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The influence of androgenic steroid hormones on female aggression in 'atypical' mammals

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Dimorphism on dominance and agonistic behaviour in mammals tends to be strongly biased toward males. In this review, we focus on a select few species of mammals in which females are as or more aggressive than males, and/or are dominant to males, and explore the role of androgenic hormones in mediating this important difference. While the data are not as clear-cut as those published on traditional laboratory mammals, our review highlights important endocrine substrates for both organizational and activational influences of steroids on female aggressive behaviour. We highlight areas in which further observations and experiments are crucial, especially the potential facilitative effects of androgens on female aggression. Finally, new and innovative techniques, including molecular genetics and receptor pharmacology, portend important insights into the ways in which androgenic hormones regulate aggressive behaviour in 'atypical' female mammals.

1. Introduction

When one views the approximately 5000 species in Class Mammalia writ large, few would contest the statement that, among the species in this class, males are more aggressive than females. This truism is reflected in prominent class-wide sexual dimorphisms in the rate of aggressive interactions, in differences in time and energy budgets allocated to aggressive and dominance interactions, variation between the sexes in the proportion of aggressive elements in the behavioural repertoire and in the evolution of traits (e.g. large body size or morphological adaptations for fighting such as large canines, horns and antlers [1,2]). Each of these traits facilitates aggressive competition among males. Traditional explanations for this prominent pattern of mammalian sexual dimorphism, vis-à-vis aggressive behaviour and its associated morphological weaponry, rely primarily on the impact of sexual selection for male traits that facilitate or promote intrasexual competitive ability, and hence reproductive success [3]. However, recent analyses have strongly suggested that this explanation is neither exhaustive nor definitive (cf. [4,5]).

As with all biological 'truisms', however, there are notable exceptions to the general rule, and exploring the causes and consequences of female aggression in species where females are as aggressive or even more aggressive than males can provide important insights into the proximate and ultimate mechanisms that underlie these differences. Ralls [6] was among the first to bring to the attention of biologists those curious cases of species in which typical mammalian sexual dimorphism in size did not hold true. This work has stimulated detailed analyses of the phylogeny, evolution and ecological contexts that are associated with mammalian species in which females are, on average, the same size or larger than males, and research on other species in which females are as or more aggressive than males (see reviews in [7,8]; [9]). To the extent that selection has favoured female aggression among mammals, one would expect to see 'fingerprints' of this selection in the underlying proximate physiological mechanisms that promote and facilitate aggression in females. Given the conservative nature of evolution, we would expect that selection for female aggression

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would operate on common or similar neurobiological pathways that are well known to regulate both enhanced male aggression (relative to females) and individual differences among males in the display of aggressive behaviour.

Given the importance of androgenic steroid hormones in both the ontogenetic organization of neural structures underlying sex-typical behaviour in males, including aggression [10], and in activating or facilitating aggressive behaviour in adult males [11,12], we focus on the impact of androgens on female aggressive phenotypes. Our goal in this paper is to highlight historical and current research on the proximate neuroendocrine mechanisms underlying female aggression in a select group of mammals. Criteria for selection of these species are twofold: first, we selected species in which mammalian-typical male-biased aggression is either absent or even sex-reversed. We included cases in which intrasexual aggression in females is conspicuous and also cases where females are more aggressive than males, and generally win fights with male conspecifics and/or exhibit dominance over males. Second, we limited our review to species for which published data on the impact of gonadal steroids on female aggression are sufficiently detailed to allow a careful assessment of the mechanisms that contribute to this atypical (for mammals) pattern.

2. Spotted hyaena (*Crocuta crotuta*)

Ever since Aristotle's formal writings over 2000 years ago on the observation that 'every [spotted] hyaena is furnished with the organ both of the male and the female' (cited in [13, p. 349]), the attention of biologists has been drawn to this species as a model for endocrine substrates for sexual differentiation of genital morphology. The conspicuously high levels of aggression in adult females and almost universal female dominance over males in clan societies also provide an exemplary natural model for exploring the differentiation of aggressive behaviour and its endocrine correlates. There is a vast and contentious literature on the ontogenetic origins of genital differentiation (which will certainly make an interesting study for historians of science; e.g. [13]), but for the purposes of this review, we limit our discussion to the role of endocrine exposure at distinct developmental stages and the link to aggressive behaviour and dominance. Below we briefly explore the evidence for differential prenatal, early postnatal and adult hormone milieus in hyaenas, and their implications for sex differences in the brain nuclei associated with social behaviour, and aggression and dominance within hyaena clans.

(a) Prenatal hormone exposure

In spotted hyaenas, levels of sex steroids rise throughout pregnancy in adult females, with levels of androgens (testosterone: T; androstenedione: A_4) peaking in the last trimester of gestation [14]. A high placental level of the steroidogenic enzyme 17β-HSD converts A_4 to T, providing a rich uterine environment with potent bioactive androgens [14,15]. This gestational androgen environment would appear to set the stage for potential masculinization of female cubs.

The most detailed evidence that prenatal hormone exposure alters patterns of postnatal aggressive behaviour comes from analyses of individual differences in maternal androgens and later-life behaviour in wild hyaenas. Dloniak *et al.* [16] confirmed the general observation of elevated maternal androgens in pregnancy, particularly in the second half of gestation. Androgen levels in this period, during which behavioural masculinization is most likely, were higher in dominant females than in subordinates. Female and male cubs exposed to higher levels of maternal androgen in the second half of pregnancy had higher levels of aggression and male-like sexual mounting than cubs exposed to lower levels of androgens, even when controlling for maternal rank and gestational glucocorticoid exposure [17]. Further analyses of lifetime rates of aggression in females revealed that rates of intersexual aggression were significantly and positively associated with gestational androgen exposure, but intrasexual aggression by females was not related to prenatal androgen exposure [18]. These data strongly implicate late-pregnancy androgen exposure as a critical determinant of both juvenile and adult female aggression.

A series of studies on the spotted hyaena have addressed organizational effects of androgens by administering a potent androgen receptor blocker (flutamide) and an aromatase inhibitor (finasteride) to pregnant females, a treatment regimen that effectively minimizes exposure of fetal hyaenas to androgenic hormones. When sampled as adults, both male and female hyaenas in the prenatal antiandrogen treatment had lower levels of T, higher levels of oestradiol (E_2) and an enhanced (i.e. more 'feminine') luteinizing hormone response to gonadotropin-releasing hormone stimulation [19]. There is an unpublished observation that prenatal antiandrogen treatment alters aggressive behaviour in females, in the form of reduced female–female neonatal sibling fights (cited in [20]), fights that are common and quite often fatal [21,22].

(b) Prepubertal hormone exposure

Female and male spotted hyaenas experience highly variable steroid hormone environments as they approach puberty. Glickman *et al.* [23] documented normative levels of sex steroids in male and female hyaenas from two to 60 months of age, and interesting sex differences emerged. First, and not surprisingly, levels of E_2 rose throughout prepubertal development in females, but not in males. Early in postnatal life, T levels did not differ between males and females, but levels increased in males beginning at 20 months of age. By 60 months of age, male T levels were five to sixfold higher than those in females. Conversely, A_4 levels showed the reverse pattern: levels were higher in females than in males beginning early in life and these sex differences persisted up to 60 months of age. Although these normative patterns of steroid exposure have not been explicitly linked with either subadult or adult aggression, removing the source of the hormones in females alters both steroid and behavioural characteristics. Neonatally ovariectomized (OVX) females displayed low or undetectable levels of both T and A_4, confirming the ovary as the source of these male-typical sex steroids in females [23]. An unpublished study implicates variation in postnatal steroid levels and aggressive behaviour in females: prepubertal ovariectomy reduced adult female aggression toward males (cited in [13]). These results are consistent with a postnatal endocrine influence on female aggression, but stronger tests of this hypothesis are required.

(c) Hormones and aggression in adult females

There is consensus in the literature that T levels in adult males (regardless of whether they live in their natal clan or

have dispersed to a new clan) are higher than those in adult females; thus, female sex-biased aggression and dominance cannot be directly traced to elevated T [14,16,24]. By contrast, levels of the important T precursor, A_4, are similar in adult male and female hyaena [14,24]. Without the knowledge of activity of the enzyme 17β-HSD that converts A_4 to T, and more importantly, sex differences in the bioactivity of this enzyme, it is impossible to determine whether T derived from A_4 conversion could potentially account for the prominent atypical sex difference in aggression and dominance in spotted hyaenas. Studies of brain structure in adult spotted hyaenas reveal departures from more traditional mammals that hint at neural substrates for enhanced aggression. The medial preoptic area and anterior hypothalamus play important roles in mediating male-typical behaviour in mammals, and the sexually dimorphic nucleus of this region, normally substantially larger in male than female mammals, was only slightly smaller in female hyaenas than in males [25]. Further, in typical mammals, males have higher densities of vasopressin-containing neurons in the lateral septum (which mediates aggressive behaviour), but in a small sample of adults, female hyaenas tended to have more vasopressin neurons than males [26]. In spite of the potential for steroids (particularly A_4) to serve an activating role for female aggression, and neural structures that are consistent with a female brain primed for aggressive behaviour, there has yet to be a good empirical test of the activational role of androgens in female spotted hyaenas. Although a facilitative effect of engaging in high levels of agonistic behaviour in elevating future levels of androgens has not been explicitly addressed as of yet in females, levels of androgens in male hyaenas are more strongly predicted by their rates of interaction with more aggressive and dominant females than by rates of male–male aggression [27]. Exploring the extent to which androgen levels in adult females vary as a consequence of rates of agonistic interactions, and subsequently alter thresholds for future aggression, would thus appear to be a promising area for additional research.

More recent evidence on spotted hyaena biology highlights the notion that circulating levels of hormones *alone* may not explain the masculinization of female genital morphology and the sex-reversed pattern of aggressive behaviour and dominance in this species. Browne *et al.* [28] report that the histomorphology of male and female gonads are distinct as early as day 30 of gestation (GD30), with male gonads expressing anti-Mullerian hormone (AMH) and three enzymes crucial for androgen biosynthesis (P450c17, cytochrome b5 and 3β−HSD). By contrast, female gonads at GD30 lacked AMH and all three enzymes. However, by GD95, ovarian tissue displayed immunohistochemical staining for all three androgen-synthesizing enzymes. Thus, there appear to be two different windows for fetal androgen synthesis for males and females. In males, the enzymatic substrates are available for androgen synthesis as early as GD30, potentially contributing to masculinization of male genitalia early in development and brain/behaviour later in gestational development. For females, by contrast, the lack of substrates for androgen synthesis in the ovary at GD30 may facilitate the growth and elaboration of accessory reproductive structures along a 'female' path (e.g. development of urethral retractor muscles and the pleating and elasticity of the 'future' birth canal). The presence of the ovarian androgen-synthesizing enzymes later in gestational development (GD90) could provide the endocrine substrates for neural masculinization (see above) underlying both neonatal and adult aggression in female hyaenas.

There are also important differences in carrier proteins for steroid hormones in the spotted hyaena that may contribute to the unique process of sexual differentiation in this species (box 1). This carrier protein (sex hormone-binding globulin; SHBG) is critical not only for increasing the half-life of steroid hormones in circulation, but also renders steroids biologically inactive while they are bound to it. Gene sequences for SHBG in spotted hyaenas were contrasted with those in three closely related species that display mammal-typical patterns of male dominance and aggression and dimorphic external genitalia [36]. Relative to the 'typical' hyaenids, spotted hyaenas have two missing amino acids and a single amino acid substitution in the SHBG secretion signal polypeptide. Modification of the spotted hyaena SHBG gene to resemble the structure of other hyaenid genes increases SHBG production, suggesting that the molecular differences in the spotted hyaena SHBG gene reduce SHBG production (and hence increase the availability of bioactive steroids to act on target tissues). Further, Hammond *et al.* [36] demonstrated that while females in the typical hyaenids have substantially higher titres of SHBG than males (and hence are 'protected' from excess androgen exposure), female spotted hyaenas had low circulating levels of SHBG, which were not statistically distinguishable from those of males. Thus, mutations in the genes regulating SHBG in spotted hyaenas, relative to other hyaenids, appear to enhance exposure of females to elevated levels of bioactive steroids, including androgens of ovarian origin, and these steroid carrier proteins may, therefore, be important contributors to variation in steroid hormone exposure.

3. Lemurs (*Lemur* spp.)

The ring-tailed lemurs (*Lemur catta*) serve as a fascinating model to explore proximate mechanisms for female aggression. Female lemurs are unique in that they exhibit unconditional dominance over adult males and sometimes over other females. This female-dominant social structure in ring-tailed lemurs was first reported by Jolly [37], and patterns of female dominance were later described in other lemur species [38–40]. Adult male lemurs will exhibit spontaneous submissive behaviours toward mature female conspecifics [41,42] and this relationship of female dominance over males occurs in reproductively mature lemurs only [43]. Moreover, females are generally intolerant of other conspecifics and fail to provide support to others during conflicts [44]. Nearly all lemurs are seasonal breeders and aggression peaks during times of peak mating and birth seasons [45], and female lemur aggression is intimately linked with patterns of feeding [46,47].

Male-like traits in female lemurs are evident in their genital morphology. Female lemurs possess masculinized genitalia including an elongated, enlarged clitoris [48], and there are potential indications that this 'masculinization' could be influenced by fetal androgen exposure [49]. For instance, lemurs pregnant with female offspring possess higher androgen/oestrogen ratios, and, consequently, the relative and not absolute values of prenatal androgens may be partially responsible for the masculinization seen among female lemurs [50]. While the endocrinology of pregnancy in female ring-tailed lemurs seems appropriate for the

Box 1. Issues in the measurement of androgens in female mammals.

There are several methodological issues that must be considered when measuring steroid hormones in female mammals, some of which are unique to females and others that are equally applicable to males and females.

Source of hormone. Multiple tissues in females are capable of synthesizing and secreting androgen hormones, including the ovary, placenta, fetus and adrenal cortex. While ovaries are the primary sources of oestrogens and progestins, thecal cells have the potential to produce androgens and, depending upon ovarian levels of aromatase, are either converted to oestrogens or secreted as androgens [29]. Conceptive ovarian cycles in a number of mammalian species, including dogs, marmosets, baboons and humans, are characterized by elevated androgens likely of luteal origin (see references in Fite *et al.* [30]). The placenta is a steroid-rich tissue, and androgens and their metabolic precursors can be produced from this source [14]. Testicular production of androgens by male fetuses can cross the fetal–placental barrier and enter general circulation in pregnant females, although this does not inevitably occur in all species (literature reviewed in French *et al.* [31]). Finally, androgens of adrenal origin, although typically secreted at substantially lower levels than those of gonadal origin, can nonetheless contribute to overall circulating androgen levels [32].

Biological substrates. Traditionally, behavioural endocrinologists have relied on measurements of circulating androgens in plasma or serum, as circulating hormones are those that have the opportunity to interact with target tissues, such as steroid-sensitive neuronal circuits in the brain. Non-invasive sampling regimens have been developed in both laboratory and especially field contexts, and steroids are now measured in a variety of biological matrices, such as urine, faeces, hair, saliva and milk. This creates two issues for the interpretation of concentrations. First, clearance rates for steroids differ depending upon the biological sample, in the order of minutes in saliva, minutes-to-hours in urine and hours-to-days in faeces [33]. Quantifying the clearance rates of labelled steroids can clarify these temporal issues. Second, significant metabolism of steroids occurs from secretion to excretion, particularly in the liver [34], thus the hormone measured in excreta can differ from the hormone acting on target tissues. Again, administration of labelled hormone and characterization of the chemical structure of the labelled excreted hormone can identify important metabolic transformation of the hormone of interest.

Cross-reactivity of antibody. As with any immune-based quantitative assay, immunoassays for androgenic steroids use antibodies of varying specificity for steroids. Antibodies with high specificity (low cross-reactivity) for a specific steroid provide the confidence that the resulting quantitative measures are selective for that steroid, but antibodies with higher cross-reactivity with chemically similar steroids (and hence less selectivity) provide quantitative data that represent the sum of multiple steroid concentrations, adjusted by the degree of cross-reactivity for steroids other than the target hormone against which the antibody has purportedly been generated. For investigators interested in the potential actions of specific steroids on behavioural outputs, antibodies with high specificity or sample purification with chromatographic separation are essential. On the other hand, antibodies with high cross-reactivity can provide information not only on the hormone of interest, but also on important levels of precursors and biologically active downstream metabolites of the hormone of interest. For example, Dloniak *et al.* [16] used an antibody directed against testosterone to measure androgens in spotted hyaena faecal samples. Chromatographic separation of androgens by polarity and subsequent immunoassay with the T antibody revealed high cross-reactivity with both androstenedione (an important prohormone for T) and dihydrotestosterone (a biologically active metabolite of T; [16]). Thus, the lack of high specificity of the T antibody in fact provides a more integrative measure of potential and available steroid levels. Clarity and caution regarding the resulting measures in these cases is required: the concentrations are accurately expressed as 'faecal androgens' as opposed to 'faecal T'.

Carrier proteins. Steroids often circulate in the blood while bound to glycoproteins known as sex hormone-binding globulin (SHBG). In the event that steroids are complexed with SHBG, they are incapable of crossing cell membranes, do not interact with nuclear steroid hormone receptors, and hence do not trigger transcriptional changes in the nucleus, which is the primary mechanism of steroid hormone action [35]. Quantification of the per cent of circulating androgens that are bound to SHBG or are circulating in a free form is crucial in interpreting the potential pool of bioactive hormones that can interact with and alter brain function.

behavioural masculinization of female fetuses (e.g. high levels in the third trimester of pregnancy, regardless of litter sex ratio [49]), specific organizational effects of androgens on behavioural profiles have yet to be demonstrated.

Given the strong and pervasive link of aggression and dominance in male mammalian species and increased androgen production [12,51], it seems likely that female lemurs with increased T levels might engage more frequently in dominant and aggressive behaviours. While a wealth of data are available for evaluating the organizational and activational hormonal contexts of female dominance found among spotted hyaenas (*Crocuta*, discussed in this article), only a few studies have examined the relationship between female dominance, aggression and androgen production in female lemurs [52,53]. Both aggression and circulating T in females peak during the lemur breeding season [45,54] and, consequently, it is easy to speculate that circulating T (and other androgens) may be facilitating the breeding season peak in female aggressive behaviour. Von Engelhard *et al.* [53] found that while both faecal T and female aggression in lemurs increased twofold during the breeding season, individual female aggression rates and individual indices of female dominance were not correlated with androgen concentrations. Additionally, female lemurs did not have higher levels of androgens relative to males, despite being the dominant sex and showing higher rates of aggression. Drea [52] documented similar seasonal

patterns of androgen production in lemurs, with peak levels of androgens also occurring during the breeding season. In this study, both T and A_4 concentrations in female lemurs were lower than that of males (larger sex differences were apparent in T than in A_4). There were also major sex differences in the ratios of these two steroid hormones. T levels in males were twofold higher than A_4 but in females A_4 levels were approximately tenfold higher than T.

In lemurs, then, there is clear evidence of a temporal correspondence between elevated androgens during the breeding season and increased aggressive behaviour in females. In addition, A_4/T ratios are proportionately higher in female lemurs than in males, suggesting that, like hyaenas, A_4 may serve as an important prehormone for androgenic effects on female aggression and dominance. However, the absence of a strong correlation between variation in levels of T and individual differences in female aggression and dominance is not consistent with the strong support for an exclusively activational role for T in lemurs.

4. Callitrichine primates (marmosets and tamarins)

Marmosets and tamarins offer a useful model for evaluating organizational and activational effects of steroid hormones on female aggressive behaviour. Callitrichine primates show a socially monogamous structure, including close heterosexual bonds [55]. Callitrichine social systems are also characterized by low rates of intragroup aggression, yet adult females are typically dominant over adult males using standard food competition paradigms [56]. Encounters between different groups, both in captivity and in the wild, are often characterized by aggressive territorial behaviour, especially between same-sex conspecifics [57], in which adult females play an active role. Behavioural responses of resident females to other female intruders can include physical attacks, but non-contact aggressive and threat displays by females are also common across species of this group ([57], see review in [58]). Thus, marmosets conform to our criteria for this paper: high levels of normative female aggression (in this case, in intergroup encounters) and dominance of females over males in adults.

(a) Prenatal androgen exposure

As marmosets and tamarins most often give birth to fraternal twins, initial speculation was that females would be less sensitive to androgens during gestation as they would run the risk of being overly masculinized by androgens secreted by their male womb-mates in the 50% of twin litters that were mixed sex. The impact of having a male versus female co-twin on female behavioural development has not been systematically examined to date. However, it is clear that marmoset fetuses are exposed to high and varying concentrations of maternally derived androgens *in utero* [31], with significant effects on later development. We demonstrated that differences in gestational exposure of fetuses to androgens influenced both morphological and behavioural development. First, offspring born to mothers with high gestational androgen levels in the first trimester were smaller at birth and had accelerated postnatal, especially juvenile, growth patterns [59]. In contrast to the results from the spotted hyaena that higher cub aggression and play-mounting was associated with high gestational androgen exposure [17], marmosets offspring born to mothers with higher levels of maternal urinary androgens during late gestation displayed *decreased* rough-and-tumble play as juveniles [60]. While the effect was greater in males, females also showed reduced play behaviour as a consequence of high gestational exposure to androgens. These findings suggest that prenatal exposure to androgens affects at least two components of phenotypic characteristics in female marmosets, including rough-and-tumble play. Further, it is clear that sensitive periods for androgen-mediated changes in offspring morphological and behavioural phenotypes occur at different stages of gestation (growth: early gestation; behaviour: late gestation).

(b) Postnatal androgens

A single study has addressed the impact of immediate postnatal androgen exposure on later adult behavioural phenotypes in marmosets. Female common marmosets treated with T for the first 50 days of postnatal life showed higher rates of rough-and-tumble play during the juvenile period if they had a female co-twin (but not a male co-twin), suggesting that neonatally administered androgens may masculinize juvenile female behaviour. However, differences in the effects of androgen treatment as a function of the sex of the potential play partner indicate that there are social influences that modulate the behavioural consequences of elevated postnatal androgens in females [61,62]. These neonatal androgen manipulations also impacted sex-typical sexual behaviour. When neonatally T-treated females were tested as adults, they showed higher rates of male-typical courtship and copulatory behaviour with a receptive female marmoset than untreated females. Postnatal steroid environments also impact aggressive behaviour in tamarins. Female tamarins ovarectomized (OVX) prior to puberty exhibited fewer threat displays in an intruder paradigm test than intact females [63]. However, OVX and intact females showed no differences in physically injurious agonistic behaviour during intruder trials with unfamiliar females.

(c) Adult hormones and female aggressive behaviour

To our knowledge, only one study has addressed activational effects of androgens on aggression in adult female callitrichine primates. In Wied's black tufted-ear marmosets, baseline levels of androgens in adult females did not predict levels of aggression during an intruder trial with an unfamiliar female [64]. However, post-aggression levels of androgen measured at 6–24 h post-encounter were altered by the quality of the aggressive interaction by resident females. Those resident females that displayed high levels of aggression toward female intruders showed significant elevations in post-encounter urinary T, whereas females that showed low or no aggression toward the intruder had unaltered T levels. This aggression-dependent elevation in T is similar to the response of resident male marmosets to intruders in the post-encounter period [65] and suggests a facilitative or 'challenge-like' response [66] of androgen to high levels of aggression in both adult male and female marmosets.

5. Rodents

(a) Naked mole-rat (*Heterocephalus glaber*)

The mammalian species with perhaps the most dramatic system of female dominance and aggression is the naked

mole-rat. This subterranean eusocial mammal lives in colonies of up to 300 individuals, and breeding is typically monopolized by a single female (queen) and from one to three males [67]. Aggression and dominance is expressed primarily by intraburrow 'shoving', head-to-head contests of strength that results in the subordinate being displaced backward in the colony tunnels. Queens perform the vast majority of shoving (and never receive shoving from any colony member), breeding males show substantially lower rates and non-breeders of both sexes rarely engage in shoving. The queen's death or removal is associated with increased shoving and the rapid onset of ovarian function by the successor queen [68]. Consistent with an activational hypothesis, during queen transition periods, urinary T levels are threefold higher in aggressive females than in non-aggressive females [69]. Observations from a captive colony in which several non-breeding females engaged in shoving further supports the activational hypothesis, because urinary T levels among these females were positively correlated with aggressive shoving [69]. Areas of the brain that are typically sexually dimorphic in mammals (ventromedial nucleus of the hypothalamus, paraventricular nucleus and medial amygdala) are similar in size in breeding male and female naked mole-rats, and are larger in breeders of both sexes than in non-breeders [70]. Unfortunately, no data are available on pre- and postnatal hormone concentrations in this species, but the lability of ovarian function and subsequent steroid hormone *milieu* as a consequence of social status [68] suggests significant postpubertal plasticity in these otherwise sexually dimorphic brain regions.

(b) Cricetidae

Females of several seasonally breeding rodent species of the family Cricetidae display conspecific social aggression (in addition to maternal aggression; see Bosch [71]) as the result of organizational programming of androgens. Typically, female rodents exhibit significantly lower levels of aggression and androgens than males do. In typically male-dominant rodent species (e.g. brown rat), there are numerous pharmacological studies to suggest that gonadal steroid exposure plays a role in female aggressiveness [72,73]. Treatment with T and E_2 together contributes to the greatest increases in aggressiveness [74]. However, this treatment regimen is not sufficient to increase aggressiveness in typically non-aggressive female rats [75]. This suggests that a highly aggressive phenotype is influenced by gonadal steroids in male-dominant rodents species, but that females with phenotypically low aggression do not appear to be as strongly influenced by gonadal steroid metabolism. In rodent species where females naturally exhibit aggressive behaviour (i.e. Syrian hamsters and California mice), the relative influence of gonadal steroids on aggressive behaviour is dissimilar to gonadal steroid–aggression relationships in females of male-dominant rodent species.

(c) Syrian hamster (*Mesocricetus auratus*)

Female Syrian hamsters not only display significantly more aggressive behaviour than males [76], but also tend to dominate males in heterosexual encounters [77]. This sex difference may be attributable to relative differences in steroid metabolism between the sexes [76]. Neonatal androgen and oestrogen exposure, as well as variation in steroid and neuropeptide levels in adulthood, have been implicated in the expression of female intrasexual social aggression. Gonadal steroid exposure during development can have a modulatory effect on behavioural phenotypes in females later in life. Female Syrian hamsters treated with T on the second day postbirth showed increased aggressiveness relative to control females [78]. This suggests that T exposure for females during development leads to a higher expression of aggressive behaviour later in life, thus providing support for an organizational hypothesis.

Additionally, changes in oestrous state have pronounced effects on the expression of aggressive behaviour in Syrian hamsters, suggesting that ovarian hormones can alter female aggressiveness. Non-oestrous females display high levels of conspecific aggression, while aggressive behaviour is essentially absent in oestrous females [79]. These fluctuations in aggressive behaviour across the oestrus cycle are attributable to changes in oestrogen and progesterone (P_4). In females treated with both E_2 and P_4, aggression is completely suppressed [79,80]. However, this reduction in aggressive behaviour is only seen when E_2 and P_4 levels are increased in concert, and is not seen when these hormones are replaced individually [79,80]. The relative contributions of gonadal steroids in these atypical rodent species appear to influence female aggression differently than in more typical male-dominant rodents. However, aggressive encounters between unisexual pairs of female Syrian hamsters were unaffected by gonadectomy, and treatment with T [81] and oestrogen [82]. Thus, despite the evidence suggesting that female aggression is influenced by hormonal state, the aggression-inducing effect of gonadal steroids on females does not appear to apply across all contexts.

(d) California mouse (*Peromyscus californicus*)

Female California mice show a similar aggression phenotype as males, with increased aggression during winter-like short days [83]. There are important consequences of androgen exposure on the development of neural mechanisms for aggression [84]. Female mice that were exposed to T neonatally, and then treated with T in adulthood displayed increased levels of aggression, relative to females that did not receive neonatal treatment [84]. Furthermore, neonatal treatment with oestrogen also greatly increased the expression of female aggression in adulthood, mirroring the effect seen from neonatal T treatment [85]. Neonatal females treated with T during development appear to have their neural system primed for aggression, by increasing androgen receptor sensitivity to T in adulthood [84]. This increased sensitivity to T leads to higher levels of female aggression displayed during adulthood.

There is also substantial evidence that indicates androgens have an activational effect on the expression of aggression in female rodents. In female California mice, T facilitates baseline aggression, as expected, but E_2 inhibits aggressive behaviour, as oestradiol treatment given to ovariectomized female mice did not induce aggressive behaviour [86]. Thus, it may be that the E_2/T ratio is more important than either hormone alone as an endocrine modulating factor for baseline aggression in this species [87]. Furthermore, female California mice displayed a decrease in P_4 levels, and the P_4/T ratio, following an intruder encounter, suggesting that P_4 inhibits female intrasexual aggression. It was also suggested that a post-encounter decrease in the P_4/T ratio may serve to modify aggression in future encounters [87].

Recent work has found that the underlying neuroendocrine mechanism responsible for increased aggression

owing to T treatment is the downregulation of neurosteroid biosynthesis of allopregnanolone (AP). Predictably, female Swiss Webster mice that were administered T displayed an increase in the expression of aggression. However, this long-term treatment with T also led to a downregulation of brain AP levels [88]. Interestingly, this T-induced aggression can be reversed by selectively increasing AP content [88] suggesting that endogenous pregnane steroids play an important modulatory role in attenuation of female aggression. Attention to androgen modulation of neurosteroid synthesis and function might provide additional insights into mechanisms of agonistic behaviour in mammals with sex-reversed patterns of aggression.

6. Rock hyrax (*Procavia capensis*)

Rock hyrax social structure is characterized by multi-male–multi-female groups, with a small number of females 'monopolized' for reproduction by a territorial male. Females do engage in cooperative rearing of offspring, although relatedness among cooperating females is not known. Some aggression within hyrax groups occurs in intrasexual contexts, but the majority of agonistic interactions, including threat displays, chases and avoidance, occurs between males (both territorial and non-group bachelor males) and females. In spite of the reproductive 'monopolization' by territorial males, adult female rock hyrax in most wild groups under observation are behaviourally dominant to males [89].

Across populations of rock hyrax, levels of T in females (measured in hair samples) are similar to or higher than levels in males [89,90] suggesting that T may serve an activational role in female aggression. However, while T was a strong predictor of within-group differences in male dominance status (and body size), individual differences in female aggression rates and dominance were independent of T levels. This finding is not consistent with the potential activational link between androgens and female aggression. Accounting for high T concentrations in female rock hyrax poses a dilemma, because there appear to be significant costs for females without apparent benefits. Females with elevated androgens have a larger proportion of skin absent of hair, a proxy measure for ectoparasite load and/or susceptibility to infection, yet other than female dominance over males, individual differences in T are not correlated with enhanced rates of aggression or reproductive success in females, as measured by litter size [90].

7. Summary, conclusion and future directions

This review has highlighted the role of androgens in shaping mammalian female aggressive patterns and female dominance in a cluster of species that depart from typical male bias in both these measures. An overall summary of the empirical data presented in this paper is highlighted in table 1. It is clear from this review that androgenic steroids play an important, but not exclusive, role in shaping these phenotypes. It is equally clear that our understanding of the role of these steroid hormones in both organizing and activating female aggression is far from complete. None of the species listed in this review have the breadth and depth of research on these issues that characterizes more traditional mammals, especially those that are studied extensively in the laboratory or in captive situations (e.g. rodents: [93]; macaque monkeys: [94]).

Table 1 Summary of female aggression, dominance, and organizational and activational effects of androgen hormones in 'atypical' mammals.

species	aggression	dominance	organizational effect	activational effects	facilitative effects	T	A_4	comments	references
hyena	F > M	F > M	+	n.a.	n.a.	M > F	F > M		[17,18,21,24]
lemur	F > M	F > M	+	–	n.a.	M > F	M ≧ F	mixed results for activation of aggression and dominance	[49,50,52,53]
marmoset	M ≧ F	F > M	+	–	+	M > F	n.a.		[60–64]
naked mole-rat	F > M	F > M	n.a.	+	n.a.	M > F	n.a.	dominant female appears to suppress subordinate reproductive hormones	[68,69]
Syrian hamster	F > M	F > M	+	–	n.a.	M > F	n.a.	mixed results for gonadal steroid exposure effect on aggression. Photoperiod important as well	[78,81,82]
California mouse	M ≧ F	n.a.	n.a.	+	+	M > F	n.a.	E_2/T ratio more important than either hormone alone as contributors influencing baseline aggression	[87,91,92]
rock hyrax	F > M	F > M	n.a.	–	n.a.	F > M	n.a.		[89,90]

The potential for significant peripubertal organizational effects of androgens on female aggression, as highlighted in the work of Sisk and co-workers [95], in more conventional mammalian species has yet to be explored. Further, the potential for androgens to play a facilitative role in future aggression in females as a consequence of aggression-induced rises in these hormones [66] has only been addressed in a limited number of these species. Additional species that depart from mammalian norms regarding sexual dimorphism in aggression, although not to the extent to which our focal species do, are also worthy of further study. For instance, unlike male-dominant and -aggressive chimpanzees (*Pan troglodytes*), male and female roles in bonobos (*Pan paniscus*) are relatively egalitarian, and females tend to be dominant over males, although without the overt aggression associated with species like spotted hyaenas [96]. Comparative analyses of androgen levels in adults show that male : female ratios of urinary androgens are highly male-biased in chimpanzees, but more similar between the sexes in bonobos [97]. However, these differences appear to be attributable primarily to elevated T in male chimpanzees, rather than elevated T in female bonobos. A recent analysis of digit ratios (2D : 4D; a proxy for *in utero* exposure to androgens) in chimpanzees and bonobos failed to find evidence for elevated prenatal exposure to androgens in female bonobos [98], shedding doubt on an organizational effect on the propensity toward female dominance in this species. However, further work is required to clarify both activational and organizational effects of androgens in bonobos and other species with less dramatic but still female-biased dominance and aggression.

Finally, more sophisticated analyses of endocrine effects on brain structure underlying agonistic behaviour [26,70], increased knowledge of the enzymatic substrates that mediate important transitions from less active to more active steroid moieties (and vice versa; [15,28]) and an appreciation that variation in carrier peptides is critical for estimating the actual exposure of steroid-sensitive brain mechanisms to hormones [36] will enrich our understanding of gonadal influences on aggressive behaviour. What is perfectly clear, however, is that the kind of variation in mechanism–behaviour relationships revealed in this review, which has its roots in the operation of natural selection on these systems, can provide us with knowledge as rich and informative as data gleaned from induced genetic variation from steroid receptor knock-out models and other laboratory constructs [99,100].

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