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Fetal death sex ratios: a test of the economic stress hypothesis

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Background The ratio of male to female live births (i.e. the sex ratio) reportedly falls when populations suffer rare and extreme ambient stressors such as the collapse of national economies. This association has been attributed to the death of male fetuses and to reduced conception of males. We assess the validity of the first of these mechanisms by testing the hypothesis that the fetal death sex ratio varies positively over time with the unemployment rate. Using the unemployment rate also allows us to determine if ambient economic stressors less extreme than collapsing national economies affect the fetal death sex ratio.

Methods We test our hypotheses by applying time-series methods to monthly counts of fetal deaths and the unemployment rate from the state of California beginning January 1989 and ending December 2001. The methods control for trends, seasonal cycles, and other forms of autocorrelation that could induce spurious associations.

Results Results support the fetal death mechanism in that male fetal deaths increased above the values expected from female deaths and from history in months in which the unemployment rate also increased over its expected value.

Conclusions Our findings suggest that ambient stressors as common as increasing unemployment elevate the risk of fetal death among males. We discuss the social, economic, and health costs borne by parents and communities afflicted with these fetal deaths.

Keywords Fetal death, sex ratio, unemployment rate, population distress

The ratio of male to female live births (i.e. the sex ratio) reportedly falls when populations suffer ambient stressors. The British sex ratio declined during London's infamous air pollution episode in 1952.¹ The sex ratio dropped in a population potentially exposed to a widely reported release of toxic pollutants in Italy.² The sex ratio among residents of Kobe, Japan fell following the earthquake of 1995.³ The sex ratio in East Germany reached a historic low with the collapse of that state's economy in 1991.⁴

The literature offers three mechanisms to explain the associations such as those listed above. The first, or fetal death mechanism, assumes that maternal stress triggers processes that, for reasons as yet unclear, jeopardize male more than female fetuses. Suspicion centres on the possibility that the faster growth rate of male fetuses increases their risk of abnormalities that lead to fetal death.^{5–8} Reports that male

fetuses react more strongly than females to the glucocorticoids produced by stressed mothers support this mechanism.⁹ These hormonal challenges may act as 'tests' of male fetal development that become more frequent and more sensitive during stressful periods. Such testing would be consistent with the long-standing yet controversial argument that natural selection has conserved mechanisms that allow stressed mothers to spontaneously abort weak male fetuses whose reproductive success would fall below that of daughters.^{6,10,11}

The second mechanism offered as an explanation for the findings alluded to the above assumption that stressed fathers produce sperm less likely to yield males. Sperm samples taken before and after the Kobe earthquake, for example, reportedly showed lower motility one month after the disaster.¹² Because of greater mucosal penetrability of Y sperm compared with X sperm, it is believed that reduced motility may attenuate the advantage of Y sperm to reach the ovum, and therefore reduce the chance of conceiving males.¹³

A third mechanism arises from the fact that both men and women seek coitus less frequently when stressed. Indeed, the

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defining characteristics of the stress response in humans include reduced libido.¹⁴ Researchers have reported that coitus early in the menstrual cycle favours the conception of males whereas that at the middle stage (i.e. ovulation) yields more females.^{15–17} Circumstances, including stress, that decrease coital frequency should reduce the likelihood of conception early in the menstrual cycle and, therefore, could reduce the sex ratio of fetuses in gestation.

The existing research does not offer empirical evidence that discriminates among these three mechanisms. Empirically testing these mechanisms would not only add to our understanding of temporal variation in the sex ratio, but also clarify how much attention this variation deserves from the public health profession. Most attention would seem warranted if the fetal death mechanism were at work because, unlike reduced libido or lower sperm motility, fetal deaths induce somatic and psychological pain in parents, as well as sympathetic pain in families and the community.

We offer a direct test of the fetal death mechanism. More specifically, we test the hypothesis that the monthly incidence of male fetal deaths in California will be higher than expected from its own history and from the incidence of female fetal deaths when the unemployment rate in the State increases over the level expected from secular trends and seasonal cycles.

We chose the widely reported monthly unemployment rate as our independent variable because much theory as well as empirical research report that populations yield increased incidence of stress related illness when the economies that support them stagnate or contract.¹⁸ Moreover, the theoretical¹⁰ and empirical literature concerned with the secondary sex ratio cites economic contraction as an environmental antecedent of lower ratios.^{4,19}

This ‘economy as stressor’ literature invokes the long standing theory that adapting to changes in the social, organizational, and physical environment can induce the physiological stress response and its health sequelae.²⁰ Much research makes these changes operational in the form of ‘stressful life events’.²¹ Empirical study has separated these events into ‘undesirable job and financial events’ (e.g. loss of work hours and income, inability to pay bills, and foreclosures) and others and found that the first occur more frequently when economic indicators fall.²² More importantly for our work, the research also reports that the experience of undesirable job and financial events increases the likelihood of subsequently experiencing other stressors such as changing residences, trouble with spouse or partners, and problems with children.²³

Methods

Our test period, determined by the availability of monthly fetal death data based on constant definitions, began January 1989 and ended December 2001 (the most recent data available at the time of the analyses). We acquired the data from the Maternal and Child Health Branch of the State of California Department of Health Services.

The State of California defines a fetal death as a ‘death prior to the complete expulsion or extraction from its mother of a product of human conception. The death is indicated by the fact that the fetus does not breathe or show any other evidence of

life such as beating of the heart, pulsation of the umbilical cord, or definite movement of voluntary muscles.’²⁴

California’s Health and Safety Code requires that ‘each fetal death, excluding induced abortions, in which the fetus has advanced to or beyond the twentieth week of uterogestation shall be registered with the local registration district within eight days following the delivery and prior to any disposition of the fetus. Only fetal deaths where the fetus has advanced to or beyond the twentieth week of uterogestation shall be registered.’²⁴ We cannot test our hypothesis for fetal deaths before the 20th week of gestation because these are not recorded.

Research from the National Center for Health Statistics suggests that fetal deaths, especially those during the 20–27 weeks of gestation, remain underreported.^{25,26} No evidence, however, suggests a gender bias in the reporting or under-reporting of fetal deaths.

Although some variables on the fetal death records frequently have missing values (e.g. parental occupation and prenatal care),²⁷ fetal gender rarely has no score. From 1989 to 2001, only 228 of 48 551 (0.47%) reported fetal deaths were classified as undetermined sex. To avoid potential misclassification of sex status, we excluded these deaths from the analysis.

The monthly unemployment rate is defined as the percentage of the labour force looking for but not having a job. The labour force is the sum of employed and unemployed persons. Unemployed persons include those looking for work but not having a job. The US Bureau of Labor Statistics estimates both populations from the monthly Current Population Survey.²⁸

Analyses

We, of course, cannot randomly assign couples of childbearing age to economies that we can manipulate. Therefore, tests such as ours must be observational. In essence, such tests are based on whether the observed number of male fetal deaths exceeds the statistically expected value when the unemployment rate moves away from its secular trend and seasonal cycle. Researchers typically assume that the statistically expected value of any variable is its mean. Time series, however, often exhibit trends, cycles, and the tendency to remain elevated or depressed after high or low values. These patterns complicate observational tests because the expected value of a patterned series is not its mean. No one, in other words, would expect the next value of a series to be its mean if previous values exhibited a cycle or trend.

Researchers dating at least to Fisher and his 1920 study of crop variation have solved the autocorrelation problem by ‘decomposing’ time series into temporally predictable and residual components.²⁹ This approach removes patterns from the dependent variable before testing the effect of avoiding spurious associations due to shared trends and cycles.

Epidemiologists have offered an alternative method that measures the dependent variable in a comparison population and uses the series as a control variable in the test equation.³⁰ This provides the benefit of the purely empirical approach in that it removes patterns in the dependent variable induced by forces also at work in the comparison population. The approach also controls unspecified variables that affect both populations but exhibit no patterns.

We combine the decomposition and comparison population approaches. We model male fetal deaths as a function of female fetal deaths. We inspect the residuals of the model for patterns. We assume that forces affecting males, but not females, induce any remaining patterns. We remove remaining patterns in the time series of male fetal deaths by including, as in the decomposition approach, the appropriate lags of the dependent variable among the predictors. We then add the unemployment variable to the equation. The coefficients of the unemployment variable are net of shared patterns as well as of any confounding effects of phenomena that affect both males and females but exhibit no patterns.

The tests proceeded through the following steps.

- (1) We modelled male fetal deaths as a function of female fetal deaths.
- (2) We inspected the residuals from step 1 for a mean and patterns. We added a constant to the equation if the mean was twice its standard error. Patterns, if any, in the residuals from step 1 were identified and specified using the augmented Dickey–Fuller test³¹ to detect for secular trends and seasonal cycles and the strategy attributed to Box and Jenkins³² as well as Ljung and Box³³ to test for and model tendencies to remain elevated or depressed, or to oscillate, after high or low values. The strategy, Auto Regressive, Integrated, Moving Average (i.e. ARIMA) modelling, allows any of a large family of possible models to be empirically fit to serial measurements. ARIMA models mathematically express various filters through which series without patterns can pass. Each filter imposes a unique pattern. The Box–Jenkins approach uses a model-building process by which the researcher infers the filter that imposed the observed pattern. The differences between the values predicted by the inferred model and the observed series are assumed to be the unpatterned values that were filtered.
- (3) We removed secular trends and seasonal cycles, both confirmed with the augmented Dickey–Fuller test,³¹ from the unemployment rate over the test period. We removed trends by taking the first differences of the series (i.e. values at month $t - 1$ subtracted from values at month t) and removed seasonal cycles by taking the 12th differences (i.e. values at month $t - 12$ subtracted from values at month t).
- (4) We added the detrended and deseasonalized unemployment rate to the equation resulting from steps 1 and 2. The test equation that emerges from step 4 is as follows.

$$\nabla^d Y_t = \omega_0 \nabla^d X_{1t} + \omega_1 X_{2t} + \omega_2 X_{2t-1} + \frac{(1 - \theta_1 B - \theta_2 B^2 - \dots - \theta_q B^q)}{(1 - \phi_1 B - \phi_2 B^2 - \dots - \phi_p B^p)} a_t$$

where ∇^d is the difference operator that indicates a series was differenced at lag d (i.e. values at t subtracted from values at lag $t-d$) to remove secular trends or cycles detected by the Dickey–Fuller test.³¹

Y_t is the number of male fetal deaths in month t .
 X_{1t} is the number of female fetal deaths in month t .
 ω_0 is the estimated parameter for female fetal deaths.
 X_{2t} is the detrended and deseasonalized unemployment rate in month t .

ω_1 is the estimated parameter for the unemployment variable.

X_{2t-1} is the detrended and deseasonalized unemployment rate in month $t - 1$.

ω_2 is the estimated parameter for the unemployment variable in month $t - 1$.

B^n is the ‘backshift operator’ that yields the value of the unemployment series at month n . We set $n = 2$ to ensure that any associations that took up to 2 months to appear would be discovered.

θ is the moving average parameter.

ϕ is the autoregressive parameter.

B^p and B^q are backshift operator for the autoregressive and moving average patterns, respectively. Seasonality in a series would result in p or q values of 6 or 12.

a_t is the error term for month t .

- (5) We estimated Equation 1 and inspected the error terms for temporal patterns. If any were found, we added ARIMA parameters to the equation and estimated the resulting equation.
- (6) We measured the association between the error terms of the equation and the unemployment variable to ensure they were not related.
- (7) We rejected the null hypothesis if the coefficient for the unemployment variable exceeded twice its standard error.

Results

Table 1 shows the mean, standard deviation of the mean, and minimum and maximum values for the variables for the 156 test months. The mean of monthly male and female fetal deaths was 151 and 139, respectively.

Steps 1 and 2 above yielded the following equation for male fetal deaths.

$$Y_t = 1.081X_{1t} + (1 - 0.1972B^{11})a_t$$

Both parameters were at least twice their standard errors and the Ljung–Box test³³ confirmed the residuals exhibited no autocorrelation through 24 lags. The parameter for female fetal deaths (i.e. 1.081) implies that temporal variability in the fetal death sex ratio oscillated around approximately 108 males per 100 females. The moving average parameter (i.e. -0.1972) suggests that an ‘echo’ followed a high or low value in male fetal deaths by 11 months, but did not carry to the 22nd month. The ‘echo’ had values opposite the earlier value.

Steps 4–6 above resulted in the following final parameters.

$$Y_t = 1.0794X_{1t} + 17.4289X_{2t} + 15.8041X_{2t-1} + (1 - 0.2475B^{11})a_t$$

All the parameters were at least twice their standard errors. The Ljung–Box test³³ detected no autocorrelation through 24

Table 1 Mean, standard deviation of the mean, and minimum and maximum values of male and female fetal deaths as well percentage unemployed in California for the 156 months beginning January, 1989

	Mean (SD)	Minimum value	Maximum value
Male fetal deaths	151.5 (19.5)	109	226
Female fetal deaths	139.1 (20.7)	99	191
Percentage unemployed	6.7 (1.6)	4.4	10.3

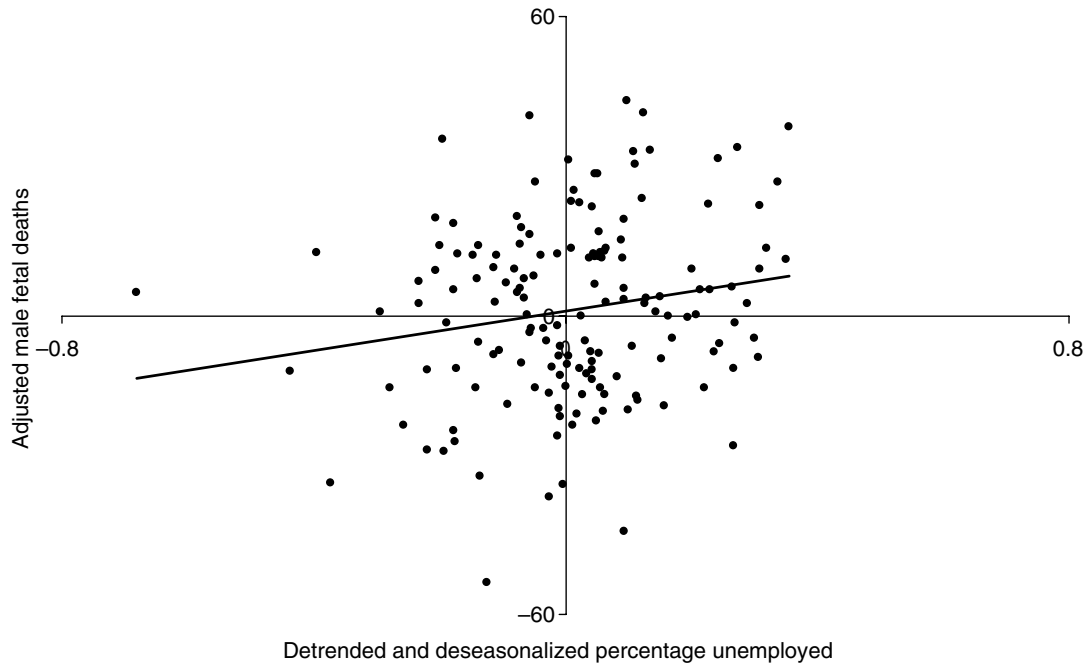


Figure 1 Scatter diagram and regression line of male fetal deaths (adjusted for female fetal deaths and autocorrelation) and detrended, deseasonalized percentage unemployed in California for 156 months beginning January 1989

lags of the residuals of the model. The residuals were not related to either of the predictor variables. Adding the unemployment rate variable to the equation resulting from step 2 increased the R^2 from 15 to 20%. Figure 1 shows a scatter diagram of male fetal deaths adjusted for female fetal deaths and autocorrelation and the detrended and deseasonalized unemployment rate at time t . A diagram of the association in which fetal deaths follow the unemployment rate by 1 month (i.e. the unemployment variable at time $t-1$) would appear very similar to Figure 1.

We added the unemployment rate variable at $t-2$ to the test equation to determine if the association continued into a third month. The coefficient for the added month fell within its 95% confidence interval (two-tailed test) whereas those for months t and $t-1$ remained above their confidence intervals.

The coefficients for the unemployment rate at t and $t-1$ sum to 33.3229 implying that approximately 33 male fetuses died for every 1% increase in the detrended and deseasonalized unemployment rate during the test period. The sum of increases in the detrended and deseasonalized unemployment rate over the 70 months in which it increased was 11.2. Therefore, we estimate that about 370, or 3.4%, of the 10 710 male fetal deaths in the 70 months, could be statistically attributed to economic contraction.

Discussion

Our results support the fetal death mechanism in that male fetal deaths increased above the values expected from female deaths and from history in months in which the detrended and deseasonalized unemployment rate increased. However, our findings do not detract from either the reduced sperm motility or reduced libido arguments. All three could connect ambient stressors to the secondary sex ratio.

We could not devise a satisfying post hoc explanation of the moving average parameter discovered at the 11th lag. The 'echo' is unlikely to reflect the seasonality in fetal deaths because male and female fetal deaths exhibit very similar seasonality and the discovered parameter survived regressing male on female fetal deaths. Moreover, the parameter is negatively signed whereas seasonality implies a positive association.

We wondered whether the inverse 'echo' at 11 months might instead be owing to women who suffer fetal deaths delaying, or seeking prenatal care for, subsequent pregnancies. However, these connections require that delaying pregnancy or seeking prenatal care be more likely among women who suffered male fetal deaths than among those who suffered female fetal deaths. We know of no reason why this would be true.

Our analyses do not include any maternal risk factors (e.g. age and smoking) for adverse obstetric outcomes raising the question of whether an omitted variable could have led us to a type I error.^{34,35} However, we note that any omitted variable that affects female fetal death could not have spuriously induced our findings. Nor could our discovered association be attributed to any omitted variable that exhibits trends, seasonality, and the tendency to remain elevated or depressed after high or low values because we removed autocorrelation from the dependent variables. Therefore, any argument that we falsely rejected the null hypothesis would have to identify an omitted variable that exhibits no autocorrelation, affects only male fetuses, and does not result from unemployment.

Our analyses have other limitations. We cannot, for example, know if the results generalize in time and space beyond California over the 156 months analysed. Only replication can establish the external validity of the findings.

We also caution that although we use the unemployment rate as a gauge of ambient economic circumstances, the temptation

remains to view our work as an ecological test of the effect of personal unemployment on obstetric outcomes. Our analyses should be considered similar to dose–response studies of the effect of air pollution or noise on health. Studies as such ours have their own weaknesses including the fact that they tell us nothing of the individual level response to the stressor at any dose.

As noted at the outset, testing the fetal death mechanism has implications for public health because unlike the reduced libido or lower sperm motility mechanisms, fetal deaths induce somatic and psychological pain in parents, as well as sympathetic pain in families and the community. It is also the case that fetal death affects the timing of a woman's other pregnancies. Women in the western world increasingly delay childbearing, want fewer children, and desire to become pregnant over a shorter interval.^{36,37} Research suggests that a poorly timed pregnancy may impose a social and economic burden on the parents and increase the risk of late initiation of prenatal care.^{38,39}

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