AN-08 Hormones and behaviour in animals

Introduction: A Tripartite Framework of Animal and Human Behaviour

The study of neuroendocrinology and its influence on behaviour requires navigating a complex matrix of biological determinism, environmental plasticity, and cognitive override. As a foundation for understanding how men and women can best live and work together in couples, families, and organizations, it is imperative to systematically dissect how hormones influence behaviour in non-human animals, and critically evaluate the extent to which these findings transfer to humans.

To achieve this rigorous synthesis, this report adopts a tripartite analytical framework consisting of three distinct but mutually interacting layers:

1. **Physiology (The Measurable Layer):** This encompasses the absolute, quantifiable neurobiological and endocrine metrics. It includes circulating hormone titers, neural receptor densities, enzymatic conversions (such as aromatase converting androgens to estrogens in specific brain nuclei), and genetic mechanisms. This layer is deterministic in its mechanics but highly dynamic in its responsiveness to environmental cues.
2. **Behaviour (The Statistical Layer):** Behavioural outputs driven by hormones are rarely binary (i.e., male versus female). Rather, they exist as overlapping statistical distributions between sexes and among individuals. Hormones do not strictly dictate a specific action; they alter the probability, threshold, and intensity of a behavioural response to a specific environmental stimulus.
3. **Culture and Cognition (The Variable Layer):** In higher primates and particularly in humans, the vast expansion of the neocortex allows for the continuous cognitive evaluation of social contexts, future planning, and the establishment of cultural norms. This layer exerts top-down control, possessing the unique capacity to suppress, amplify, or completely redirect hormonally primed behavioural impulses.

By examining foundational experimental literature—ranging from fetal androgen manipulation and castration paradigms to oxytocin receptor knockouts and sex-typed toy preferences—this report synthesizes the strongest available evidence. It distinguishes established, replicated findings from contested hypotheses, separates population averages from individual variances, and ultimately evaluates the transferability of animal models to human social organization.

1. Fetal Formatting: The Organizational-Activational Hypothesis

The foundational paradigm of modern behavioural neuroendocrinology was established in a landmark study published in *Endocrinology* in 1959 by Charles H. Phoenix, Robert W. Goy, Arnold A. Gerall, and William C. Young[1]. Prior to this publication, the prevailing scientific consensus posited that mammalian brains were inherently bisexual or blank slates, with male or

female mating behaviours driven almost entirely by the secretions of adult gonads[4].
Phoenix and colleagues fundamentally overturned this view by administering varying doses of testosterone propionate to pregnant female guinea pigs and systematically observing the adult mating behaviour of their female offspring[4]. The researchers utilized two distinct prenatal dosing strategies: a high dose that fully masculinized the genitalia of female fetuses in utero, rendering them externally indistinguishable from male littermates, and a lower dose that altered the brain without noticeably impacting the external genitalia[4]. The experimental protocol in adulthood involved castrating the offspring to remove endogenous hormones, followed by the administration of targeted hormone therapies to test behavioural capacities.
The researchers demonstrated two distinct, permanent neurobehavioural phenomena in these prenatally androgenized genetic females:
First, they observed profound **defeminization**. When ovariectomized in adulthood and primed with exogenous estradiol benzoate and progesterone, the prenatally treated females showed a significantly reduced capacity to display the female-typical lordosis reflex (a concave arching of the back that facilitates intromission) compared to control females[2]. Crucially, the lordosis responses were triggered by manual flank stimulation from a human hand, ensuring that the behavioural deficit was not due to altered pheromonal communication with stimulus males, but rather a permanent change in how the subjects' hypothalamic neural circuits responded to ovarian steroids and somatosensory inputs[3].
Second, they documented significant **masculinization**. When given exogenous testosterone in adulthood, these same genetic females displayed male-typical mounting behaviour at frequencies equivalent to those of genetic males, and significantly higher than untreated control females[1].
From these massive effect sizes, the authors proposed the "Organizational-Activational Hypothesis." They posited that exposure to steroidal hormones during a critical fetal or neonatal window permanently *organizes* the neural architecture of the developing brain (the measurable physiological layer). Later in life, adult hormones act upon this sexually differentiated neural substrate to *activate* specific behaviours (the statistical behavioural layer)[1].

Extension to Non-Human Primates

To determine whether these phenomena were merely rodent artifacts or highly conserved mammalian mechanisms, the organizational-activational hypothesis was subsequently tested in non-human primates. In a series of longitudinal studies spanning from the 1960s to the 2000s, Goy, Wallen, Thornton, and colleagues administered prenatal androgens to pregnant rhesus macaques (*Macaca mulatta*)[7].
The resulting data demonstrated that prenatal neuroendocrine environments exert a profound, permanent influence on primate behavioural predispositions well outside of strict copulatory reflexes. The genetic female offspring exhibited highly masculinized juvenile behaviours, including increased frequencies of rough-and-tumble play and peer mounting[7]. This body of research confirms that the organizational effects of prenatal androgens format the neural circuits governing complex social interactions.

Primate Cohort (Rhesus Macaques)	First Report / Primary Authors	Hormone Manipulation	Treatment Period	Behavioural Outcome
Proof of Principle	Young et al. (1964)	Testosterone Propionate (TP)	Gestational Days 40–90 ($n = 2$)	Permanent masculinization of juvenile social behaviour.
Cohort 1	Phoenix et al. (1968), Goy (1968)	Testosterone Propionate (TP)	Gestational Days 40–111 (Total $n = 8$)	Increased male-typical mounting and delayed lordosis.
Cohort 2	Goy (1981)	TP and Dihydrotestosterone (DHTP)	Gestational Days 40–120 (Total $n = 33$)	DHTP failed to fully replicate TP effects, implicating neural aromatization of TP to estradiol.
Cohort 4	Herman et al. (2000)	Testosterone Enanthate, Flutamide	Gestational Days 35–145 (Total $n = 38$)	Flutamide (androgen blocker) demasculinized male juvenile play.

Table 1: Summary of key longitudinal cohorts demonstrating the organizational effects of prenatal androgens in rhesus macaques, adapted from Thornton (2011)[7].

2. Baseline Hormones and Individual Sensitivity: The Castration Paradigm

A persistent misconception in both popular culture and rudimentary sociological models is that

higher levels of circulating adult testosterone directly equate to higher levels of male-typical behaviour, such as sexual motivation or physical aggression. The rigorous experimental literature fundamentally rejects this linear dose-response fallacy, demonstrating a crucial distinction between group averages (where males generally exhibit higher testosterone and different behavioural profiles than females) and individual variance.

In a classic study published in the *Journal of Comparative and Physiological Psychology*, Grunt and Young (1953) meticulously observed the baseline sexual behaviour of individual male guinea pigs, categorizing them statistically into high, medium, and low copulators based on their spontaneous behavioural frequencies[1]. Following surgical castration, all males exhibited a precipitous and uniform decline in mating behaviour, their activity dropping to near zero. This confirmed Aristotle's ancient observation and established the baseline scientific fact that gonadal hormones are strictly necessary for the activation of these specific behaviours[11].

The critical insight, however, emerged during the hormone replacement phase of the experiment. The researchers administered the exact same, standardized replacement dose of testosterone propionate to all castrated males. If behaviour were a simple linear function of circulating hormone levels, all males receiving the identical dose should have converged on a uniform, intermediate level of sexual behaviour. Instead, the males rapidly segregated back into their exact pre-castration categories of high, medium, and low copulators[1].

This foundational finding was heavily reinforced decades later by Damassa, Smith, Tennent, and Davidson (1977) in a highly replicated study published in *Endocrinology*. Utilizing advanced silastic capsule implants to precisely control circulating hormone levels, they demonstrated that in castrated male rats, administering merely one-tenth (10%) of the normal physiological concentration of circulating testosterone was entirely sufficient to fully restore and maintain pre-castration levels of copulatory behaviour[11].

Mechanisms of Individual Variance

1. **The Threshold Effect:** The experimental data conclusively demonstrate that testosterone operates on a physiological threshold basis rather than a linear dose-response curve. Once a minimal circulating threshold is met, the behavioural circuitry is fully activated. Adding additional exogenous hormone above this maintenance concentration fails to increase the frequency or intensity of the behaviour[11].
2. **Receptor Density Over Fluid Concentration:** Individual differences in behaviour are not driven by the absolute amount of fluid hormone in systemic circulation (the physiological layer). Instead, behavioural variance is dictated by the brain's inherent sensitivity to that hormone[11]. This sensitivity is determined by the density, distribution, and transcription rate of androgen and estrogen receptors localized within the medial preoptic area (MPOA) and related hypothalamic structures, which were permanently organized during fetal development[11].

Therefore, distinguishing between population averages and individuals is vital. While males, as a statistical population, have higher testosterone and display different behavioural profiles than females, one cannot measure the circulating baseline testosterone of two healthy adult males and accurately predict which will be more sexually motivated or aggressive.

3. Seasonal Testosterone, Aggression, and the Challenge Hypothesis

The relationship between testosterone and aggression further illustrates the intricate interaction between the physiological, behavioural, and environmental layers. To contextualize the evolutionary role of testosterone in aggression, Wingfield, Hegner, Dufty, and Ball (1990) published the "Challenge Hypothesis" in *The American Naturalist*, based primarily on extensive field observations of seasonally breeding birds[16].

The original Challenge Hypothesis proposed an elegant three-level model of testosterone secretion:

- **Level A (Baseline):** A very low level of circulating testosterone maintained during the non-breeding season.
- **Level B (Breeding Baseline):** A moderate physiological increase at the onset of the breeding season to support basic reproductive functions, such as spermatogenesis and the development of secondary sexual characteristics.
- **Level C (Physiological Maximum):** A rapid, transient spike in testosterone occurring strictly in response to intense social challenges, particularly male-male territorial intrusions and mate-guarding conflicts[16].

The hypothesis successfully explained the evolutionary trade-off between the undeniable benefits of aggression (securing mates and territory) and its severe physiological costs (increased metabolic rate, reduced immune function, heightened predation risk, and the suppression of essential paternal caregiving behaviours)[16]. According to this model, organisms keep testosterone relatively low to permit paternal care and preserve energy, spiking it to physiological maximums only when fitness is acutely threatened by a rival.

The DHEA Anomaly in Non-Breeding Aggression

However, glaring anomalies in the animal kingdom forced a re-evaluation of this systemic model. In species like the North American song sparrow (*Melospiza melodia*), males remain highly territorial and aggressively defend their territories during the autumn non-breeding season. This behaviour occurs despite their gonads being completely regressed and systemic circulating testosterone being nearly undetectable, even following intense simulated territorial intrusions[19].

Research by Soma and colleagues (2000), published in the *Proceedings of the Royal Society B*, resolved this neuroendocrine paradox. They discovered that during the non-breeding season, the brain utilizes dehydroepiandrosterone (DHEA), an otherwise inert prohormone secreted by the adrenal glands[20]. Using silastic implants over a 14-day period, they demonstrated that exogenous DHEA significantly heightened aggression and decreased the latency to attack in non-breeding male song sparrows[20].

The mechanism relies on neurosteroidogenesis. The brain locally metabolizes this circulating DHEA directly into active testosterone and estradiol via the enzyme aromatase, specifically within targeted neural circuits governing aggression (such as the preoptic area)[20]. Thus, the animal maintains highly aggressive behaviour to defend resources while successfully sparing

the peripheral body the systemic, immunosuppressive costs of high circulating gonadal testosterone[21].

Challenge Hypothesis 2.0

In 2019, Goymann, Moore, and Oliveira published the "Challenge Hypothesis 2.0" in *BioScience*, arguing that the original 1990 model fundamentally overemphasized male-male competition[23]. A systematic meta-analytic review across diverse vertebrate taxa demonstrated that male-female interactions—such as courtship, mate acquisition, and mate guarding—are frequently more potent stimulators of transient testosterone pulses than male-male aggression[23]. This revision clarifies that testosterone is fundamentally a hormone of *reproduction and status acquisition*, rather than merely a hormone of violence.

Challenge Hypothesis Iteration	Primary Focus	Trigger for Testosterone Spike	Physiological Cost Mitigated	Key Theorists
Original (1990)	Mating effort vs. Paternal care	Male-Male territorial intrusion	Reduced paternal care, immunosuppression	Wingfield et al.[16]
Brain Aromatization (2000)	Non-breeding territoriality	Adrenal DHEA converted locally in brain	Systemic toxicity of circulating androgens	Soma et al.[20]
Version 2.0 (2019)	Reproductive opportunity	Male-Female interaction & Courtship	Energetic trade-offs in mate acquisition	Goymann et al.[23]

Table 2: Evolution of the Challenge Hypothesis and the neuroendocrine regulation of aggression and testosterone pulses.

4. The Neuroendocrinology of Care: Maternal Hormones and Sensitization

If testosterone represents the neuroendocrinology of competition, the biological mechanisms underpinning caregiving and social bonding represent the neuroendocrinology of affiliation. Research in this domain reveals a remarkable degree of neural plasticity, demonstrating how environmental stimuli can completely override baseline physiological states.

Maternal Sensitization: Environmental Override

In standard laboratory rodent models, virgin (nulliparous) females and adult males typically ignore neonates, or actively avoid them due to neophobia. However, Jay S. Rosenblatt (1967), in a seminal and highly replicated paper in *Science*, demonstrated the phenomenon of "maternal sensitization"[27].

Rosenblatt placed 5- to 10-day-old foster pups in cages with non-pregnant adult rats. By continuously exposing these naive adults to the pups over a period of 5 to 7 days, Rosenblatt successfully induced full maternal behaviours (retrieving, crouching over the pups, nest-building) in virgin females and, remarkably, in adult males[27]. The sample sizes were robust for the era, with 33 of 67 virgin females showing spontaneous retrieval, and the remaining subjects reliably exhibiting sensitization[27].

This sensitization occurred without any internal hormonal intervention, proving definitively that the basic neural circuitry required for intensive caregiving exists in all members of the species, regardless of sex or reproductive status. The biological hardware for parenting is universally distributed; it can be activated purely by persistent environmental stimuli and social proximity (pup cues)[28].

Endocrine Priming: The Fast Track to Care

While environmental sensitization takes nearly a week, the physiological events of pregnancy prime the brain for immediate, short-latency caregiving at the moment of parturition. Robert S. Bridges (1984), publishing in *Endocrinology*, established a precise quantitative endocrine model demonstrating how the sequence and duration of pregnancy hormones reorganize the brain's motivational state[31].

Using ovariectomized rats and precise silastic implants to mirror the exact hormonal titers of pregnancy, Bridges demonstrated that maternal behaviour is triggered by a highly specific cascade: a sustained, multi-day elevation of progesterone (simulating gestation), followed by a precipitous decline in progesterone and a sharp rise in estradiol (simulating impending birth), capped by an infusion of pituitary prolactin[31].

When virgin females were subjected to this precise hormonal regimen, their latency to display full maternal care dropped from the 5-7 days required for sensitization down to mere hours[31].

Lesion studies conducted by Numan (1974), published in the *Journal of Comparative and Physiological Psychology*, successfully identified the Medial Preoptic Area (MPOA) of the hypothalamus as the critical neural node that integrates these hormonal fluctuations with incoming sensory information to trigger caregiving[34].

Furthermore, research by Kendrick et al. (1997) utilizing ewes demonstrated that vaginocervical stimulation during the birthing process triggers a massive central release of oxytocin, which acts directly on the brain to facilitate immediate maternal bonding and selective recognition of the neonate[36].

5. The Oxytocin and Vasopressin Knock-Out Controversy

The nonapeptides oxytocin (OXT) and arginine vasopressin (AVP) are globally recognized in both scientific literature and popular media as the primary neurochemicals modulating

maternal bonding, social recognition, and monogamous pair-bonding.

The Foundation of the Oxytocin Dogma

The central role of oxytocin in social cognition was heavily solidified by Ferguson, Young, and Insel (2000) in a highly influential study published in *Nature Genetics*[39]. The researchers utilized male mice engineered to lack the oxytocin gene (Oxt -/-). In a standardized social recognition paradigm—measuring the time spent investigating an ovariectomized female over four successive 1-minute trials—wild-type (Oxt +/+) mice showed an approximately 70% reduction in investigation time by the second trial, indicating they remembered the female[39].

In contrast, the Oxt -/- knockout mice exhibited "social amnesia," continuing to investigate the female as if she were a total stranger on every trial[39]. Crucially, the researchers proved this deficit was not a generalized cognitive or sensory failure; the knockout mice exhibited perfectly intact spatial memory (in Morris water mazes) and intact non-social olfactory habituation[39]. The social amnesia was entirely rescued by injecting oxytocin directly into the medial amygdala prior to the encounter, cementing oxytocin's indispensable role in processing social memory[40].

For decades subsequently, the prairie vole (*Microtus ochrogaster*) has served as the premier animal model for social monogamy. A vast, replicated literature base has suggested that oxytocin receptor (OXTR) signalling in the nucleus accumbens, alongside vasopressin signalling in the ventral pallidum, is strictly and uniquely required for the formation of enduring pair bonds[41].

The CRISPR Disruption

In one of the most significant and fiercely debated disruptions in modern behavioural neuroendocrinology, Berendzen, Manoli, and colleagues (2023) published a study in *Neuron* utilizing advanced CRISPR/Cas9 technology to generate OXTR-null mutant prairie voles (with highly robust sample sizes of $n = 15$ to 16 pairs per behavioural cohort)[41].

In stark contradiction to 30 years of pharmacological literature, these genetically modified OXTR-knockout voles *still formed enduring pair bonds*, displayed robust partner preference in three-chamber assays, and exhibited normal parental care[43]. The only significant deficit observed was physiological: knockout mothers showed severe deficits in milk ejection, resulting in roughly 50% lower pup survival rates compared to wild-type mothers[44].

The Controversy and Critics: This finding has fractured the field. Leading researchers who established the original oxytocin models (including Larry Young and colleagues) argue that genetic knockouts present from the moment of conception allow the highly plastic developing mammalian brain to deploy massive compensatory physiological mechanisms[43]. Therefore, the absence of a behavioural deficit in an adult knockout does not prove oxytocin is uninvolved in wild-type animals; it merely proves the brain can reroute around the deficit.

Furthermore, follow-up analyses by Berendzen's own group (2025) indicate a more nuanced reality: while long-term bonding occurs, OXTR-knockouts fail to show partner preference on acute, short-term timescales (e.g., they fail to bond after only 6 hours of cohabitation, whereas wild-types bond rapidly)[41].

The strongest, most robust insight to emerge from this controversy is the principle of *biological*

redundancy. Behaviours that are absolutely critical to the evolutionary survival of a species—such as social attachment, mating, and infant care—are almost never dependent on a single, non-redundant molecular pathway. The physiology is highly robust, ensuring that the behaviour persists even in the face of severe genetic or environmental disruption.

6. Sex-Typed Juvenile Behaviours: Play and Toy Preferences

One of the most heavily debated areas of behavioural transferability between non-human animals and humans involves sex differences in juvenile play styles and object preferences.

Rough-and-Tumble Play

Across diverse mammalian taxa, ranging from rodents and carnivores to non-human primates and humans, juvenile males engage in rough-and-tumble play at significantly higher frequencies and intensities than juvenile females[47]. Meaney and Stewart (1981), publishing in *Animal Behaviour*, meticulously documented this in rats, demonstrating that rough-and-tumble play—characterized by wrestling and pinning—peaks predictably between 30 and 40 days of age[48].

Crucially, the sex difference in rough-and-tumble play is strictly dependent on neonatal androgen exposure, representing a classic organizational effect. Female rats injected with testosterone neonatally exhibit male-typical play frequencies, while castration of males neonatally permanently suppresses it[47]. This is a highly replicated, exceptionally robust finding indicating that the statistical distribution of aggressive play styles is biologically formatted prior to any possible social interaction.

Object and Toy Preferences in Non-Human Primates

In humans, boys show massive statistical preferences for mechanical and wheeled toys, while girls show preferences for plush toys or dolls. A systematic review and meta-analysis of 75 studies encompassing 113 effect sizes found these sex differences to be highly significant, with boys preferring boy-related toys yielding an effect size of $d \geq 1.60$, a magnitude rarely seen in psychological research[52]. Social constructivist critics frequently argue that these preferences are entirely the result of cultural conditioning, patriarchal socialization, and gendered marketing[53].

To rigorously test this hypothesis, researchers introduced human toys to non-human primates entirely lacking human cultural socialization. Alexander and Hines (2002), publishing in *Evolution and Human Behavior*, studied a captive colony of vervet monkeys ($N = 88$; 44 males, 44 females)[55]. They found that males spent significantly more time contacting typically masculine toys (a car and a ball), while females preferred typically feminine toys (a doll and a cooking pot)[55].

Hassett, Siebert, and Wallen (2008) replicated and refined this methodology in a troop of 135 rhesus macaques, publishing in *Hormones and Behavior* (analyzing 11 males and 23 females that engaged with the objects)[53]. When presented simultaneously with wheeled toys (wagons, dump trucks) and plush toys (Winnie-the-Pooh, armadillo), male macaques showed a strong,

statistically significant preference for wheeled toys in both frequency and mean duration of interaction[54]. Female macaques exhibited greater variability, playing with both toy types, exactly mirroring the broader flexibility in toy preference seen in human girls[54].

The Controversy and Critics: Constructivist critics point out that these primate studies suffer from limitations: relatively small sample sizes for the interacting subjects (e.g., 11 males in Hassett et al.), implementation in single captive colonies which may lack broad ecological validity, and the inherent anthropomorphism in classifying a monkey touching a truck as a "masculine" preference[57].

However, the animal data align tightly with human clinical data. Sheri Berenbaum and Melissa Hines (1992) studied young girls with Congenital Adrenal Hyperplasia (CAH), a genetic condition resulting in abnormally high prenatal exposure to adrenal androgens[60]. Despite being explicitly socialized as girls by their parents, girls with CAH exhibit significantly masculinized toy preferences, shifting their behavioural distribution closer to that of boys[62].

The strongest current scientific synthesis avoids the naturalistic fallacy by positing that prenatal androgens do not encode a genetic preference for a "truck" per se. Rather, androgens organize the visual and motor cortices to prefer objects offering mechanical motion, predictable trajectories, and high-contrast spatial dynamics[54]. Human culture subsequently channels these broad biological biases into heavily gendered commercial toy markets.

7. Cortical Control and Transferability to Humans

When translating experimental neuroendocrinology from animal models to humans, it is critical to recognize the evolutionary shift in the locus of behavioural control. Kim Wallen (1999), in *Hormones and Behavior*, articulated the vital concept of "emancipation"[63].

In lower mammals, gonadal hormones strictly and deterministically dictate the *ability* to mate (e.g., a female rodent is only physically receptive during the hormonal window of estrus; without estrogen and progesterone, mating is physically impossible)[63]. In higher primates and humans, the physical ability to engage in socio-sexual behaviour has been completely emancipated from strict hormonal control. Humans can physically engage in sexual or social behaviour at any point in the endocrine cycle, independent of current hormone titers[63].

Because the physiological layer in humans dictates *motivation* rather than compulsory motor reflexes, the cultural and cognitive layers possess immense veto power[63]. Hormones in humans no longer act as binary "on/off" switches; they act as volume knobs for desire and motivation.

The Meta-Analytic Reality of Human Testosterone

The attenuation of hormonal determinism in humans is most evident in the aggression literature. While animal models demonstrate robust causal links between testosterone manipulation and violence, human data show immense variability.

John Archer's extensive meta-analyses (1988, 2006) and a recent, highly comprehensive meta-analysis by Geniole et al. (2020) analyzing 45 independent studies involving $N = 9760$ participants reveal startlingly weak effects[65].

Testosterone	Correlation with	95% Confidence	Significance /

Variable in Humans	Aggression (r)	Interval	Notes
Baseline Testosterone (Overall)	0.054	[0.028, 0.080]	Very weak, but statistically significant
Baseline Testosterone (Men only)	0.071	[0.041, 0.101]	Weak positive association
Dynamic Change in T (Overall)	0.108	[0.041, 0.174]	Stronger than baseline, highly context-dependent
Manipulated (Exogenous) T	0.046	[-0.015, 0.108]	Not statistically significant

Table 3: Meta-analytic summary of the relationship between testosterone and human aggression (data from Geniole et al., 2020)[65].

Experimental administrations of exogenous testosterone in humans do not reliably induce aggression. Rather, they alter the salience of status-relevant cues, occasionally resulting in *prosocial* behaviours if cooperation or generosity is the most effective means to enhance social status in a given cultural context[67]. A human male may experience a transient testosterone spike in response to a status challenge (physiology), feel a surge of competitive motivation (behavioural impulse), but cognitively evaluate the social consequences and choose to negotiate calmly or walk away (cultural override)[64].

Therefore, while animal models accurately map the biological hardware and the origins of our motivational drives, human behaviour is ultimately a negotiated outcome between subcortical hormonal priming and neocortical executive function.

8. Summary of Robust Findings

1. Fetal exposure to androgens permanently organizes neural architecture, heavily influencing the future statistical distribution of sex-typical behaviours across mammalian species.
2. Circulating adult hormones activate these pre-organized neural circuits, but do so on a threshold basis; individual behavioural differences are driven by neural receptor sensitivity, not by the absolute volume of circulating hormones.
3. Testosterone does not obligatorily cause physical violence; it modulates the salience of status and competitive motivation in response to environmental challenges.

4. The neural circuitry required for intensive caregiving exists in both sexes and can be activated non-hormonally through persistent environmental exposure and proximity (sensitization).
5. Pregnancy hormones (estradiol, progesterone, prolactin) act synergistically to rapidly prime the brain for maternal care, dropping the behavioural latency from days to mere hours.
6. Rough-and-tumble play is a biologically organized, sexually dimorphic behaviour heavily dependent on prenatal/neonatal androgens.
7. Sex-typed object preferences have measurable biological roots linked to prenatal hormone exposure organizing visual and motor cortices, which then interact with social conditioning.
8. Vital socio-reproductive behaviours, such as pair-bonding, rely on redundant physiological pathways, demonstrating immense resilience even when primary genetic targets (like oxytocin receptors) are deleted.
9. Evolutionary advancement in primates shifts hormonal function from dictating physical behavioural *ability* (compulsory reflexes) to modulating behavioural *desire* (motivation).
10. In humans, the vastly expanded neocortex allows for continuous cognitive evaluation, ensuring that culture and context can override, suppress, or redirect hormonally primed impulses.

9. Contested or Open Questions

- **The Necessity of Oxytocin in Bonding:** Does the ability of CRISPR OXTR-knockout voles to pair-bond reflect an inherent irrelevance of oxytocin for attachment, or does it merely demonstrate massive developmental compensation by redundant neural networks during gestation?
- **The Challenge Hypothesis 2.0:** To what extent are transient testosterone spikes driven by aggressive male-male territoriality versus the energetic and motivational demands of male-female courtship and mate guarding?
- **Translation of Primate Toy Preferences:** Are the sex differences observed in vervet and rhesus monkey object manipulation truly homologous to human toy preferences, or are they artifacts of captivity, small sample sizes, and anthropomorphic human interpretation?
- **Non-Breeding Aggression:** What are the exact neurochemical pathways that allow adrenal DHEA to be converted locally into estrogens in the brain to fuel aggression, and does a homologous mechanism regulate human aggression?
- **Testosterone and Human Prosociality:** Under what specific social conditions does testosterone promote prosocial, cooperative behaviour in humans as a mechanism for status enhancement, rather than antisocial competition?

10. Important Sources to Read in Full

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11. Implications for Living Together

The integration of rigorous neuroendocrinology into our understanding of human social structures—whether optimizing harmony in couples, structuring families, or managing dynamics within small groups and organizations—requires careful philosophical navigation to avoid the naturalistic fallacy. The empirical observation that a behaviour is biologically "natural" or hormonally primed does not mean it is morally "good," socially desirable, or inevitable. Biology provides the baseline probability, not the boundary condition.

For couples and families, the extensive literature on maternal sensitization is profoundly optimistic. The physiological capability for deep nurturing and caregiving is not locked behind a biological barrier of gestation or strictly limited to females; it is latent in all individuals. It is activated through proximity, routine, and behavioural investment. Fathers, adoptive parents, and non-biological caregivers undergo measurable neuroendocrine shifts—including alterations in oxytocin and prolactin networks—simply by engaging in the active behaviour of care. In the realm of family dynamics, behaviour shapes biology just as robustly as biology shapes behaviour.

Regarding conflict and ambition within families or organizations, understanding testosterone as a modulator of status-seeking rather than a deterministic engine of physical violence fundamentally reframes how we manage competition. If biological drives are fundamentally geared toward securing status, resources, and reproductive opportunity, organizations and societies possess the agency to consciously construct environments where cooperation, mentorship, and prosocial achievements are the primary metrics of high status. Because humans are evolutionarily emancipated from strict hormonal control, we have the cognitive architecture to design cultures that channel biological motivations into constructive, rather than destructive, societal outcomes.

Biology formats our starting distributions, but culture, sustained by human cognition and voluntary action, determines our destinations.

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