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Hormonal Evidence Supports the Theory of Selection *in Utero*

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Abstract

Objectives—Antagonists in the debate over whether the maternal stress response during pregnancy damages or culls fetuses have invoked the theory of selection *in utero* to support opposing positions. We describe how these opposing arguments arise from the same theory and offer a novel test to discriminate between them. Our test, rooted in reports from population endocrinology that human chorionic gonadotropin (hCG) signals fetal fitness, contributes not only to the debate over the fetal origins of illness, but also to the more basic literature concerned with whether and how natural selection *in utero* affects contemporary human populations.

Methods—We linked maternal serum hCG measurements from prenatal screening tests with data from the California Department of Public Health birth registry for the years 2001–2007. We used time series analysis to test the association between the number of live born male singletons and median hCG concentration among males in monthly gestational cohorts.

Results—Among the 1.56 million gestations in our analysis, we find that median hCG levels among male survivors of monthly conception cohorts rise as the number of male survivors falls.

Conclusions—Elevated median hCG among relatively small male birth cohorts supports the theory of selection *in utero* and suggests that the maternal stress response culls cohorts in gestation by raising the fitness criterion for survival to birth.

Keywords

hCG; pregnancy; sex ratio; population endocrinology

1. INTRODUCTION

Demographers (e.g., Poston and Bouvier, 2010) assume that birth, death, and migration determine the size and characteristics of geographically defined human populations. Research into the latter two determinants has traditionally studied the transition of

observable humans from living to dead and from settled to migrant. Research into birth, however, has substituted fecundity (i.e, the transition from non-parent to parent) for the transition from conceptus to infant (e.g., Olsen and Andersen, 1999). This substitution persists despite the fact that fewer than 25% of human conceptions yield a live birth (Boklage 1990; Roberts and Lowe, 1975). Understanding the mechanisms by which conceptuses transition to birth should help us better anticipate the characteristics, and perhaps needs, of human populations.

No one argues that conceptuses surviving to infancy represent the whole of their conception cohort. On the contrary, much literature argues that natural selection has conserved mechanisms by which women spontaneously abort fetuses that, if born, would require relatively great maternal investment to survive to reproductive age and yield grandchildren (Baird, 2009; Forbes, 1997; Møller, 1997; Navara, 2010; Stearns, 1987; Trivers and Willard 1973; Wells, 2000).

The relative frailty of sons suggests that this selection *in utero* should affect male more than female fetuses. The few data we have describing the fitness of human gestations show that women in relatively stressful environments who disproportionately bore sons had fewer grandchildren than other women (Gabler and Voland 1994; Lummaa 2001). The comparatively high likelihood of death among males before reaching reproductive age accounted for most of the low fitness of sons. That high likelihood continues in contemporary populations. Male infants have higher death rates than any other male or female age group under reproductive age in all societies and all years for which we have dependable data (Human Mortality Database 2010). This frailty among male infants persists, moreover, despite the fact that mothers invest more energy in sons than in daughters (Clutton-Brock 1991; Helle, Lummaa, and Jokela 2002; Powe, Knott, and Conklin-Brittain 2010).

Evidence for male susceptibility to selection *in utero* includes that the ratio of male to female live births (i.e., secondary sex ratio) falls in populations encountering ambient stressors (Boklage, 1990; Catalano and Bruckner, 2006B; Catalano et al., 2009; Wells, 2000). Such stressors raise the risk of morbidity and death more among male than among female infants (Bruckner et al., 2010; Catalano et al., 2008; Drevenstedt et al., 2008; Fukuda et al., 1998; Lyster, 1974; Navara, 2010; Wells, 2000). Natural selection would conserve any mechanism that enabled a woman to abort a frail male fetus during stressful times because she could then conceive a hardier offspring (Lahdenpera et al., 2004; Lummaa, 2001; Lummaa et al., 1998; Lummaa et al., 2001; Rickard et al., 2007; Wells, 2000).

Selection *in utero* assumes not only that a mother autonomically assess environmental threats to infants, but also that the fetus somehow signal its hardiness to her. Research into fetal signaling suggests a complex, perhaps redundant, set of channels that vary over the course of gestation (Erlebacher, 2010; Sales et al., 2011; Vigano et al., 2003). Among the most frequently tested signals, a relatively low level of gestational human chorionic gonadotropin (hCG) in maternal serum best predicts spontaneous abortion (Cole, 2010; Dugoff et al., 2004; Goetzl et al., 2004; Jelliffe-Pawlowski et al., 2010; Kirkegaard et al., 2010; Sasaki et al., 2008). As important, and consistent with the relative frailty of sons compared to daughters, gestations of males yield endemically lower hCG levels in maternal serum than those of females (Cowans et al., 2009). This difference has been observed as early as the 4th week of gestation and continues to birth among surviving fetuses (Cowans et al., 2009, Yaron et al., 2001).

The association between low hCG and premature termination of gestation appears so strong that clinicians have proposed, and human subjects regulatory mechanisms have approved,

use of the hormone to treat women with histories of spontaneous abortion (Devaseelen, Fogarty, and Regan 2010). The low efficacy of these treatments leads to the inference that low hCG signals, but does not cause, low fetal fitness.

The literature suggests two mechanisms by which hCG signaling could connect ambient stressors to male fetal loss. The first, or shifting-distribution mechanism, assumes that the maternal stress response includes elements (e.g., production of glucocorticoids, changes in diet) that disrupt normal gestation and thereby damage fetuses (Barker, 1994; Gluckman et al., 2008). This assumption arises, at least in part, from the epidemiologic literature reporting that stressed mothers exhibit elevated risk of spontaneous abortion and other adverse birth outcomes (Nakamura et al., 2008). Under this assumption, stressful times would shift downward the distribution of fitness, and presumably hCG, in gestational cohorts. Fetuses in ranks near but above the criterion for spontaneous abortion in benign times would fall below the criterion in stressful times. Shifting the distribution of ranks downward moves more males than females below the criterion because males disproportionately inhabit the lower ranks of fetuses on fitness (Lahdenpera et al., 2004; Lummaa, 2001; Lummaa et al., 1998; Lummaa et al., 2001; Rickard et al., 2007; Wells, 2000).

The second, or shifting-criterion mechanism, assumes that the maternal stress response includes raising the criterion for abortion, causing the termination of gestations that would have survived had the environment remained comparatively benign (Forbes, 1997; Stearns, 1987; Wells, 2000). An upward shifting criterion predicts an effect on the secondary sex ratio similar to that of a downward shifting distribution. More male than female fetuses expire when the criterion for spontaneous abortion rises because males disproportionately inhabit the lower ranks of fitness (Catalano and Bruckner, 2006b; Catalano et al., 2009).

Although the shifting-distribution and shifting-criterion mechanisms both predict lower secondary sex ratios in stressed populations, they imply very different circumstances for males who survive to birth. A downward shifted distribution implies a "damaged" cohort in which males who survive to birth will, on average, suffer more mortality before and during reproductive age than those in other cohorts. An upward shifted criterion implies a "culled" cohort in which males surviving to birth will, on average, exhibit less mortality before and during reproductive age than males in other cohorts.

Two attempts to empirically determine which prediction better fits available demographic data appear in the literature. One test found, consistent with the shifting-criterion mechanism, an inverse association between male cohort life expectancy (i.e., realized life span) and the sex ratio of annual birth cohorts in Sweden from 1751 through 1912 (too many males remain alive in post-1912 birth cohorts to estimate cohort life span) (Catalano and Bruckner, 2006b). A second test replicated the Swedish results in Denmark as well as in England and Wales (Catalano and Bruckner, 2006a).

Although these studies find demographic patterns consistent with a shifting-criterion, they focus on only one prediction implied by the mechanism – that the secondary sex ratios of birth cohorts will vary inversely with male longevity. The external validity of these tests remains, moreover, unknown and difficult to estimate given their dependence on scarce data measuring the life span of cohorts born, at latest, nearly a century ago.

We offer an alternative test rooted in the tradition and methods of population endocrinology (Silverin, 1984). Unlike the demographic tests based on male longevity, our test focuses on an observable characteristic of contemporary gestations for which the competing mechanisms make opposite and testable predictions. The shifting-criterion mechanism predicts that the median level of maternal serum hCG in monthly conception cohorts of

males who survive to birth will, adjusting for hCG levels among females in the cohort and other correlates, vary inversely with the number of live born males in the cohort. The shifting-distribution explanation predicts a positive association.

California's Genetic Disease Screening Program assesses fetal risk of chromosomal abnormalities and birth defects using blood analytes, including second trimester maternal serum hCG. We linked the hCG scores from the screening program with data from the California Department of Public Health birth registry for the years 2001–2007. We determine whether the shifting-criterion or shifting-distribution prediction better describes 2,057,433 singleton births from monthly gestational cohorts conceived in California from May 2001 through March 2007.

2. MATERIALS AND METHODS

2.1 Data

All women in prenatal care by the 140th day of gestation have, by law, the opportunity to participate in California's Genetic Disease Screening Program (GDSP). The program assesses the risk of chromosomal abnormalities using several blood analytes, including maternal serum hCG, in an annual average of 350,000 gestations. GDSP contracts with regional private laboratories to analyze blood samples from women who opt for testing. The labs follow a uniform assay protocol that uses an automated analytical system. They submit results to GDSP for quality control, risk calculation, and communication of results to medical providers (Cunningham and Tompkinison, 1999; Kazerouni et al., 2009).

We linked the hCG scores from the screening program with data describing live born infants (i.e., survivors of gestation) from the California Department of Public Health birth registry for the years 2001–2007. The probabilistic linking procedure used combinations of mother's/father's/child's names (first two letters, NYSIIS phonetic codes, and whole names), mother's social security number, street address, phone numbers, residential ZIP code, mother's birth date (year only and whole dates), time of birth, facility name, and child's birth date/estimated date of delivery.

2.2 Variables

We calculated sex-specific median levels of maternal serum hCG (measured in international units per liter), assayed in the 14th through 21st weeks of gestation, among live-born members of each of the 71 monthly cohorts conceived from May 2001 through March 2007. We used gestational age, derived primarily from ultrasound tests at the time of blood draw, to assign pregnancies to month of conception. If ultrasound results were not available, gestational age was estimated from date of last menstrual period or physical exam (Dietz et al., 2007; Pearl et al., 2007). Approximately 65% of live births linked to prenatal screening records, yielding 2,057,433 singleton births. We included only singleton gestations in the study because conventions for assigning gestational hCG to survivors of multiple gestations have not been set, maternal serum screening of multiple gestations appears much less common than for singleton gestations (53% compared to 65%), and sex-specific selection *in utero* among twins appears different than that among singletons (Catalano et al., 2009).

We used the median hCG score among live male births from each of the 71 conception cohorts as our dependent variable. We used the median, rather than mean, score because the shifting-criterion mechanism assumes that the range of male scores, not just the distribution of males across a fixed range of scores, will vary inversely with the number of live born males in conception cohorts.

We derived our independent variable from the number of live born males from each of the monthly conception cohorts. As described below, satisfying the assumptions of statistical tests required us to transform the time series of male births into the number of males above or below values expected from trends, cycles, and the tendency to oscillate or to remain elevate or depressed after high or low values.

We included nine covariates in our test equations. We included median hCG for live born females to ensure that no hCG measurement artifact (e.g., changes in test kits over time) or other confounding variables affecting both male and female gestations could induce false rejection of the null hypothesis. Because hCG levels vary over gestation, we included mean gestational age, in days, at time of maternal blood draw in each cohort. We included percentage of insulin-dependent mothers of male fetuses in each cohort because diabetes reportedly increases the production of hCG. We also included mean maternal weight at time of the hCG test and mean maternal age at birth for mothers of males in each cohort. The test equation also included cohort percentage of mothers reporting Hispanic, non-Hispanic white, non-Hispanic African American, and Asian American race or ethnicity.

2.3 Tests

Statistical tests of association essentially measure the degree to which two variables differ from their expected values in the same cases. The tests typically assume that the expected value of any observation is the mean of all observations. Variables measured over time, however, often violate this assumption because they exhibit "autocorrelation" in the form of secular trends, cycles, or the tendency to remain elevated or depressed, or to oscillate, after high or low values. The expected value of an observation in such a series is not the mean of all observations but rather the value predicted by autocorrelation.

Consistent with earlier research in this field (Catalano et al., 2009), we use Box-Jenkins ARIMA modeling to detect and model autocorrelation in our independent and, after adjusting for covariates, dependent variable (Box et al., 1994). More specifically, we test our hypotheses through the following steps.

1. We used Box-Jenkins ARIMA routines to decompose the number of males in the cohorts into statistically expected and residual (i.e., observed minus expected) values.

2. We regressed the median hCG score among live male births in each of the 71 conception cohorts on the nine covariates described above.

3. We applied Box-Jenkins routines to the residuals of the model estimated in Step 2 to detect autocorrelation and added indicated ARIMA parameters.

4. We estimated the model formed by adding, as a predictor variable, the residuals yielded in Step 1 to model developed in Step 3. We inspected the residuals from the model for autocorrelation and, if we detected any, added the appropriate ARIMA parameters.

3. RESULTS

Table I compares live births from the screened gestations with all births in the test years. The groups appear similar although the screened group had a lower proportion of older women and a higher proportion of privately insured women. These differences probably result from older women being referred directly for amniocentesis rather than for the blood screening test, and uninsured women less frequently obtaining prenatal care and therefore less likely receiving any screening.

Our test proceeded through four steps. First, we used Box-Jenkins ARIMA modeling (Box et al., 1994) to decompose the number of males (in 100's) in the conception cohorts who survived to birth into expected and residual values. The following model best fit the series.

$$\nabla_{12} Y_t = \frac{\left(1 + .4596B^3 - .6471B^{12}\right)}{(1 - .5329B)} a_t \qquad [1]$$

Y_t is the number of males from the cohort conceived in month t that survived to birth. ∇_{12} is the difference operator indicating the variable was transformed subtracting its value at time t from its value at t-12 and assigning the difference to time t-12. B^n is the backshift operator or value of the variable at time t-n. The model implies that the number of males surviving from cohorts conceived in months t-12, t-3, and t-1 predict the number surviving from the cohort conceived in month t.

Second, we regressed median level of gestational hCG among male survivors of conception cohorts on nine covariates (median hCG for live born females, mean gestational age for each cohort, percentage of insulin-dependent mothers, mean maternal weight in pounds within one month of the hCG test, mean maternal age at delivery, and cohort percentage of mothers reporting Hispanic, non-Hispanic white, non-Hispanic African American, and Asian American race or ethnicity) and found that median hCG among female survivors of the conception cohorts, mean gestational age at time of blood collection, and percent insulin dependent mothers significantly (i.e., p<0.05, two-tailed tests) shared unique variance with the dependent variable. We carried these and other covariates forward to the final test to ensure control of their combined association with the dependent variable.

Third, we applied Box-Jenkins ARIMA routines to the residuals of the model estimated in step 2 and detected a pattern in which high or low median levels of hCG among males repeated, adjusting for covariates, with similar, but smaller, high or low values 16 months later. This pattern implies that we could not predict hCG for cohorts conceived before September 2002. Our final test, therefore, used 55 cohorts conceived from September 2002 through March 2007. These cohorts yielded 795,631 live male, and 760,000 live female, births.

Figure 1 shows the observed and expected values from the combined regression and ARIMA modeling of residuals. As suggested by Table II, the close "fit" between the observed data and the regression line arises, at large part, from the fact that gestations of males and females share many causes (e.g., measurement methods such as "kit" type) of temporal variation in hCG.

The final step required estimating the equation formed by adding the residuals from equation 1 above (i.e., the ARIMA model for the number of males surviving to birth from each conception cohort) to the equation derived in steps 2 and 3. As shown in Table II, the coefficients for median hCG in female gestations, mean maternal weight, and percent insulin dependent mothers exceeded twice their standard errors, as did the negatively signed estimate for the unexpected number of males in the cohort (i.e., the residuals from equation 1).

Consistent with the shifting criterion argument, we found an inverse "dose response" in which the median level of gestational hCG among male survivors of conception cohorts differs from the level expected from covariates and autocorrelation by 0.03 international units per liter (IU/L) with each 100 more or fewer surviving males than statistically expected. The monthly differences from the expected number of males ranged from 478

fewer to 514 more, implying corresponding hCG differences ranging from 0.1542 to −0.1434 IU/L.

We assessed the robustness of our findings in several ways. We applied routines devised to determine if outliers in the dependent variable could have inflated the confidence intervals of our residuals, thereby leading to false acceptance of the null for several covariates (Chang et al., 1988; Hillmer, 1984). We detected no outliers. Next, we used an iterative process to pare non-significant (i.e., p > 0.05; 2-tailed test) parameters from the final equation (Liu and Hudak, 1992). The four parameters with coefficients twice their standard errors in the full test, as well as the constant and ARIMA parameter, survived.

We also estimated the sensitivity of our findings to several artifacts of our test. We re-estimated the equation using mean, rather than median, hCG for the dependent variable and the covariate for females. Results did not change. In two separate tests, we made the ratio of male to female hCG as well as their difference the dependent variable and removed female hCG from the covariates. The results from both tests supported the shifting criterion hypothesis. We also estimated the final equation without the 16-month autoregressive parameter. Results again supported the shifting criterion hypothesis, as they did when we transformed the dependent variable and covariate for females to their natural logarithms.

4. DISCUSSION

Our results contribute to the debate over whether temporal variation in selection *in utero* results primarily from a shifting distribution of fitness or a shifting criterion for spontaneous abortion (Catalano and Bruckner, 2006b). This study represents the first test of selection *in utero* using biomarkers during pregnancy, and the results support the shifting criterion mechanism. Unlike earlier research based on vital statistics describing cohorts born nearly a century ago, our test uses observable components of the mechanisms in contemporary populations. More specifically, we argue that the mechanisms imply different levels of hCG measured during gestation, in contrast to differences in longevity, which require many generations to observe and which may respond to many other exogenous circumstances (e.g., wars, political change, weather, medical care) (Catalano et al., 2008; Elder et al., 2009; Nobles et al., 2010; Nolte et al., 2002).

Strengths of our analysis include the very large and ethnically diverse population from which we obtained maternal serum hCG during pregnancy. In addition, unlike studies that use clinical samples, our use of second trimester hCG from the majority of pregnancies in California allows us to gauge the population-level manifestation of selection *in utero*. Indeed no other study, to our knowledge, in population endocrinology has as many participants. Our methods, moreover, rule out the rival explanation that an unmeasured factor that affects hormone levels in all gravid mothers accounts for the findings because we controlled for hCG of female gestations in all our tests.

Using California data has the added benefit of testing our hypothesis in a population and period in which earlier research found the overarching association, assumed by the shifting criterion argument, between population stressors and indicators of selection *in utero*. This research reports that secondary sex ratios fell in birth cohorts exposed in approximately the 20th week of gestation to terrorist events (Catalano et al., 2005b) and unexpected contraction of the labor force (Catalano et al, 2010). The sex ratio of fetal deaths, moreover, reportedly increased soon after unexpected contraction of the labor force (Catalano et al., 2005a).

Weaknesses in our study include that the "dose response" relationship described above has little intuitive clinical meaning. We believe, however, that the findings contribute to the broader debate over whether contemporary society subjects pregnant women to stress loads

that affect future generations (Center on the Developing Child, 2007). The popular manifestation of that debate appears much influenced by the argument that maternal stress "programs" fetuses for later life morbidity (Paul, 2010). Our findings, however, suggest that the maternal stress response may, via selection *in utero,* raise the average fitness of exposed cohorts. Although stressful environments increase the risk of morbidity among individuals directly exposed, it remains unclear how such exposures affect the biology of generations *in utero* during times of stress.

Our findings do not apply to the subset of older mothers at increased risk of delivering chromosomally abnormal infants, because many, although not all, of these mothers seek amniocentesis rather than blood screening tests. Gestations among these women likely exhibit hCG signatures different from those we observed because the chromosomal abnormalities found more frequently among offspring of older mothers often yield "dishonest" hCG signals (Cole, 2010; Knofler, 1999).

Our findings cannot contribute to the debate over which of several mechanisms may affect the primary sex ratio (ratio of male to female conception). These mechanisms include the effect of stress on the quality of sperm (Fukuda et al., 1998), the frequency of coitus (James, 1971; Renkonen, 1970; Segraves, 1998), and parental hormonal concentrations at the time of conception (James, 1996; James, 2004; James, 2008a; James, 2008b; James, 2010).

Whether readers find our findings compelling depends on the relative parsimony of our argument and its rivals. We cannot, for example, rule out that some mechanism may decrease the primary sex ratio (i.e., sex ratio at conception) as well as raise the level of hCG among males in our conception cohorts. Such a mechanisms would, of course, have to explain not only our findings, but also those that hCG predicts spontaneous abortion, that males from low sex ratio birth cohorts live longer than males in high sex ratio cohorts, that monthly secondary sex ratios do not vary with population stressors 8, 9, or 10 months earlier but rather with those 3 or 4 months earlier, and that the fetal death sex ratio varies positively with population stressors. We can think of no theory other than a shifting criterion that would parsimoniously explain these observations.

We extend the shifting criterion hypothesis to predict patterns in population endocrinology. We find support for the prediction in observed data. We argue that the same extension of the shifting distribution hypothesis makes a different prediction not supported by the data. We can, however, imagine a rival extension of the shifting distribution hypothesis that could predict our findings. That rival makes five assumptions. First, hCG signals genotypic but not phenotypic hardiness. Second, phenotypic hardiness emerges as fetal damage, induced by the maternal stress response, decreases genotypic hardiness. Third, phenotypic hardiness has its own signal as yet undetected by science. Fourth, the ranking of gestations on the two signals correlate strongly even if the distance between their medians grow (i.e., as the phenotypic signal declines with stressors on the population). Fifth, the mechanisms that trigger spontaneous abortion "read" the phenotypic signal and compare it to a constant (i.e., not shifting) survival criterion. This combination of circumstances could produce the result that we attribute to a shifting survival criterion. We note, however, that a shifting criterion would appear the more parsimonious explanation because it need not assume circumstances for which we have no evidence (e.g., signals of phenotypic hardiness, rank correlations with hCG), and it alone explains other reports that males from low sex ratio cohorts enjoy extended longevity (Catalano and Bruckner, 2006a; b; Catalano et al., 2008) likely due to low death rates before reproductive age (Catalano et al., 2009b; Bruckner and Catalano, 2007).

Future research should search for phenotypic signals of fetal hardiness. Research should, moreover, directly test the hypothesis that males from high hCG gestations suffer lower morbidity and mortality before reproductive age than other males. This hypothesis arises from combining our findings with earlier reports that males from low sex ratio cohorts exhibit relatively low death rates before reproductive age (Catalano et al., 2009b; Bruckner and Catalano, 2007) and comparatively high life expectancy (Catalano and Bruckner, 2006a; b; Catalano et al., 2008).

We used population endocrinology (Silverin, 1984) to determine whether a shifting distribution or shifting criterion better predicts the level of maternal serum hCG in conception cohorts of male infants. Our results support the shifting criterion and, in doing so, provide an example of how natural selection affects which potential humans transition into birth and thereby into the population further determined by death and migration.

Acknowledgments

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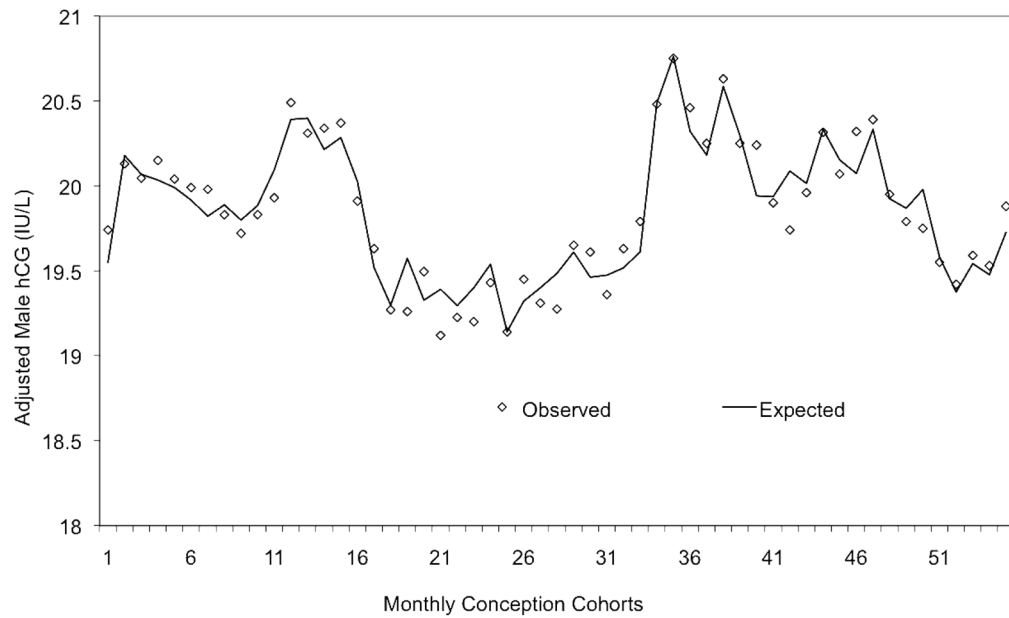
Figure 1.
Observed and expected median values of second trimester serum hCG (in IU/L), adjusted for potentially confounding covariates and autocorrelation, in pregnancies resulting in live born males by monthly conception cohort (N=55 months starting September 2002 and ending March 2007)

Table 1

Comparison of study sample to all California births, 2002–2007.

	Study sample (May 2001-March 2007)	California births 2002–2007
Singletons	2,057,443	3,203,026
% Male	51.1	51.2
Race/ethnicity		
% White	26.8	29.0
% African American	5.5	5.6
% Asian	12.1	12.2
% Hispanic	53.9	51.5
Maternal Age (years)		
% <20	8.8	9.6
% 20–24	22.9	23.2
% 25–34	55.4	50.6
% >34	12.9	16.7
Payment source (delivery)		
% Public	45.5	45.6
% Private	51.4	48.7
% Uninsured	1.1	2.3
% Other/Unknown	2.0	3.3
Month prenatal care began		
% 1–2	72.1	66.9
% 3–4	23.5	23.9

Table 2

Coefficients and standard errors for equation predicting median maternal serum hCG (IU/L) among male survivors to birth from 55 monthly gestational cohorts conceived in California from September 2002 through March 2007.

	Coefficient	Standard Error
Constant	6.8783	7.9337
Observed minus expected male survivors of conception cohort(in 100's)	−.0198 *	.0081
Median hCG (in IU/L) among female survivors of conception cohort	.8236 **	.0573
Mean gestational age (in days) at blood draw	.6852	.4331
Mean maternal age at birth	.1104	.2111
Mean maternal Weight within one month of blood draw	−.1168 **	.0411
Percentage insulin dependent mothers	77.0579 *	30.4587
Percentage Hispanic mothers	−.9969	1.5888
Percentage non-Hispanic white mothers	−2.6322	2.6285
Percentage non-Hispanic African American mothers	7.9315	7.8103
Percentage Asian mothers	−1.6396	5.0770
Autoregressive parameter at 16 months	−.66823 **	.1394

*
p < .05; 2-tailed test

**
p< .01; 2-tailed test