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Original Article

Maternal stress to partner quality is linked to adaptive offspring sex ratio adjustment

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Female birds have been shown to have a remarkable degree of control over the sex ratio of the offspring they produce. However, it remains poorly understood how these skews are achieved. Female condition, and consequent variation in circulating hormones, provides a plausible mechanistic link between offspring sex biases and the environmental and social stresses commonly invoked to explain adaptive sex allocation, such as diet, territory quality, and body condition. However, although experimental studies have shown that female perception of male phenotype alone can lead to sex ratio biases, it is unknown how partner quality influences female physiological state. Using a controlled within-female experimental design where female Gouldian finches (*Erythrura gouldiae*) bred with both high- and low-quality males, we found that partner quality directly affects female hormonal status and subsequent fitness. When constrained to breeding with low-quality males, females had highly elevated stress responses (corticosterone levels) and produced adaptive male-biased sex ratios, whereas when they bred with high-quality males, females had low corticosterone levels and produced an equal offspring sex ratio. There was no effect of other maternal hormones (e.g., testosterone) or body condition on offspring sex ratios. Female physiological condition during egg production, and variation in circulating hormones in particular, may provide a general mechanistic route for strategic sex allocation in birds.
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INTRODUCTION

In response to a number of social and environmental variables, female birds have been shown to have a remarkable degree of control over the sex ratio of the offspring they produce (Komdeur et al. 1997; Sheldon et al. 1999; Pryke and Griffith 2009a). To date, however, it remains unknown how these skews are achieved (reviewed in Pike and Petrie 2003; Alonso-Alvarez 2006). One idea gaining increasing attention is that hormones circulating in the breeding female around the time of sex determination could affect sex adjustment (Krackow 1995; Pike and Petrie 2003). Elevated maternal glucocorticoids during egg production have been linked to female-biased primary sex ratios (Pike and Petrie 2005, 2006; Bonier et al. 2007) and male-biased embryo mortality (Hayward and Wingfield 2004; Love et al. 2005; Love and Williams 2008). Increased levels of reproductive hormones in breeding females have also been associated with male-biased sex ratios, including maternal testosterone (Veiga et al. 2004; Pike and Petrie 2005; Rutkowska and Cichon 2006), prolactin (Badyaev et al. 2005), and progesterone (Correa et al. 2005).

Variations in maternal hormones are likely to be related to the social and environmental variables that have commonly been associated with offspring sex adjustments. For example, relative body weight (Nager et al. 1999; Kalmbach et al. 2001; Thuman and Griffith 2005), maternal diet (Nager et al. 1999; Rutstein et al. 2004), age (Blank and Nolan 1983), dominance status (Leonard and Weatherhead 1986), territory quality (Komdeur 1996), and breeding season (Dijkstra, Daan, and Buker 1990; Lessells et al. 1996) can influence a female's condition and physiological state. Nevertheless, it is unclear if similar mechanistic pathways underlie sex ratio adjustment when females bias offspring sex in relation to the phenotype of their partner. In socially monogamous birds, partner quality can affect the quality of the territory that a female inhabits, and thus can influence female condition (Griffith and Pryke 2006). However, although experimental demonstrations have shown that extreme sex ratio biases can result from the physical appearance of the partner alone (e.g., Ellegren et al. 1996; Sheldon et al. 1999; Pryke and Griffith 2009a), the influence on female physiological state is unclear.

Here, we examine the underlying mechanism of extreme sex ratio adjustments in the socially monogamous Gouldian finch (*Erythrura gouldiae*), where the quality of the male is unrelated to extrinsic factors, such as territory or food quality. Instead, mate quality is signaled through genetically determined head color (black or red), where there are predictable and severe sex-specific fitness effects associated with breeding with a genetically incompatible partner of the alternative head-color morph (Pryke and Griffith 2009b). In particular, genetically incompatible pairs have 40.2% greater mortality of sons and 83.8% greater mortality of daughters (i.e., from egg to sexual maturity) than broods produced from genetically compatible pairs of the same head-color morphs (Pryke and Griffith 2009b). Nevertheless, despite these fitness costs, and genetically determined morph-assortative mate preferences (Pryke 2010), mixed morph mating is not uncommon in wild populations (20–30%) because of limitations on mate availability (Pryke and Griffith 2007). However, females constrained to breeding with incompatible partners compensate for increased daughter mortality by overproducing sons (up to 81.2%; Pryke and Griffith 2009a). Recent work has also shown that females paired with incompatible mates have elevated stress responses (i.e., elevated plasma corticosterone; Griffith et al. 2011). Using a within-female experimental

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design, unconfounded by precopulatory mate choice and environmental sources of variation, we investigated whether the stress levels of females breeding with both low- and high-quality mates underlies adaptive offspring sex adjustment.

MATERIALS AND METHODS

Experimental breeding design

Experiments were run between January and April 2010 (Martinsville, New South Wales, Australia). Wild-type Gouldian finches used in these experiments were naive (virgin) birds of the same age (all in their first year of adult plumage; Pryke 2007) and of known genotypes. Sex-linked genotypes of red and black morphs can be directly inferred from the phenotype of females (i.e., red females are Z^R and black females are Z^r) and black males (i.e., homozygous recessive: Z^rZ^r). Because heterozygous red males (Z^RZ^r) are genetically incompatible with red females (Pryke and Griffith 2009b), only homozygous dominant males (Z^RZ^R) were used in these experiments (determined from established pedigrees).

In total, 50 red females were initially paired to either a red ($n = 25$) or black male ($n = 25$), and 50 black females were paired to either a red ($n = 25$) or black male ($n = 25$). At the completion of the first brood (i.e., independent offspring; offspring 60 days old), the adult male and offspring were removed from the cage. After an isolation period of 18 days (to exclude the possibility that female's store sperm; Birkhead and Møller 1993), females were then paired with a male of the alternate morph. Consequently, each female ($n = 100$) was paired with a partner of their own phenotype (compatible pair) and a partner of a different phenotype (incompatible pair). This experimental design ensured that any effects of variation in maternal hormonal responses were not confounded by genetic differences between females. To preserve the conservative within-individual experimental design, only females that bred with both male morphs were included in analyses ($n = 86$; 43 compatible and incompatible pairs).

All pairs were placed in single visually isolated breeding cages (1.2 m^3) with nest-boxes (details in Pryke and Griffith 2010). Nest-boxes were checked each morning (between 06:00 and 08:00 h), and freshly laid eggs were individually marked with a nontoxic marker. There were no temporal gaps in the laying sequence (i.e., all females laid one egg per day), although females in incompatible pairs laid smaller clutches than those in compatible pairs (see also Pryke and Griffith 2009a). On the day the first egg hatched, we checked nests every 2–3 h (during daylight hours) to determine which hatchling came from which egg and marked newly hatched chicks by clipping one of their toenails.

The sex of all offspring was determined from a small blood sample (2–3 drops) taken from chicks or tissue (stored in alcohol) from dead chicks (<2 days) by polymerase chain reaction amplification of sex-linked markers P2 and P8 following standard protocols (Griffiths et al. 1998). All unhatched eggs that developed a visible embryo were successfully sexed.

Hormone and body condition measures

To examine individual variation in plasma corticosterone and testosterone levels of females, we took blood samples in the early morning (05:00–06:00 h) on the day each female laid their second egg. Because circulating corticosterone increases rapidly in birds after capture (Wingfield 1994), we collected blood samples (ca. 75 μl) within 2 minutes of capture (49.4 ± 5.9 s) to ensure we measured baseline levels. There were no differences in capture times between females paired to compatible and incompatible males ($F_{1,85} = 0.37$, $P = 0.73$) and there was no detectable effect of time after capture (within 0- to 2-min interval) on plasma corticosterone levels ($F_{1,85} = 0.53$, $P = 0.59$). Blood samples were centrifuged and plasma removed and stored at −20 °C.

Maternal body condition is associated with offspring sex ratio biases in a number of species (e.g., Nager et al. 1999; Kalmbach et al. 2001), and we therefore examined within-individual changes in body mass (i.e., measure of body condition) when females bred with compatible and incompatible males. Body mass (to the nearest 0.1 g) was measured on the mornings (05:00–06:00 h) that the female laid her second egg (immediately after blood samples were taken).

Hormone assays

Corticosterone was measured in duplicate from plasma samples using a Cayman Enzyme Immunoassay kit (no. 500651; Ann Arbor, MI; details provided in Pryke et al. 2007). Kit instructions were followed for steroid extraction, but each sample was also initially spiked with a trace amount of radiolabeled corticosterone (Amersham [1,2,6,7-3H]) to determine and optimize percentage recovery (tritium activity measured using a scintillation counter). After extraction in dichloromethane, samples were reconstituted in buffer dilutions optimized for the standard curve (1:20). Final hormone values were adjusted for individual sample recovery (mean recoveries were 88.5 ± 1.8%). Intra-assay variation was 6.9% and inter-assay variation was 10.2%, assessed using a chicken plasma pool.

Testosterone was analyzed in duplicate using a Cayman Enzyme Immunoassay kit (no. 582701; Ann Arbor, MI; details provided in Pryke et al. 2007). Briefly, each sample was initially spiked with tritiated testosterone (Amersham [1,2,6,7-3H]) to allow the calculation of recoveries following extraction in diethyl ether, and samples were redissolved in buffer dilutions optimized for the standard curve (1:20). Percentage recovery was 85.6% ± 2.6%, and intra-assay variation and inter-assay variation were 7.4% and 11.6%, respectively. Testosterone was quantified for a random subsample ($n = 60$) of 30 individual females (15 red, 15 black) when breeding with both compatible and incompatible males.

Statistical analyses

Data were analyzed in GENSTAT 9 (Rothamsted Experimental Station, Harpendon, UK). Within-individual differences in female hormonal responses and body condition between the 2 breeding contexts (i.e., compatible and incompatible) were analyzed using repeated-measures analysis of variance with a structured blocking function (i.e., repeated measures for within-individual comparisons). All variables were tested for normality; those that did not meet the requirements of normality were successfully normalized by transformation.

Offspring sex ratios (number of males/total number of sexed eggs), as well as offspring survival at hatching (number of nestlings hatching/total number embryos) and fledging (number of nestlings fledging/total number hatching) were analyzed at the brood level by fitting generalized linear mixed models. The error distribution was binomial with a logit link, weighted by brood size (i.e., to account for differences in brood sizes between compatible and incompatible pairs). Female identity was included as the random repeated subject but did not constitute a significant random component in any model ($P > 0.24$). The significance of explanatory terms in these models was assessed by the Wald statistics, which are distributed as χ^2. Significant probability values were derived

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from having all relevant terms fitted in the final model together, whereas those of nonsignificant terms were obtained from having all significant terms in the final model and each nonsignificant term fitted individually. Means ± standard deviation are presented throughout.

RESULTS

Maternal response to male quality

Females constrained to breeding with incompatible mates had significantly higher plasma corticosterone levels during egg production compared with when they bred with compatible males ($F_{1,85} = 32.17$, $P < 0.001$; Figure 1). These within–female differences in circulating levels of corticosterone to male quality were evident in both black and red females (female morph × male morph: $F_{2,84} = 0.41$, $P = 0.69$; female morph: $F_{1,85} = 0.09$, $P = 0.81$) and there no effects of the order in which a female was paired with a compatible or incompatible partner ($F_{1,85} = 0.52$, $P = 0.47$).

Testosterone levels of individual females did not differ between breeding with compatible and incompatible males ($F_{1,59} = 1.68$, $P = 0.19$; Table 1). There were also no morph-specific differences to mate quality (female morph × male morph: $F_{2,58} = 0.56$, $P = 0.57$; female morph: $F_{1,59} = 0.31$, $P = 0.72$) or treatment order effects ($F_{1,59} = 0.51$, $P = 0.64$). Similarly, despite the considerable stress-related increase in corticosterone when breeding with incompatible males, individual female body weight did not change between the 2 breeding contexts ($F_{1,85} = 1.21$, $P = 0.24$; Table 1), irrespective of the order in which females bred with compatible or incompatible males ($F_{1,85} = 0.91$, $P = 0.37$).

Maternal hormones influence offspring sex ratios

Consistent with previous findings (Pryke and Griffith 2009a), individual females breeding with compatible mates produced an unbiased primary sex ratio (48.7% male; $\chi^2 = 1.74$, $P = 0.16$, $n = 44$ families, 234 offspring) but when breeding with incompatible males overproduced sons (82.2%; $\chi^2 = 23.72$, $P < 0.001$, $n = 42$ families, 149 offspring). There was no relationship between offspring sex ratio and position within the laying order ($\chi^2 = 0.63$, $P = 0.42$, $n = 88$ families, 430 eggs) for either compatible or incompatible breeding pairs ($\chi^2 = 1.27$, $P = 0.26$), and thus sons were produced throughout the laying sequence.

Table 1

Mean (±standard deviation) within-female differences in body weight, corticosterone, and testosterone levels for females breeding with both compatible and incompatible males

Measure	Compatible male	Incompatible male
Corticosterone (ng/ml)	**19.36 ± 25.91**	**67.95 ± 42.53**
Testosterone (ng/ml)	0.12 ± 0.11	0.15 ± 0.14
Body weight (g)	13.92 ± 1.23	13.07 ± 1.48

Only corticosterone levels (in bold) differed between pairings.

Primary offspring sex ratios differed with circulating levels of maternal corticosterone ($\chi^2 = 48.76$, $P < 0.001$) but not with testosterone ($\chi^2 = 1.43$, $P = 0.24$). In particular, females with relatively higher corticosterone levels produced significantly more sons in their broods than those with lower corticosterone levels (Figure 2). The influence of maternal corticosterone on sex ratio biases is evident in both compatible and incompatible breeding contexts, as demonstrated by the nonsignificant interaction term (breeding context × corticosterone: $\chi^2 = 2.08$, $P = 0.15$; Figure 2).

In 47 eggs (9.2% of eggs produced), there were no visible signs of embryonic development (despite normal incubation) and we were unable to sex these eggs (i.e., infertile or suffered very early embryo mortality). Clutches produced by incompatible pairs had significantly more unsexed eggs (8.4%) than compatible pairs (0.8%; $\chi^2 = 17.63$, $P < 0.001$). However, there was no relationship between maternal corticosterone levels and the number of unsexed eggs ($\chi^2 = 0.86$, $P = 0.36$) in either breeding context (compatible or incompatible: $\chi^2 = 0.09$, $P = 0.77$).

In total, 126 of 383 nestlings died between hatching and fledging. Proportionately more offspring from incompatible nests (32.4%) died than from compatible nests (2.3%) ($\chi^2 = 23.42$, $P < 0.001$), and this effect was particularly severe for daughters (offspring sex: $\chi^2 = 1.01$, $P = 0.32$; genetic parents × offspring sex: $\chi^2 = 8.76$, $P < 0.001$; see also Pryke and Griffith (2009b) for specific mortality rates throughout juvenile development). Similar to findings for infertility and prehatching mortality, maternal corticosterone was unrelated

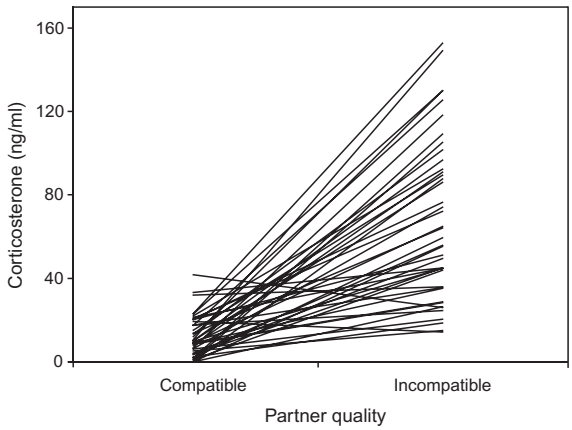

Figure 1
Within-female changes in stress levels (circulating corticosterone concentrations) at egg production for individual females breeding with compatible and incompatible males. Treatment order (i.e., first breeding with compatible or incompatible mate) was randomized in the experiments.

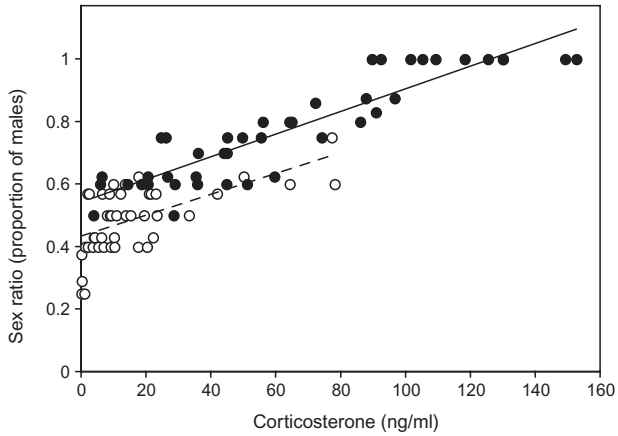

Figure 2
Relationship between primary offspring sex ratio and corticosterone concentrations (ng/ml) of egg-laying females breeding with compatible (open circles; $y = 0.0034x + 0.4301$, $r^2 = 37.7\%$) and incompatible males (closed circles; $y = 0.0035x + 0.5423$, $r^2 = 82.2\%$).

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to offspring mortality at hatching ($\chi^2 = 1.85$, $P = 0.17$, $n = 86$ broods) or fledging ($\chi^2 = 1.81$, $P = 0.18$, $n = 75$ broods). Furthermore, circulating levels of maternal testosterone were unrelated to either hatching ($\chi^2 = 0.48$, $P = 0.49$, $n = 59$ broods) or fledging success ($\chi^2 = 0.12$, $P = 0.73$, $n = 51$ broods).

DISCUSSION

Corticosterone is the major avian glucocorticoid released in response to stress (Harvey et al. 1984) and the 3.5-fold increase in corticosterone levels of females paired to incompatible males suggests that females experience considerable stress when breeding with males that they perceive as incompatible or low quality (Griffith et al. 2011). These elevated maternal stress hormones, in turn, may play a mechanistic role in the sex manipulating process. Females with relatively higher circulating corticosterone concentrations during egg laying produced significantly more sons. Although this is consistent with a growing number of studies that report a relationship between maternal corticosterone and sex-biased broods (Love et al. 2005; Pike and Petrie 2005; Bonier et al. 2007), our results contrast with these studies in being biased toward sons rather than daughters. This sex-related discrepancy is likely related to the fitness gains from strategic offspring sex manipulation. In contrast to most studies of sex allocation that predict that females should overproduce sons when breeding with attractive partners (Burley 1986; Sheldon 1999; West and Sheldon 2002), in the Gouldian finch system, there is a selective advantage to females of overproducing sons when paired with a nonpreferred male morph (Pryke and Griffith 2009a, 2009b).

It is also possible that corticosterone may influence sex ratios through its affects on other hormones. For example, testosterone has been associated with male-biased ratios (Veiga et al. 2004; Pike and Petrie 2005; Rutkowska and Cichon 2006; Goerlich et al. 2009) and corticosterone may have inhibitory effects on testosterone production (DeNardo and Sinervo 1994). We found no evidence that testosterone was related to the observed male-biased sex ratios or that the levels of maternal testosterone varied significantly with corticosterone levels. However, the interactions between corticosterone and testosterone are complex and could be obscured by other factors (Wingfield et al. 1992) and variation in other reproductive hormones (e.g., progesterone and prolactin).

Elevated corticosterone may also have negative effects on other physiological functions (Sapolsky 1992; Munck et al. 1994; Wingfield and Ramenofsky 1999; Sapolsky et al. 2000; Cyr and Romero 2007). Although decreases in body weight are typically associated with stress (e.g., Gray et al. 1990; Romero and Wikelski 2001) and are often related to offspring sex ratio biases (e.g., Nager et al. 1999; Kalmbach et al. 2001), there was no difference in female body weight (at egg laying) between the 2 breeding contexts, despite large variations in corticosterone levels and extreme sex ratio adjustment. This may be a result of the constant availability of high-quality food for the captive birds, but it eliminates low body weight as a confounding factor in this experiment.

Birds have no known physiological or genetic mechanisms for skewing offspring sex ratios (Krackow 1995; Oddie 1998; Sheldon 1998; Komdeur and Pen 2002). However, recent studies suggest the potential involvement of maternal hormones (reviewed in Pike and Petrie 2003; Alonso-Alvarez 2006). In many species, for example, increased maternal hormones may influence sex-specific embryo mortality (Pike and Petrie 2003; Hayward and Wingfield 2004; von Engelhardt et al. 2004; Love et al. 2005; Love and Williams 2008). In Gouldian finches, however, elevated corticosterone levels are not responsible for the higher embryo and offspring mortality of incompatible pairings, which appear to be primarily determined by genetic incompatibilities between head-color morphs (see Pryke and Griffith 2009b).

In birds, females are the heterogametic sex (producing Z and W bearing ova), and thus females may potentially control offspring sex prior to ovulation (Oddie 1998; Pike and Petrie 2003). Follicles developing in the ovary contain both sets of chromosomes until the first meiotic division during which one of them is consigned to the polar body (i.e., discarded) and the other remains in the ovum, effectively determining the sex of the egg (Romanoff and Romanoff 1949; Johnson 2000). If the ovulated egg is fertilized, it undergoes the second meiotic division and develops into a male or female embryo (Romanoff and Romanoff 1949). In several bird species, chromosome segregation has been shown to occur shortly (0.5–3 h) before ovulation (Birenkott et al. 1988; Johnson 1996), and thus fine-tuned adjustment of offspring sex may be possible, even between different eggs within the clutch. Potentially, females could selectively abort unwanted sex follicles and ovulate only follicles of the desired sex (Emlen 1997; Pike and Petrie 2005). However, this mechanism is likely to induce frequent laying sequence gaps (ca. 24 h; Emlen 1997), which would be evident in species laying large clutches, such as Gouldian finches (3–8 eggs per clutch). Therefore, this mechanism is unlikely to explain the observed sex biases in this study because there were no temporal delays in the laying sequence in this study ($n = 86$ pairs) or in previous studies with similar breeding designs ($n = 324$ pairs; Pryke and Griffith 2009a). Furthermore, there are no intraclutch variations in egg and chick size (Pryke and Griffith 2009a, 2010), which might be expected if targeted follicles are ovulated prematurely, resulting in eggs with smaller yolks (Pike and Petrie 2003; Alonso-Alvarez 2006). Instead, high circulating levels of corticosterone may directly influence sex chromosome segregation at the first meiotic division (Petrie et al. 2001) or lead to differential maturation rates of sex-specific ova (Krackow 1995). Variation in circulating levels of corticosterone in egg-laying females could therefore potentially provide a dynamic physiological mechanism for females to control primary (preovulation) offspring sex ratios.

Most sex allocation studies have examined female adjustment in relation to social and environmental factors that are likely to directly affect maternal condition, such as food availability or diet (Nager et al. 1999; Rutstein et al. 2004), increased egg production (Nager et al. 1999; Pike 2005), helpers at the nest (Komdeur et al. 1997), breeding stage (Dijkstra, Bult, et al. 1990), and habitat quality (Wiebe and Bortolotti 1992; Komdeur 1996). Corticosterone typically fluctuates rapidly in response to social and environmental stresses (Schwabl et al. 1997; Gil et al. 1999; Whittingham and Schwabl 2002), and corticosterone is often elevated in birds that are stressed or in poor condition (e.g., Kitaysky et al. 1999; Schoech et al. 1999; Williams et al. 2008). Consequently, corticosterone may mediate physiological responses to stressful events (Wingfield et al. 1998; Sapolsky et al. 2000), which may directly or indirectly (e.g., via other hormones) affect female condition and physiological state. Although the link between environmentally induced stress (e.g., food and habitat quality) and female physiological state is perhaps more obvious, socially induced stresses may also affect maternal physiological condition. In Gouldian finches, females constrained to breeding with low-quality males are highly stressed (elevated corticosterone levels), both on initial pairing with incompatible males and during egg production (Griffith et al. 2011). Thus, female physiological condition, mediated through corticosterone (Pike and Petrie 2005, 2006; Bonier et al. 2007) and perhaps other interacting maternal hormones (e.g., Badyaev et al. 2005; Correa

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et al. 2005; Pike and Petrie 2005), may be a general mechanism underlying adaptive sex ratio biases in birds.

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