidemiology 27 (4), 657–659.

Grinblatt, M., Keloharju, M., Linnainmaa, J., 2011. IQ and stock market participation. Journal of Finance 66 (6), 2121–2164.

Guiso, L., Monte, F., Sapienza, P., Zingales, L., 2008. Culture, gender, and math. Science 320, 1164–1165.

Hack, M., Flannery, D. J., Schluchter, M., Cartar, L., Borawski, E., Klein, N., 2002. Outcomes in young adulthood for very-low-birth-weight infants. New England Journal of Medicine 346 (3), 149–157, pMID: 11796848.

Hauser, H., Gandelman, R., 1983. Contiguity to males in utero affects avoidance responding in adult female mice. Science 220 (4595), 437–438.

Heaton, J., Lucas, D., 2000. Portfolio choice and asset prices: The importance of entrepreneurial risk. Journal of Finance 55, 1163–1198.

Hsin, A., 2012. Is biology destiny? birth weight and differential parental treatment. Demography 49 (4), 1385–1405.

Knüpfer, S., Rantapuska, E., Sarvimäki, M., 2014. Labor Market Experiences and Portfolio Choice: Evidence from the Finnish Great Depression. Working paper, London Business School.

Lombardo, M. V., Ashwin, E., Auyeung, B., Chakrabarti, B., Lai, M.-C., Taylor, K., Hackett, G., Bullmore, E. T., Baron-Cohen, S., 2012a. Fetal programming effects of testosterone on the reward system and behavioral approach tendencies in humans. Biological Psychiatry 72 (10), 839–847.

Lombardo, M. V., Ashwin, E., Auyeung, B., Chakrabarti, B., Taylor, K., Hackett, G., Bullmore, E. T., Baron-Cohen, S., 2012b. Fetal testosterone influences sexually dimorphic gray matter in the human brain. The Journal of neuroscience 32 (2), 674–680.

Malmendier, U., Nagel, S., 2011. Depression babies: Do macroeconomic experiences affect risk taking? Quarterly Journal of Economics 126 (1), 373–416.

Malmendier, U., Nagel, S., 2014. Learning from inflation experiences. Working Paper, UC Berkley.

Merton, R. C., 1969. Lifetime portfolio selection under uncertainty: The continuous-time case. Review of Economics and Statistic 51, 247–257.

Metcalfe, N. B., Monaghan, P., 2001. Compensation for a bad start: grow now, pay later? Trends in Ecology & Evolution 16 (5), 254 – 260.

Paul, A. M., 2011. Origins: How the Nine Months Before Birth Shape the Rest of Our Lives. Free Press.

Petronis, A., 2010. Epigenetics as a unifying principle in the aetiology of complex traits and diseases. Nature 465 (7299), 721–727.

Ravelli, G.-P., Stein, Z. A., Susser, M. W., 1976. Obesity in young men after famine exposure in utero and early infancy. New England Journal of Medicine 295 (7), 349–353.

Robson, A. J., 2001a. The biological basis of economic behavior. Journal of Economic Literature 29, 11–33.

Robson, A. J., 2001b. Why would nature give individuals utility functions? Journal of Political Economy 109, 900–914.

Rosen, H. S., Wu, S., 2004. Portfolio choice and health status. Journal of Financial Economics 72, 457–484.

Ryan, B. C., Vandenbergh, J. G., 2002. Intrauterine position effects. Neuroscience & Biobehavioral Reviews 26 (6), 665–678.

Samuelson, P. A., 1969. Lifetime portfolio selection by dynamic stochastic programming. Review of Economics and Statistics 51, 239–246.

Sanders, N. J., 2012. What doesnt kill you makes you weaker prenatal pollution exposure and educational outcomes. Journal of Human Resources 47 (3), 826–850.

Sapienza, P., Zingales, L., Maestripieri, D., 2009. Gender differences in financial risk aversion and career choices are affected by testosterone. Proceedings of the National Academy of Sciences 106 (36), 15268–15273.

Slutske, W. S., Bascom, E. N., Meier, M. H., Medland, S. E., Martin, N. G., 2011. Sensation seeking in females from opposite-versus same-sex twin pairs: Hormone transfer or sibling imitation? Behavior Genetics 41 (4), 533–542.

Stein, Z., Susser, M., Saenger, G., 1975. Famine and human development: the Dutch hunger winter of 1944-1945. Oxford Medical Publications.

Sundén, A. E., Surette, B. J., 1998. Gender differences in the allocation of assets in retirement savings plans. American Economic Review 88, 207–211.

Tapp, A. L., Maybery, M. T., Whitehouse, A. J., 2011. Evaluating the twin testosterone transfer hypothesis: A review of the empirical evidence. Hormones and Behavior 60 (5), 713–722.

van de Beek, C., Thijssen, J. H., Cohen-Kettenis, P. T., van Goozen, S. H., Buitelaar, J. K., 2004. Relationships between sex hormones assessed in amniotic fluid, and maternal and umbilical cord serum: What is the best source of information to investigate the effects of fetal hormonal exposure? Hormones and Behavior 46 (5), 663–669.

vom Saal, F. S., Bronson, F., et al., 1980. Sexual characteristics of adult female mice are correlated with their blood testosterone levels during prenatal development. Science 208 (4444), 597–599.

Zak, P. J., Kurzban, R., Ahmadi, S., Swerdloff, R. S., Park, J., Efremidze, L., Redwine, K., Morgan, K., Matzner, W., 2009. Testosterone administration decreases generosity in the ultimatum game. PLoS ONE 4 (12), 8330.

Table 1
Summary Statistics: Socioeconomic Characteristics

Panel A: Twin Testosterone Transfer Sample (Fraternal Twins)

	Female with Female Co-Twin (F_F) (N = 61,099)		Female with Male Co-Twin (F_M) (N = 63,042)		Male (N = 106,975)	
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
Age	57.399	15.770	56.724	13.862	55.941	14.129
Birth Order	1.599	1.275	1.679	1.430	1.688	1.422
Number of Siblings	1.150	1.385	1.274	1.518	1.309	1.500
Net Worth (ln)	12.619	1.520	12.664	1.518	13.001	1.459
Volatility of Labor Income	14.089	13.646	13.603	13.075	13.473	13.596
Business Owner	0.014	0.119	0.016	0.126	0.034	0.181
Years of Education	9.368	4.957	10.070	4.451	9.741	4.392
Missing Education Data	0.162	0.369	0.103	0.304	0.109	0.311
Poor Health	0.174	0.379	0.204	0.403	0.126	0.332
Single	0.210	0.407	0.207	0.405	0.267	0.442
Divorced	0.120	0.324	0.124	0.330	0.103	0.304
Number of Children	0.586	1.034	0.592	1.030	0.662	1.076
Retired	0.408	0.491	0.370	0.483	0.354	0.478
Disposable Income (ln)	12.268	0.596	12.277	0.586	12.475	0.692

Panel B: Birth Weight Sample (Identical Twins)

	Lowest Birth Weight Quartile (N = 5,140)		Highest Birth Weight Quartile (N = 2,581)		Entire Sample (N = 17,510)	
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
Birth Weight (g)	1,759.685	239.742	3,308.627	210.667	2,413.858	566.986
Birth Weight (ln)	7.462	0.151	8.102	0.062	7.760	0.246
Age	59.354	9.692	56.727	8.800	57.854	9.274
Birth Order	1.462	0.772	2.071	1.133	1.688	0.964
Number of Siblings	1.109	1.345	1.506	1.379	1.252	1.340
Net worth (ln)	13.019	1.405	13.209	1.416	13.122	1.390
Volatility of Labor Income	0.121	0.111	0.132	0.114	0.119	0.109
Business Owner	0.024	0.152	0.038	0.190	0.024	0.154
Years of Education	10.311	4.427	11.070	4.203	10.844	4.279
Missing Education Data	0.094	0.292	0.067	0.251	0.076	0.265
Poor Health	0.175	0.380	0.199	0.399	0.175	0.380
Single	0.117	0.322	0.106	0.308	0.119	0.323
Divorced	0.160	0.366	0.149	0.356	0.155	0.362
Number of Children	0.495	0.895	0.621	0.947	0.579	0.957
Retired	0.357	0.479	0.272	0.445	0.312	0.463
Disposable Income (ln)	12.367	0.633	12.339	0.712	12.357	0.629

Table 1 Panel A provides summary statistics for several socioeconomic characteristics for the fraternal twins used in the Twin Testosterone Transfer analyses, separately for women with a female co-twin (F_F), women with a male co-twin (F_M), and for men. Table 1 Panel B provides summary statistics for the identical twins used in the Birth Weight analyses, separately for the lowest birth weight quartile, the highest quartile, and the entire sample. All variables are defined in detail in Appendix Table A1. N provides the total number of twin-year observations.

Table 2
Summary Statistics: Financial Risk Taking

Panel A: Twin Testosterone Transfer Sample (Fraternal Twins)

	Female with Female Co-Twin (F_F)			Female with Male Co-Twin (F_M)			Male		
	Mean	Std. Dev.	*N*	Mean	Std. Dev.	*N*	Mean	Std. Dev.	*N*
Risky Share	41.555	38.340	61,099	42.686	38.537	63,042	43.901	38.298	106,975
Portfolio Volatility	14.251	11.471	26,690	14.706	11.911	28,203	18.296	14.179	49,748
Proportion Stocks	21.969	35.598	44,658	23.579	36.491	46,864	35.784	41.183	83,231

Panel B: Birth Weight Sample (Identical Twins)

	Lowest Birth Weight Quartile			Highest Birth Weight Quartile			Entire Sample		
	Mean	Std. Dev.	*N*	Mean	Std. Dev.	*N*	Mean	Std. Dev.	*N*
Risky Share	43.269	37.478	5,140	47.510	38.773	2,581	44.959	37.494	17,510
Portfolio Volatility	14.925	12.356	1,329	14.703	11.434	763	15.263	12.323	4,926
Proportion Stocks	29.965	39.115	3,368	29.331	39.787	1,774	28.636	38.408	11,744

Table 2 Panel A reports summary statistics for measures of financial risk taking for the fraternal twins used in the Twin Testosterone Transfer analyses, separately for women with a female co-twin (F_F), women with a male co-twin (F_M), and for men. Table 2 Panel B provides similar measures for the identical twins used in the Birth Weight analyses, separately for the lowest birth weight quartile, the highest quartile, and the entire sample. All variables are defined in detail in Appendix Table A1. *N* provides the total number of twin-year observations.

Table 3
The Effect of Having a Male Co-twin

	Risky Share	Portfolio Volatility	Proportion Stocks
Male Co-Twin (F_M)	1.242**	0.386***	2.984**
	(0.046)	(0.010)	(0.016)
Age less than 35	21.004***	2.790***	-13.702***
	(0.000)	(0.001)	(0.000)
Age less than 50	16.332***	3.563***	-2.715
	(0.000)	(0.000)	(0.350)
Age less than 66	12.483***	2.284***	3.732*
	(0.000)	(0.000)	(0.087)
Number of Sibling	-0.743	0.061	-0.432
	(0.188)	(0.651)	(0.526)
Birth Order	0.400	-0.150	-0.740
	(0.330)	(0.156)	(0.294)
Intercept	23.761***	12.496***	-7.053***
	(0.000)	(0.000)	(0.004)
N	124,141	54,893	91,522

Table 3 reports results from Tobit regressions of annual measures of financial risk taking of female fraternal twins between 1999 and 2007 onto an indicator variable for women with a male co-twin ("Male Co-Twin") and additional controls. For each model, we report the coefficient estimates as well as the corresponding *p*-values. *p*-values are based on double-clustered standard errors, robust for correlation across years within same individuals and across individuals within the same year. All variables are defined in detail in Appendix Table A1. *N* provides the number of observations used in each estimation. Levels of significance are denoted as follows: * if $p<0.10$; ** if $p<0.05$; *** if $p<0.01$.

Table 4
Having a Male Co-twin and the Gender Gap

	Risky Share	Portfolio Volatility	Proportion
Male Co-Twin (F_M)	1.273**	0.380**	2.931**
	(0.034)	(0.012)	(0.013)
Male	3.299***	3.923***	26.190***
	(0.000)	(0.000)	(0.000)
Age less than 35	19.378***	3.005***	-12.190***
	(0.000)	(0.001)	(0.000)
Age less than 50	15.654***	4.151***	0.336
	(0.000)	(0.000)	(0.885)
Age less than 66	11.477***	2.604***	3.451**
	(0.000)	(0.000)	(0.039)
Number of Sibling	-0.775	-0.057	-0.345
	(0.123)	(0.655)	(0.445)
Birth Order	0.327	-0.012	-0.443
	(0.330)	(0.832)	(0.332)
Intercept	24.828***	12.139***	-6.574***
	(0.000)	(0.000)	(0.000)
N	231,116	104,641	174,753

Table 4 reports results from Tobit regressions of annual measures of financial risk taking of female and male fraternal twins between 1999 and 2007 onto an indicator variable for women with a male co-twin ("Male Co-Twin"), an indicator variable for men ("Male"), and additional controls. For each model, we report the coefficient estimates as well as the corresponding *p*-values. *p*-values are based on double-clustered standard errors, robust for correlation across years within same individuals and across individuals within the same year. All variables are defined in detail in Appendix Table A1. *N* provides the number of observations used in each estimation. Levels of significance are denoted as follows: * if $p<0.10$; ** if $p<0.05$; *** if $p<0.01$.

Table 5
Interactions Later in Life

Panel A: Twins with Fewer Contacts

	Risky Share	Portfolio Volatility	Proportion Stocks
Male Co-Twin (F_M)	2.591**	0.400	0.996
	(0.017)	(0.133)	(0.640)
Additional Controls	Yes	Yes	Yes
N	45,733	20,628	34,552

Panel B: Twins with Longer Travel Distance

	Risky Share	Portfolio Volatility	Proportion Stocks
Male Co-Twin (F_M)	1.553*	0.537**	2.660
	(0.063)	(0.011)	(0.113)
Additional Controls	Yes	Yes	Yes
N	62,733	28,075	46,941

Panel C: Twins with Low Portfolio Overlap

	Risky Share	Portfolio Volatility	Proportion Stocks
Male Co-Twin (F_M)	1.396**	0.324*	1.242
	(0.017)	(0.062)	(0.380)
Additional Controls	Yes	Yes	Yes
N	43,774	27,048	42,955

Table 5 reports results from Tobit regressions of annual measures of financial risk taking of female fraternal twins between 1999 and 2007 onto an indicator variable for women with a male co-twin ("Male Co-Twin"). Additional controls are the same control variables used in Table 3. In Panel A, we include only female twins that have below median contacts. In Panel B, we include only female twins with above median travel distance between their primary residences. In Panel C, we include only female twins with less than 50 percent portfolio overlap. For each model, we report the coefficient estimates as well as the corresponding *p*-values. *p*-values are based on double-clustered standard errors, robust for correlation across years within same individuals and across individuals within the same year. All variables are defined in detail in Appendix Table A1. *N* provides the number of observations used in each estimation. Levels of significance are denoted as follows: * if $p<0.10$; ** if $p<0.05$; *** if $p<0.01$.

Table 6
The Effect of Male Siblings

Panel A - Twin Sample

	Risky Share	Portfolio Volatility	Proportion Stocks
Male Co-Twin (F_M) **[A]**	1.253**	0.388***	3.008**
	(0.037)	(0.010)	(0.013)
Non-twin Male Sibling **[B]**	2.434*	0.045	-1.407
	(0.081)	(0.857)	(0.441)
Non-twin Female Sibling **[C]**	1.000	0.243	1.695
	(0.357)	(0.379)	(0.349)
Additional Controls	Yes	Yes	Yes
[B] - [C]	1.434	-0.198	-3.101
	(0.186)	(0.397)	(0.103)
[A] - {[B] - [C]}	-0.181	0.586**	6.109***
	(0.885)	(0.041)	(0.008)
N	124,141	54,893	91,522

Panel B - Siblings Sample

	Risky Share	Portfolio Volatility	Proportion Stocks
Any Male Sibling **[A]**	0.330	-0.177	-1.553
	(1.226)	(0.309)	(2.511)
Any Female Sibling **[B]**	0.660	-0.075	-0.390
	(1.403)	(0.349)	(2.766)
Additional Controls	Yes	Yes	Yes
[A] - [B]	-0.330	-0.102	-1.164
	(0.843)	(0.818)	(0.728)
N	85,572	38,988	64,490

Table 6 reports results from Tobit regressions of annual measurs of financial risk taking between 1999 and 2007 onto an indicator variable for women with a male co-twin ("Male Co-Twin"). Additional controls are the same control variables used in Table 3. Panel A includes female fraternal twins. Panel B includes the siblings of the twins of Panel A. For each model, we report the coefficient estimates as well as the corresponding *p* -values. *p* -values are based on double-clustered standard errors, robust for correlation across years within same individuals and across individuals within the same year. All variables are defined in detail in Appendix Table A1. *N* provides the number of observations used in each estimation. Levels of significance are denoted as follows: * if $p<0.10$; ** if $p<0.05$; *** if $p<0.01$.

Table 7
The Effect of Birth Weight

	Risky Share		Portfolio Volatility		Proportion Stocks	
	(1)	(2)	(3)	(4)	(5)	(6)
Birth Weight (ln)	4.950**	5.958*	-0.298	-2.859***	-5.752*	-12.061**
	(0.042)	(0.095)	(0.741)	(0.000)	(0.098)	(0.011)
Twin Pair Fixed Effects	No	Yes	No	Yes	No	Yes
N	17,510	17,510	4,926	4,926	11,744	11,744
R-squared	0.001	0.417	0.025	0.460	0.033	0.581

Table 7 reports results from linear regressions of annual measures of financial risk-taking of identical twins between 1999 and 2007 onto birth weight ("Birth Weight (ln)"). In columns 2, 4, and 6, we include twin pair fixed effects. In the models without twin fixed effects, we add an indicator variable for women ("Female"). For each model, we report the coefficient estimates as well as the corresponding *p* -values. *p* -values are based on double-clustered standard errors, robust for correlation across years within same individuals and across individuals within the same year. All variables are defined in detail in Appendix Table A1. *N* provides the number of observations used in each estimation. *R-squared* denotes the coefficient of determination. Levels of significance are denoted as follows: * if $p<0.10$; ** if $p<0.05$; *** if $p<0.01$.

Table 8
The Effect of Birth Weight on Additional Outcomes

	Net Worth (ln)		Volatility of Labor Income		Business Owner		Years of Education		Poor Health	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Birth Weight (ln)	0.155	0.202*	0.006	-0.022*	0.006	-0.016	1.522***	0.689***	0.028	-0.064**
	(0.110)	(0.097)	(0.485)	(0.091)	(0.558)	(0.399)	(0.000)	(0.006)	(0.257)	(0.031)
Twin Pair Fixed Effects	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
N	17,222	17,222	22,972	22,972	23,646	23,646	23,646	23,646	23,646	23,646
R-squared	0.020	0.586	0.003	0.596	0.005	0.510	0.009	0.787	0.011	0.418

Table 8 reports results from linear regressions of socioeconomic outcomes of identical twins onto birth weight ("Birth Weight (ln)"). In columns 2, 4 , 6 , 8, and 10, we include twin pair fixed effects. In the models without twin fixed effects, we add an indicator variable for women ("Female"). For each model, we report the coefficient estimates as well as the corresponding *p* -values. *p* -values are based on double-clustered standard errors, robust for correlation across years within same individuals and across individuals within the same year. All variables are defined in detail in Appendix Table A1. *N* provides the number of observations used in each estimation. *R-squared* denotes the coefficient of determination. Levels of significance are denoted as follows: * if $p < 0.10$; ** if $p < 0.05$; *** if $p < 0.01$.

Table 9
The Direct and Indirect Effect of Birth Weight

	Risky Share		Portfolio Volatility		Proportion Stocks	
	(1)	(2)	(3)	(4)	(5)	(6)
Birth Weight (ln)	3.862	5.920*	-0.741	-3.457***	-7.175**	-13.267***
	(0.128)	(0.078)	(0.406)	(0.001)	(0.039)	(0.005)
Net Worth (ln)	0.466	-1.201**	0.523*	-0.067	3.848***	1.724***
	(0.484)	(0.038)	(0.066)	(0.884)	(0.000)	(0.001)
Volatility of	-7.864	0.901	7.614***	5.846*	25.766***	26.554***
Labor Income	(0.218)	(0.903)	(0.006)	(0.020)	(0.005)	(0.002)
Business Owner	-4.812	-3.295	-2.416	-1.721	3.335	7.880*
	(0.309)	(0.398)	(0.146)	(0.162)	(0.629)	(0.099)
Years of Education	0.706***	0.186	0.288**	-0.027	0.157	-0.214
	(0.002)	(0.535)	(0.023)	(0.836)	(0.614)	(0.586)
Poor Health	-0.150	0.510	0.288	-0.492	-0.094	0.712
	(0.922)	(0.494)	(0.648)	(0.605)	(0.963)	(0.533)
Additional Controls	Yes	Yes	Yes	Yes	Yes	Yes
Twin Pair Fixed Effects	No	Yes	No	Yes	No	Yes
N	15,767	15,767	4,543	4,543	10,754	10,754
R-squared	0.016	0.452	0.051	0.492	0.067	0.605

Table 9 reports results from linear regressions of annual measures of financial risk taking of identical twins onto birth weight. In columns 2, 4, and 6, we include twin pair fixed effects. Additional controls include: Female, Single, Divorced, Number of Children, Retired, Disposable Income (ln) and Missing Education Data. All variables are defined in detail in Appendix Table A1. For each model, we report the coefficient estimates as well as the corresponding *p*-values. *p*-values are based on double-clustered standard errors, robust for correlation across years within same individuals and across individuals within the same year. *N* provides the number of observations used in each estimation. *R-squared* denotes the coefficient of determination. Levels of significance are denoted as follows: * if $p<0.10$; ** if $p<0.05$; *** if $p<0.01$.

Appendix Table A1
Definition of all Variables

Variable	Definition
Types of Twins	
Fraternal Twins	Twins that on average have a genetic correlation of 50%, also called dizygotic or non-identical twins. Fraternal twins can be of the same sex or of opposite sex. Zygosity is determined by the Swedish Twin Registry based on questions about intrapair similarities in childhood.
Identical Twins	Twins that are genetically identical, also called monozygotic twins. Zygosity is determined by the Swedish Twin Registry based on questions about intrapair similarities in childhood.
Measures of Financial Risk Taking	
Risky Share	The fraction of financial assets invested in equity either directly (individual stocks) or indirectly (equity mutual funds). The ratio is computed annually using end-of-year market values as reported by Statistics Sweden. Financial assets include checking, savings, and money market accounts, (direct and indirect) bond holdings, (direct and indirect) equity holdings, investments in options and other financial assets such as rights, convertibles, and warrants.
Portfolio Volatility	Using twelve monthly return observations, we calculate the annualized, value-weighted portfolio return volatility for each twin and year. The portfolio consists of the holdings of risky (i.e., equity) assets and missing if for individuals that do not hold risky assets.
Proportion Stocks	The fraction of risky (i.e., equity) holdings invested in individual stocks as opposed to mutual funds, as reported by Statistics Sweden. This measure is computed annually and is missing for for individuals that do not hold risky assets.
Determinants of Financial Risk Taking	
Male Co-Twin (F_M)	An indicator variable that is one if a female twin has a male co-twin and zero otherwise.
Non-twin Male (Female) Sibling	An indicator that is one if a female fraternal twin has a Male (Female) non-twin sibling and zero otherwise.
Any Male (Female) Sibling	An indicator that is one if a female non-twin has any male (female) siblings and zero otherwise.
Birth Weight (ln)	The natural logarithm of the birth weight (measured in grams (g)) as reported by the Swedish Twin Registry.
Net Worth (ln)	The difference between the end-of-year market value of an individual's assets and her liabilities (for each year an individual is included in our sample), as reported by Statistics Sweden. We compute the natural logarithm of net worth, originally expressed in nominal Swedish Krona (SEK).
Volatility of Labor Income	The time-series standard deviation of the log growth rate of an individual's labor income between 2000 and 2007 (as reported by Statistics Sweden). The variable is missing if four or more of the log growth rates are missing. The top and bottom one percentile of the log growth rate distribution is set to missing.
Business Owner	An indicator that is one if in a given year an individual has income from active business activity that exceeds 50% of the labor income. The indicator is zero otherwise. Income data are from Statistics Sweden.
Years of Education	Years of Education is based on the highest completed degree. For a subset of the sample, the variable is obtained from the Swedish Twin Registry. We use a linear regression model to extend the variable to the rest of our sample. Specifically, we regress the years of education onto variables indicating the higest degree obtained (e.g., high school, college) (avaialable for most individuals in our data set from Statistics Sweden) and then predict years of education out of sample.
Poor Health	An indicator variable that equals one if in a given year an individual an individual receives payments due to illness, injury, or disability and zero otherwise. Data on payments are obtained from Statistics Sweden.
Additional Controls	
Age	The age for every year an individual is included in our sample. Age is obtained from the Statistics Sweden. In our analyses, we use indicator variables for those younger than 35 (*Age less than 35*), between 35 and 49 (*Age less than 50*), and between 50 and 65 (*Age less than 66*).
Birth Order	The order of birth within the family. First-born siblings are assigned value equal to one. Twins are assigned the same birth order number.
Number of Siblings	The number of siblings (brothers and sisters) of the twin in the family of origin. The count includes the co-twin.
Male	An indicator variable that equals one if an individual is male and zero otherwise. Gender is obtained from Statistics Sweden.
Female	An indicator variable that equals one if an individual is female and zero otherwise. Gender is obtained from Statistics Sweden.
Missing Education Data	An indicator variable that equals one if no educational data are available for an individual, zero otherwise. Educational information is obtained from Statistics Sweden.
Single	An indicator variable that equals one if an individual is single in a given year and zero otherwise. Marital status information are obtained from the Statistics Sweden.
Divorced	An indicator variable that equals one if an individual has divorced in the past (and has not re-married since) and zero otherwise. Marital status information is obtained from the Statistics Sweden.
Number of Children	The number of children living in the same household in a given year. Family data are from Statistics Sweden.
Retired	An indicator variable that equals one if an individual is retired and zero otherwise. Occupational data are obtained from Statistics Sweden.
Disposable Income (ln)	The natural logarithm of individual disposable income for every year an individual is included in our sample, as defined by Statistics Sweden, that is, the sum of income from labor, business, and investment, plus received transfers, less taxes and alimony payments, originally expressed in nominal Swedish Krona (SEK). The data are obtained from Statistics Sweden.
Measures of Intra-twin Pair Interaction	
Contact Intensity	The number of contacts per year between twins. The number is calculated as the average of the numbers reported by both twins. If only one twin provides a number, this number is used. The data are obtained from the Swedish Twin Registry.
Travel Distance	The driving distance in kilometers between the municipalities of the twins' primary residence. The distance is obtained from Google Maps.
Portfolio Overlap	The sum of the absolute value of portfolio weight differences across the two twins. This measure ranges between zero (identical portfolios) and two (non-overlapping portfolios). A value equal to one corresponds to a 50-percent portfolio overlap.