HEDG
HEALTH, ECONOMETRICS AND DATA GROUP
THE UNIVERSITY of York

WP 15/18

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Eskil Heinesen, Susumu Imai & Shiko Maruyama

September 2015

http://www.york.ac.uk/economics/postgrad/herc/hedg/wps/

In-Utero Social Interaction of Twins

Eskil Heinesen*, Susumu Imai† and Shiko Maruyama‡

August 3, 2015

Abstract

We model pre-birth twins' competition for maternal resources inside the womb. When the innate endowment affects both birth weight and the post-birth outcome directly, pre-natal social interaction leads to bias in the standard twin fixed-effects estimator. We propose a test of social interaction that is based on data on triplets, and find some evidence for social interaction. We then use an instrumental-variable estimation strategy that recovers consistently the returns to birth weight. Our estimation results indicate that the returns to birth weight are closer to the sibling-based estimates than the twin fixed-effects estimates reported in the previous literature.

*Rodkwool Foundation Research Unit, e-mail:esh@rff.dk

†Economics Discipline Group, University of Technology Sydney, e-mail:Susumu.Imai@uts.edu.au

‡Economics Discipline Group, University of Technology Sydney, e-mail:Shiko.Maruyama@uts.edu.au

1 Introduction

A large body of literature in the medical and social sciences has documented worse future outcomes of infants with lower birth weight. Researchers have found this robust correlation not only in a wide range of health and clinical outcomes, such as infant mortality, various morbidity, neurological impairment, and physical growth, but also in various socio-economic outcomes such as test scores (Maruyama and Heinesen, 2014), welfare assistance (Oreopoulos et al., 2008), health care costs (Almond et al., 2005), risk-taking behavior (Hack et al., 2002), behavioral problems (Hultman et al., 2007), criminal tendencies (Tibbetts and Piquero, 1999; Maruyama and Heinesen, 2014), and even the birth weight of the next generation (Royer, 2005; Black et al., 2007; Currie and Moretti, 2007). The effect of unobserved endowment on birth weight and post-birth outcomes, however, causes correlation between the birth weight and the error term of the outcome equation, and thus leading to endogeneity bias. The methodology widely used in this literature to overcome this bias is the twin fixed-effects estimation strategy (Behrman and Rosenzweig, 2004; Almond et al., 2005; Miller et al., 2005; Conley et al., 2006; Black et al., 2007). The method relies on within-twin variations and has been considered among researchers as effective in dealing with the endogeneity of birth weight. The twins-fixed effects estimator differences away common parental background and the common part of their genetic endowment. Moreover the monozygotic twins have the same genes as well. Even though dizygotic twins do not share the same genes, the difference can be considered reasonably close to be random. Therefore, by differencing the twins' birth weight and the outcomes, one obtains variation that is orthogonal to the parental background, and thus, regression analysis based on them has been considered not to suffer from endogeneity bias.

The findings from the twin fixed-effects studies can be summarized into three points. First, their findings tend to be unstable and often mixed, depending on data, countries, and empirical design.[1] Second, the majority of these studies nevertheless find significant long-term birth weight effects on health, cognitive, and socio-economic outcomes, with

[1]For example, Miller et al. (2005) study twins in Australia and find no strong birthweight effect on schooling and earnings, which is inconsistent with most other studies. Royer (2009) discusses instability in the results of Black et al. (2007).

magnitudes often similar to ordinary least squares (OLS) estimates. Third, when infant mortality and infant health are studied, the use of twin fixed effects substantially reduces the estimates of birth weight effect relative to their OLS counterparts (Almond et al., 2005; Conley et al., 2006; Black et al., 2007; Oreopoulos et al., 2008; Royer, 2009). The latter two observations imply that the OLS correlation between birth weight and infant health is largely non-causal, but birth weight does have a latent effect that alters one's life much later.

These findings might be broadly understood in terms of the fetal origins hypothesis. This widely-cited hypothesis claims that the intrauterine environment, particularly nutrition, "programs" the fetus to have particular metabolic characteristics, which can lead to future adult diseases even if there is no immediate impact (Barker, 1990; Barker, 1995).[2] Another interpretation follows the recent literature on the early childhood human capital investment literature by Cunha and Heckman (2008) and others which suggest human capital production function where ability endowment and human capital investment are complements. Then, the post-natal investment dynamics would propagate even a small change in infant outcome resulting from low birth weight to a large long term differences in outcome. On the other hand, in the medical literature, there is a large body of physiological and clinical knowledge about how low birth weight causes neonatal complications and morbidity. For example, respiratory failure is a major cause of morbidity and mortality in low birth weight infants, and the introduction of a treatment called surfactant therapy in the 1990s has substantially reduced mortality and morbidity in this population (Wilson-Costello et al., 2005).

In this paper, we consider a simple model of twin competition for common resources in the womb, and formally show that the pre-birth social interaction between the twin pairs leads to bias in the estimated returns to endowment or returns to fetal environment. Thus we argue that the twin fixed-effects model does not estimate the returns to birth weight consistently, regardless of whether it is interpreted as the returns to endowment or returns to fetal environment. This could be the reason for the twin fixed-effects literature finds birth weight effects to be much lower than those of the OLS.

[2]See Almond and Currie (2011A) for a review of the growing literature on the hypothesis from the economics perspective.

In this paper, we propose to test the hypothesis of social interaction versus the standard twins fixed effects model using data on triplets. We follow the twins literature that exploits the following unique aspect of the the standard models that describe behavior of singletons and twins: except for the assumptions that twins share the same parental background and similar genetic background when they are dizygotic and the same genetic background when they are monozygotic, the rest of the model is the same for both singletons and twins. That is why data on twins can be used to difference away the observed and unobserved parental and genetic characteristics and thus obtain consistent estimates of the behavioral model, which is common to both singletons and twins. That is, data on twins are used to infer about the behavior of singletons. We extend the idea and use data on triplets to investigate the behavioral model of twins. In particular, we test the existence of social interactions among twins. Exploiting the triplets, we take difference of the log birth weight of two infants, just like the first differencing that is done in twins estimation, and estimate the correlation between the above first difference and the log birth weight of the third infant. If the model of birth weight is conventional, then the differenced log birth weight should not contain any variation common to all the triplets, and since in standard models, the residuals after taking away the common component is assumed to be uncorrelated, the above correlation should be zero. We detect significant correlation and thus reject the hypothesis that the standard behavioral model describes the data on triplets. The theoretical analysis imply that we can interpret the correlation of the residuals as the social interaction, which includes competition of twins (triplets) for maternal resources or hormone transfer among different gender twins (triplets).

We then propose an instrumental-variable (IV) estimation strategy that estimates the effects of birth weight on infant mortality and the number of days in hospital, which we interpret to measure the returns to improvements in fetal environment. This estimator provides consistent estimates even when there is pre-birth social interaction between the twin pairs. For this purpose, our IV exploits the exogenous change of the environment of a twin member. Specifically, we use the gender of the dichorionic twin partner as an instrument. The gender of the twin partner is known to be random, and we know from the literature and from inspection of the data that the birth weight of a twin who has a male partner is smaller than the one with a female twin partner.

The preliminary evidence indicates that our IV estimates are larger than the twin fixed-effects estimates and close to the conventional OLS estimates for twins and singletons as well as to the mother fixed-effects estimates for siblings. What this result implies is that the long run effect of low birth weight could be the persistence of the short run effect, not necessarily due to complex mechanism such as genetic programming or post-natal dynamic propagation mechanisms.

We also investigate the possibility of the results being generated by a mechanism other than the one through competition for maternal resources. In particular, the hormone transfer hypothesis argues that the female hormones in male-female twin pair are also partially transferred to male twin, resulting in feminization of males, and similarly, male hormones are partially transferred to female twin, resulting in masculinization of females. Those hormonal allocations would improve the infant health for males and reduce it for females. In this paper, we conduct a formal test of the hormone transfer hypothesis and show that the hypothesis of no bias of our results due to hormone transfer cannot be rejected.

In Chapter 2 Section 1, we briefly review the twin fixed effects literature and then, formally show the source of bias if we allow for the social interaction of twins in utero. In Section 2, we propose the IV strategy that uses the gender of the twin partner as an instrument. In Section 3, we formally discuss the Identification and the Estimation strategy.

2 Data

2.1 Danish administrative data and population selection.

We use data from Danish administrative registers. The birth registry contains the population data for newborns in Denmark, providing information on birth date, unique personal identifiers of the newborns and biological parents, and a range of clinical variables about the mother and infant, including birth weight. The birth registry is considered to be of high quality in regard to both validity and coverage (Blenstrup and Knudsen, 2011). Blenstrup and Knusden (2011) also confirm the validity of the reference linkage between

parents and children. Using the personal identifiers of newborns and parents, we match birth records to other Danish registers.

The following are excluded from our analysis: multiple pregnancy of quadruples or more, stillbirths, children whose mother is not identified in the birth registry, children born overseas (because of the lack of birth-related information), and adopted children and their biological siblings. These exclusions account only less than 1% of the population. Children whose father is not identified are retained in our population (1% of our final sample); we include an indicator for missing father information when necessary. A very small number of observations with missing values, highly unrealistic values, and other data problems are discarded.

2.2 Outcome and control variables.

We study the effect of birth weight on four health outcomes: one year mortality, 4 weeks mortality, APGAR score taken within 5 minutes after birth, and the number of days in hospital between the date of birth and the 2nd birthday.

Our regression analysis includes a number of control variables. It is standard in the literature to use control variables that are defined at birth, but we use control variables defined at conception when it is possible and appropriate. This is because birth weight is strongly related to the timing of birth, so that control variables defined at birth may cause endogeneity bias. The estimated date of conception is available for most observations. For a very few cases in which gestational age is unavailable (0.6% of the entire sample), we infer the gestation length from a regression on birthweight, gender, maternal age, and other variables.

We construct from the birth registry the following explanatory variables: the gender of the child, year- and month-of-birth dummies, indicators for the mother's age at conception (one indicator for every two years), an indicator for being the first child, birth order, indicators for past pregnancies (1, 2, 3, and 4+), the number of past cesarean sections, and also indicators for the mother's past spontaneous and induced abortions (1 and 2+), past stillbirths, and smoking habits. From the hospital admission registry, we construct a variable for the number of days spent by the mother in the hospital during the 180 days

around the conception, except for pregnancy-related admissions. This variable captures the aspects of the mother's general health that are not the consequence of pregnancy-related conditions.

The other demographic and socioeconomic variables are constructed from various administrative registers: indicators for the mother's highest education completed (less than 9 years, 9 years, upper secondary, low and medium tertiary, and high tertiary education); indicators for formal marital status and whether the biological mother and father are living together on the January 1st prior to the birth; an indicator for the immigrant status of either the mother or father; the mother's working status (an indicator for whether she was working most of the year) and income in the previous year, and interaction terms between these variables and conception month dummies (to account for the effect of pregnancy on the previous year's labor supply); the father's information (an indicator for missing information on the biological father, age at conception, income and working status in the previous year, and highest education completed as grouped as the mother's education indicators above); and county dummy variables. These controls are included in all regressions.

We use data from 1981 to 2013. Later, when we conduct IV estimation on twins, we control for the chorionicity or zygosity of the twins. Those data are only available from the year 2003. Therefore, for those analyses, we use the data from year 2003 to 2013.

3 Model of Bodyweight Growth of Twins

3.1 Econometric Specification of twin fixed effects models

We first review the conventional model of birth weight and infant health outcome, and then, the properties of the OLS and twin fixed effects estimator. Here, we mostly follow Behrman and Rosenzweig (2004). Consider a linear model of log birth weight of twins:

$$BW_{ki} = X_k\beta + \alpha_k + u_{ki}$$

where BW_{ki} is log birth weight of twin i in twin pair k, α_k is the twin pair-specific term and u_{ki} is the child-specific term (which is the deviation of nutrient intake of twin ki from

the average nutrient intake of the two twins in the model of Behrman and Rosenzweig, 2004). Suppose the equation for the outcome Y_{ki} is specified as

$$Y_{ki} = \mathbf{X}_k \boldsymbol{\eta}_X + u_{ki}\eta_B + \xi_k + \epsilon_{ki} = \mathbf{X}_k \boldsymbol{\delta}_X + BW_{ki}\eta_B + \zeta_k + \epsilon_{ki}$$

where ξ_k is the effect on the outcome of the unobserved family background/twin pair endowment and ϵ_{ki} is the twin i specific idiosyncratic shock affecting the outcome, whose mean is zero. Furthermore,

$$\boldsymbol{\delta}_X = \boldsymbol{\eta}_X - \boldsymbol{\beta}\eta_B,\ \zeta_k = \xi_k - \alpha_k \eta_B$$

The literature argues that cross section or siblings based longitudinal analysis are problematic because of the likely correlation between the log birth weight BW_{ki}, and the error term, because the former includes α_k and the latter ζ_k. Both are unobserved twin pair-specific terms and one cannot rule out correlation between the two. That is, as sample size goes to infinity,

$$\begin{aligned} \hat{\eta}_{OLS} &\equiv \frac{\widehat{Cov}\left(BW, Y\right)}{\widehat{Var}\left(BW\right)} = \frac{\widehat{Var}\left(BW\right)\eta_B + \widehat{Cov}\left(BW, \zeta\right)}{\widehat{Var}\left(BW\right)} \\ \hat{\eta}_{OLS} &\xrightarrow{p} \eta_B + \frac{Cov\left(\alpha, \xi - \alpha\eta_B\right)}{Var\left(BW\right)} \neq \eta_B. \end{aligned}$$

Therefore, the OLS is not a consistent estimator of the returns to log birth weight. On the other hand, by the very nature of their birth process, twins have the same pre-natal parental socioeconomic background and similar genetic endowment. Indeed, for the monozygotic twins, they are known to have the same genes. Therefore, the within twin difference in log birth weight can be considered as the variation in birth weight that does not contain any difference in pre-natal environment and small difference in genes (or none if they are monozygotic twins) and thus, would be close to a pure random shock of birth weight. In this case, if we denote $\Delta BW_k = BW_{k1} - BW_{k2}$ as the difference between log birth weight of twins,

$$\Delta BW_k = BW_{k1} - BW_{k2} = u_{k1} - u_{k2} = \Delta u_k$$

and similarly for outcome

$$\Delta Y_k = Y_{k1} - Y_{k2} = \Delta BW_k \eta_B + \Delta \epsilon_k$$

Hence, by taking within twin difference in log birth weight and in outcomes, then one can difference away any observed and unobserved common parental and genetic background, which was the source of bias in the OLS. Then, assuming that Δu_k and $\Delta \epsilon_k$ are orthogonal, the twin fixed-effects estimator can be derived as follows:

$$\hat{\eta}_{TFE} = \frac{\widehat{Cov}\left(\Delta BW, \Delta Y\right)}{\widehat{Var}\left(\Delta BW\right)} = \frac{\widehat{Var}\left(\Delta BW\right)\eta_B + \widehat{Cov}\left(\Delta BW, \Delta \epsilon_k\right)}{\widehat{Var}\left(\Delta BW\right)} \xrightarrow{P} \eta_B$$

as the sample size goes to infinity. That is, under the above model specification, the twins fixed-effects estimator consistently estimates the returns to log birth weight. For the monozygotic twins, both of them share the same parental background as well as the same genes, and thus, assumption of orthogonality between Δu_k and $\Delta \epsilon_k$ is likely to hold. However, for the dizygotic twins, they share the same parental background but only about 50 percent of the genes. Therefore, orthogonality is not guaranteed to hold for them. However, many papers such as Black et. al. (2007) and others report very similar results for the twin fixed effects estimators for the dizygotic and monozygotic twins.

One of the implicit assumptions that is used in the twins research is that the behavioral model is the same for singletons, twins and triplets. Then, it follows that returns to log birth weight estimated by using twins is informative on the returns to log birth weight of singletons. In the similar manner, returns to log birth weight estimated by using triplets should be informative on the returns to log birth weight of twins. Unlike the case of singletons and twins, where one can obtain the consistent estimate on twins, but not on singletons, we can obtain the consistent estimates on both twins and triplets, by using twins or triplets fixed effects. Therefore, we can compare the estimates of twins and triplets to see whether they are similar, as the standard behavioral model predicts. In other words, by using separately the data on twins and triplets, we exploit the fact that in both twins and triplets, we have a group of siblings who have the same within twin (triplet) parental background and similar genetic endowment, but potentially different environment. Then, one can infer whether maternal environment can affect the returns to log birth weight, which standard behavioral models abstract from.

Table 1 shows the mean birth weight of singletons, twins and triplets. First, we can see that the singleton birth weight is the highest, followed by the twins and then the triplets. This indicates the possibility of limited maternal resources that need to be share among

siblings in utero, and thus the competition for in utero resources among infants. Second, males have higher birth weight than females, for singletons, twins and triplets. Third, there are some differences between twins and triplets when we look at the birth weight for different gender partners. For twins, the birth weight is higher when the partner gender is different, but for triplets, birthweight when the partner gender is the same is higher than when they are different. In Tables 2, 3 and 4, we also report sample statistics of one year and 4 weeks infant mortality APGAR score and the number of hospital days before 2nd birty day. We can also see the same pattern, albeit in the opposite direction, with respect to infant mortality. That is, 1) Singletons have by far the lowest infant mortality, followed by the twins, and then triplets. 2) Overall, males have higher infant mortality than females. But there are some exceptions as well. 3) For twins, infant mortality is lower for males with female partner than for male partner, but for females, infant mortality is higher for male partner twins than for female partner ones. However, for triplets, different gender pairs have higher infant mortality. In some cases, the differences are striking. Male infant mortality in one male two female triplet is 0.083 and the same for female infant mortality is 0.071. APGAR score results and the results for the hospital days are similar as well.

Next, in Tables 5, 6, 7 and 8, we report the results where we estimated the returns to log birth weight using conventional twins and triplets fixed effects. As we can see, the twin fixed effects results are conventional. If we look at the returns to birth weight on infant mortality, the singleton fixed effects estimates are around -0.137 for the mortality within one year and -0.123 for the mortality within four weeks. Since the coefficients on the interaction term between log birth weight and female dummies are relatively small, female infant mortality estimates are also similar to the male ones mentioned above. On the other hand, twins fixed effects estimates are around -0.04 for the mortality within one year and -0.03 within four weeks. All of them are significant. Again, interaction term coefficients indicate that the returns for females are similar. If we then look at the triplet fixed effects, then the estimates are -0.103 for both one year mortality and four weeks mortality, and they are both significant at the 10 percent level. Those returns to log birth weight estimated using the triplets fixed effects are about 2.5 to 3 times as high as the twins fixed effects estimates. The interaction term coefficient is positive and large, around

0.068 for the one year mortality and 0.065 for the 4 weeks mortality, albeit insignificant. Hence, returns to birth weight for females of triplets could be small, but the inferred magnitude would still be larger than the twins estimate. For the returns to log birth weight on APGAR score, the twins fixed effects estimator is 0.381 and significant, but the triplets fixed effects estimator is 0.148, a little more than one third of the twin fixed effects estimator, and insignificant. Given the negative coefficient on the interaction term for the twins and the positive coefficient for the triplets, the returns to log birth weight for females in twins and triplets are likely to be closer to each other. If we take a look at the returns to log birth weight on hospital days, then the twins fixed effects estimate is -4.965 and significant, but the triplets fixed effects estimate is -15.92 and significant, about three times of the twins fixed effects estimate. The relatively small interaction term coefficients indicate that the returns to log birth weight coefficients show simlar patterns for females.

As we can see above, even though many of the triples fixed effects estimates are insignificant due to the small sample size, one can tentatively conclude that the twins fixed effects estimates and the triple fixed effects estimates are not similar, and thus there are some possibility for the difference in maternal environment affecting the results. There is one alternative explanation for the discrepancy between twins and triplets returns to birth weight estimates: nonlinearity. That is, even for twins, lower birth weight pairs may have higher returns to birth weight estimates than others. In Table 9, we present the twins fixed effects estimators where the average birth weight of the twin pair is less than 2000 gram, which we argue roughly corresponds to the birth weight distribution of the triplets. The returs to birth weight on infant mortality is about -0.136 for one year and -0.109 for 28 days mortality, which is close to the -0.105 or -0.103 estimated from the triplet fixed effects. The interaction term of log birth weight and female dummy are about 0.65 for the triplets and for the twins with birth weight less than 2000 gram, -0.02 for the one year infant mortality and 0.02 for the 4 weeks infant mortality. Those coefficients make the returns to birth weight for females to be similar for the triplets and the low birth weight twins. On the other hand, for APGAR score, the twins fixed effect estimate is 0.904, much higher than the triplets fixed effects estimate of 0.148, and the relatively small interaction term for the low birth weight twins and triplets make the results to

be similar for the females. For the hospital days, twins fixed effects estimate is -8.293, which is about half the estimate of the triplets fixed effects estimate -15.92. However, the positive interaction term coefficient for the triplets and the negative coefficient for the low birth weight twins could make the difference much smaller for females in twins. Overall, there still are substantial differences between the twins fixed effects estimates of twins with average birth weight being less than 2000 grams and the triplets fixed effects estimates. Hence, one could conclude that nonlinearities would not explain all the differences between the twins fixed effects and the triplets fixed effects, and thus, differences in maternal environments could play some role in explaining the difference. There are two reasons why maternal environment of twins and triplets are different. First, observed and unobserved characteristics of parents who have twins and those who have triplets are likely to be different. This is because triplets are much more frequent among parents who use fertility treatments than who don't and they typically are older and higher educated and have higher income. Second, having one partner versus having two partners are likely to influence the maternal environment as well. However, in a standard twins (triplets) fixed effects models, those differences in maternal environment should have been differenced away by using the fixed effects estimator.

In this paper, we investigate alternative ways where singletons, twins and triplets may differ. That is, we focus on the fact that in multiple birth, twins and triplets share the same maternal womb. In contrast to the conventional twins literature that only takes up the aspect that they thus have the same parental and genetic background, we explicitly consider the possibility that the infants who share the maternal womb interact with each other. We explicitly model the interaction of the twins and triplets in the maternal womb, and through that, piont out potential reasons why the twins and triplets returns to birth weight estimates differ, and thus, why the twins fixed effects estimates may systematically and substantially diverge from the true returns to birth weight of singletons.

To do so, we make some modifications to the above birth weight and outcome equations. We argue that due to the resource constraint in the maternal womb, the ability in

nutrient intake by the twin pair j, u_{kj} reduces own log birth weight by $\gamma_B u_{kj}$,[3] i.e.

$$BW_{ki} = \mathbf{X}_k\boldsymbol{\beta} + \alpha_k + u_{ki} - u_{kj}\gamma_B = \mathbf{X}_k\boldsymbol{\beta} + \alpha_k + (1+\gamma_B)\, u_{ki} - (u_{ki} + u_{kj})\, \gamma_B$$

That is, for a twin, the higher nutrient intake by his/her twin partner worsens his/her fetal environment. Notice that the effect of the nutrient intake by twin partner is similar to the adverse impact on the fetal environment considered in the fetal origins hypothesis (Almond and Curie 2011). Then, by taking the twins difference, we obtain

$$\Delta BW_k = BW_{k1} - BW_{k2} = (1+\gamma_B)\,(u_{k1} - u_{k2}) = (1+\gamma_B)\,\Delta u_k$$

We also modify the outcome equation so that it both includes the nutrient intake of own and partner twins.

$$Y_{ki} = \mathbf{X}_k\boldsymbol{\eta}_X + \xi_k + u_{ki}\eta_{B1} - u_{kj}\gamma_B\eta_{B2} + \epsilon_{ki} = \mathbf{X}_k\boldsymbol{\eta}_X + \xi_k + u_{ki}\,(\eta_{B1} + \gamma_B\eta_{B2}) - (u_{ki} + u_{kj})\,\gamma_B\eta_{B2} + \epsilon_{ki}$$

As we can see, η_{B1} is the parameter that measures the effect of own determinants of birth weight, such as higher own nutrient intake, etc., on the outcome, whereas η_{B2} measures the effect of adverse fetal environment due to the higher nutrient intake by the twin pair on the outcome. So far, existing maternal health policies have mostly been trying to improve the fetal environment, and have not been targeting the ability to take in nutrients per se. Therefore, for policy makers, η_{B2} is the policy relevant parameter of interest, not η_{B1}. Furthermore, notice that, it is easier to interpret η_{B2} than η_{B1} because by constriction, η_{B1} measures all the other effects of birth weight on infant outcome, which could include temporary changes in birth weight that may not be related to infant health outcome. Therefore, from now on, we will mostly focus on the parameter η_{B2} as the effect of birth weight on outcome.

Then, by taking the twin difference of log birth weight, we can difference away the common component $\mathbf{X}_k\boldsymbol{\beta} + \alpha_k - (u_{ki} + u_{kj})\,\gamma_B$ to obtain:

$$\Delta BW_k = BW_{k1} - BW_{k2} = (1+\gamma_B)\,(u_{k1} - u_{k2}) = (1+\gamma_B)\,\Delta u_k$$

[3]It is not hard to find heuristic evidence for the competition of twins for the common maternal resources in utero. It is well known that males have on average higher birth weight than females. As we have seen, in our data the birth weight of male twins with female partners is on average higher than the birth weight of those with male partners. Similarly, the birth weight of female twins with male partners is on average even lower than the birth weight of those with female partners.

Similarly, by taking the twins difference of outcome, we can difference away the common component $\mathbf{X}_k\boldsymbol{\beta} + \alpha_k - (u_{ki} + u_{kj})\,\gamma_B\eta_{B2}$ to obtain:

$$\Delta Y_k = Y_{k1} - Y_{k2} = (\eta_{B1} + \gamma_B\eta_{B2})\,\Delta u_k + \Delta\epsilon_k$$

Then, as sample size goes to infinity, the twins fixed effects estimator satisfies:

$$\hat{\eta}_{TFE} = \frac{\widehat{Cov}\,(\Delta BW, \Delta Y)}{\widehat{Var}\,(\Delta BW)} \xrightarrow{p} \frac{\eta_{B1} + \gamma_B\eta_{B2}}{1 + \gamma_B}$$

which does neither recover η_{B1}, returns to birth weight, nor η_{B2}, returns to change in in-utero environment,.

The low short run returns to birth weight estimated by twin fixed effect can be consistent with a large value of η_{B2} as long as γ_B is positive and relatively small, and η_{B1} has a small value. The higher twin fixed effects estimated long term returns to birth weight could be either due to an increase in η_{B1} or η_{B2}. In particular, even if the already high value of η_{B2} at birth remains constant, as long as the value of η_{B1} increases with age, then we would get the twin fixed effects estimated long term returns to birth weight to be high. It is actually reasonable to think that at infancy, the fetal environment would be the primary factor determining health outcome, but with age, the returns to other determinants of birth weight, such as innate ability would increase. In contrast, the conventional explanation of the low short term returns and high long term returns to birth weight is an increase in the value of η_{B2} with age. It is a somewhat intriguing explanation because one would presume that fetal environment would be more important, or at least not less important, for infants than for children or adults.

The important point we make in this paper is: so far, twins studies have been extensively used in many areas that includes labour economics and health economics primarily because of the attractive premise that by using twins who share the same maternal environment, the common maternal environment can be differenced away. However, we argue that because of the very fact that those twins share the same maternal womb, the ability to extract maternal resources of the partner would change the own resource extraction, and thus constitutes an important part of the maternal environment he/she is in. Because the twins in a pair are known to have substantially different birth weights, one can infer that their ability in extracting maternal resources are also different, and thus, automatically

so are the abilities of their partners. Therefore, each member of the twin pair is facing a different maternal environmen and thus, the impact of maternal environment cannot simply be differenced away. Therefore, in any twins analysis, to conduct it properly, one may have to carefully design a strategy that could identify the social interaction of the twins, just like in the empirical industrial organization literature that carefully estimates multiple heterogenous firms who share the same market, where each firm is facing a different opponent, and thus, different market environment, or in the schooling literature where researchers carefully study the social interaction of students in a class.

Another, somewhat related model we consider is based on hormone transfer hypothesis. The hormone transfer hypothesis argues that the female hormones in a male-female twin pair are also partially transferred to male twin, resulting in feminization of males, which includes a reduction of birth weight, and similarly, male hormones are partially transferred to female twin, resulting in masculinization of females, which includes an increase in birth weight. Those hormonal allocations would improve the infant health for males and reduce it for females. The evidence on this hypothesis is mixed at best, and even if the effect is tested to be significant, its size is in general small. Right now, the literature is more concerned with the male to female hormone transfer because research has shown that the pre-natal sex difference is formed primarily by male hormone which female unborns receive very little from the mother unless there is a male twin partner. Then, a very simple extension of the above model to include hormone transfer hypothesis would be

$$BW_{kMF} = \mathbf{X}_k\boldsymbol{\beta} + \alpha_k + u_{kM} - u_{kF}\gamma_B - h_{kF}$$

$$BW_{kFM} = \mathbf{X}_k\boldsymbol{\beta} + \alpha_k + u_{kF} - u_{kM}\gamma_B + h_{kM}$$

where BW_{kMF} is defined to be the male birth weight of the male-female twin and BW_{kFM} is defined as the female birth weight of the male-female twin. $h_{kF} \geq 0$ denotes a measure of the female hormones transferred from female twin partner to the male twin, which reduces birth weight, and $h_{kM} \geq 0$ is the male hormone transferred from male twin partner to the female twin, which increases birth weight.

4 Testing social interaction using triplets

It is in general impossible to point identify the social interaction parametr γ from the birth weight equation alone if we were to use the twins data. More concretely, we assume that α_k, v_k are normally distributed, and one can regress away the term $X_k\beta$ and hence, we will ignore the term. We also specify the correlation to be $-\gamma$. That is, we consider the residual

$$BW_{ki} = \alpha_k + u_{ki} - \gamma u_{kj}.$$

Then,

$$Var\left(BW_{ki}\right) = Var\left(\alpha_k\right) + \left(1+\gamma_B^2\right) Var\left(u_{ki}\right),\ Cov\left(BW_{k1}, BW_{k2}\right) = Var\left(\alpha_k\right) - 2\gamma_B Var\left(u_{ki}\right)$$

. Since there are only two moment equations and three unkowns: $Var\left(\alpha_k\right)$, γ_B and $Cov\left(BW_{k1}, BW_{k2}\right)$, they cannot be separately identified.

To test the existence of social interaction, we use triplets. Consider the extension of the twin social interaction model to triplets. Consider the triplets where the first infant two infants are males and the third infant is female. That is, we denote bw_{kM1} the log birth weight of the first infant who is male. Similarly for bw_{kF3}, which is the log birth weight of the third infant who is female. We also denote the social interaction parameter for male γ_M and for female γ_F to be different. That is,

$$bw_{kM1} = \alpha_k + u_{kM1} - h_{kF} - \gamma_M\left(u_{kM2} + u_{kF3}\right).$$

$$bw_{kM2} = \alpha_k + u_{kM2} - h_{kF} - \gamma_M\left(u_{kM1} + u_{kF3}\right).$$

$$bw_{kF3} = \alpha_k + u_{kF3} + h_{kM} - \gamma_F\left(u_{kM1} + u_{kM2}\right).$$

Now,

$$bw_{kM1} - bw_{kF3} = u_{kM1}\left(1+\gamma_F\right) - \left(\gamma_M - \gamma_F\right) u_{kM2} - u_{kF3}\left(1+\gamma_M\right) - h_{kF} - h_{kM}.$$

Hence,

$$Cov\left(bw_{kM2}, bw_{kM1} - bw_{kF3}\right) = \left[-\left(1+\gamma_F\right)\gamma_M + \left(1+\gamma_M\right)\gamma_M - \left(\gamma_M - \gamma_F\right)\right] Var\left(u\right)$$

$$+Cov\left(bw_{kM2}, -h_{kF} - h_{kM}\right) + Cov\left(-h_{kF2}, bw_{kM1} - bw_{kF3}\right).$$

$$= (\gamma_M - \gamma_F)(\gamma_M - 1) Var(u) + Cov(bw_{kM2}, -h_{kF} - h_{kM}) + Cov(-h_{kF2}, bw_{kM1} - bw_{kF3}).$$

Notice that in order for the covariance to be nonzero, $\gamma_M \neq \gamma_F$ needs to hold. Notice that without social interaction, i.e $\gamma_M = \gamma_F = 0$, then,

$$Cov(bw_{kM1} - bw_{kF3}, bw_{kM2}) = Cov(bw_{kM2}, -h_{kF} - h_{kM}) + Cov(-h_{kF}, bw_{kM1} - bw_{kF3})$$

where we only have the effect from the hormone transfer, and if we assume no hormone transfer, then

$$Cov(bw_{kM2}, bw_{kM1} - bw_{kF3}) = 0.$$

Similar results hold if we allow for $\sigma_M^2 = Var(u_M) \neq Var(u_F) = \sigma_F^2$. Then,

$$Cov(bw_{kM1} - bw_{kF3}, bw_{kM2}) = -[\gamma_F \gamma_M + 2\gamma_M - \gamma_F] \sigma_M^2 + (1 + \gamma_M) \gamma_M \sigma_F^2$$

$$+ Cov(bw_{kM2}, -h_{kF} - h_{kM}) + Cov(-h_{kF}, bw_{kM1} - bw_{kF3})$$

whose non-hormone transfer part also is zero if $\gamma_M = \gamma_F = 0$.

Therefore, we can run the following regression

$$bw_{kM2} = \beta_0 + \beta_1 (bw_{kM1} - bw_{kF3}) + \epsilon_{kM2}$$

and test the hypothesis $\beta_1 = 0$. If the hypothesis is rejected, then there is social interaction, which potentially includes both competition for maternal resources and hormone transfer. Notice that the test is still valid if we include some nonlinearity in the birth weight equation. That Let us modify the model to include nonlinearity as follows.

$$bw_{kM1} = \alpha_k + u_{kM1} - \gamma_{M1}(u_{kM2} + u_{kF3}) + \gamma_{M2}\alpha_k (u_{kM2} + u_{kF3}) - h_{kF}.$$

$$bw_{kM2} = \alpha_k + u_{kM2} - \gamma_{M1}(u_{kM1} + u_{kF3}) + \gamma_{M2}\alpha_k (u_{kM1} + u_{kF3}) - h_{kF}.$$

$$bw_{kF3} = \alpha_k + u_{kF3} - \gamma_{F1}(u_{kM1} + u_{kM2}) + \gamma_{F1}\alpha_k (u_{kM1} + u_{kM2}) + h_{kM}.$$

$$bw_{kM1} - bw_{kF3} = u_{kM1}(1 + \gamma_{F1}) - (\gamma_{M1} - \gamma_{F1}) u_{kM2} - u_{kF3}(1 + \gamma_{M1})$$

$$+\alpha_k [u_{kM1}(1 + \gamma_{F2}) - (\gamma_{M2} - \gamma_{F2}) u_{kM2} - u_{kF3}(1 + \gamma_{M2})] - h_{kF} - h_{kM}.$$

Then, as before, if $\gamma_{M1} = \gamma_{F1} = \gamma_{M2} = \gamma_{F2} = 0$, and $h_{kF} = h_{kM} = 0$, then

$$bw_{kM1} - bw_{kF3} = u_{kM1} - u_{kF3} + \alpha_k (u_{kM1} - u_{kF3}).$$

$$Cov\left(bw_{kM1} - bw_{kF3}, bw_{kM2}\right) = Cov\left(u_{kM1} - u_{kF3} + \alpha_k\left(u_{kM1} - u_{kF3}\right), \alpha_k + u_{kM2}\right) = 0.$$

As we can see from Table 10, we reject the hypothesis of $\beta_1 = 0$ for male triplets at 5 percent significance and the same for female triplets at about 6 percent significance level. Therefore, we reject the hypothesis using the triplets data that the model of log birth weight has a conventional specification of $\gamma_M = \gamma_F = 0$, $h_{kF} = h_{kM} = 0$.

5 Instruments

It is well known that males are born heavier than females. The sex of a fetus is determined by sex chromosome from its father, which is considered to be completely random. Hence, the sex of a child has been used in past studies as an IV. However, the sex of a fetus itself is not a valid instrument for our purposes because it not only affects own birth weight but also directly influences the survival of the fetus and potentially many other outcomes. We instead use the sex of the partner twin.

However, there is one important pathway that correlates the sex of the twin partner and the in-utero environment, more specifically, the chorionicity. The chorion is one of the membranes that exist between the fetus and mother, and twins may share the same chorion (monochorionic twins) or develop in two separate chorions (dichorionic twins). Monochorionic twins have a higher mortality risk than dichorionic twins because monochorinoic twins share the same placenta, causing a risk of twin-to-twin transfusion syndrome. Although the sex of a zygote is determined completely randomly[4], the correlation between the sex of the partner twin and the chorionicity arises because of the zygosity.

As illustrated in Figure 1, dizygotic twins (aka fraternal twins) always have separate chorions[5], and the sex combination of dizygotic twins is randomly determined. On the other hand, monozygotic twins (aka identical twins) always have the same sex, and around 25% of them share the same chorion. For this reason, we focus our empirical analysis on dichorionic twins so that our IV estimates are not influenced by the effect of chorionicity.

[4]Except for the case of IVF

[5]Except for the case of IVF

The chorionicity of monozygotic twins depends on the timing of zygote separation: if a zygote splits within the first four days, they become dichorionic. The timing of split is just as random as the occurrence of monozygotic twin, and hence our focus on dichorionic twins is not a concern for the validity of our instrument.

In the analysis below, we report the results where we use the dichorionic twins without controlling for zygosity, and the one that controls for zygosity. Because monozygotic twin pair have identical gender, monozygotic twins only exist in dichorionic same gender twins, and not in mixed gender twin pairs. This could create bias in our results. To deal with this issue, we propose to use dichorionic twin samples data on twins generated from the IVF (In-Vitro Fertilization) procedure as an effective alternative to control for zygosity. It is known in medical research that IVF involves fertilisation of multiple eggs, guaranteeing that the chance of monozygotic twins in the data is minimal. We chose to do so rather than use the conventional twin survey that includes information on zygosity. In the survey, zygosity is determined by the similarity in appearance, which is subject to classification errors. More importantly, since it is survey based and not register based, it could contain endogeneity issues in reporting and also has relatively small sample for the more recent twins, for which we have data on chorionicity.

5.1 Identification and estimation strategy

Next, we consider the consistency of the estimator using the instrument z, The assumptions for the IV estimation is

1) $E\left[\alpha_k|\mathbf{z}_k, \mathbf{x}_k\right] = E\left[\alpha_k|\mathbf{x}_k\right]$.

2) $E\left[u_{ki}|\mathbf{z}_k, \mathbf{x}_k\right] = E\left[u_{ki}|\mathbf{x}_k\right]$, $i = 1, 2$.

3) $E\left[u_{ik}|\mathbf{z}_k\right] = E\left[u_{ik}\right]$.

1) implies that the twin pair specific unobservables are orthogonal to the instrument $\mathbf{z}$ given $\mathbf{x}$. 2) implies that the conditional mean of the twin unobservable term is orthogonal to $\mathbf{x}$. 3) implies that twin i specific unobservables are also orthogonal to the instrument $\mathbf{z}$ and $\mathbf{x}$. We use the gender of the twin partner as the instrument. That is, $z = 1$ if the partner gender is male and $z = 0$ otherwise. It is well known in the medical literature

that the gender of a twin is purely random. Assumption 1) is justified because of the randomness of the partner gender. For assumption 2), it is straightforward to show that one can normalize u_{ki}, $i = 1, 2$ to not be a function of $\mathbf{x}_k$ by including $\mathbf{x}_k$s as controls for the birth weight equation. In 3) we impose the exclusion restriction that own ability u_{ki} is orthogonal to the gender of the twin partner.[6]

Then, the mean log birth weight conditional on the instrument and $\mathbf{x}$ is,

$$E\left[BW_i|z,\mathbf{x}\right] = \mathbf{x}'\boldsymbol{\beta} + E\left[\alpha|\mathbf{x}\right] + E\left[u_i\right] - \gamma E\left[u_j|z\right].$$

Then, for different values of the instrument z and z', the difference in conditional mean of the log birth weight and outcome are:

$$E\left[BW_{ki}|z,\mathbf{x}\right] - E\left[BW_{ki}|z',\mathbf{x}'\right] = -\left[E\left(u_{ki}|z\right) - E\left(u_{ki}|z'\right)\right]\gamma_B$$

$$E\left[Y_{ki}|z,\mathbf{x}\right] - E\left[Y_{ki}|z',\mathbf{x}'\right] = -\left[E\left(u_{kj}|z\right) - E\left(u_{kj}|z'\right)\right]\gamma_B\eta_{B2}$$

Therefore,

$$\eta_{\mathbf{x}} \equiv \frac{E\left[Y_{ki}|z,\mathbf{x}\right] - E\left[Y_{ki}|z',\mathbf{x}'\right]}{E\left[BW_{ki}|z,\mathbf{x}\right] - E\left[BW_{ki}|z',\mathbf{x}'\right]} = \eta_{B2}$$

The above result leads to a 2SLS estimator with the first stage: $\widehat{BW}_{ki} = \widehat{C}\left(\mathbf{x}_{ki}\right) + \widehat{D}\left(\mathbf{x}_{ki}\right)z_{ki}$ where $\widehat{C}\left(\mathbf{x}_{ki}\right)$ and $\widehat{D}\left(\mathbf{x}_{ki}\right)$ are polynomials of $\mathbf{x}_{ki}$. The 2nd stage outcome equation is $Y_{ki} = \widehat{G}\left(\mathbf{x}_{ki}\right) + \widehat{D}\left(\mathbf{x}_{ki}\right)z_{ki}$, where again, $\widehat{G}\left(\mathbf{x}_{ki}\right)$ is a polynomial of $\mathbf{x}_{ki}$. wlog, we let $z = 1$ and $z' = 0$. Then, for all twins with $z_{ki} = 0$,

$$\widehat{BW}_{ki} = \widehat{C}\left(\mathbf{x}_{ki}\right) \xrightarrow{p} E\left[BW_{ki}|0,\mathbf{x}_{ki}\right]$$

and for all twins with $z_{ki} = 1$,

$$\widehat{BW}_{ki} = \widehat{C}\left(\mathbf{x}_{ki}\right) + \widehat{D}\left(\mathbf{x}_{ki}\right) \xrightarrow{p} E\left[BW_{ki}|1,\mathbf{x}_{ki}\right].$$

Then, the IV estimator given $\mathbf{x}$ is

$$\eta_{IVx} = \frac{Cov\left(\widehat{BW}_{ki}, Y_{ki}|\mathbf{x}\right)}{Var\left(\widehat{BW}_{ki}|\mathbf{x}\right)} = \frac{E\left[Y_{ki}|1,\mathbf{x}\right] - E\left[Y_{ki}|0,\mathbf{x}\right]}{E\left[BW_{ki}|1,\mathbf{x}\right] - E\left[BW_{ki}|0,\mathbf{x}\right]} = \eta_{B2}$$

Therefore, by taking the expectations over $\mathbf{x}$, we obtain, $\eta_{IV} \xrightarrow{p} \eta_{B2}$.

[6]Later, we will test the Assumption 3 for specific deviations from the exclusion restrictions that are predicted by the hormone transfer hypothesis. That is, in utero, male hormones are transmitted from female pair and vice versa.

5.2 Hormone transfer hypothesis.

In order for the exclusion restriction to hold, we need to assume that the partner's sex affects the outcome only through its effect on the dichorionic twin's birth weight. If there are hormone transfers between opposite gender twins, then that exclusion restriction won't hold. As discussed above, so far, there has not been any strong evidence in favor of the hormone transfer hypothesis. Even then, below, we explicitly derive the potential bias due to the hormone transfer hypothesis and propose a formal test of the hormone transfer hypothesis in the context of the relationship between birth weight and the infant health outcome, our research interest.

Denote MF be the twin whose own gender is male and the partner gender is female. MM, FM, and FF are similarly defined. Then,

$$E\left[BW_{ki}|MF,\mathbf{x}\right]-E\left[BW_{ki}|MM,\mathbf{x}\right]=E\left[u_{ki}|MF\right]-E\left[h_{kF}|MF\right]-\left[E\left[u_{kj}|MF\right]-E\left[u_{kj}|MM\right]\right]\gamma_B$$

$$\leq-\left[E\left[u_{k}|FM\right]-E\left[u_{k}|MM\right]\right]\gamma_B$$

This is because female hormone transfer from female twin partner reduces birth weight. Also, both the LHS and the RHS are positive. Furthermore,

$$E\left[Y_{ki}|MF,\mathbf{x}\right]-E\left[Y_{ki}|MM,\mathbf{x}\right]=E\left[u_{ki}|MF\right]\eta_h-\left[E\left[u_{kj}|MF\right]-E\left[u_{kj}|MM\right]\right]\gamma_B\eta_{B2}-E\left[h_{kF}|MF\right]\eta_h$$

$$\leq-\left[E\left[u_{kj}|MF\right]-E\left[u_{kj}|MM\right]\right]\gamma_B\eta_{B2}$$

if female hormones transferred to male twin improves health outcomes. Then, because both the LHS and RHS are negative, we obtain

$$\frac{E\left[Y_{ki}|MF,\mathbf{x}\right]-E\left[Y_{ki}|MM,\mathbf{x}\right]}{E\left[BW_{ki}|MF,\mathbf{x}\right]-E\left[BW_{ki}|MM,\mathbf{x}\right]}\leq\eta_{B2}$$

which implies that the possibility of hormone transfer between mixed gender twins would make the gender IV estimator to be the lower bound of the true η_{B2}. Hence, a strong impact of hormone transfers between mixed gender twins could be the real reason for the high gender IV estimated returns to birth weight (i.e. maternal health). That is why it is important to properly assess the impact of the hormone transfers on the estimated birth weight effect.

We next explain the test of the bias that the hormone transfer may cause. To do so, we exploit the asymmetry in the way hormone transfer affects the birth weight and the

infant health outcomes. That is, both male hormone transfer to female twin and female hormone transfer to male twin are nonnegative. Hence, in the twin pair where female birth weight is large relative to male birth weight, on average there is large male (female) hormone transfer from male (female) to female (male) twins. On the other hand, since negative amount of hormone transfers are not feasible, in the twin pair where male birth weight is large relative to female birth weight, hormone transfers are on average small, i.e. close to zero. Hence, by running the twin fixed effect estimates for the above two situations, i.e. the case where demeaned male birth weight is larger than the demeaned female birth weight, and the case where the opposite is true, and test whether those two results are the same, we can test for the bias due to the hormone transfer hypothesis. More formally, with the possibility of hormone transfers from male to female and female to male twins, the birth weight equation becomes

$$BW_{kM} = BW^*_{kM} - h_{kF},\ BW_{kF} = BW^*_{kF} - h_{kM},\ h_{kF} \geq 0,\ h_{kM} \geq 0$$

where BW_{kM} denotes the log birth weight of the male twin of the male female twin pair, and BW_{kF} is also similarly defined. h_F is the female hormone transferred from female twin to male twin, and h_M is the male hormone transferred from male twin to female twin. Furthermore,

$$BW^*_{kM} = \mathbf{X}_k\boldsymbol{\beta} + \alpha_k + u_{kM} - u_{kF}\gamma_B$$

$$BW^*_{kF} = \mathbf{X}_k\boldsymbol{\beta} + \alpha_k + u_{kF} - u_{kM}\gamma_B.$$

Both BW^*_{kM} and BW^*_{kF} are the parts of birth weight that is not affected by the hormones. Both are assumed to be nonnegative. Furthermore, male hormones are not transferred from female to male twins, and female hormones are not transferred from male to female twins. Then, taking the twin difference, we obtain

$$BW_{kM} - BW_{kF} = BW^*_{kM} - BW^*_{kF} - (h_{kM} + h_{kF})$$

The modified outcome equation is defined as

$$Y_{kM} = \mathbf{x}_k\eta_x + \xi_k + u_{kM}\eta_{B1} - u_{kF}\gamma\eta_{B2} + h_{kF}\eta_h + \epsilon_{kM}$$

$$Y_{kF} = \mathbf{x}_k\eta_x + \xi_k + u_{kF}\eta_{B1} - u_{kM}\gamma\eta_{B2} - h_{kM}\eta_h + \epsilon_{kM}$$

where η_h measures the effect of hormone transfer on outcome. by taking the twins difference, we obtain

$$Y_{kM} - Y_{kF} = (u_{kM} - u_{kF})(\eta_{B1} + \gamma\eta_{B2}) + (h_{kM} + h_{kF})\eta_h$$

Since transferred female hormones improve infant health (reduce male infant mortality and hospital days), and transferred male hormones reduce infant health (increase female infant mortality and hospital days), $\eta_h \leq 0$ is expected.

In order to test for the hormone transfer hypothesis, we focus on the mixed gender twins, and subdivide the twins into two groups. Let $\overline{BW}_M$ be the average of the log birth weight of male infants of the mixed gender twins, and $\overline{BW}_F$ be the same for the females of the mixed gender twins. Then, the first group are the twin pairs that satisfy $lnBW_{kMi} - \overline{BW}_M \geq lnBW_{kFi} - \overline{BW}_F$. The second group is the twin pair satisfying $lnBW_{kMi} - \overline{BW}_M < lnBW_{kFi} - \overline{BW}_F$.

Notice that in the 2nd group, the male birth weight is relatively lower than the female birth weight in the same mixed gender pair. This implies that in the 2nd group, higher birth weight difference implies higher birth weight of female infant relative to male infant, which makes it likely that more female hormones were transferred to male twin, thereby reducing the male birth weight and more male hormones were transferred to female twin, thereby increasing female birth weight. Then, if we consider infant mortality, because male hormones increase mortality and female ones reduce it, the higher hormone transfer increases mortality for females and reduces them for males, thereby reduces the male-female difference in infant health. Thus, in order to test the effect of hormone transfer hypothesis on the effect of birth weight on health outcomes of twin infants, we test whether the estimated birth weight effect on infant outcomes using twin fixed effects on group 1 data is the same as group 2. In contrast, in group 1 male birth weight is larger relative to female birth weight. This makes the large female hormone transfer to male twin and large male hormone transfer to females unlikely. Therefore, overall, the transferred hormone levels are smaller, and thus, changes in male to female birth weight is not accompanied by large changes in hormone transfer, which reduces the importance of hormone transfer on the infant health outcome. More formally, taking the twin difference, we obtain

$$BW_{kM} - BW_{kF} = BW^*_{kM} - BW^*_{kF} - (h_{kM} + h_{kF}).$$

Hence, for $A \geq 0$,

$$E\left[h_{kM} + h_{kF} | BW_{kM} - BW_{kF} = A\right] = \frac{\int_0^\infty H f(A+H) g(H) dH}{\int_0^\infty f(A+H) g(H) dH}$$

$$E\left[h_{kM} + h_{kF} | BW_{kM} - BW_{kF} = -A\right] = \frac{\int_0^\infty H f(-A+H) g(H) dH}{\int_0^\infty f(-A+H) g(H) dH}$$

Where $f()$ is the density function of $BW^*_{kF} - BW^*_{kM}$ and $g()$ is a density function of $H = h_{kM} + h_{kF}$, which is nonnegative. Assume that $f()$ has a peak at 0 and is log concave, and assume that $g()$ has a peak at zero and is log concave as well. Then, one can show that for equation (1) decreases with an increase in A and (2) increases with an increase in A. In particular, for equation (1), as A increases, the average H converges to zero. Furthermore, notice that

$$Y_{kM} - Y_{kF} = (BW_{kM} - BW_{kF}) \frac{\eta_{B1} + \gamma_B \eta_{B2}}{1 + \gamma_B} + (h_{kM} + h_{kF}) \left(\eta_h + \frac{\eta_{B1} + \gamma_B \eta_{B2}}{1 + \gamma_B}\right) + \epsilon_{kM} - \epsilon_{kF}.$$

Then, for large positive values of $BW_{kM} - BW_{kF}$, it is more likely that both hormone tranfers from females to males that reduces BW_{kM} and hormone transfers that increases BW_{kF} are small, and thus the conditional mean of $h_{kM} + h_{kF}$ given $BW_{kM} - BW_{kF}$ becomes small, thus,

$$Y_{kM} - Y_{kF} \approx (BW_{kM} - BW_{kF}) \frac{\eta_{B1} + \gamma_B \eta_{B2}}{1 + \gamma_B}$$

and thus, the hormone transfer hypothesis does not cause major bias to the twin fixed effects estimate. On the other hand, for a large negative $BW_{kM} - BW_{kF}$, a decrease of the above birth weight difference would further increase $h_{kM} + h_{kF}$, thus could cause some sizeable bias to the twin fixed effects estimator. This logic suggests a test which compares the twin fixed effects estimator for the twin pairs with relatively large positive values of $BW_{kM} - BW_{kF}$ and the estimator for the twin pairs with large negative values of $BW_{kM} - BW_{kF}$. What we proposed above follows the logic and compares the twin fixed effects for positive birth weight twin difference and the one using the negative twin birth weight difference.

Another possible test is to compare the twins fixed effects estimates for the same gender twins and the estimates for the different gender twins. There are no hormone transfers between same gender twins. Therefore,

$$Y_{k1} - Y_{k2} = (BW_{k1} - BW_{k2}) \frac{\eta_{B1} + \gamma_B \eta_{B2}}{1 + \gamma_B} + \epsilon_{k1} - \epsilon_{k2}.$$

On the other hand, for the mixed gender twins with hormone transfer, we have

$$Y_{kM} - Y_{kF} = (BW_{kM} - BW_{kF}) \frac{\eta_{B1} + \gamma_B \eta_{B2}}{1 + \gamma_B} + (h_{kM} + h_{kF}) \left(\eta_h + \frac{\eta_{B1} + \gamma_B \eta_{B2}}{1 + \gamma_B} \right) + \epsilon_{kM} - \epsilon_{kF}.$$

where $E(h_{kM} + h_{kF} | BW_{kM} - BW_{kF})$ is a decreasing function of $BW_{kM} - BW_{kF}$. Therefore, by running the below regression, we can test the hormone transfer hypothesis.

$$Y_{k1} - Y_{k2} = \beta_0 + \beta_{10} (BW_{k1} - BW_{k2}) + \beta_{11} (BW_{k1} - BW_{k2}) I_{MG,k} + \epsilon_k.$$

where $I_{MG,k}$ is the indicator for the twin pair to have mixed gender. The hypothesis of no hormone transfer is $\beta_{11} = 0$.

5.3 IV Estimates

In table 11, we report the sample statistics of the dichorionic twins. In the first row, we report the sample statistics of the birth weight. As we can see, on average, male twins have higher birth weight than females, which was also the case with the sample statistics of all twins. On the other hand, both males and females have on average higher birth weight when their twin partner is a female than when the partner is male. This indicates in utero competition for maternal resources since male partners have higher birth weight, thus less resources may be left than if the partners were females. Next, we take a look at 1 year and 4 months infant mortality, whose sample statistics are shown in rows 2 and 3, respectively. There, male infant mortality is higher than the femals, and both males and females have lower infant mortality when their partner is female than when their partner is males. It is well known in the medical literature that males have higher infant mortality. Furthermore, the fact that female twin partners make the twins have lower mortality could be either due to the lower resource consumptions of their female partners, or due to the transfer of female hormones to males, which tends to reduce male mortality

or in the opposite case, transfer of male hormones to females, which tends to increase the female infant mortality. The sample statistics of APGAR score and hospital days show similar pattern as well. That is, males are less healthy than females and twins with female partners are healthier than the ones with male partners. The difference between the sample statistics for all twins and the ones for dichorionic twins arise because of the former including both monochorionic as well as dichorionic twins in the same gender twins, but only dichorionic twins in the mixed gender twins, and the same gender twins in general have lower birth weight and lower health status than the mixed gender twins.

In Table 12, we report the results of the dichorionic twins's returns to birth weight. We see that the OLS estimated returns to birth weight on one year infant mortality for the dichorionic twins are similar to the ones estimated for the twins reported in Table 5. The former is -0.173 for males and -0.154 for females, both of which are significant whereas for the latter, it -0.192 and significant for males and 0.180 (0.192-0.0128) for females, which we derived from the female dummy. As in the literature, the twins fixed effects estimated returns to birth weight reported in Table 12, where the one for male-male twins is -0.0249 and insignificant, the one for female-female twins -0.0461 and for the mixed gender twins, -0.0491. These again are similar to the results for the twins in Table 5 which are -0.0387 for males and -0.0201 for females, both of which are much smaller than OLS estimates. On the other hand, the gender IV estimates in Table 12, where the estimates are -0.102 for males and -0.182 for females, both of which are significant. Similar results are obtained for the returns to log birth weight on 28 days infant mortality, again, reported in Table 12. The twins fixed effects estimates of the returns to log birth weight are one third or less of the OLS and gender IV ones. The estimates of the returns to log birth weight on APGAR score also have the similar pattern: large and significant OLS and gender IV estimates:(OLS estimates:1.260 for males and 1.180 for females and Gender IV estimates: 1.666 for males and 1.370 for females) but small and often insignificant twins fixed effects estimates: 0.389 for male only, 0.116 for female only and 0.164 for mixed gender twins. Furthermore, similar results are also obtained for the number of hospital days until the 2nd birth date. The returns to log birth weight for hospital days are quite high for OLS and gender IV estimates, both around -50 to -60, but for the twins fixed effects, they are -3.866 for male only, -2.357 for female only and -2.941 for mixed gender twins. As we

discussed earlier, the low twins fixed effects estimator could be due to the fact that they may be primarily recovering the returns to ability estimates, whereas the OLS and gender IV estimates the returns to maternal environment.

Also, what we can see is that the difference between the twins fixed effects of all males, all females and mixed gender twins are small. Then, if we consider that hormones are transferred only between different gender twins, we can infer that the effect of hormone transfer is small. We next follow the logic and formally test the hormone transfer hypothesis, by running the following twins fixed effects regression.

$$Y_{k1} - Y_{k2} = \beta_0 + \beta_{01} I_{MG} + \beta_{10} \left(BW_{k1} - BW_{k2} \right) + \beta_{11} \left(BW_{k1} - BW_{k2} \right) I_{MG} + \epsilon_k \qquad (1)$$

In Table 13, we report the results. The first two columns are the results for the twins fixed effects estimation for the same gender twins, which include male only and female only ones. The second two columns are for the mixed gender twins. As we can see, the estimated returns to log birth weight are very similar. The same gender estimate being -0.0363 and the mixed gender estimate being -0.0466. In the last two columns, we report the estimates of the equation (1). Then, the null hypothesis of no hormone transfer is $\beta_{11} = 0$, i.e. that the coefficient on the interaction term between the differenced log birth weight and the mixed gender twins dumy to be zero. As we can see from the last two columns, the coefficient of the interaction term is estimated to be -0.0104, which is about a third of the returns to log birth weight estimate, and is insignificant. Therefore, we cannot reject the hypothesis of $\beta_{11} = 0$, no hormone transfer between opposite gender. Similarly, we report results for the returns to birth weight on hospital days. The estimate for the same gender twins is -3.075 and significant, and the mixed gender estimate is -2.246, and again is significant. They are fairly close and as we can see, the coefficient on the interaction term is 0.8290, and insignificant. Therfore, again the hypothesis of no hormone transfer cannot be rejected.

6 Concluding Remarks

In this paper, we have theoretically and empirically analyzed the model of twins where twins interact in utero before birth. First, we theoretically demonstrate that the social

interaction between twins in utero would potentially be a source of bias in the traditional twins fixed effects estimator. This is because the ability of the twin partner to extract maternal resources is likely to negatively affect own resource consumption. Therefore, the ability of the twin partner is also an important component of the maternal environment. Since each twin has a partner with different ability, which is an important component of the maternal environment, maternal environment cannot be differenced away.

To test the existence of the social interaction in utero among multiple birth infants, we use data on triplets. The test rejects the standard model of birth weight, and thus favors the model of social interaction.

We then use an IV strategy to consistently estimate the returns to birth weight that represents the returns to maternal environment. It is much higher than the twins fixed effects estimates obtained in the literature. We then test one likely source of the bias of the IV estimator, the hormone transfer between different gender twins, and the test did not reject the hypothesis of no hormone transfer.

In this paper, we have primarily focused on the returns to birth weight, but in utero social interaction would be a potential source of bias for any other analysis such as returns to schooling. This is because it invalidates the premise that by taking difference of the variables between the twins, one can difference away any observed and unobserved maternal environment and most of the genetic background.

Because of the potential bias, we suggest that researchers should interpret the results of the twins fixed effects estimates more carefully than has been done in the literature. We also believe that the use of instruments or the use of triplets would be useful additional procedures to assess the potential bias of the twins fixed effects estimators. Also, literature on the social interactions, where researchers analyse the interactions among menbers in a group, or the empirical literature on oligopoly, where researchers study the equilibrium interactions of firms in a market, could be helpful in the identification of the degree and the effect of social interaction in utero.

Overall, the potential of a bias and its difficulty to fully deal with it makes the twins analysis less attractive than before relative to the direct empirical analysis of singletons. In that sense, for the returns to birth weight literature, more focus should be given to the research that exploits exogenous or near exogenous variation in birth weight, such as

Almond et. al. (2011A), Maruyama and Heinesen (2014) and others.

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Figure 1: Chorionicity and Zygocity

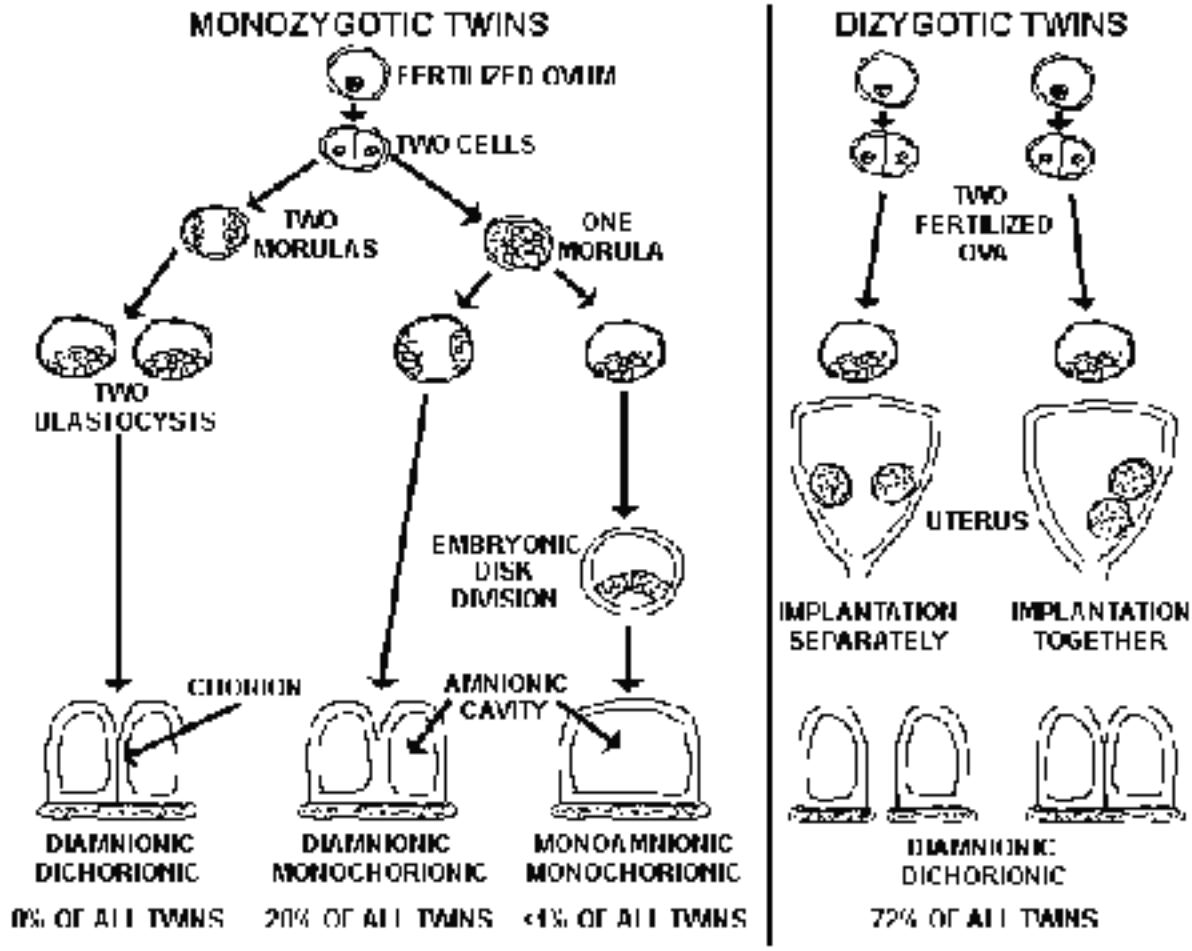

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Table 1: Sample statistics: birth weight

	Birth Weight		Log Birth Weight		Sample Size
M singlet	3552.5	579.3	8.160	.1896	998738
F singlet	3430.1	546.2	8.125	.1844	946562
M in MM	2516.5	619.6	7.790	.3105	21126
M in 1M1F	2588.3	602.1	7.823	.2917	12287
F in 1M1F	2477.0	583.0	7.778	.2960	12287
F in FF	2442.5	581.5	7.764	.2980	20328
M in MMM	1844.6	496.9	7.479	.2968	321
M in 2M1F	1798.9	522.3	7.441	.3543	372
M in 1M2F	1796.7	568.4	7.433	.3767	142
F in 2M1F	1743.9	543.8	7.400	.3917	186
F in 1M2F	1707.0	564.2	7.375	.3944	284
F in FFF	1712.3	447.2	7.406	.2976	300

Table 2: Sample statistics: infant mortality

	Infant Mortality 1 year		Infant Mortality 4 weeks		Sample Size
M singlet	.00390	.06233	.00324	.05679	724668
F singlet	.00323	.05673	.00256	.05058	687351
M in MM	.01950	.13831	.01909	.13683	17070
M in 1M1F	.01340	.11500	.01197	.10875	10595
F in 1M1F	.01501	.12159	.01319	.11409	10595
F in FF	.01412	.11800	.01437	.11903	16428
M in MMM	.02593	.15921	.01572	.12460	270
M in 2M1F	.04573	.20922	.04570	.20911	328
M in 1M2F	.08333	.27754	.07746	.26827	120
F in 2M1F	.03659	.18832	.04301	.20343	164
F in 1M2F	.07083	.25708	.05282	.22406	240
F in FFF	.02564	.15840	.01667	.12823	234

Table 3: Sample statistics: APGAR score

	Apgar Score		Sample
M singlet	9.8566	.64893	991489
F singlet	9.8840	.58974	939937
M in MM	9.6621	1.0890	20752
M in 1M1F	9.6951	1.0202	12121
F in 1M1F	9.7060	1.0182	12108
F in FF	9.6751	1.0956	19978
M in MMM	9.7905	.59769	315
M in 2M1F	9.6713	.94429	356
M in 1M2F	9.4928	1.4859	138
F in 2M1F	9.5	1.3557	180
F in 1M2F	9.4	1.7023	275
F in FFF	9.6062	1.2180	292

Table 4: Sample statistics: Hostpital days

	Hospital days		Sample
M singlet	5.8268	11.817	933959
F singlet	5.2481	10.948	886512
M in MM	16.156	21.693	19226
M in 1M1F	15.637	21.732	11145
F in 1M1F	15.064	22.717	11128
F in FF	15.335	20.419	18514
M in MMM	35.795	33.688	307
M in 2M1F	35.738	29.550	340
M in 1M2F	37.286	30.024	126
F in 2M1F	37.183	33.518	172
F in 1M2F	36.564	34.044	256
F in FFF	38.946	27.989	270

Table 5: Returns to log birthweight on infant mortality (from birth to 365 days)

	Single: OLS	Single: FE	Twin:OLS	Twin: FE	Triple: OLS	Triple FE
lnBW	-0.0807****	-0.137****	-0.192****	-0.0387****	-0.315****	-0.105*
	(0.00157)	(0.00246)	(0.00804)	(0.00875)	(0.0419)	(0.0590)
female	-0.0868****	-0.105****	-0.109	0.144**	-0.270	-0.520
	(0.0178)	(0.0221)	(0.0816)	(0.0709)	(0.354)	(0.419)
lnBW fem	0.0102****	0.0121****	0.0128	-0.0186**	0.0338	0.0681
	(0.00217)	(0.00270)	(0.0104)	(0.00898)	(0.0469)	(0.0554)
R^2	0.0462	0.0735	0.175	0.00442	0.297	0.0115
N	1944760	1576013	66004	66004	1602	1602

Standard errors are in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$, $^{****}p < 0.001$.

Table 6: Returns to log birth weight on 4 week infant mortality (from birth to 28 days)

	Single: OLS	Single: FE	Twin:OLS	Twin: FE	Triple: OLS	Triple FE
lnBW	-0.0715****	-0.123****	-0.181****	-0.0286****	-0.294****	-0.103*
	(0.00152)	(0.00240)	(0.00805)	(0.00784)	(0.0424)	(0.0571)
female	-0.0779****	-0.0899****	-0.121	0.0660	-0.289	-0.496
	(0.0172)	(0.0223)	(0.0813)	(0.0656)	(0.350)	(0.418)
lnBW fem	0.00920****	0.0104****	0.0144	-0.00849	0.0360	0.0648
	(0.00211)	(0.00261)	(0.0103)	(0.00832)	(0.0463)	(0.0552)
R^2	0.0541	0.0870	0.180	0.00256	0.321	0.0146
N	1944703	157944	65995	65988	1602	1602

Standard errors are in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$, $^{****}p < 0.001$.

Table 7: Returns to log birth weight on APGAR score

	Single: OLS	Single: FE	Twin:OLS	Twin: FE	Triple: OLS	Triple FE
lnBW	0.564****	0.835****	1.274****	0.381****	0.682***	0.148
	(0.0107)	(0.0168)	(0.0570)	(0.0654)	(0.218)	(0.236)
female	0.451****	0.515****	0.637	0.725	-3.751**	-0.743
	(0.121)	(0.147)	(0.573)	(0.483)	(1.663)	(1.608)
lnBW fem	-0.0497****	-0.0560***	-0.0748	-0.0896	0.488**	0.0864
	(0.0148)	(0.0180)	(0.0730)	(0.0613)	(0.220)	(0.213)
R^2	0.0296	0.0400	0.117	0.00290	0.210	0.00647
N	1931426	1558992	64959	64548	1556	1524

Standard errors are in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$, $^{****}p < 0.001$.

Table 8: Returns to log birth weight on hospital days before the 2nd birthday.

	Single: OLS	Single: FE	Twin:OLS	Twin: FE	Triple: OLS	Triple FE
lnBW	-21.11****	-25.99****	-54.77****	-4.965****	-72.27***	-15.92**
	(0.174)	(0.237)	(0.677)	(0.773)	(5.077)	(6.777)
female	-11.45****	-11.83****	-6.012	4.164	-26.38	-25.47
	(1.989)	(2.189)	(7.388)	(7.771)	(36.03)	(28.18)
lnBW fem	1.248****	1.269****	0.434	-0.624	3.094**	3.271
	(0.243)	(0.268)	(0.938)	(0.985)	(4.750)	(3.699)
R^2	0.121	0.105	0.456	0.00486	0.534	0.0218
N	1820471	1466348	60013	59396	1471	1401

Standard errors are in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$, $^{****}p < 0.001$.

Table 9: Returns to log birth weight, twins fixed effects for less than 2000 grams.

	365 Infant mortality		28 Infant mortality		APGAR score		Hospital days	
	OLS	FE	OLS	FE	OLS	FE	OLS	FE
lnBW	-0.526****	-0.136****	-0.508****	-0.109****	2.968****	0.904****	-77.16****	-8.293***
	(0.0174)	(0.0323)	(0.0177)	(0.0299)	(0.157)	(0.239)	(2.304)	(3.048)
female	-0.297*	0.164	-0.319*	-0.0102	1.916	2.354*	17.23	36.19
	(0.167)	(0.181)	(0.170)	(0.171)	(1.474)	(1.351)	(25.17)	(36.34)
lnBW fem	0.0383*	-0.0211	0.0415*	0.0223	-0.251	-0.319*	-2.852	-5.063
	(0.0227)	(0.0245)	(0.0231)	(0.0231)	(0.201)	(0.184)	(3.436)	(4.964)
R^2	0.360	0.0123	0.358	0.00725	0.260	0.00745	0.402	0.00861
N	8368	8368	8360	8354	7866	7732	6790	6460

Standard errors are in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$, $^{****}p < 0.001$.

Table 10: Test of social interactions using triplets.

Dep. Variable	bw_{kM2}			bw_{kF2}	
Const	7.4398****	(0.0185)	Const.	7.3794****	(0.0235)
$(bw_{kM1} - bw_{kF3})$	-0.1968**	(0.0811)	$(bw_{kM1} - bw_{kF3})$	-0.1918*	(0.1018)
Sample Size	404		Sample Size	306	

Standard errors are in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$, $^{****}p < 0.001$.

Table 11: Sample Statistics, dichorionic twins

	Male in MM		Male in MF		Female in MF		Female in FF	
Birth weight	2498	(630.6)	2539	(605.4)	2420	(585.5)	2437	(585.6)
Infant mortality (1 year)	0.0178	(0.1324)	0.0132	(0.1140)	0.0141	(0.1177)	0.0129	(0.1130)
Infant mortality (4 weeks)	0.0154	(0.1232)	0.0117	(0.1076)	0.0114	(0.1063)	0.0109	(0.1039)
APGAR score (5 min)	9.669	(1.158)	9.678	(1.074)	9.687	(1.097)	9.683	(1.173)
Hospital days (2 years)	16.28	(20.55)	15.89	(22.20)	15.46	(21.53)	14.74	(18.85)

Table 12: Returns to log birth weight, dichorionic twins.

		OLS		TFE			Gender IV	
		Male	Female	MM	FF	MF	Male	Female
Infant Mortality (365 days)	$lnBW$	-0.173****	-0.154****	-0.0249	-0.0461*	-0.0491***	-0.102***	-0.182**
		(0.0159)	(0.0159)	(0.0283)	(0.0249)	(0.0172)	(0.0321)	(0.0813)
	female dummy					-0.00157		
						(0.00180)		
	R^2	0.201	0.184	0.000929	0.00546	0.00638	0.162	0.146
	N	7506	7356	4090	3940	6832	7258	7136
Infant Mortality (28 days)	$lnBW$	-0.160****	-0.138****	-0.0237	-0.0399*	-0.00526	-0.153***	-0.231****
		(0.0160)	(0.0156)	(0.0259)	(0.0231)	(0.00976)	(0.0516)	(0.0548)
	female dummy					-0.000555		
						(0.00153)		
	R^2	0.202	0.177	0.00115	0.00488	0.00100	0.202	0.109
	N	7504	7353	4086	3938	6830	7504	7353
APGAR score	$lnBW$	1.260****	1.180****	0.389*	0.116	0.164	1.666****	1.370****
		(0.123)	(0.122)	(0.234)	(0.255)	(0.141)	(0.395)	(0.491)
	female dummy					0.0143		
						(0.0211)		
	R^2	0.141	0.114	0.00282	0.000286	0.000634	0.124	0.0916
	N	7410	7268	4014	3878	6718	7181	7067
		OLS		TFE			Gender IV	
		Male	Female	MM	FF	MF	Male	Female
Hospital days (2 year from birth)	$lnBW$	-60.20****	-56.85****	-3.866**	-2.357*	-2.941***	-49.58****	-56.66****
		(1.340)	(1.487)	(1.570)	(1.382)	(0.989)	(7.064)	(10.08)
	female dummy					-0.438***		
						(0.146)		
	R^2	0.647	0.619	0.00725	0.00404	0.00673	0.629	0.146
	N	5935	5729	3239	3048	5287	5935	5729

Standard errors are in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$, $^{****}p < 0.001$.

Table 13: Test of hormone transfer hypothesis.

	same gender		mixed gender		all dich. twins	
Dep Variable	1 year infant mortality					
Const.	0.0	(0.0013)	0.0	(0.0013)	0.0	(0.0013)
mixed gender					0.0	(0.0019)
$\Delta lnBW$	-0.0363****	(0.0080)	-0.0466****	(0.0072)	-0.0363****	(0.0075)
$\Delta lnBW\times$ mixed gender					-0.0104	(0.0108)
R^2	0.0025		0.0062		0.0039	
N	8030		6832		14862	
Dep Variable	Hospital days until 2nd birth day					
Const.	0.0	(0.0866)	0.0	(0.0975)	0.0	(0.0879)
mixed gender					0.0	(0.1300)
$\Delta lnBW$	-3.075****	(0.5192)	-2.246****	(0.5436)	-3.075****	(0.5268)
$\Delta lnBW\times$ mixed gender					0.8290	(0.7505)
R^2	0.0056		0.0032		0.0045	
N	6286		5286		11572	

Standard errors are in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$, $^{****}p < 0.001$.