Sex ratios in California following the terrorist attacks of September 11, 2001

Ralph Catalano1, Tim Bruckner, Jeff Gould, Brenda Eskenazi and Elizabeth Anderson

School of Public Health, University of California at Berkeley, Berkeley, CA 94720, USA
1To whom correspondence should be addressed. E-mail: rayc@berkeley.edu

BACKGROUND: Natural and man-made disasters as well as declining economies appear to coincide with reduced odds of male live births among humans (i.e. lower secondary sex ratio). This association has been attributed to excess death of males in gestation and to reduced conception of males. We attempt to empirically discriminate between these two attributions by testing the hypotheses that the attacks of September 11, 2001 were followed in California first by higher fetal death sex ratios and later by lower sex ratios among very low weight births and total live births. METHODS: We apply interrupted time-series methods to the fetal death, very low birth weight, and secondary sex ratios. The methods control for trends, seasonal cycles, and other forms of autocorrelation that could induce spurious associations. RESULTS: Findings support the excess death explanation in that the fetal death sex ratio reached its highest level in the 6 year test period in October and November of 2001, while the very low weight birth sex ratio dropped to its lowest level in 14 years in December of 2001. The secondary sex ratio exhibited its second lowest value in 14 years in December of 2001. No support was found for the reduced conception explanation in that the sex ratio did not differ from expected values 9, 10 or 11 months after the attacks. CONCLUSIONS: We infer support for the excess death explanation at the expense of the reduced conception explanation. We also describe the implications of our findings for public health planning.

Introduction

The human secondary sex ratio (i.e. the ratio of male to female live births) appears to fall when natural and man-made disasters (Lyster, 1974; Mocarelli et al., 1996; Fukuda et al., 1998) as well as declining economies (Catalano, 2003; Catalano and Bruckner, 2005) disrupt the regular functioning of human communities. These reports converge with the finding from case-controlled studies that stressful experiences decrease the likelihood that pregnant women will deliver males (Hansen et al., 1999). This and related work, described below, suggests that the events of September 11, 2001 could have affected sex ratios in the USA. We test this possibility in the hope of contributing to the scholarly debate concerning the nature and causes of temporal variability in sex ratios and to the practical efforts to prepare for similar events in the USA and elsewhere.

Explanations of the reported association between population stressors and the human sex ratio typically build on the observation that males exceed females among embryonic and fetal deaths (Mizuno, 2000). The literature, however, remains vague on why the excess deaths of males in gestation apparently increase during periods of maternal stress. Speculation includes that the response of embryos and fetuses to maternal glucocorticoids determines whether or not gestation continues (Erickson et al., 2001). These hormones perturb males in gestation more than females (Owen and Matthews, 2003). Pregnant women produce more glucocorticoids when stressed (Hobel et al., 1999), raising the possibility that some characteristic of embryonic or fetal response to high doses of the hormones increases the risk of death among weaker males (Erickson et al., 2001).

The excess death theory implies that males lost to elevated maternal stress would have been at risk of premature delivery and, therefore, very low birthweight (<1500g) had the environment remained constant. The theory explicitly posits that gestations likely to yield weak male infants have an elevated risk of spontaneous abortion. Premature, very low weight infants certainly must be considered among the weakest of newborns, given their elevated risk of death as well as of ongoing physical and behavioural deficits (Ross et al., 1991; Kiely et al., 1994). Indeed, few would survive without considerable medical intervention. The risk of very low birthweight, moreover, increases with maternal stress and the sex ratio of very low weight infants significantly exceeds that of other live births (Hall and Carr-Hill, 1982; Cooperstock and Campbell, 1996; Vatten and Skjaerven, 2004).

Although the work cited above assumes that the sex ratio drops during stressful times, controversy remains over
The empirical and theoretical bases for the assumption. Empirical controversy arises from the fact that sex ratios reportedly increase during wars (MacMahon and Pugh, 1954; Graffelman and Hooekstra, 2000), although decreases have been observed during recent conflicts (Ansari-Lari and Saa-dat, 2002; Zorn et al., 2002). The literature also reports that the sex ratio did not decline in rural Costa Rica during 19th century epidemics (Madrigal, 1996). The chronic stress presumably induced by low socio-economic status, moreover, does not appear universally associated with low sex ratios (Marleau and Saucier, 2000).

The theoretical literature does not agree on whether the termination of gestations likely to yield weak males is the purpose, or a side-effect, of the stress mechanism supposedly conserved by natural selection among females of sexually dimorphic species (Trivers and Willard, 1973; Brown and Silk, 2002; Krackow, 2002). Not all the literature, moreover, assumes that excess death of males in gestation induces the presumed decline in the secondary sex ratio during periods of environmental stress. Other explanations assume that temporal variation in the secondary sex ratio reflects earlier variation in the primary sex ratio (ratio of conceived males to females). Stress reportedly reduces sperm motility and could thereby reduce the sex ratio at conception (Fukuda et al., 1996). Stress also reduces the frequency of coitus. Researchers have reported that coitus early in the human menstrual cycle favours the conception of males whereas that at the middle stage (i.e. ovulation) yields more females (Martin, 1997; James, 1999; Lazarus, 2002). Circumstances, including cognitive stress, that decrease coital frequency should reduce the likelihood of conception early in the menstrual cycle and, therefore, reduce the primary sex ratio.

The events of September 11, 2001 as stressors of the US population

Much literature suggests that the events of September 11 induced anxiety and bereavement among Americans via the witnessing of harm (Galea et al., 2002; Schlenker et al., 2002). Effects have been noted not only among Americans living near the attack sites but throughout the country (Schuster et al., 2004; Whalen et al., 2004). These reports converge with research demonstrating that witnessing harm to others induces biological responses in the witness that resemble those in the persons harmed (Singer et al., 2004). These responses can include the production of glucocorticoids alluded to above as those implicated in the spontaneous abortion of males (Irwin et al., 1987; Cohen et al., 1998; Fukuda et al., 2000).

Anxiety, fear, and their biological components could also trigger the reduced conception mechanisms. The reduction of sperm motility reported among men witnessing the effects of earthquakes could plausibly proceed from the events of September 11, 2001 (Fukuda et al., 1996). These events also could have reduced libido among bereaved males and females and thereby triggered the reduced coitus mechanism (National Center for Post Traumatic Stress Disorder, 2004).

Hypotheses

The following hypotheses were tested.

(1) The secondary sex ratio was lower than expected among cohorts born from September through December 2001 (i.e. infants in gestation $\geq 20$ weeks in September 2001).

(2) The secondary sex ratio was lower than expected among cohorts born from May through July 2002 (i.e. infants conceived in September through November, 2001).

(3) The sex ratio among very low weight births was lower than expected among cohorts born from September through December 2001 (i.e. infants in gestation $\geq 20$ weeks in September 2001).

(4) The sex ratio among fetal deaths was higher than expected among cohorts born from September through December 2001.

The argument that population stressors reduce the secondary sex ratio would be supported if either or both hypotheses 1 and 2 were accepted. The fetal loss mechanism would be supported to the exclusion of the reduced conceptions mechanism if hypotheses 1, 3 and 4 were accepted but 2 rejected. The reduced conceptions mechanism would be supported to the exclusion of fetal loss if hypothesis 2 were accepted and the others rejected. The mechanisms do not exclude each other, implying that both would be supported if all the hypotheses were accepted.

Materials and methods

Data

We used California data for our tests because we regularly subject them to time-series modelling for quality assurance purposes. The Maternal and Child Health Branch of the State of California Department of Health Services provides these data for us. While research on the psychological effects of September 11, 2001 (Schuster et al., 2004; Whalen et al., 2004) suggests that the events of that day stressed all Americans, a dose–response varying with distance remains possible for somatic effects. As discussed below, the results of our test may not generalize to other states and could underestimate effects in populations closer to the attack sites.

We test the first three hypotheses with time series beginning January 1989 and ending December 2002. These were the longest series with consistent definitions available to us at the time of the tests. Due to changes in definition (e.g. exclusion of therapeutic abortions), we tested hypothesis 4 with a time series that began January 1996. We end the series in December 2001 because the fetal loss theory offers no prediction for fetal deaths beyond that point.

A very low weight infant weighs $<1500$ g at birth (Kiely et al., 1994). Nearly all such infants are premature. A fetal death is defined by the State of California 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’ (California Department of Health Services, 1996).

California’s Health and Safety Code requires that ‘each fetal death, excluding induced abortions, in which the fetus has advanced to or beyond the 20th week of uterogestation shall be registered with the local registration district within 8 days following the delivery and prior to any disposition of the fetus. Only fetal deaths where
the fetus has advanced to or beyond the 20th week of uterogestation shall be registered’ (California Department of Health Services, 1996). Our tests, therefore, do not apply to fetal deaths before the 20th week of gestation.

Any direct test of the excess death mechanism must use fetuses ≥20 weeks in gestation because the primary sex ratio (i.e. the ratio of male to female conceptions) cannot be known and deaths before 20 weeks cannot be accurately counted in California or elsewhere. Our test of hypothesis 4, therefore, may underestimate the association between the events of September 11 and the spontaneous abortion of males.

Monthly male and female fetal deaths averaged 151 and 139 respectively over the 72 months for which we had data. Monthly very low weight male and female births averaged 183 and 167 respectively over the 168 months of data available to us. Figures 1 and 2 show the secondary and very low weight birth sex ratios plotted over the test period. Figure 3 shows the fetal death sex ratio.

**Design and analyses**

Our analyses follow in the tradition of the interrupted time-series quasi experiment (Box *et al.*, 1994). The null hypothesis for such tests is that the post-interruption values of the dependent variable do not differ from the values expected from the pre-interruption series.

Time series often exhibit autocorrelation, such as trends, cycles, and the tendency to remain elevated or depressed after high or low values that complicate estimating the post-interruption values. Researchers dating at least to Fisher (1921) and his 1920 study of crop variation have solved the autocorrelation problem by ‘decomposing’ time series into temporally predictable and residual components. The predictable values become those expected under the null hypothesis. Contemporary applications of Fisher’s logic (e.g. Box *et al.*, 1994) typically decompose a time series by expressing autocorrelation as an effect of earlier values, or ‘lags’, of the series itself. If an equation includes the correct lags of the dependent variable, the residuals exhibit no autocorrelation. The analyst can

---

**Figure 1.** California secondary sex ratio (male/female live births) for 168 months from January 1989 to December 2002. Last four months of 2001 shown with boxes.

**Figure 2.** California sex ratio (male/female) of very low weight births for 168 months from January 1989 to December 2002. Last four months of 2001 shown with boxes.
than add the independent variable or variables to the equation to determine if their coefficients differ from zero in the hypothesized direction.

We used the strategy attributed to Dickey and Fuller (1979) as well as Box and Jenkins (Box et al., 1994) to identify and model autocorrelation in male fetal deaths and very low weight births for the months January, 1996 through August 2001. The Dickey–Fuller test detects trends and seasonal cycles as well as their combination (e.g. upwardly or downwardly trending sine waves). The Box and Jenkins routines allow the researcher not only to model trends and cycles detected by the Dickey–Fuller test, but also to identify and model the tendency of a series to remain elevated or depressed, or to oscillate, after high or low values. The Box and Jenkins approach uses ‘autoregressive’ and ‘moving average’ parameters to model such tendencies. Autoregressive parameters best describe patterns that persist for relatively long periods, while moving average parameters parsimoniously describe less persistent patterns.

We converted the sex ratios into their natural logarithms to allow us to express our results in the familiar ‘effect on odds’ format. The general form of the Box–Jenkins equation applied to the natural logarithm of a sex ratio is as follows.

\[
\left( \frac{m_t}{f_t} \right)^e = c + \left(1 - \theta_1 B - \theta_2 B^2 - \cdots - \theta_p B^p \right) \left(1 - \phi_1 B - \phi_2 B^2 - \cdots - \phi_q B^q \right) \alpha_t,
\]

(1)

\(m_t\) and \(f_t\) are male and female births (either all or very low weight), or fetal deaths in California in month \(t\). \(c\) is a constant. \(0\) is the moving average, or ‘short memory’, parameter. \(\phi\) is the autoregressive, or ‘long memory’, parameter. \(B\) indicates that either \(0\) or \(\phi\) acts on the value of the error term ‘\(\alpha\)’ at month \(t - q\) or \(t - p\), and \(\alpha_t\) is the error term at month \(t\).

We added a binary variable scored 1 for September, 2001 and 0 otherwise to the best fitting Box–Jenkins equations. We specified the variable such that we could detect associations with cohorts born not only in September 2001 but, depending on the hypothesis, up to 10 months later. Testing hypotheses 1 and 2, for example, required that we specify up to 10 months because the fetal loss mechanism would predict decreased sex ratios among cohorts likely in gestation in September (i.e. those born from September, 2001 through April 2002) while the reduced conception mechanism predicts reduced sex ratios in June and July, 2002. Reduced sex ratios in May 2002 could be attributed to either mechanism.

Hypothesis 3 arises from the fetal loss mechanism and, therefore applies only to birth cohorts in gestation in September 2001. The test, therefore, requires that we specify the binary variable for 8 months in addition to September 2001. Hypothesis 4 concerns the fetal death sex ratio. Since public health surveillance systems register fetal deaths only after the 19th week of gestation, testing hypothesis 4 requires that the binary variable be specified for only 3 months after September 2001.

We estimated test equations that took the following general specification.

\[
\left( \frac{m_t}{f_t} \right)^e = c + \omega B^n I_t + \left(1 - \theta_1 B - \theta_2 B^2 - \cdots - \theta_p B^p \right) \left(1 - \phi_1 B - \phi_2 B^2 - \cdots - \phi_q B^q \right) \alpha_t,
\]

(2)

\(I_t\) is a binary variable scored 1 for September 2001 and 0 for all other months. \(\omega\) is the effect parameter of which the antilog is the ‘effect on odds’ associated with the events of September 11, 2001. ‘\(n\)’ in \(B^n\) indicates how many monthly cohorts beyond September 2001 we tested. ‘\(n\)’ is 0–10 for hypotheses 1 and 2, 0–8 for hypothesis 3, and 0–3 for hypothesis 4.

We estimated final models by deleting from the initial models any variables with estimated parameters inside their 95% confidence interval (two-tailed test). We inspected the residuals of each model for autocorrelation and added Box–Jenkins parameters to the equation to express any that we discovered.

**Results**

Table I shows the results for hypotheses 1 and 2 concerning the secondary sex ratio (i.e. the odds of male to female live births). The parameters for the September 11 binary variable support hypothesis 1 but not hypothesis 2. The secondary sex ratio was lower than expected in the cohort born in December 2001 but not in the cohorts born in May, June or July 2002. The antilog of the estimated parameter for December 2001 implies that the odds of a male live birth dropped by 2% in this month.
The antilog of the constant indicates that the average secondary sex ratio over the test period was consistent with the literature (i.e. 1.049). The fact that the secondary sex ratio exhibited no autocorrelation over the test period accounts for the absence of Box–Jenkins parameters in the test equations.

Table II shows the results for hypothesis 3 concerning the sex ratio of very low weight births. The findings support hypothesis 3 in that the sex ratio was unexpectedly low in December 2001. The antilog of the coefficient suggests that the odds of very low weight birth being male dropped by 29% in December.

The antilog (i.e. 1.10) of the constant (i.e. 0.0960) comport with the literature in that the sex ratio of very low weight births is higher than that for all live births. The moving average parameter at 5 suggests that the very low weight sex ratio exhibits an ‘echo’ such that high or low values were followed 5 months later by similarly high or low values, albeit declining in absolute size. These ‘echoes’ do not carry to the 10th month.

Table III shows the results for hypothesis 4 concerning the sex ratio of fetal deaths. The results shown in Table III support hypothesis 4 in that the fetal death sex ratio exceeded expected values in October and November of 2001. The antilogs of these parameters suggest that the odds of a fetal death being male increased in October and November by 24 and 23% respectively.

The constant implies that, as expected from the wider literature, the sex ratio of fetal deaths (i.e. 1.10) is higher than that for live births. The autoregressive parameter at 11 suggests that the fetal death sex ratio exhibits an ‘echo’ such that a high or low value repeats at increments of 11 months. Unlike moving average ‘echoes’ such as that detected in the sex ratio of very low weight births, autoregressive ‘echoes’ continue, though with declining size, throughout the time series.

We conducted three additional tests suggested by our primary findings. The fetal loss mechanism assumes that more male, but not female, fetuses die than expected during periods of maternal stress. To ensure that our sex ratio findings arose from excess male deaths rather than unexpectedly few deaths among females, we modelled male and female fetal deaths separately. Consistent with the fetal loss mechanism, and with the findings for the fetal death sex ratio, significantly more (i.e. \( \omega = 23; SE = 11 \)) male fetuses than expected died in October 2001 and the increase remained elevated into November. Fewer female fetuses than expected died in October but the difference was not significant (i.e. \( \omega = -13.1844; SE = 13.2197 \)).

We also modelled the natural logarithm of the sex ratio of infants weighing > 1499 g to determine if our findings for the secondary sex ratio resulted, as the fetal loss theory implies, primarily from losses of fetuses that, had they survived to birth, would have been very low weight. As with the secondary sex ratio, and the sex ratio for very low weight births, the sex ratio for other than very low weight births showed a lower than expected value in December, 2001. That value, however, did not fall below the 95% confidence interval (i.e. \( \omega = -0.0175; SE = 0.0100 \)).

Discussion

The results of our tests support the fetal death explanation of the association between ambient stressors and the secondary
The welcome rarity of natural and man-made disasters raises the question of whether findings such as ours generalize to other, more common phenomena that perturb entire populations. As noted above, for example, most of the war literature (e.g. Graffelman and Hoekstra, 2000) is at odds with that concerning disasters. While more empirical work should be done to estimate how much of the divergence results from methodological artefacts, we also need a theory that explains the divergent findings. Could it be, for example, that wars vary in the degree to which they are anticipated, promise relief from other adversity or unite populations, and therefore in the degree to and manner in which populations habituate to them? These and similar considerations raise the question of whether fetal loss theory applies to wars.

As noted in Materials and methods, any test of the excess death mechanism must use fetuses ≥ 20 weeks in gestation. The loss of males in gestation due to maternal stress could exceed our estimates because the mechanism, at least in theory, applies to all males in gestation. Our tests of hypotheses 1 and 3, however, did not show decreased sex ratios for cohorts born between January and May 2002, suggesting that the events of September 11, 2001 did not induce significant loss of males in gestation < 20 weeks.

Changes in the secondary sex ratio may not warrant public health concern, but the public health community should be concerned with mechanisms that induce such change. Increases in the incidence of male fetal deaths, for example, suggest that the biology of at least women of childbearing age can be altered by population stressors.

Those responsible for public health in the USA have responded to the attacks of September 11, 2001 by preparing for similar events (Institute of Medicine, 2002). These efforts understandably focus primarily on the populations most exposed to toxins, explosions, and other mayhem. The Institute of Medicine (2003), however, has urged planning for the needs of those who would be cognitively, rather than physically, exposed to such attacks. Our findings, if replicated in other states distant from the attacks, should add impetus to this planning.

**References**


James WH (1999) The status of the hypothesis that the human sex ratio at birth is associated with the cycle day of conception. Hum Reprod 14,2177–2178.


R.Catalano et al.
Sex ratios following September 11, 2001


Submitted on May 17, 2004; resubmitted on September 9, 2004; accepted on January 7, 2005