Title
Exogenous shocks to the human sex ratio: the case of September 11, 2001 in New York City.

Journal
Human reproduction (Oxford, England), 21(12)

ISSN
0268-1161

Authors
Catalano, R
Bruckner, T
Marks, A R
et al.

Publication Date
2006-12-26

Peer reviewed

Human Reproduction Vol.21, No.12 pp. 3127–3131, 2006 doi:10.1093/humrep/del283
Advance Access publication August 26, 2006.

Exogenous shocks to the human sex ratio: the case of September 11, 2001 in New York City

R.Catalano[1], T.Bruckner, A.R.Marks and B.Eskenazi

School of Public Health, University of California, Berkeley, Berkeley, CA, USA

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

BACKGROUND: The human secondary sex ratio reportedly falls in populations subjected to exogenous stressors such as earthquakes or political and social upheavals. Explanations of the association include reduced conception of males and increased fetal deaths among males. The latter explanation has been supported by research reporting that the sex ratio in California fell 3 months, but not 8, 9 or 10 months, after the terrorist attacks of September 11, 2001. California's distance from the attacks raises the questions of whether the results arose from chance and would be found elsewhere. We contribute to the literature by testing the association between the secondary sex ratio and the events of September 11 in New York City. METHODS: We replicate the California tests by applying interrupted time-series methods, which control for secular trends, seasonality and other forms of autocorrelation, to 91 cohorts born in New York City during 28-day periods from January 1996 to June 2002. RESULTS: As hypothesized, the sex ratio in New York City in the period 1 January to 28 January 2002 fell to 1, which was the lowest observed value during the test period and significantly (i.e. $P < 0.01$, two-tailed test) below the value expected from history. CONCLUSIONS: Our findings support the male fetal loss explanation of the association between exogenous population shocks and the secondary sex ratio.

Key words: fetal death/population distress/pregnancy/sex ratio

Introduction

The human secondary sex ratio (i.e. odds of a male birth) reportedly falls in populations subjected to exogenous stressors such as earthquakes (Fukuda *et al*., 1998), environmental toxins (Lyster, 1974; Mocarelli *et al*., 1996), political and social upheavals (Catalano, 2003) and contracting economies (Catalano and Bruckner, 2005). Other research reports that gravid women who experience stressful life events deliver fewer males than expected (Hansen *et al*., 1999). High levels of treated anxiety in Swedish women, moreover, predict low sex ratios in monthly birth cohorts (Catalano *et al*., 2005a).

Explanations of these findings fall into two groups. One group assumes that exogenous shocks decrease the odds of male conceptions (Pyrzak, 1994; Fukuda *et al*., 1996). The anxiety induced by population stressors presumably reduces the frequency of coitus in the population. Reduced coitus, particularly early in the human menstrual cycle, reportedly reduces the conception of males (James, 1999; Martin, 1997; Lazarus, 2002). Anxiety induced by population stressors may also reduce sperm motility and thereby reduce the sex ratio at conception (Fukuda *et al*., 1996).

A second group of explanations posits that population stressors cause endocrine changes in females that induce spontaneous abortion of small or weak male fetuses (Forchhammer, 2000; Owen and Matthews, 2003; Catalano *et al*., 2005b,c). One branch of this literature argues that natural selection conserved this mechanism because extinguishing weak male fetuses increases the chance that females in stressful environments will have grandchildren (Trivers and Willard, 1973). Another branch accepts that the endocrinal response to population stressors disproportionately extinguishes male fetuses but disputes the claim of evolutionary benefits (e.g. Krackow, 2002).

Knowing whether reduced male conceptions or increased male fetal death better fits available data would have applied as well as theoretical value. Fetal deaths induce somatic and psychological pain in parents, as well as sympathetic pain in families and the community. Indeed, many public health and medical interventions focus on reducing fetal deaths (Centers for Disease Control, 2004). Intuition suggests that these interventions may be made more effective if those who provide them could anticipate when and where risk will most likely increase.

The scholarly literature reports that the terrorist attacks of September 11 induced stress and anxiety in many Americans (American College of Obstetricians and Gynecologists, 2001; Schlenger *et al*., 2002; Knudsen *et al*., 2005). Consistent with this literature and the male fetal loss argument, the secondary sex ratio in California reportedly fell below its expected value 3 months after the terrorist attacks of September 11, rather than

7 or more months as suggested by the explanations that assume reduced conceptions of males (Catalano *et al*., 2005c). California's geographic distance from the attacks raises the obvious question of whether the association appeared by chance.

We contribute to the literature by testing the association between the secondary sex ratio and the events of September 11 in New York City using the methods applied in the California test and in other reports of reduced sex ratios following population shocks (i.e. Graffelman and Hoekstra, 2000; Catalano, 2003). We test the hypothesis for all fetuses in gestation on September 11, 2001, but, based on the California findings, hypothesize a lower than expected sex ratio in one or more of the 3 months centred, as described below, on December 2001.

Materials and methods

After approval by the Committee for the Protection of Human Subjects at the University of California, Berkeley, the Office of Vital Statistics at the New York City Department of Health and Mental Hygiene provided us anonymous birth certificate data for infants born to New York City residents for the period 9 January 1996 to 17 June 2002. Excluding multiple births and infants born to mothers with diabetes (Leipold *et al*., 2005), as well as records with missing data on essential variables such as infant sex and birth date, left us 717 026 cases.

Because September 11, 2001 was a Tuesday, we separated our data into 28-day periods each beginning on a Tuesday. This procedure yielded 91 periods, the 75th of which began on September 11, 2001. The fetal death argument, combined with the California findings, implies that pregnancies ending with live births in one or more of periods 77 (i.e. 6 November to 3 December 2001), 78 (i.e. 4 December to 31 December 2001) or 79 (i.e. 1 January to 28 January 2002) would yield a lower sex ratio than expected from patterns over time. The reduced conceptions argument implies the ratio would be lower in one or more of the periods 82 (26 March to 22 April 2002), 83 (23 April to 20 May 2002) or 84 (21 May to 17 June 2002).

The sex ratio may exhibit seasonality and other patterns over time (i.e. autocorrelation) that could 'schedule' predictably few male births in Novembers, Decembers or Januarys and thereby induce a type I error in our test. We controlled for this possibility by adopting the methods used in the California test described above as well as in other tests of the effect of population shocks on the secondary sex ratio (i.e. Graffelman and Hoekstra, 2000; Catalano, 2003). These methods, typically referred to as 'interrupted time-series modeling', use the approach of Box and Jenkins to detect and express autocorrelation (Box *et al*., 1994).

The Box–Jenkins strategy requires that we estimate the following test equation:

$$\frac{M_t}{T_t - M_t} = c + \left(\omega_0 + \omega_1 B + \omega_1 B^2 + \cdots + \omega_9 B^9\right) I_t + \left(\frac{1-\theta B^q}{1-\phi B^p}\right) a_t, \quad (1)$$

where M_t is the number of male infants born in period t, T_t is the total number of infants born in period t, c is a constant, I_t is a binary variable scored 1 for the 28-day period beginning with September 11, 2001 and 0 otherwise, ω_0 to ω_9 are estimated parameters (estimating these 10 parameters allow us to determine if the sex ratio dropped 3, 4 or 5 months after the events of September 11 as predicted by the California findings and fetal loss theory or 8, 9 or 10 months later as would be predicted by the reduced male conceptions theory), ϕ is an autoregressive parameter (autoregressive parameters measure a series' tendency to remain above or below its means after a perturbation), θ is the 'moving average' parameter (moving average parameters measure the tendency of perturbations to be present for more than one period), B^n is the 'backshift operator' or value of the conditioned variable at period $t - n$ and a_t is the error term at year t.

The analyses proceeded through the following steps. First, we used Box–Jenkins methods to identify and model autocorrelation in the sex ratio. Second, we added the binary variable (i.e. I) to the Box–Jenkins equation and estimated the variable's association with the sex ratio in periods 75–84. Last, we inspected the residuals from the step 2 to insure they exhibited no autocorrelation.

Results

Figure 1 shows the observed sex ratio over the test period. The observed value (i.e. 0.9995) in the 79th period (1 January to 28 January 2002) was the lowest secondary sex ratio in the 91 test periods.

A comparison of the sex ratio of each of the 10 birth cohorts in gestation during, or conceived immediately after, the events of September 11 with the mean of the remaining 81 cohorts supports our hypothesis. The mean secondary sex ratio of the 81 unexposed cohorts was 1.0510 with a standard deviation of 0.0234. Of the 10 exposed cohorts, those born in period 79 exhibited a secondary sex ratio (i.e. 0.9995) below the 95% confidence interval (CI) (i.e. lower bound is 1.0051; two-tailed test) of the mean of all the 81 unexposed cohorts. As noted above, however, this comparison could lead to a type I error if the sex ratio manifests patterns over time that would yield a low value in January 2002 regardless of the events of September 2001. Values in Januarys, for example, may always be low.

To test the 'low-Januarys' possibility, we compared the sex ratio of period 79 with those of the seven other 28-day periods comprised mostly of Januarys. The sex ratio for period 79 (i.e. 0.9995) fell below the 95% CI (i.e. lower bound is 1.014; two-tailed test) of the mean for the other periods that roughly correspond to Januarys.

Because patterns other than consistently low Januarys could have induced a type I error, we estimated Equation (1) using the Box–Jenkins method. This approach identifies and controls all forms of autocorrelation.

Table I summarizes the results of estimating the test equation. The sex ratio exhibited autocorrelation in which a value in period t could be predicted by the value 2 and 3 periods earlier. As hypothesized, the sex ratio in New York City in the period 1 January to 28 January 2002 fell significantly below ($P < 0.05$; two-tailed test) the value expected from history. Inconsistent with the reduced conceptions argument, the sex ratios 8, 9 and 10 months after the terrorist attacks did not fall.

We conducted three additional tests to determine whether our results could have been induced by analytic artefacts. We transformed the sex ratio to its natural logarithm and estimated the test equation again to ensure that variation in variability over time did not distort our estimates. The results of the tests did not change.

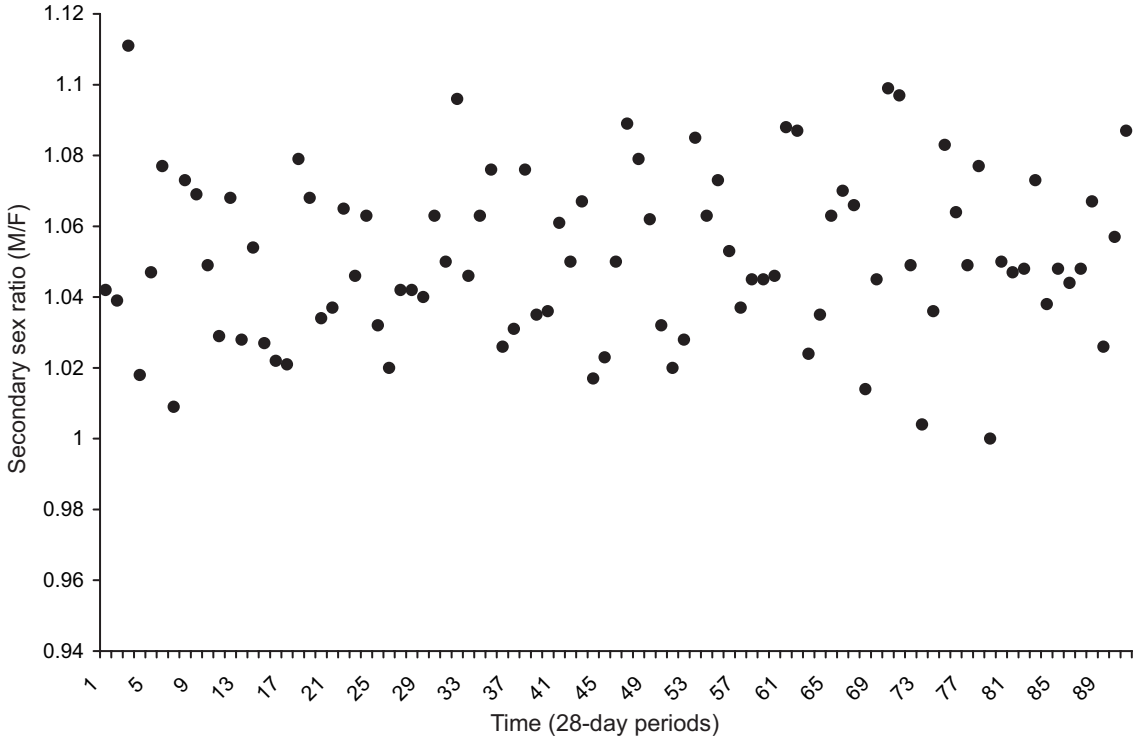

Figure 1. Observed secondary sex ratio (i.e. M/F) for New York City for 91 28-day periods beginning Tuesday 9 January 1996. Period 75 begins with September 11, 2001.

Table I. Coefficients (standard errors in parentheses) for model of the secondary sex ratio (i.e. M/F) in New York City for 91 28-day periods beginning Tuesday 9 January 1996

Predictor	Estimated coefficients
Constant	1.0509* (0.0012)
Moving average parameter	B^2 = 0.3592* (0.1077)
Autoregressive parameter	B^3 = –0.2462* (0.1135)
Period effects	
Period 75 (begins 9/11/2001)	0.0240 (0.0191)
Period 76	0.0015 (0.0194)
Period 77	0.0055 (0.0203)
Period 78	0.0282 (0.0207)
Period 79	–0.0484* (0.0207)
Period 80	0.0009 (0.0206)
Period 81	–0.0044 (0.0207)
Period 82	–0.0038 (0.0202)
Period 83	0.0197 (0.0191)
Period 84	–0.0144 (0.0190)

*$P < 0.05$; two-tailed test.

The natural logarithm transformation allowed us to express our findings in the familiar 'effect on odds' metric. Taking the antilog of the coefficient (i.e. –0.0494) for period 79 yielded the estimate that the events of September 11, 2001 reduced the secondary sex ratio in New York City by ~5%.

Using the methods devised by Chang *et al*. (1988), we also assessed whether outliers at periods other than those we tested could have led us to misestimate the CIs for our parameters. We found no outliers other than that at period 79.

We deleted the periods without significant coefficients from our test equation and estimated the remaining parameters to ensure that the presence of statistically non-significant parameters did not distort our estimates. Results remained the same.

It could be argued that mothers who gave birth in New York City during January of 2002 included an unusually high fraction of low socio-economic status or African American women. The literature (e.g. Teitelbaum and Mantel, 1971; James, 1984; Lazarus, 2002) reports that these mothers may deliver fewer males than expected from the population. We, therefore, added two variables, the odds of mother having less than a high-school education and of being African American, to our test equation and estimated it again. The results of our test did not change in that the coefficient for period 79 remained statistically significant ($P < 0.05$, two-tailed test).

Discussion

Our findings support the male fetal loss explanation of the association between exogenous population shocks and the secondary sex ratio. The analyses controlled for trends, seasonality and other forms of autocorrelation in the sex ratio that could have induced a type I error.

Limitations of our study include that we could not test the hypothesis, implied by our findings, that the sex ratio of fetal deaths rose significantly between 11 September 2001 and 1 January 2002. No state in the United States requires reporting fetal deaths before the 20th week of gestation.

We cannot reconcile the findings that sex ratios in neither California nor New York City declined 8, 9 or 10 months after the terrorist attacks, with the report (Hansen *et al*., 1999), based on excellent data describing a large sample in Scandinavia, that gestations begun in stressed families yield fewer males than expected. *Post hoc* explanations of the discrepancy include that the effect of September 11 on sperm motility or the frequency of coitus may have been too small or spread too

broadly in time for methods such as those used in California and New York City to detect.

Although our findings converge with those from California, important issues remain unresolved. Why, for example, did only those cohorts in ~20–24th week of gestation appear vulnerable to the shock? The answer to this question may be related to the finding that the fetal response to maternal stressors appears strongest in the second half of gestation (Van den Bergh *et al*., 2005) and that mothers may use that response as a test of male fetal robustness (Owen and Matthews, 2003). Cortisol shocks produced by maternal stressors late in gestation may, moreover, increase the incidence of premature delivery (Bolt *et al*., 2002; Hobel, 2004). Our findings would be consistent with the net effect of these two circumstances in that sex ratios would drop primarily among cohorts shocked late in the second and early in the third trimesters.

Empirical issues that need to be addressed include the shape and determinants of sex ratio dose response to environmental shocks. Should, for example, places geographically between New York City and California have exhibited declines in the sex ratio greater than California's 2% but less than New York City's 5%? Or should the pattern reflect social rather than geographic distance? Would telephone traffic between New York City and another community during the 77th period of our study predict dose response better than geographic distance?

Other issues worthy of study include whether the health of males born in January 2002 differs from that expected from less stressed cohorts. Recent research (Catalano and Bruckner, 2006) raises the issue of whether cohorts of males stressed *in utero* suffer damage and, therefore, relatively high prevalence of morbidity or are culled of their weakest members and exhibit relatively low morbidity.

We see our work as informed by, and contributing to, the larger sex ratio literature thoroughly reviewed by James (1987) and Lazarus (2002). We, for example, controlled for the fraction of African American and poorly educated mothers because research in the United States converges on the suggestion that these mothers give birth to fewer males than expected (Teitelbaum and Mantel, 1971; James, 1984; Lazarus, 2002). We, moreover, applied the methods of Box and Jenkins because other literature implies that season of birth as well as the timing of fertilization during the mother's cycle may induce autocorrelation in the secondary sex ratio (James, 1987).

As noted at the outset, testing the male fetal death mechanism has implications not only for theory but also for public health. Fetal deaths induce somatic and psychological pain in parents, as well as sympathetic pain in families and the community. Fetal death also affects the timing of a woman's other pregnancies. Women in the developed world increasingly delay childbearing, want fewer children and attempt to 'schedule' pregnancies (Abma *et al*., 1997; Hall, 1999). A fetal death may lead to less optimally timed pregnancies that impose a social and economic burden on parents and increase the risk of late initiation of prenatal care (Piccinino, 1994; Kost *et al*., 1998).

References

Abma J, Chandra A, Mosher W, Peterson L and Piccinino L (1997) Fertility, family planning and women's health: new data from the 1995 National Survey of Family Growth. Vital Health Stat 19,1–114.

American College of Obstetricians and Gynecologists (2001) ACOG report: assessing the impact of September 11 on pregnancy. Washington, DC.

Bolt RJ, van Weissenbruch MM, Lafeber HN and de Waal HA (2002) Development of the hypothalamic-pituitary-adrenal axis in the fetus and preterm infant. J Pediatr Endocrinol Metab 15,759–769.

Box G, Jenkins G and Reinsel G (1994) *Time Series Analysis: Forecasting and Control*, 3rd edn. Prentice Hall, London.

Catalano R (2003) Sex ratios in the two Germanies: a test of the economic stress hypothesis. Hum Reprod 9,1972–1975.

Catalano RA and Bruckner T (2005) Economic antecedents of the Swedish sex ratio. Soc Sci Med 60,537–543.

Catalano R and Bruckner T (2006) Secondary sex ratios and male lifespan: Damaged or culled cohorts. Proc Natl Acad Sci USA 103,1639–1643.

Catalano R, Bruckner T, Hartig T and Ong M (2005a) Population stress and the Swedish sex ratio. Paediatr Perinat Epidemiol 19,413–420.

Catalano R, Bruckner T, Anderson E and Gould JB (2005b) Fetal death sex ratios: a test of the economic stress hypothesis. Int J Epidemiol 15,944–948.

Catalano R, Bruckner T, Gould J, Eskenazi B and Anderson E (2005c) Sex ratios in California following the terrorist attacks of September 11, 2001. Hum Reprod 20,1221–1227.

Centers for Disease Control and Prevention (2004) Racial/Ethnic Trends in Fetal Mortality — United States, 1990–2000 (http://www.cdc.gov/od/oc/media/mmwrnews/n040625.htm).

Chang I, Tiao G and Chen C (1988) Estimation of time series parameters in the presence of outliers. Technometrics 30,193–204.

Forchhammer M (2000) Timing of foetal growth spurts can explain sex ratio variation in polygynous mammals. Ecol Lett 3,1–4.

Fukuda M, Fukuda K, Shimizu T, Yomura W and Shimizu S (1996) Kobe earthquake and reduced sperm motility. Hum Reprod 11,1244–1246.

Fukuda M, Fukuda K, Shimizu T and Moller H (1998) Decline in sex ratio at birth after Kobe earthquake. Hum Reprod 13,2321–2322.

Graffelman J and Hoekstra R (2000) A statistical analysis of the effect of warfare on the human secondary sex ratio. Hum Biol 72,433–445.

Hall DM (1999) Children in an ageing society. BMJ 319,1356–1358.

Hansen D, Moller H and Olsen J (1999) Severe periconceptional life events and the sex ratio in offspring: follow up study based on five national registers. BMJ 319,548–549.

Hobel CJ (2004) Stress and preterm birth. Clin Obstetrics Gynecol 47,856–880.

James WH (1984) The sex ratios of black births. Ann Hum Biol 11,39–44.

James WH (1987) The human sex ratio. Part 1: a review of the literature. Hum Biol 59,721–752.

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.

Knudsen HK, Roman PM, Johnson JA and Ducharme LJ (2005) A changed America? The effects of September 11th on depressive symptoms and alcohol consumption. J Health Soc Behav 46,260–273.

Kost K, Landry DJ and Darroch JE (1998) Predicting maternal behaviors during pregnancy: does intention status matter? Fam Plann Perspect 30,79–88.

Krackow S (2002) Why parental sex ratio manipulation is rare in higher vertebrates (invited article). Ethology 108,1041–1056.

Lazarus J (2002) Human sex ratios: adaptations and mechanisms, problems and prospects. In Hardy I (ed.) *Sex Ratios Concepts and Research Methods*. Cambridge University Press, Cambridge, UK, pp. 287–311.

Leipold H, Worda C, Schwindt J, Kautzky-Willer A, Bancher-Todesca D and Husslein PW (2005) Severe diabetic fetopathy despite strict metabolic control. Wien Klin Wochenschr 117,561–564.

Lyster W (1974) Altered sex ratio after the London smog of 1952 and the Brisbane flood of 1965. J Obstet Gynaecol Br Commonw 81,626–631.

Martin JF (1997) Length of the follicular phase, time of insemination, coital rate and the sex of offspring. Hum Reprod 12,611–616.

Mocarelli P, Brambilla P, Gerthoux PM, Patterson DG Jr and Needham LL (1996) Change in sex ratio with exposure to dioxin. Lancet 348,409.

Owen D and Matthews SG (2003) Glucocorticoids and sex-dependent development of brain glucocorticoid and mineralocorticoid receptors. Endocrinology 144,2775–2784.

Piccinino LJ (1994) Unintended Pregnancy and Childbearing. In Wilcox L and Marks J (eds) *From Data to Action: CDC's Public Health Surveillance for Women, Infants, and Children*. US Department of Health and Human Services, Public Health Service, Hyattsville, MD, pp. 73–82.

Pyrzak R (1994) Separation of X- and Y-bearing human spermatozoa using albumin gradients. Hum Reprod 9,1788–1790.

Schlenger WE, Caddell JM, Ebert L, Jordan BK, Rourke KM, Wilson D, Thalji L, Dennis JM, Fairbank JA and Kulka RA (2002) Psychological reactions to terrorist attacks: findings from the National Study of Americans' Reactions to September 11. JAMA 288,581–588.

Teitelbaum MS and Mantel N (1971) Socio-economic factors and the sex ratio at birth. J Biosoc Sci 3,23–41.

Trivers R and Willard D (1973) Natural selection of parental ability to vary the sex ratio of offspring. Science 179,90–92.

Van den Bergh BR, Mulder EJ, Mennes M and Glover V (2005) Antenatal maternal anxiety and stress and the neurobehavioural development of the fetus and child: links and possible mechanisms. Neurosci Biobehav Rev 29,237–258.

Submitted on November 11, 2005; resubmitted on January 31, 2006, April 18, 2006; accepted on May 8, 2006