THE ROYAL SOCIETY

Received 30 March 2004
Accepted 15 April 2004
Published online 8 July 2004

Facultative adjustment of mammalian sex ratios in support of the Trivers–Willard hypothesis: evidence for a mechanism

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Evolutionary theory predicts that mothers of different condition should adjust the birth sex ratio of their offspring in relation to future reproductive benefits. Published studies addressing variation in mammalian sex ratios have produced surprisingly contradictory results. Explaining the source of such variation has been a challenge for sex-ratio theory, not least because no mechanism for sex-ratio adjustment is known. I conducted a meta-analysis of previous mammalian sex-ratio studies to determine if there are any overall patterns in sex-ratio variation. The contradictory nature of previous results was confirmed. However, studies that investigated indices of condition around conception show almost unanimous support for the prediction that mothers in good condition bias their litters towards sons. Recent research on the role of glucose in reproductive functioning have shown that excess glucose favours the development of male blastocysts, providing a potential mechanism for sex-ratio variation in relation to maternal condition around conception. Furthermore, many of the conflicting results from studies on sex-ratio adjustment would be explained if glucose levels *in utero* during early cell division contributed to the determination of offspring sex ratios.

Keywords: Trivers–Willard hypothesis; sex allocation; sex ratio; maternal investment

1. INTRODUCTION

Variation in the production of sons and daughters is a key variable in life-history strategies and evolutionary theory. The Trivers–Willard hypothesis (TWH) proposes that where one sex has more variable reproductive success (males in polygynous species), mothers in good condition would be advantaged by producing more of that sex, whereas mothers in poor condition would be advantaged by producing more of the reproductively stable sex (Trivers & Willard 1973). The hypothesis was originally demonstrated using a hypothetical polygynous ungulate. Trivers and Willard argued that mothers in good condition would produce a son that would outcompete a daughter, because a highly successful son would leave many more grandchildren for the mother than even the most successful daughter, who is constrained to a low reproductive rate (usually one or two offspring a year in ungulates). On the contrary, a mother in poor condition would produce a daughter that would outcompete a son, as most females that survive to adulthood produce at least some offspring, whereas an unsuccessful male in a polygynous species may never breed. Therefore, the mother would leave more grandchildren from her daughter than from her son.

Despite the age of the hypothesis (30 years and around 1000 citations), the number of citations per year continues to grow, and recent theoretical articles and, in particular, criticisms are numerous (e.g. Frank 1990; Hiraiwa-Hasegawa 1993; Festa-Bianchet 1996; Kojola 1998; Hewison & Gaillard 1999; Packer *et al.* 2000; Brown 2001). The hypothesis has been tested on a variety of taxa, and studies have investigated both variation in birth sex ratios, and the extended prediction that maternal investment should vary in relation to maternal condition. However, over 50% of all studies are on mammals, and Trivers and Willard originally proposed the hypothesis using mammals as an example. It is therefore surprising that despite extensive theoretical considerations and empirical tests, few studies have provided conclusive evidence either for or against the TWH, and that the results are seemingly so inconsistent between, and even within, species. Consequently, most authors only agree that empirical studies of the TWH produce results that are difficult to interpret because of their inconsistency (Clutton-Brock & Iason 1986; Frank 1990; Festa-Bianchet 1996; Kojola 1998; Hewison & Gaillard 1999; Brown 2001).

The lack of a known mechanism for sex-ratio adjustment magnifies the problem of interpreting results. Most studies on sex-ratio manipulation mention that there is no known mechanism by which sex ratios could be adjusted (e.g. Cockburn *et al.* 2002), although several hypotheses have been proposed (e.g. Krackow 1995*a*,*b*; James 1996; Forchhammer 2000; Krackow *et al.* 2003). Many of these mechanisms focus on differences around conception or are related to early development (e.g. Krackow 1995*a*; Krackow & Burgoyne 1998; Forchhammer 2000). In addition, some studies have suggested that sex-ratio adjustment may occur around conception (e.g. Cameron *et al.* 1999; Enright *et al.* 2001), and a recent meta-analysis on ungulates showed that measures taken around conception provide the most consistent support for the TWH (Sheldon & West 2004).

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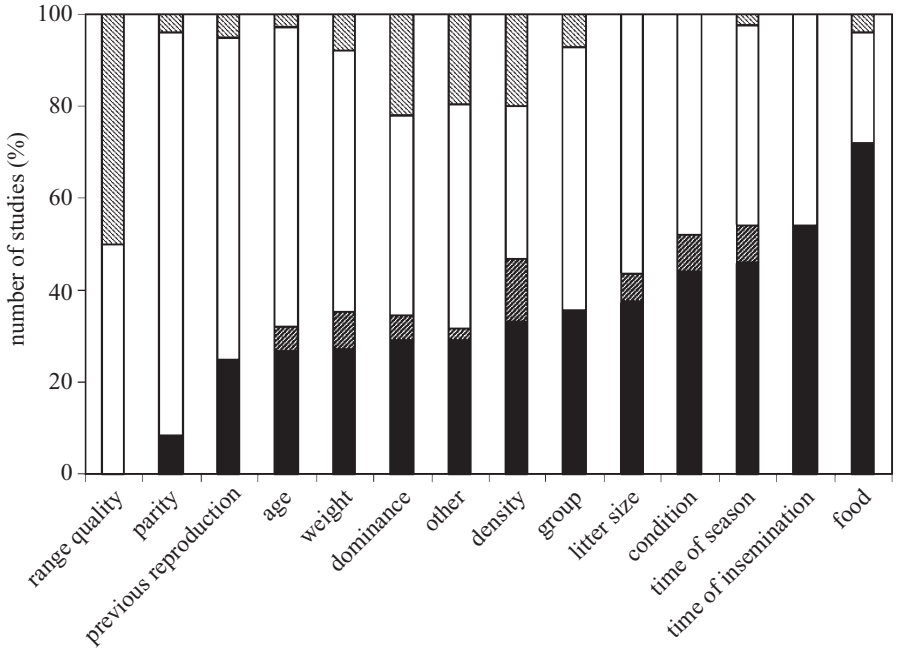

Figure 1. Studies of the TWH in relation to condition measure that show significant support (black), a non-significant trend to support (light hatching), no significant support (white) or a significant result in the opposite direction (dark hatching).

I therefore performed a meta-analysis (Hedges & Olkin 1985) on previous studies, investigating sex ratios in all mammalian taxa (except humans) to determine if studies that measured condition near the time of conception showed more support for the TWH than studies using condition measures at other times of the reproductive cycle.

2. METHODS

I attempted to locate all the studies that investigated sex-ratio variation in mammals by searching for citations of Trivers & Willard (1973) in the Science Citation Index and the Social Sciences Citation Index. To prevent a positive publication bias (i.e. studies that found results supporting TWH are more likely to cite TWH (Festa-Bianchet 1996)), I also searched for all papers that mentioned 'sex ratio' in the abstract in the Science Citation Index and Current Contents. Studies were included up to 15 April 2003. This search yielded almost 1000 studies. Out of these, I judged the studies in terms of the taxa on which the study focused. After locating all studies on mammals, I judged from the abstract whether papers presented data on sex ratios or later aspects of maternal investment. All those investigating sex ratios in mammals (except humans) were collected. I then isolated all the studies that involved an empirical test of the hypothesis. From this collection I excluded those papers that tested for sex-ratio biases on a population level, as Trivers and Willard made explicit predictions about individuals, not populations (Trivers & Willard 1973; Sheldon & West 2004). Studies were used in the general analysis but excluded from the meta-analysis if there was insufficient presentation of the statistical analysis for conducting an analysis. For the meta-analysis I needed a test statistic or *p*-value and the sample size. In some cases I was able to calculate these from the data presented and these analyses were used in the analysis. All the studies used are listed in electronic Appendix A.

Studies were classified into the categories of measures identified by the authors, and support for the TWH was attributed on the basis of the author's conclusions. Although it is not immediately obvious why some measures would be an appropriate test of the TWH, the authors' conclusions were followed for categorization of support and for conducting the meta-analysis. The studies were further categorized by the timing of the condition measure, where this was specific to a time in the reproductive cycle. Such studies were categorized as 'around conception', 'mid-gestation' and 'around birth'.

The meta-analysis was conducted using META, v. 5.3 (Schwarzer 1989). A random effect model was used, which allows real differences in effect size and sampling error to be considered simultaneously. Random effects models are probably more appropriate for ecological meta-analyses (Gurevitch & Hedges 1999).

3. RESULTS

Out of 422 tests, only 34% support TWH, with a further 5% showing a non-significant trend in support. Most studies produced results that were not significant, whereas a few (8.5%) showed a significant result opposite to that predicted by TWH. Each of the measures produced very different results (figure 1), with some showing support for the TWH (e.g. time of season 46%), whereas others demonstrated little support (e.g. maternal parity 8%, previous reproduction 25%).

The meta-analysis confirmed the initial analysis. Although there was an overall significant relationship supporting the TWH, the predictive power was low (weighted $Z = 5.42$, one-tailed: $p < 0.0001$, $n_{\text{studies}} = 381$, $n_{\text{data points}} = 3\,995\,262$). Most importantly, the data were significantly heterogeneous, suggesting that the studies are not measuring the same thing ($\chi^2 = 1077$, $p < 0.0001$). Therefore, the conclusion of previous authors (that there is little consistent support for the TWH) is supported when condition measures are considered equivalent.

When I performed a meta-analysis on each of the condition measures individually, it became apparent that the

Table 1. Meta-analysis of studies investigating birth sex ratios in relation to indices of maternal condition.

measure	$n_{studies}$	percentage support	$n_{data\ points}$	$Z_{weighted}$	effect size (r)	heterogeneity (χ^2)
all measures	381	34	3995262	5.42***	0.0027	1050.28***
age	68	27	600218	4.84***	0.0063	104.87**
condition	25	44	17850	3.94***	0.0295	29.17
density	13	33	12376	8.17***	0.0730	62.87***
dominance	45	29	7440	2.12*	0.0246	254.56***
food	23	73	10431	2.25*	0.0220	54.01***
group	12	36	1783	5.22***	0.1236	43.36***
litter	44	37	2262302	3.27**	0.0022	72.41**
parity	23	8	500687	1.27	0.0018	22.22
previous year's reproduction	16	25	3207	1.65*	0.0292	31.67**
range quality	10	0	3093	0.37	0.0066	32.07***
time of insemination	9	50	20736	0.67	0.0047	20.71**
time of season	35	46	516526	3.24**	0.0045	56.82**
weight	29	27	8207	0.38**	0.0042	119.59***
other (fewer than five studies each)	27	35	17539	0.48	0.0037	54.91**

*$p < 0.05$; **$p < 0.01$; ***$p < 0.0001$.

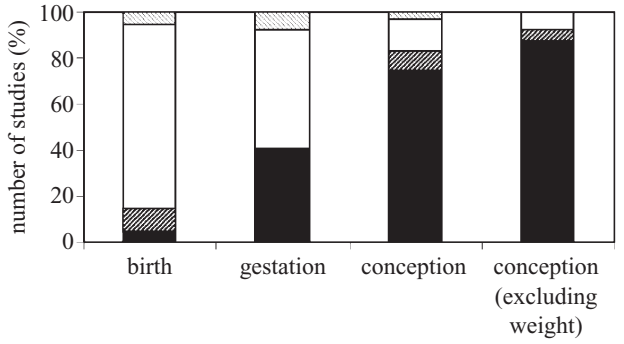

Figure 2. Studies of the TWH that measured body condition at a specific time in the reproductive cycle. A significant support (black), a non-significant trend to support (light hatching), no significant support (white) or a significant result in the opposite direction (dark hatching). Condition measures included are body weight, body condition and food availability.

measure itself was a source of considerable variance. Condition measures varied in whether there was significant support for the hypothesis, the effect size and the heterogeneity of data (table 1). Only maternal body condition significantly supported the hypothesis and had homogenous data.

To test if there was stronger support if the measure is taken close to conception, I examined studies that investigated condition around conception, rather than at other times during the reproductive cycle (figure 2). When body condition, weight or food are measured or manipulated around conception, 74% of studies support the TWH, whereas measures taken during gestation (41% support) or at birth (5%) show little relationship with sex ratio. Interestingly, three out of the four studies around conception time that did not support the hypothesis used weight as a condition measure, which can be confounded by body size; such that a heavier individual may not always be in better physical condition (e.g. Stamps 1990; Cameron *et al.* 1999; Georges & Guinet 2001). When I excluded weight measures of condition around conception the relationship was further strengthened; 88% of the 25 studies showed significant support for the TWH, with one study additionally showing a non-significant trend to support (92%). The meta-analysis of these studies shows a significant relationship (table 2; $Z = 6.32$, $p < 0.00001$), with a larger effect size ($r = 0.08$), and, most importantly, no significant heterogeneity of data, suggesting the data are measuring the same effect ($\chi^2 = 13.53$, $p = 0.96$). Therefore, the TWH is consistently supported if the measure used relates to variation in maternal condition around conception.

4. DISCUSSION

My analysis suggests that sex-ratio adjustment occurs at or near implantation. The analysis does not preclude adjustment at other times, but does suggest that inconsistencies in results may be caused by the appropriateness of the condition measure, rather than because adjustment is not occurring. If sex-ratio adjustment occurs around implantation, variation is not because of differential loss of less viable or more costly offspring during gestation. The observed variation is consequently more likely to be a facultative adjustment rather than a side effect of accelerated male growth rates in sexually dimorphic species, particularly as some studies have shown significant results in relatively monomorphic species (e.g. Cameron *et al.* 1999). The analysis suggests a more general applicability of the TWH than is currently thought, and emphasizes the importance of appropriate methods for hypothesis testing in studies of sex-ratio variation in mammals, with the most valuable studies being designed to test the TWH, rather than retrospective analysis of datasets collected for alternative purposes.

(a) *A potential mechanism?*

Recent research on embryos raised *in vitro* has identified a potential mechanism for sex-ratio adjustment by demonstrating two important characteristics of conceptuses. First, there is sexual dimorphism in the expression of interferon-tau (which signals pregnancy to the mother (Bazer *et al.* 1997)), as early as the expanding blastocyst

Table 2. Meta-analysis of sex-ratio variation in relation to condition measures (food, body condition, weight) taken at different times during the reproductive cycle.

measure	$n_{studies}$	percentage support	$n_{data\ points}$	$Z_{weighted}$	effect size (r)	heterogeneity (χ^2)
birth measures	18	5	8746	0.74	0.0079	38.05**
gestation measures	25	41	19939	3.33**	0.0236	86.32***
conception measures	31	74	7518	6.04***	0.0697	64.01***
conception (excluding weight)	25	88	6504	6.13***	0.0772	14.18

stage (Larson *et al.* 2001). Consequently, males and females are distinct from a young age, and mothers are signalled differently, potentially providing plasticity in embryo selection during pregnancy establishment (Flint *et al.* 1997; Larson *et al.* 2001). The concern that mothers would be unable to adjust sex ratios facultatively may be alleviated if blastocysts signal their presence differentially. Second, an increase in glucose in a culture medium inhibited the development of the female conceptus from divided cells into expanded blastocysts (Larson *et al.* 2001), supporting other research that suggested that glucose influenced the development of male and female blastocysts differently (Gutiérrez-Adán *et al.* 2001). The role of glucose in determining sex ratios explains a frequently observed phenomenon; embryos become increasingly male-biased as they develop from differentiated cells into expanded blastcysts *in vitro* (where glucose-enriched media are used for development), but not *in vivo* (e.g. Gutiérrez-Adán *et al.* 1996; Catt *et al.* 1997; Dominko & First 1997; Pegoraro *et al.* 1998; Hasler *et al.* 2002). A similar pattern emerges with human blastocysts used for *in vitro* fertilization; more differentiated cells result in a male-biased sex ratio (e.g. Mercader *et al.* 2001; Milki *et al.* 2003). The glucose added to *in vitro* cultures enhances male conceptus growth and development, but inhibits female conceptus growth and development, causing the difference in sex ratios between conceptuses raised *in vivo* and *in vitro* (Larson *et al.* 2001).

A recent study lends support to the hypothesis that circulating glucose during early cell division may alter sex ratios, showing that significantly more males were produced when mothers were fed a high-fat diet at conception compared with a low-fat diet, with the same total calorific value (Rosenfeld *et al.* 2003). The study supports earlier suggestions that fatty acids play a role in favouring male offspring (Austad & Sunquist 1986; Crawford *et al.* 1987). A high-fat diet can result in higher levels of circulating glucose (e.g. Folmer *et al.* 2003), thereby supporting the hypothesis that glucose may be contributing to sex determination.

Furthermore, several confusing results could be explained if glucose influences the development of males and females differently.

(i) Socially stressed animals typically produce more sons than daughters (see, for example, Krackow & Hoeck 1989; Pratt & Lisk 1989; Pratt *et al.* 1989; Perret 1990). The adaptive value of producing sons under social stress is not known. However, high stress results in increases to circulating glucose. Interestingly, the stress-related litter changes can be prevented by the administration of dexamethasone (Pratt & Lisk 1990), which, among other actions, inhibits glucose transport and lowers plasma glucose (Hahn *et al.* 1999; Shamey *et al.* 2000; Buren *et al.* 2002; Seematter *et al.* 2002), without a major impact on follicular development.

(ii) A study using mice as an animal model for human diabetes found that an increase in circulating glucose resulted in a male-biased sex ratio (Machado *et al.* 2001). Conversely, insulin-dependent human mothers, who have low glucose availability, show a sex ratio significantly biased towards daughters (Rjasanowski *et al.* 1998).

(iii) Offspring sex ratio tends to vary with timing of insemination (e.g. Verme & Ozoga 1981; Krackow & Hoeck 1989; Hedricks & McClintock 1990; Huck *et al.* 1990). Glucose levels vary throughout the cycle, and are important for reproductive activity (e.g. Diskin *et al.* 2003; Reist *et al.* 2003), such that differences in sex ratio in relation to timing of insemination could, in principle, be explained by differences in blood glucose. Further research is required to determine if sex ratios that vary with timing of insemination are related to fluctuations in glucose availability.

Interestingly, the relationship between dominance and sex ratios is particularly inconsistent (e.g. Brown & Silk 2002). Out of the 56 studies on the effect of maternal dominance on sex ratios reviewed here, only 29% support the hypothesis, 43% yielded non-significant results and 21% had significant results opposite to the predictions of the TWH, and the inconsistency was apparent in the 45 studies used in the meta-analysis (table 1). Several studies have shown a U-shaped relationship between dominance and sex ratios, with both low-ranked and high-ranked females producing more sons (e.g. Berman 1988). Higher stress levels could result from being both dominant and subordinate. Such confusing patterns could arise if both stress and nutrition contributed to sex-ratio determination through an interaction with blood glucose.

Therefore, there is evidence that glucose levels during early cell division may contribute to biased sex ratios, as increased levels of glucose inhibit the development of female blastocysts (Larson *et al.* 2001). Glucose influences a range of reproductive variables, such as the expression of growth hormone receptor (Brameld *et al.* 1999), gonadotropin-releasing hormone release and the timing of the luteinizing hormone pulse (Diskin *et al.* 2003), and so may influence blastocyst development directly, or indirectly through interactions with other factors. Other

hypotheses concentrate on early gestation as a time for an adjustment mechanism, particularly the developmental asynchrony hypothesis (Krackow 1995*a*,*b*, 1997; Krackow & Burgoyne 1998). The glucose hypothesis does not preclude this hypothesis. Nonetheless, some studies showing marked sex-ratio variation have been on relatively monomorphic species (e.g. horses (Monard *et al.* 1997; Cameron *et al.* 1999)), suggesting that sex differences in growth may be insufficient to explain all the trends observed. The role of glucose does not depend on sexual-size dimorphism. Further studies designed to directly test the role of glucose in determining sex ratios in mammals are required.

The ideas expressed here benefited from discussions with Trish Fleming, Wayne Linklater, Ed Minot and Kevin Stafford. The manuscript was improved by comments from Bill Bateman, Neil Jordan, George Potter, Robert Trivers and anonymous referees.

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