The Effect of Testosterone on the Sense of Agency

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Abstract

Sensorimotor agency, or simply, the sense of agency (SoA), can be defined as the subjective experience that one is initiating, executing and controlling one’s own volitional actions in the environment. Such experiences have been found to play a major role in motivated learning and instrumental voluntary behaviours. Recent research suggests that higher-order psychological experiences characterised by the feeling of having control over social events and which are associated with trait dominance, may be rooted in basic brain mechanisms associated with sensorimotor agency. The hormone, testosterone, has been consistently implicated in a variety of social behaviours linked to the acquisition of social dominance, positioning it ideally as a potential modulator of SoA. This study therefore examined the influence of testosterone on the SoA by measuring its effects on intentional binding in a repeated measures, placebo-controlled experiment (n=26). Intentional binding is an implicit measure of agency whereby one experiences a subjective compression in time between voluntary actions and the sensorimotor consequences of these actions. More specifically, intentional binding estimates were derived from calculated perceptual shifts in relation to both the action event (action-binding) and the tone consequence (tone-binding). Findings indicate a significant effect of testosterone on the SoA. Specifically, this effect appears to occur as a result of the facilitation of the intention to act more so than via processes linked to retrospective reappraisal, as demonstrated in its significant enhancement of action-binding. Moreover, these results further imply that through an embodied SoA, testosterone can ultimately modulate higher-order experiences of social power and goal-directed behaviour.
The Effects of Testosterone on the Sense of Agency

The human mind is an intrinsically complex system, being both structurally intricate and functionally diverse. Our understanding of psychological experience is burdened by this sheer complexity, compounded by the inherent subjectivity of the phenomenon under study. A prevalent theoretical approach termed, “embodied cognition”, has been used to identify the basic mechanisms upon which complex, higher-order aspects of consciousness are founded (Borghi & Cimatti, 2010; Witt, 2011). Such theories claim that sensory-motor processes, engendered by the physical body interacting with the environment, are intrinsically involved in the emergence of higher-order mental functions. This reflects a deconstructivist approach to the organisation of the nervous system, in which more complex, abstract aspects of consciousness are built upon simpler, concrete functions (Gallese, 2000).

Recently, it has been suggested that the psychological experience of agency may be grounded in lower-level sensorimotor agency (Barlas & Obhi, 2013). That is, the feeling that one is an autonomous agent in the environment, capable of exerting control through performing behaviours that produce desired consequences, may emerge from basic brain mechanisms related to sensory-motor processing (Obhi, Swiderski, & Brubacher, 2012). Sensorimotor agency, or simply, the sense of agency (SoA), can be defined as the subjective experience that one is initiating, executing and controlling one’s own volitional actions in the environment (Gentsch, Weiss, Spengler, Synofzik, & Schutz-Bosbach, 2015). The SoA is described as having a “thin” phenomenology, due to the implicit and pre-reflective nature of awareness (Tsakiris, Schutz-Bosbach, & Gallagher, 2007).

However, the minimal nature of the SoA should not be taken to mean that the experience itself derives from a simple brain mechanism. Recent literature demonstrates that the SoA encompasses several lower-level sensorimotor and emotional processes, which together create a highly meaningful mode of consciousness that appears to be fundamental to instrumental learning and distinguishing between self-generated and other-generated behavioural consequences (Gentsch, Kathmann, & Schutz-Bosbach, 2012; Stanton & Schultheiss, 2009). “Initiated” or “volitional” action implies that the behaviour under question is goal-directed, meaning that the SoA, as per its definition, is driven by intentions and thus motivated. Motivation invokes the notion of subjective emotions since emotional
states imbue the environment with an immediate sense of value, which act to promote specific responses (Panksepp & Biven, 2012). In this way, stimuli or events in the environment can be understood as lying on a continuum from pleasurable to unpleasurable, which directs approach or avoidant behaviours respectively. The definition of the SoA further emphasises the experience of “control”. This suggests that attributing the cause of an event to the self and perceiving oneself as an influential entity is fundamental in the emergence of SoA (Moore & Obhi, 2012).

To this effect, studies on the neural mechanisms underlying the SoA have demonstrated that the SoA involves both retrospective and prospective processes (Haggard & Clark, 2003; Moore & Haggard, 2008). The retrospective inferential model suggests that the SoA emerges out of the post-hoc matching of actions and the action consequences experienced (Haggard & Clark, 2003). Specifically, the SoA is thought to develop subsequent to processing of the actual consequences of an action, which is consciously viewed in light of the actual action taken. Moreover, if actual consequences are retrospectively perceived to be in line with actions taken, the SoA emerges. This process strongly relies on information received from the sensory modalities (Moore & Haggard, 2008). Further, insula activation has consistently been shown to occur in association with the experience of SoA, and may specifically contribute to retrospective mechanisms (Farrer et al., 2003; Mutschler et al., 2009; Tsakiris, Longo, & Haggard, 2010). For example, Farrer and colleagues (2003) demonstrated that insula activation strongly correlates with the degree of match between an action and the sensory feedback. In contrast to the retrospective account, the prospective account refers to the intention-action link, suggesting that a SoA is generated prior to the actual consequence of the action and thus precedes conscious processing of the consequence (Moore & Obhi, 2012). This predictive mechanism has been associated with neural activity in the pre-supplementary motor cortex (Moore, Ruge, Wenke, Rothwell, & Haggard, 2010b). For example, research conducted by Fried and colleagues (1991) demonstrated that electrical stimulation of this region in a human sample generated an internal “urge to act”. Moreover, Lau, Rogers, Haggard and Passingham (2004) demonstrate that activation of this area can be brought about through merely concentrating on the intention to act.

Now a standard methodology in the field, Haggard, Clark and Kalogerias (2002) developed an implicit measure of agency known as “intentional binding” to
assess pre-reflective, implicit attributions of actions and action consequences (Barlas & Obhi, 2013; Obhi et al., 2012). Intentional binding refers to the subjective compression of time between a voluntary action and the sensory consequences (Moore & Obhi, 2012). Importantly, this compression of time only occurs when one attributes the cause of the event to oneself.

However, the kinds of attributions made are highly dependent on the context and are suggested to be dependant on the affective nature of the event. Specifically, self-attributions are particularly prevalent when the events are perceived as positive or beneficial, and less prevalent when the events are perceived as negative (Penton, Thierry, & Davis, 2014; Taylor & Brown, 1994). This pattern of attributions is referred to as a self-serving bias, which reflects a tendency for healthy individuals to attribute positive events to internal, personal factors and negative events to external factors (Krusemark, Keith Campbell, & Clementz, 2008; Obhi et al., 2012). Further, this pattern of attribution is considered to be evolutionarily significant, acting to protect pre-existing self-models to facilitate approach-related behaviour (Taylor & Brown, 1994). In turn, a link can be made between self-serving biases and the SoA, given their shared connections with intentional binding (Penton et al., 2014). The role of self-serving biases in SoA highlights the contribution of prospective accounts of SoA, specifically that the emergence of the SoA is vitally contingent upon top-down, intentional mechanisms.

Further support for the prospective, intentional model comes from findings demonstrating that by increasing the availability of choice and invoking a deliberate intention, the SoA increases (Leotti, Iyengar & Ochsner, 2010). This suggests that the mere potential for action, accompanied by an intention to act, generates implicit feelings of control. Furthermore, potential actions appear to be selected based on their predicted outcomes. A priming study conducted by Chambon, Sidarus and Haggard (2014) provides evidence that subliminal primes drive the selection of compatible actions, generating a fluency in action selection. It is as a result of this selection fluency that a SoA emerges.

Despite the recent growth of research in this area, few studies have paid due attention to the emotional component of the SoA. This is surprising, given that the SoA is motivational as conceptualised in its very definition and modulated by intentions or desires. The role of motivational processes in SoA contrasts earlier
understandings of SoA as an objective attribution based on weighting various sensorimotor cues according to their reliability, as suggested by early models of Bayesian optimal cue integration theory (Knill & Pouget, 2004). Rather, motivational factors can be seen to play a central role in modulating SoA. Neuro-imaging findings demonstrate that the prefrontal cortex and striatum, brain regions rich in dopaminergic receptors associated with motivation, are involved in the SoA, and other findings suggest that the administration of dopamine enhances intentional binding (Moore et al., 2010a). Similarly, schizophrenic patients, whose psychopathology is thought to relate to an excess in dopamine, exhibit increased levels of agency compared to control groups (Abi-Dargham et al., 1998; Haggard, Martin, Taylor-Clarke, Jeannerod, & Franck, 2003). In contrast, depressed patients, who possess characteristically reduced levels of dopaminergic activity, exhibit pathologically low experiences of agency (Gentsch et al., 2015). Patients with damage to medial forebrain bundle, a key region of the mesolimbic dopamine pathway, commonly present with adynamia, a syndrome characterised by an almost complete lack of expression of intention (Zillmer, Spiers, & Culbertson, 2008). These findings underscore the motivational component to the SoA, in contrast to its earlier conceptualisations as an objective attribution.

In a recent study, Gentsch and Synofzik (2014) found that this affective dimension relates to motivations to assume or discard responsibility in a given context, such as motives to enhance or protect one’s self-esteem by modulating the weighting of different signals that inform SoA. This finding highlights the contribution of affect and motivation in brain mechanisms that generate the SoA and specifically, that these motives may override the desire to ascertain an unbiased representation of one’s abilities (Gentsch & Synofzik, 2014). This motivational-based distortion in attribution reflects healthy individuals’ tendency towards adopting a self-serving bias (Taylor & Brown, 1994).

Following an embodied account of the mind, lower-level feelings of agency may be important in more complex psychological experiences in the social world, particularly those related to higher-order feelings of self-efficacy and social control (Barlas & Obhi, 2013). Such higher-order feelings are considered adaptive as they encourage active and sustained engagement with the social environment (Galinsky, Gruenfeld, & Magee, 2003). Pfister, Obhi, Rieger and Wenke (2014) showed that the
principles underlying the SoA for actions and effects also apply to social actions, that is, the execution of an intention by another social agent. They suggest that tendencies to exhibit social approach behaviours are facilitated by accumulated experiences of the SoA, which are fundamentally involved in instrumental learning in both social and material environments. The SoA acts to reinforce instrumental behaviours, such that expectations developed over time about behaviour-outcome contingencies may drive experiences of SoA regardless of the outcome (Stanton & Schultheiss, 2009). Over time, repeated experiences of the SoA may then act to facilitate the development of an internal working model (Bowlby, 1969). Thus, given the prominent role of emotion in the SoA, we might reason that the neuro-chemistries that promote motivation in social contexts contribute prominently to the basic sensorimotor processes that engender SoA when actions are initiated, and specifically, that this lower-level SoA provides the platform upon which a disposition towards feeling socially powerful is founded. This is in line with a hierarchical model of the mind, where complex functions are built upon basic processes (Gallese, 2000).

Importantly, the acquisition of social power has been linked to the hormone testosterone, suggesting that fluctuations in this hormone may contribute to the SoA in interpersonal interactions. Eisenegger, Haushofer and Fehr (2011) highlight several approach-related, socio-emotional states and behaviours that are highly associated with testosterone and that may aid in the promotion of social dominance behaviour, which refers to the incentive for acquiring or maintaining a high level of social status or influence relative to others (Eisenegger et al., 2011). These include: increased self-esteem, competitiveness, egocentrism, decreased ability to empathise, fearlessness, threat vigilance and instrumental learning after victory in social challenges (Cashdan, 1995; Eisenegger et al., 2011; Hermans, Putman, & van Honk, 2006; Stanton & Schultheiss, 2009; Wright et al., 2012). Given these findings, dominance is considered to be a socially adaptive mechanism, as it is through status enhancement that individuals benefit from greater access to resources and opportunities (Galinsky et al., 2003).

Several bodies of data provide support for the idea that testosterone may modulate the SoA, particularly in social interactions. Firstly, both testosterone and the SoA are linked to the experience of power. Though sensorimotor agency is generally thought of as merely an implicit, low-level form of agency, recent evidence has
demonstrated that it is in fact closely related to higher-order feelings of power. Obhi and colleagues (2012) have shown that inducing participants to feel powerful, through power priming, enhances the SoA. Secondly, with regard to the neurochemistry involved in the SoA, the rewarding value of an increase in testosterone levels has been linked to the mesolimbic dopamine system of reward and motivation (Johnson & Wood, 2001). Specifically, testosterone increases the expression of dopamine along this brain system but its secretion is highly context specific. Only when individuals are placed in a competitive environment where there is opportunity for social reward, will testosterone levels rise in anticipation, which is thought to assist the victor in learning and to reinforce behaviour instrumental in achieving dominance over others (Mezulis, Abramson, Hyde, & Hankin, 2004). Finally, both the SoA and testosterone are intimately linked to adaptive functioning and high self-esteem, which suggests that the mechanism via which testosterone exerts its influence in social environments may be via its effect on this basic sensorimotor level (Cashdan, 1995; Leotti et al., 2010).

In sum, literature suggests that a relationship may exist between testosterone and the SoA, through shared involvement in psychological and physiological states of power that facilitate social approach behaviours by modulating systems related to motivation, cognition and learning. Despite preliminary evidence of this relationship, no studies to date have directly explored the extent and nature of this relationship.

Research Aim and Questions

The current study aimed to contribute to the embodiment literature by examining the potential link between testosterone and the implicit SoA. We intended to determine whether testosterone has a modulatory effect on intentional binding and whether this effect is related to self-serving biases.

We hypothesised that:

1. The administration of testosterone will significantly enhance intentional binding, namely action- and tone-binding.
2. SoA, as measured by intentional binding, will positively correlate with the presence of self-serving biases in the placebo condition
3. The administration of testosterone will increase the likelihood of exhibiting state-dependent self-serving biases.

Methods
Design and setting

This study formed part of a larger project, investigating how testosterone modulates the experience of the body in ways which may facilitate social dominance. The current study was experimental in design, constituting a double-blind, placebo-controlled within-group design. The double-blind procedure acted to diminish any potential for experimenter bias or social desirability biases in the results. On separate days, the participants participated in both an experimental and a control condition. Participants were randomly assigned to either condition on a given day.

The site for both administering the testosterone or placebo and subsequent data collection took place in the GCS lab in the Psychology department at the University of Cape Town (UCT). A standardised setting was used to eliminate the influence of potential location-specific variables on results. Potential time-specific confounding variables, such as those relating to circadian hormonal fluctuations or time of day on performance, were controlled for by standardising the administration and testing sessions to 9:00-9:30 and 14:00-15:30, respectively.

Participants

A power calculation was performed through the GPower program using Moore and colleagues’ (2010a) intentional binding estimates, which provided a desired sample size of 4 participants. Specifically, this calculation was based upon a Cohen’s D effect size of 1.12, a desired level of power of 0.95, an alpha of 0.05 and an allocation ratio of 1:1.

Participants for the pilot study were recruited through UCT’s Psychology undergraduate SRPP program, whilst participants making up the principal study were recruited through advertisements (Appendix A) posted on UCT departmental websites and on Gumtree (local online classifieds website).

Inclusion criteria. Females aged between 18-35, representing all racial/cultural groups were invited to take part in the study. In addition, individuals were only able to participate during the first ten days following the end of their last menstruation in order to control for hormonal fluctuations over the month.

Exclusion criteria. Males, pregnant females, females diagnosed with a psychiatric disorder, taking psychiatric medication or hormone medication, such as the pill, injection or an implant.

Measures
**Intentional binding.** Implicit, sensorimotor SoA was assessed through its operationalised measure, intentional binding, defined as the subjective compression in the time perceived between a voluntary action and the consequence or effect that this action causes (Moore & Obhi, 2012). The task required the participant to judge the time interval between a voluntary button press and an ensuing tone. A computer program was developed based on a design originally specified by Moore and Obhi (2012). This design has been extensively utilised in assessing SoA and has been shown to be reliable and considerably valid (Haggard & Clark, 2003; Hughes & Desantis, 2013; Moore & Obhi, 2012).

The task was made up of four different conditions, or “blocks”, each containing 5 practice trials and 30 test trials. The first two blocks constituted baseline blocks, which did not involve time estimates of cause-and-effect relationships. Specifically, in block one and two, participants made simple judgements about when they made a voluntary action (pressing the down-arrow key) or when they heard a tone, in isolation. In Block 1, participants were instructed to allow a random amount of time to pass before pressing the allocated button. Shortly thereafter they were prompted to estimate the position of the clock hand when they initiated this action. In Block 2, they were instructed to simply wait for the tone to occur, and then report the onset time. In contrast, the third and fourth blocks were operant, or agency, blocks, where a tone was emitted 250ms following a voluntary button press and required participants to estimate either the time of an action or tone consequence. Time judgements were made using the Libert clock paradigm (Libert, Gleason, Wright, & Pearl, 1983). This methodology required participants to report the position of a hand on a clock face numbered 5-60 (see Figure 1) - in 5-point intervals - in order to indicate, as specifically as possible, at what point they perceived a voluntary action (pressing the down-arrow key) or an auditory tone. Meanwhile, the computer program recorded the actual time at which the voluntary actions and sensory consequences actually occurred. The order in which each condition appeared was partially counterbalanced, with agency conditions always presented first to affirm the association between the action and the effect, but alternated between tone and action blocks. In this way, only two out of four possible sequence combinations of the four blocks were used.
The differences in time estimation errors between baseline and operant blocks were calculated in order to assess intentional binding. That is, the specific contribution of operant behaviour to time estimation errors was calculated by subtracting out the error from baseline conditions.

**Attribution style.** Participants’ style of attribution was assessed through the use of the Internal, Personal and Situational Attributional Questionnaire (IPSAQ; Appendix B; Kinderman & Bentall, 1996). This questionnaire consists of 32 items, 16 of which describe positive events and 16 of which describe negative events. The participant was instructed to read the scenario, and both write a causal explanation in one’s own words and select a causal explanation from 3 specified options, choosing to attribute responsibility to either oneself, another individual or to situational factors. Kinderman and Bentall (1996) found that IPSAQ demonstrated adequate reliability in normal participants, proving to be better than its predecessor, the Attribution Style Questionnaire (ASQ).

**2D4D ratio.** Participants underwent a hand-scan in order to identify their second-to-fourth digit ratio (2D4D), which serves as a rough indicator of their level of foetal testosterone exposure (Malas, Dogan, Evcil, & Desdicioglu, 2006). It is widely used in hormone research and shown to be a valid measure (Manning, Bundred, & Flanagan, 2002; van Honk et al., 2011). Specifically, levels of prenatal testosterone exposure may influence neurological pathways and may therefore modulate effects of administered testosterone, thus this must be controlled for.

**Procedure**

A pilot study without the use of hormones was conducted using an independent sample (n=40) from that of the principal study in order to validate the intentional binding task, ensure efficiency in the procedure and to identify any previously unidentified confounds in the testing process.
For the main study, potential candidates who demonstrated an interest in the study were provided with an Internet link to a survey on SoGO, where they received details of the study, provided preliminary consent, filled in personal details, and completed several questionnaires relating to the broader study. Candidates were advised on what dates they were eligible to participate, taking into consideration their menstrual cycle, and then they were provided a link to Doodle, an online scheduling service, in order to select their time-slots.

The study consisted of four sessions (two per day) over two separate days, both which needed to be scheduled, within the first 10 days following the cessation of the participants’ menstrual period. Participants were instructed not to eat, drink (anything other than water) or smoke during the hour preceding the study. In addition, participants were instructed not to consume any alcohol less than 12 hours before the session, in order to prevent contamination of the results (Shiels et al., 2009). A maximum of four people were able to sign-up for a day of testing in order to account for venue and administrative capacity. The order of administration (placebo versus testosterone) across days was randomised across participants. Upon arrival at the first session of the first day, participants completed the consent form and several online questionnaires relating to the broader study. Once this was completed, participants had their right hand scanned in order to assess their second-to-fourth digit ratio, after which they were required to provide a saliva sample for future analysis. Participants were then provided with a vial containing 0.5ml of either liquid testosterone or a placebo liquid, which they were instructed to empty under their tongue for 60 seconds before swallowing. Participants were briefed that neither they nor the researcher knew whether the vial contained testosterone or the placebo. Before leaving, participants were asked to please refrain from excessive caffeine intake or from engaging in strenuous exercise, as this may influence testosterone levels and confound results (Beaven et al., 2008; Shiels et al., 2009).

The second session began 4 hours following the first session. This is due to the fact that testosterone generally requires a period of four hours to activate (Boss, Hermans, Montoya, Ramsey, & van Honk, 2010; Tuiten et al., 2000). Participants began by filling out the IPSAQ. Detailed verbal instructions regarding the intentional binding task were then provided. Participants were instructed to keep their gaze fixed upon the clock face in the centre of the computer screen for the entire duration of each
trial until required to report their time estimates in a text-box. Across blocks, a total of 120 test trials were performed, including a total of 20 practice trials. The entire task lasted approximately 40-50 minutes. After completing the task, a second saliva sample was taken, after which the participants were free to go. Participants were reminded to return for the second day of testing. The procedure of each day was identical, however, a second version of the IPSAQ with different but semantically congruent items was administered on the second day. After completion of the second day of testing, participants were reimbursed with R500. Participants were subsequently emailed a debriefing document which detailed the aims and the hypotheses of the study.

Statistical Analysis

Intentional binding. Raw data related to the intentional binding measure was analysed on the SPSS statistical software package using a means analysis. Following Moore and Obhi’s (2012) rationale, participant time-judgement biases are known to vary across blocks. Biases can be controlled for by comparing time judgments for the same event (either action or tone-judgement) against different blocks. The difference between the time estimation errors in operant and baseline blocks was calculated to represent the “perceptual shift” in awareness that occurs during the SoA. In other words, the difference in the perceived estimate and actual time of the voluntary action in the baseline block (action in absence of a consequence) was subtracted from the difference in perceived and actual time of an action in the agency block (when both an action and consequence are present). This procedure was duplicated for the tone-judgement blocks.

This type of analysis is capable of highlighting whether there are trends in the shifting of time estimates for actions and action-effects in the agency blocks relative to baseline blocks. Intentional binding will be illustrated by a positive shift of the time estimate for an action, which reflects a delayed perception/awareness of time relative to the actual time of voluntary actions in agency blocks relative to baseline blocks, and is referred in the literature as “action-binding” (see Figure 2). In addition, intentional binding is also illustrated by a negative shift of the time estimate for a sensory consequence, where perception/awareness of the time of the tone is preemptive of the actual time of the tone emission in operant blocks relative to baseline blocks. This negative shift in temporal perception for action effects is known as “tone-
binding” (see Figure 2). In this way, action-binding shifts the temporal perception of an action towards the ensuing tone, whilst tone-binding shifts the tone towards the prior action. The combined shifts in time estimates of an action and action effect result in a perceived temporal binding of both events, such that there is a subjective compression in the time interval between the action and its action effect. However, action- and tone-binding were assessed independently in order to explore, in detail, testosterone’s influence on the binding process.

![Diagram of intentional binding breakdown](image)

**Figure 2. Intentional binding: Breakdown**

(a) The physical events – the objective time when the voluntary button-press is made and the tone occurs; (b) Action-binding – when making a voluntary-press judgement in the agency block, participants tend to perceive the voluntary-press as occurring later; temporally shifting the perceived action towards the ensuing tone; (c) tone-binding – when making a tone-judgement in the agency block, participants tend to perceive the tone as occurring earlier, shifting the perceived tone towards the voluntary-action.

Whilst a means analysis is capable of investigating significant perceptual drifts within testing conditions (testosterone and placebo), two additional 2-tailed t-tests were run in order to determine whether intentional binding, as measured through computed perceptual shifts, was significant across testosterone and placebo conditions in relation to action-binding and tone-binding. In other words, we wish to determine whether testosterone contributed to significantly greater intentional binding, not already explained by the placebo condition.
Moreover, two repeated-measures within-factors ANOVAs were conducted on time estimations relating to action blocks and tone blocks. This statistical test was utilised in order to determine whether testosterone generates any global effects on perception over and above that of placebo. More specifically, two repeated-measures ANOVAs were run; one aimed at estimating action-binding, and another estimating tone-binding. Given that each shift moves in opposite directions as predicted in previous literature, the extent of each must be analysed separately as not to cancel out the effects of one on the other.

Each ANOVA was set-up using “testing conditions” as one factor, with two levels including testosterone and placebo conditions, and a second factor defined as “testing blocks,” consisting of both operant and baseline blocks. The critical difference between the two ANOVAs was that each incorporated different testing blocks, with one specifying blocks relating to time estimation of the voluntary button-press and another based on tone consequence blocks, but both included the same testing conditions, namely testosterone and placebo. The variance of independent factors was analysed against a dependent variable, namely time estimation errors made (calculated as the discrepancy in the perceived time relative to actual time of an event, which is measured via a proxy of time, namely through the distance in units of error as viewed around the circumference of a clock face). Prior to the running of this analysis, a composite variable representing the average of the 30 trials of time estimations was calculated for each participant for both placebo and testosterone conditions. A comparison of averages is more favourable than a comparison of raw data given that the prior reduces the amount of error variance present in the sample.

Following this, planned comparisons t-tests were conducted to further investigate any potential interaction effects, in order to isolate the cause of significant results within specific levels of independent factors in relation to intentional binding. More specifically, separate calculations were run for action and tone-judgement blocks, mirroring the means analysis, paired-sample t-tests and repeated-measure ANOVA model specifications. Moreover, weighted time estimation errors for each participant were also utilised instead of raw data.

**Self-serving attributions.** Pearson correlations were run between measures of intentional binding and self-serving attribution scores for the placebo condition only. In particular, self-serving scores were calculated by sum-totalling the number of self-
attributions made in statements suggestive of positive event statements (n=16). This procedure was also followed for negative event statements (n=16). Given that separate scores were determined in relation to positive and negative events statements, this meant that separate correlations were determined for negative and positive self-attributions in relation to intentional binding. This makes intuitive sense given our prediction that negative events will be negatively associated with intentional binding, whilst positive event statements should be positively associated with intentional binding, as indicative of self-serving attributional patterns. Moreover, in reference to our third hypothesis where testosterone’s effect on self-serving biases was predicted, a repeated-measures ANCOVA was run in order to determine whether testosterone had an effect on self-serving attributions when this relationship alone was considered, and when using digit ratios as a covariate.

All data was stored on Excel spreadsheets. SPSS was subsequently utilized to clean the data, by removing influential outliers.

**Ethical Considerations**

Before the study commenced, ethical approval was obtained for the larger study on testosterone and social dominance by UCT’s Human Research Ethics Committee of Health Science Faculty, and the South African Government’s Department of Health (Appendix C). Further, ethical approval was granted by the UCT Research Ethics Committee.

All data has been collected in line with the Declaration of Helsinki (World Medical Association, 2013) and UCT’s guidelines for conducting research on human participants.

**Potential risks.** The safety and reliability of the methods and measures employed have been well established in several other studies, suggesting that the present study did not place any risk to the participants’ physical or mental well-being (Boss et al., 2010; Gentsch & Synofzik, 2014; Haggard & Clark, 2003; Hermans et al., 2006; Moore & Obhi 2012; van Honk et al., 2011). Although only a very rare side-effect, a single dose of 0.5ml of testosterone may cause headaches or nausea (Lawley Pharmaceuticals, 2014). In unlikely circumstances, cyclodextrin carriers, which are present in the liquid form of testosterone, may cause diarrhoea. Participants were informed of these potential risks before signing up and were reminded that they may exercise their right to withdraw from the study at any time. In the unlikely event
of a medical problem, a doctor was on stand-by. In line with current ethical standards, candidates were required to provide informed consent in writing before participating in the study (Appendix D).

**Potential benefits.** Participants benefited from participating through compensation of R500, and additionally from understanding their vital contribution to scientific knowledge formation. We found it reasonable to believe that the potential benefits of participation outweighed any potential risks of participation.

**Confidentiality and privacy.** All data collected was kept strictly confidential. Numbers were assigned to each participant upon arrival. These participant numbers were subsequently used to link the different tasks completed, and the saliva samples provided, to each participant over the two days. All data was stored under the participant numbers, which were dissociated from participant names. This allowed the identity of all participants to remain anonymous throughout the research. Once collected, data was stored in an electronic password-protected format, and saliva samples were stored in a locked freezer.

**Debriefing.** After data-collection, participants were sent a debriefing document via email which explained in detail the nature and aims of the study. Participants were encouraged to ask any further questions via email.

**Advertising.** As the current procedure has only been previously validated on females, males were justifiably excluded given the scientific relevance of female samples (Tuiten et al., 2000). Moreover, we proposed that R500 remuneration was considered an ethically appropriate level of compensation for participation, which we presumed would not hamper with the participant’s ability to make an informed decision to participate in the study.

**Significance**

Results obtained from this study may provide more insight into the role of testosterone in emotional functioning, especially with regard to mental illnesses characteristic of an abnormal sense of agency, such as depression and schizophrenia. Additionally, given that testosterone and the SoA are implicated in affective states, findings may enhance understanding of the mechanisms underlying pathological emotional states, such as social anxiety, depression and schizophrenia. Moreover, the use of an embodied cognition framework could offer alternative approaches to treatment.
Results

Demographic Information

Overall, 26 female participants formed the sample for this research with ages ranging between 18-35. 14 participants considered themselves as “Black”, 6 as “White”, 4 as “Coloured” and 2 as “Indian”.

Prior to conducting statistical tests on data obtained from the intentional binding measure, seven influential outliers were removed based on cook’s distance scores, as extreme outliers are presumed to represent lapses in concentration. Given the simplicity of the task in requiring merely time estimations, a fairly concrete construct, it is unlikely that the outliers are reflective of any other meaningful, unaccounted factors other than concentration. Moreover, normality of data was confirmed.

Intentional binding

Means analysis of perceptual shifts within testing conditions. One-tailed t-tests were utilized in order to obtain estimates of intentional binding in terms of the perceptual shifts in operant blocks relative to baseline blocks. More specifically, action-binding, as evidenced by a positive shift in awareness of the action towards the tone, was calculated by comparing the means of time estimation errors in the operant (Block 3) block against the baseline (Block 1) block. Moreover, tone-binding, as shown via a negative shift in awareness of the tone towards the action, was reflected by a negative shift in mean time estimation error made between the agency (Block 4) and baseline block (Block 2).

Results revealed that significant intentional binding, in the form of action- and tone-binding, occurred in both testing conditions (refer to Table 1). More specifically, significant action-binding was demonstrated in both the placebo ($M=430.06$, $SD=2764.39$, $p<.001$) and the testosterone conditions ($M=899.69$, $SD=2876.76$, $p<.001$).

<table>
<thead>
<tr>
<th>Testing Condition</th>
<th>Intentional binding: $M$ (SD)</th>
<th>$p$ (1-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Placebo</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>Action $32.48$ (2530.03)</td>
<td>776</td>
</tr>
<tr>
<td></td>
<td>Tone $208.93$ (2631.17)</td>
<td>777</td>
</tr>
<tr>
<td></td>
<td>Agency Action $462.53$ (2595.75)</td>
<td>776</td>
</tr>
<tr>
<td></td>
<td>Tone $-2090.82$ (3627.79)</td>
<td>777</td>
</tr>
<tr>
<td><strong>Testosterone</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>Action $38.6$ (2308.84)</td>
<td>779</td>
</tr>
<tr>
<td></td>
<td>Tone $-314.8$ (2689.37)</td>
<td>773</td>
</tr>
<tr>
<td></td>
<td>Agency Action $938.29$ (2443.76)</td>
<td>779</td>
</tr>
<tr>
<td></td>
<td>Tone $-2509.35$ (3779.41)</td>
<td>773</td>
</tr>
</tbody>
</table>

Note: * reflects significance based on $\alpha < .05$
Moreover, significant tone-binding was also found within the placebo ($M=-2299.75, SD=4052.91, p<.001$) and testosterone conditions ($M=-2194.56, SD=4379.8, p<.001$). These findings are consistent with previous literature on intentional binding (Hughes & Desantis, 2013; Moore & Obhi, 2012; Moore et al., 2010a).

**Perceptual shifts across testing conditions.** Two-tailed t-tests were conducted in order to compute and compare the differences in perceptual shift values, which are indicative of intentional binding, across testosterone and placebo conditions (see Table 2). More specifically, a perceptual shift variable was created which subtracted baseline time estimations from that of operant blocks, and in turn was utilised to compare testosterone with placebo. Findings demonstrate that tone-binding across testosterone and placebo conditions was non-significant, with $t(769) = .39$ and $p=.39$. This is reasonable to assume, given that the difference in mean values across testosterone ($M=-2194.56$) and placebo conditions ($M=-2299.75$) appears insubstantial, with a difference of 105.2. Conversely, action-binding across testosterone and placebo conditions was significant with $t(774)=3.44$ and $p<.001$, where perceptual shifts of the action taken were significantly greater in testosterone relative to the placebo condition. This can be confirmed through observation of the means, which descriptively displays greater action-binding within the testosterone condition ($M=899.69$) relative to the placebo condition ($M=430.06$), and accounts for a difference of 468.136 between the two conditions.

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Paired-sample t-tests output: Perceptual shifts across testing conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perceptual shift of Action (Testosterone – placebo)</td>
<td>774</td>
</tr>
<tr>
<td>Perceptual shift of Tone (Testosterone – placebo)</td>
<td>769</td>
</tr>
</tbody>
</table>

*Note: * reflects significance based on $a < .05$

Furthermore, in order to investigate whether testosterone had any significant global effects on time perception over and above that of placebo, a repeated-measures within-factors ANOVA was conducted for instances of both action- and tone-binding. In terms of tone-binding (see Table 3), there was a non-significant main effect of testosterone relative to placebo, $F(1, 50)=1.87, p=.18$, as well as no
interaction effect, $F(1, 50)=.33, p=.57$. This suggests that testosterone does not have a significant unique effect on tone-binding that is not already explained by the placebo condition. For action-binding (see Table 4), there was no main effect of testosterone relative to placebo, $F(1, 50)=.03, p=.87$. However, a disordinal interaction effect was obtained, $F(1, 50)=5.49, p=.02$ (see Figure 3), suggesting that a particular level of one factor is interacting significantly with one level of another factor. Thus, it may be that time errors differ drastically within a certain block across testing conditions (testosterone and placebo) or that errors differ across agency and baseline blocks within a certain testing condition (testosterone or placebo). The latter is indicative of intentional binding, which has already been established via means analysis, assuming that relative error shifts move in the anticipated direction (i.e. errors in voluntary press agency block are greater than the baseline block). Planned comparison t-tests were conducted in order to determine wherein the interaction effect lies.

Table 3
*Repeated-measures ANOVA output: Tone-binding*

<table>
<thead>
<tr>
<th>Main/Interaction effects</th>
<th>Df</th>
<th>$F$</th>
<th>$p$ (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main effect (testosterone/placebo)</td>
<td>1</td>
<td>1.87</td>
<td>.18</td>
</tr>
<tr>
<td>Interaction effect</td>
<td>1</td>
<td>.32</td>
<td>.57</td>
</tr>
<tr>
<td>(Tone*testosterone/placebo)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Error</td>
<td>50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Note: * reflects significance based on $\alpha &lt; .05$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 4
*Repeated-measures ANOVA output: Action-binding*

<table>
<thead>
<tr>
<th>Main/Interaction effects</th>
<th>Df</th>
<th>$F$</th>
<th>$p$ (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main effect (testosterone/placebo)</td>
<td>1</td>
<td>.03</td>
<td>.87</td>
</tr>
<tr>
<td>Interaction effect</td>
<td>1</td>
<td>5.49</td>
<td>.02*</td>
</tr>
<tr>
<td>(Action*testosterone/placebo)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Error</td>
<td>50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Note: * reflects significance based on $\alpha &lt; .05$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Planned comparison t-tests (see Table 5 & 6) based on weighted/average time error estimates for each participant, as was utilised in the ANOVA test, revealed what the means analysis and subsequent paired-t-tests had confirmed, namely that testosterone significantly enhanced the relative difference in errors made between the operant and baseline block, $t(25)=3.88$, $p<.001$, but further demonstrated that the operant block was specifically associated with relatively higher time estimation errors made. Moreover, there was a non-significant difference between agency and baseline blocks for the placebo condition, $t(25)=.71$, $p=.49$. Whilst the placebo condition was associated with a significant difference between voluntary press operant and baseline blocks within the means analysis, the results of the planned t-test differ because the test was carried out using a weighted estimate for each participant and not the raw data (which was used in the means analysis). Furthermore, contrasting results between testosterone and placebo conditions imply that testosterone significantly enhances
intentional binding (the relative difference between blocks) to a greater extent than placebo. It is the difference between testing blocks within the testosterone condition wherein the interaction effect lies. To support this finding, additional planned comparison t-tests were conducted to investigate whether errors made in the agency block (or baseline block) significantly differed across testosterone and placebo conditions, which revealed non-significant findings of, \( t(25)=1.12, p=.28 \) and \( t(25)=-1.33, p=.19 \) respectively. These results further demonstrate that the interaction lies specifically between the voluntary button-press operant and baseline block within the testosterone condition relative to placebo. In other words, testosterone significantly enhances action-binding over and above that of the placebo.

Table 5
\textit{Planned comparison T-tests across testing conditions for voluntary press testing blocks: Exploring the interaction effect}

<table>
<thead>
<tr>
<th>Action-binding</th>
<th>Df</th>
<th>( t )</th>
<th>( p ) (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Testosterone: Agency – baseline condition</td>
<td>25</td>
<td>3.88</td>
<td>&lt;.001*</td>
</tr>
<tr>
<td>Placebo: Agency – baseline condition</td>
<td>25</td>
<td>0.71</td>
<td>.49</td>
</tr>
</tbody>
</table>

Note: * reflects significance based on \( \alpha <.05 \)

Table 6
\textit{Planned comparison T-tests across voluntary press testing blocks: Exploring the interaction effect}

<table>
<thead>
<tr>
<th>Time estimation errors</th>
<th>Df</th>
<th>( t )</th>
<th>( p ) (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agency: Testosterone-related errors – placebo-related errors</td>
<td>25</td>
<td>1.12</td>
<td>.28</td>
</tr>
<tr>
<td>Baseline: Testosterone-related errors – placebo-related errors</td>
<td>25</td>
<td>-1.33</td>
<td>.19</td>
</tr>
</tbody>
</table>

Note: * reflects significance based on \( \alpha <.05 \)

\textbf{Testosterone, Intentional binding and self-serving biases}

\textit{Intentional binding and self-serving attribution scores.} Pearson correlations were run between intentional binding scores and attribution scores (see
Table 7). Controlling for baseline time errors and testosterone, a significant, proportional correlation was found between action-binding and positive event attribution scores ($r = .61$, $p < .001$). Moreover, a positive, significant association was also found between action-binding and negative event attribution scores ($r = .45$, $p = .03$). However, time errors made in the tone agency block was non-significantly related to negative event attributions as well as positive event attributions, with $r = .21$, $p = .31$ and $r = .25$, $p = .24$, respectively.

**Testosterone and self-serving attribution scores.** A repeated-measures ANCOVA (see Table 8) was run to determine whether digit ratios mediated the effect of testosterone on self-serving attribution scores for positive event statements and negative event statements. Interaction effects were assessed first, as in accordance with our third hypothesis, we predicted testosterone would affect negative and positive event self-attributions scores in opposing ways. Using digit ratio as a covariate, the interaction effect between testosterone and event-attributions appeared non-significant, $F(1, 50) = .16$, $p = .69$. The interaction between attributions and digit ratios was also non-significant, $F(1, 50) = 1.28$, $p = .26$. Moreover, when the digit ratio was excluded from the analysis, results remained non-significant, $F(1, 50) = .36$, $p = .55$. Furthermore, main effects were additionally investigated given that significant proportional correlations were evident between intentional binding and both negative event and positive event self-attributions (see Table 7), which contradicts the predictions of self-serving biases. However, results suggest a non-significant main effect between testosterone and self-serving biases, both with and without the digit ratio used as a covariate, $F(1, 50) = .35$, $p = .56$ and $F(1, 50) = .36$, $p = .55$, respectively.

**Table 7**

<table>
<thead>
<tr>
<th></th>
<th>Negative event attributions</th>
<th>Positive event attributions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Action-binding</td>
<td>$r$ = .45, $p = .03^*$</td>
<td>$r$ = .61, $p &lt; .001^*$</td>
</tr>
<tr>
<td>Tone-binding</td>
<td>$r$ = .21, $p = .31$</td>
<td>$r$ = .25, $p = .24$</td>
</tr>
</tbody>
</table>

Note: * reflects significance based on $a < .05$
Based on accumulating evidence of the embodied nature of cognition, this study explored the potential neurochemical role of testosterone – a hormone strongly implicated in social dominance related behaviours – in modulating the experience of the SoA, based on their shared relationship with higher-order experiences of power (Eisenegger et al., 2011; Obhi et al., 2012). The main findings reported here support a role of testosterone in the SoA. To the best of our knowledge, this study is the first to investigate the endocrinological basis of intentional binding. Specifically, our findings indicate that the administration of a single 0.5ml dosage of testosterone significantly enhances action-binding compared to placebo in a sample of women. Given that action-binding is considered to be a valid index of the SoA, these findings provide the first evidence for the contribution of testosterone in subjective feelings of initiating and experiencing control over simple voluntary actions (Wolpe, Haggard, Siebner, & Rowe, 2013). However, no effect of testosterone on the manifestation of state-level self-serving biases was observed.

**Testosterone and the SoA**

That testosterone might act as a mechanism to enhance the SoA is consistent with findings linking testosterone to the insular cortex. More specifically, high levels of testosterone positively relate to insula volume and a single dosage of the hormone

---

**Table 8**  
*Repeated-measures ANCOVA output: Investigating the relationship between Testosterone and self-serving biases (with digit ratios as a covariate)*

<table>
<thead>
<tr>
<th>Main effects</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Testosterone/Placebo</td>
<td></td>
<td>.35</td>
<td>.56</td>
</tr>
<tr>
<td>Testosterone/Placebo</td>
<td></td>
<td>.36</td>
<td>.55</td>
</tr>
</tbody>
</table>

**Interaction effect**  
*With covariate*

| Attributions*Digit ratio |  | 1.28 | .26 |
| Attributions*Testosterone/placebo |  | .16 | .69 |

**Without covariate**

| Attributions*Testosterone/placebo |  | .36 | .55 |

Error | 50 |

* “attributions” refer to both negative and positive event attributions

**Discussion**

Based on accumulating evidence of the embodied nature of cognition, this study explored the potential neurochemical role of testosterone – a hormone strongly implicated in social dominance related behaviours – in modulating the experience of the SoA, based on their shared relationship with higher-order experiences of power (Eisenegger et al., 2011; Obhi et al., 2012). The main findings reported here support a role of testosterone in the SoA. To the best of our knowledge, this study is the first to investigate the endocrinological basis of intentional binding. Specifically, our findings indicate that the administration of a single 0.5ml dosage of testosterone significantly enhances action-binding compared to placebo in a sample of women. Given that action-binding is considered to be a valid index of the SoA, these findings provide the first evidence for the contribution of testosterone in subjective feelings of initiating and experiencing control over simple voluntary actions (Wolpe, Haggard, Siebner, & Rowe, 2013). However, no effect of testosterone on the manifestation of state-level self-serving biases was observed.

**Testosterone and the SoA**

That testosterone might act as a mechanism to enhance the SoA is consistent with findings linking testosterone to the insular cortex. More specifically, high levels of testosterone positively relate to insula volume and a single dosage of the hormone
has been shown to activate this brain region (Heany, van Honk, Stein, & Brooks, 2015). Given the wide recognition of insula activity in the experience of SoA, the effect of testosterone on the SoA may occur via its direct influence on the insula (Farrer et al., 2003; Mutschler et al., 2009; Tsakiris et al., 2010). Alternatively, testosterone’s effect on the SoA may be mediated by modulation of dopaminergic brain pathways. Moore and colleagues (2010a) demonstrated that the administration of L-dopa in Parkinson’s patients enhances intentional binding relative to binding in the absence of treatment. Since testosterone has been shown to up-regulate dopaminergic activity, its effect on the SoA may depend on its interaction with the mesolimbic dopamine system (Abi-Dargham et al., 1998; Haggard et al., 2003). Dopamine is known to underpin motivational behaviour in general. Hence, given that a rise in testosterone tends to occur specifically in contexts in which there is an opportunity to enhance one’s social status, and SoA too increases under such conditions of social competition and is shown to facilitate such approach-related behaviour, testosterone and SoA can be seen to be related by their shared association with socially motivated behaviours and cognitions (Cashdan, 1995; Moore et al., 2010a).

Our findings also suggest a mechanism via which power-priming increases the SoA. Obhi and colleagues (2012) recently demonstrated that priming different levels of power in individuals alters the experience of the SoA; whereby priming low-levels of power is shown to significantly reduce the experience of SoA. Given the robust link between baseline testosterone levels and high social rank, power priming may function to transiently increase testosterone levels. Corroborating this idea, Carney, Cuddy, and Yap (2010) have shown that by getting participants to pose for several minutes in “powerful” postures, such as holding a clenched fist or adopting an expansive stable stance, salivary testosterone levels tend to rise. Such research suggests a bi-directional relationship between power, testosterone and the SoA. Testosterone may increase the experience of power via its action on sensorimotor processes, while the feeling of being powerful may in turn promote the SoA.

**Action- versus tone-binding**

Findings indicated that participants tended to perceive the voluntary button-press as occurring later when the action was followed by a tone (agency block) in comparison to when it was not (baseline block), even more so within the testosterone
condition than the placebo condition. However, there appeared to be no additional effect of testosterone on temporal relations (that could not already be explained by the placebo condition) when the consequences of a voluntary action were appraised (tone-binding). A significant shift in temporal awareness exclusively within the action-binding condition suggests that testosterone influences perception differently depending on the judgement required – action versus action-effects. The significant action-binding finding in comparison to non-significant tone-binding suggests that testosterone may act to selectively enhance factors relating to voluntary button-press judgements. In other words, testosterone appears to influence the SoA by specifically modulating mechanisms involved in the monitoring and preparation of actions rather than judgments relating to sensory consequences.

The facilitating effect of testosterone on intentional binding at the level of action-binding only is supported by recent research indicating that tone-binding and action-binding may be informed by distinct mechanisms, both of which inform the SoA (Wolpe et al., 2013). For instance, Moore and colleagues (2010b) demonstrated a dissociation in action- and tone-binding through transcranial magnetic stimulation. In particular, the pre-supplementary motor area, which plays an important role in motor intention and motor preparation, has been implicated specifically in action-binding (Moore et al., 2010b). The parietal lobes have also been implicated as a potential region which may contribute uniquely to action-binding (Moore et al., 2010b). In accordance with this evidence, Waszak, Cardoso-Leite, and Hughes (2012) suggest that the activation of the parietal lobes is involved in predictive processes of action consequences.

Moore and Haggard (2008) suggest that although both retrospective and prospective mechanisms are involved in the experience of SoA, the relative contribution of each is considered to be context specific. For example, when action-effects are unpredictable, there is a greater contribution of retrospective processes, which allow one to infer authorship based on sensorimotor post-hoc analyses. In contrary, prospective mechanisms are recruited to a greater extent when the action-effect link is well established, which allow for relatively reliable predictions. In support of these claims, Wolpe and colleagues (2013) recently demonstrated that action-binding in particular is affected by the perceived reliability of sensory outcomes. More specifically, Wolpe and colleagues (2013) found that when there is
uncertainty as to whether action effects will follow actions, action-binding is
diminished. This implies that predictive mechanisms contribute uniquely to the SoA
in an action-binding paradigm. Conversely, tone-binding can be enhanced under
conditions of uncertainty. Since we observed no significant effect of testosterone on
tone-binding, but a robust influence in the action block, we can surmise that
testosterone may be exerting its effect on SoA by modulating mechanisms related to
degrees of belief in inferential processing (Friston, 2012).

According to contemporary theories of Bayesian Probabilistic Inference
(Friston, 2012), perceptual experiences emerge out of a process of the matching
between incoming sensory evidence and top-down estimates as to the most probable
cause of those events. This essentially enables efficiency in processing. These top-
down estimates or “probabilities” are fundamentally degrees of a belief or expectation
about outcomes of events and may be value-laden, that is, charged with emotion
(Paulus & Yu, 2012).

If an observation is surprising, that is, if it contradicts prior beliefs, the model
of the world, which drives estimations, is considered to be poor at predicting
observations. Thinking about the brain as a Bayesian system implies that its task is to
minimize uncertainty, and not, instead, potential negative outcomes or the subsequent
subjective discomfort such outcomes may cause (Yoshida, Seymour, Koltzenburg, &
Dolan, 2013). This is because the feeling of uncertainty, or “not knowing”, is an
inherently alarming state, probably akin to the feeling of foreboding. In contrast, the
brain does not try to attenuate discomforting subjective experiences resulting from
negative outcomes per se, as they contribute toward negative reinforcement,
promoting future adaptive behaviours (Eisenegger et al., 2011).

Though several lines of research have demonstrated that testosterone tends to
reduce feelings of anxiety, the precise mechanisms via which the hormone might
influence predictive coding remains speculative (van Honk, Peper, & Schutter, 2005).
There is, however, indirect evidence related to testosterone’s role in fostering
assertiveness to suggest that the modulation of emotional information processing
might be at play. Firstly, a large body of research has shown that increases in
testosterone after a competition, especially when the outcome entails a loss, tend to
encourage decisions to compete again for the second time (Leotti et al., 2010; Obhi et
al., 2012; Mezulis et al., 2004; Stanton & Schultheiss, 2009). This kind of optimism
must surely reflect to some extent an underlying prediction of impending victory. Similarly, testosterone’s ability to facilitate approach-oriented action sequences under conditions of uncertainty, such as aggressive behaviour in response to threat or decisions to engage in risky behaviour, may represent in part the same kind of mechanism (Eiseneggar et al., 2011). These kinds of proactive responses are most likely facilitated by an intrinsic sense of confidence in one’s decisions, otherwise, such behaviours would be counter-intuitive. Testosterone may therefore contribute to the neural processes that organize information so as to strengthen predictive models that inform motivated behavioural responses. In other words, testosterone may strengthen intentions.

This idea is supported by the modulatory role of testosterone on the mesolimbic dopaminergic system (Moore et al., 2010a). Activation of this system is strongly related to motivational states. Thus by artificially increasing testosterone levels, behavioural intentions - which are goal-directed and motivational in nature - are likely to be enhanced. This may have important influences on motor priming to prepare and organise for appropriate action (Moore et al., 2010b). Further, testosterone may act to orient the individual to task-related salient factors and situational demands. This is in line with Guinote’s (2007) Situational Focus Theory of Power, which proposes that powerful individuals differ in terms of their basic cognitive processing, which subsequently affects how one responds to situational factors. When power is primed, individuals become more attuned to the situation, thus their actions are based upon the environmental demands, which in the case of the intentional binding paradigm reflects an up-regulation of motor preparatory processes underlying the voluntary button press in order to induce the tone.

Our finding of a significant effect of testosterone on action-binding may therefore represent an increased weighting of cues deriving from the voluntary button-press action. When judgements were centred on the tone, we found no effect of testosterone. Wolpe and colleagues (2013) propose that tone-binding is shown to rely more strongly on the mere activation of predictions about the effects of actions, prior to the actual occurrence of such events. Waszak and colleagues (2012) suggest a pre-activation mechanism to explain tone-binding, whereby pre-activation of the sensory consequence allows the tone to reach the threshold of awareness faster than when such consequences are not predicted. Put more simply, the pre-activation creates a
diminished perceptual latency of the action effect. This accounts for why tone-binding occurs in such a way that time errors are pre-emptive of the time of actual action effects. This is in accordance with research presented by Voss, Ingram, Wolpert and Haggard (2008), which provides evidence that sensory attenuation, a phenomenon closely related to the experience of agency, is based on action preparation, rather than action execution. Our findings support the dissociation of agency mechanisms, suggesting that the anticipation of sensory consequences of voluntary actions and motivational factors that promote confidence in behavioural choices, derive from distinct brain mechanisms.

**Self-serving biases**

An additional hypothesis explored the proposed effect of testosterone on self-serving causal attributions. Specifically, it was predicted that increased levels of testosterone would enhance participants’ tendency to display state-dependent self-serving biases. Given testosterone’s role in promoting approach-related behaviours, it was hypothesised that this effect is underpinned in part by implicit biases in cognition that function to protect the self-concept. However, the analyses here indicates a non-significant effect of testosterone on transient self-serving biases, both when considered alone and when taking into account prenatal testosterone exposure. This may be due to the fact that the IPSAQ, the measure used to assess self-serving biases, did not sufficiently induce affective responses in participants. It may be that a significant role for testosterone on self-serving biases only emerges when there is a motivational component, that is, when event statements are positively (rewarding) or negatively (punishing) valenced in such a way that they induce affective responses.

Alternatively, the lack of an effect of testosterone may relate to other limitations of the IPSAQ. Although the questionnaire was designed to measure self-serving biases operating at the state-level, it can be argued that the items actually test for *trait*-based biases. Successful elicitation of self-serving biases relies on items that are relatively ambiguous and able to subtly elicit biases in attributions. Many of the items of the IPSAQ draw upon stereotypical or archetypal scenarios, such as common practices around birthdays or courtship. As such, it is possible that participants were responding in a way that reflects over-learned associations or culturally sanctioned behaviour. The influence of testosterone on behaviour and emotion is, however, considered to occur on a pre-reflective, implicit level, influencing behaviour and
cognition without conscious awareness (van Honk et al., 2005). Thus, the influence of testosterone on cognition and emotional behaviour may only manifest at this implicit level. Specifically, although we were interested in the transient and implicit influences of testosterone on self-serving biases, the IPSAQ instead appears to tap into explicit cognitive knowledge, that is, internal working models that have become concretised over the course of a lifetime.

Some validity, however, for the IPSAQ can be demonstrated in our finding that it is significantly correlated with action-binding. This is consistent with previous research showing that SoA and self-serving biases are positively related (Penton et al., 2014). This relationship is thought to depend on their shared connection with affect and motivation; specifically, the agency attribution underlying the experience of SoA has been shown to be influenced by motivational factors relating to enhancement of self esteem (Gentsch & Synofzik, 2014). To this end, action effects associated with positive outcomes tend to enhance the SoA whilst negative action consequences diminish the SoA, suggesting that self-serving biases may manifest via the modulation of instrumental learning that the SoA is thought to support.

Interestingly, Fast, Gruenfeld, Sivanathan and Galinsky (2009) found that by inducing the feeling of power, the illusory perception of control, which refers to the unfounded sense of control over events which are not derived from the agent, can be generated. This kind of cognition may provide one with the motivational capacity to reject negative events that are attributed to oneself. Given testosterone’s relationship to power, it was therefore surprising that, while our findings demonstrate a link between SoA and self-serving biases in the placebo condition, testosterone had no effect on the IPSAQ.

However, action-binding was also proportionately associated with negative attributions to oneself, within the placebo condition. This finding lies in contrast to previous literature which instead suggests a disproportional relationship between SoA and negative event attributions (Gentsch et al., 2015; Gentsch & Synofzik, 2014). Such contradictory results may be due to a lack of ecological validity of the measure, or may reflect the involvement of a third-variable process.

One such process may be self-focus. Earlier research has shown that the administration of testosterone promotes egocentric thinking (Wright et al., 2013). Higher scores on the action-binding task in the placebo condition may reflect a greater
preoccupation with the self as seen in narcissistic personality types, a character trait that is associated with a high degree of self-centeredness but which ultimately derives from feelings of insecurity (Hascalovitz & Obhi, 2015). Hence, the link between testosterone and the SoA, and the SoA and both positive and negative attributions, may hinge on this variable. Indeed, testosterone has been linked to higher levels of narcissistic personality traits.

**Limitations**

The current study had several limitations. As discussed above, the use of the IPSAQ may have provided misleading results; suggesting that testosterone does not significantly influence state-based self-serving biases, when the measure itself does not lend itself to an implicit, state-based assessment. Rather, the explicit nature of the measure seems to assess trait-based self-serving tendencies. A more subtle measure could better attend to state-based changes, which perhaps requires judgements on more ambiguous statements which are more sensitive to implicit, state-based biases.

Additionally, several responses on the IPSAQ were misinterpreted across participants, whereby participants tended to perceive scenarios which were intended as positive, as representing something negative. For example, the scenario, “your neighbour invited you in for a drink”, was consistently interpreted in such a way that the neighbour was perceived as threatening or maintaining ulterior motives, rather than friendly or good-hearted. This could reflect South African-based thinking, where in the context of high levels of crime and violence, the population can be seen to be vigilant for potential threats. Such misinterpretations could be indicative of cultural limitations of the IPSAQ in the South African context.

An additional limitation relates to the compensation offered. Initially, we reasoned that R500 was an appropriate amount of compensation to reimburse the participants for their time and the costs of participating, which include both travel costs and to alleviate the general unease many women have regarding ingesting testosterone – even a relatively minute amount as in the current study. However, in hindsight, R500 might have been too appealing as many candidates attempted to sign-up for any available slots, disregarded their eligibility period. Although this did not contaminate results, it became an administrative burden, requiring us to constantly monitor whether people were adhering to the study requirements.

**Future directions**
Future research could potentially investigate into greater detail, the specific mechanisms through which testosterone influences action-binding in operant conditions. One could do this by altering the uncertainty of the sensory consequence, altering the kinds of action effects experienced as well as the sensory modalities in which they are presented. Moreover, one could alter the kinds of actions produced, alternating between self-produced, externally-produced and involuntary actions. Further, as testosterone was not shown to modulate tone-binding, further research could build on the current findings and explore what potential modulatory mechanisms may underlie this phenomenon. Moreover, an alternative measure of self-serving bias, besides the IPSAQ, could be used. More specifically, one which is capable of assessing state-dependent differences. Additionally, future research should assess the cultural-relevance of the ISPAQ in the South African context and consider developing an attribution measure which is more suitable to the South African population. Furthermore, given that sensory attenuation has been used as a measure of the SoA, which was not utilised in this study, it would be beneficial to investigate whether testosterone enhances sensory attenuation in order to further confirm or disconfirm whether testosterone modulates the SoA.

The current research aimed to contribute to the embodiment literature by examining the potential link between testosterone and the implicit SoA. Several lines of research suggest that feelings of agency in complex, social settings may be founded upon more basic sensorimotor processes that reflect the experience of control over the body. This idea falls within an embodied, hierarchical account of the mind, which argues that higher facets of human consciousness emerge via their interaction with lower-level systems in the nervous system. We therefore hypothesised that testosterone, a hormone widely known to facilitate social-approach behaviour, might achieve its effects on emotional functioning via a modulatory effect on the implicit SoA. Using the intentional binding paradigm, results indicate that testosterone positively enhances the experience of SoA through enhancing action-binding. It is proposed that this reflects testosterone’s role in facilitating predictive mechanisms and strengthening motor intentions. These results suggest that natural fluctuations in testosterone may ultimately modulate higher-order experiences of social power and goal-directed behaviour, given that the hormone is known to increase specifically in socially challenging contexts in which the facilitation of sensorimotor mechanisms is
adaptive. Contrary to prediction, however, testosterone was not shown to significantly influence state-based self-serving biases, as measured by the IPSAQ. This has been suggested to reflect the inappropriateness of this measure in the current context. Specifically, the IPSAQ could be better suited to assess trait-based self-serving tendencies and future research should address this question.
References


Appendix A

Recruitment Advertisements

Advertisement for UCT departmental boards

Subject: Research Invitation - Females for Hormones and Cognition Study

Female Students are Invited to Participate in a Study on Hormones and Cognition in Exchange for R200.

Details about the study: Researchers at the Psychology department are running a study on the effects of testosterone on cognition. Due to standardisation procedures, we are only recruiting females who are NOT taking any form of hormonal contraception (pill/patch/injection/Mirena) or chronic medication. Participation will involve coming into the lab at the Psychiatry department twice on one day. At the first session you will come in and receive either a placebo or a 0.5ml dosage of liquid testosterone to be taken orally. All women have naturally circulating testosterone in the body and the dosage you may receive is less than the total amount produced during one day. It will be out of your system within a few hours and you will not experience any harmful side-effects. At session 2 you will return to the lab for 1 and a half hours where you will perform a variety of tasks. At the end you will be reimbursed with R200 cash. This procedure has been approved by the Human Research Ethics Committee of the Health Sciences Faculty, and the South African Government’s Department of Health

How to participate: If you would like to find out more about the study and sign up to participate, please follow the provided link by cutting and pasting it in a new browser and fill in your details. (paste link)

Advertisement for Gumtree

We are currently recruiting participants for a study looking at the effects of testosterone on the brain and behaviour. Participants will be required to ingest 0.5ml of liquid testosterone (or a placebo). Participants will be compensated for their time in the amount of R200.

Criteria to participate: females only (aged between 18-35), not currently on any form of hormonal contraceptive.

If you are interested in participating, reply to this advert and you will be contacted shortly.
Appendix B

Internal, Personal, and Situational Attributions Questionnaire (IPSAQ)

Please read the statements on the following pages. For each statement, please try to vividly imagine that events happened to you. Then try to decide what was the main cause of the event described in each statement. Please write down the cause you have thought of in the space provided. Then circle the appropriate letter (a, b, or c) according to whether the cause is:

a) Something about you
b) Something about another person (or a group of people)
c) Something about the situation (circumstances or chance)

It might be quite difficult to decide which of these options is exactly right. In that case, please pick one option, the option which best represents your opinion. Please pick only one letter in each case.

Thank you for your time and cooperation.

1. A friend gave you a lift home.
   What caused your friend to give you a lift home?  
   (Please write down one major cause)
   Is this:
   a. Something about you?
   b. Something about the other person or other people?
   c. Something about the situation (circumstances or chance)?

2. A friend talked about you behind your back.
   What caused your friend to talk about you behind your back?
   (Please write down one major cause)
   Is this:
   a. Something about you?
   b. Something about the other person or other people?
   c. Something about the situation (circumstances or chance)?

3. A friend said that he (she) has no respect for you.
   What caused your friend to say that he (she) has no respect?
   (Please write down one major cause)
Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?

4. A friend helped you with the gardening.

  What caused your friend to help you with the gardening?
  (Please write down one major cause)

  Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?

5. A friend thinks you are trustworthy.

  What caused your friend to think that you are trustworthy?
  (Please write down one major cause)

  Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?

6. A friend refused to talk to you.

  What caused your friend to refuse to talk to you?
  (Please write down one major cause)

  Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?

7. A friend thinks you are interesting.

  What caused your friend to think you are interesting?
  (Please write down one major cause)

  Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?

8. A friend sent you a postcard.

  What caused your friend to send you a postcard?
(Please write down one major cause)

Is this:
   a. Something about you?
   b. Something about the other person or other people?
   c. Something about the situation (circumstances or chance)?

9. A friend thinks you are unfriendly.

What caused your friend to think that you are unfriendly?
(Please write down one major cause)

Is this:
   a. Something about you?
   b. Something about the other person or other people?
   c. Something about the situation (circumstances or chance)?

10. A friend made an insulting remark to you.

What caused your friend to insult you?
(Please write down one major cause)

Is this:
   a. Something about you?
   b. Something about the other person or other people?
   c. Something about the situation (circumstances or chance)?

11. A friend bought you a present.

What caused your friend to buy you a present?
(Please write down one major cause)

Is this:
   a. Something about you?
   b. Something about the other person or other people?
   c. Something about the situation (circumstances or chance)?

12. A friend picked a fight with you.

What caused your friend to fight with you?
(Please write down one major cause)

Is this:
   a. Something about you?
   b. Something about the other person or other people?
   c. Something about the situation (circumstances or chance)?

13. A friend of yours thinks you are dishonest.
What caused your friend to think you are dishonest?
(Please write down one major cause)

Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?

14. A friend spent some time talking to you.

What caused your friend to spend time talking to you?
(Please write down one major cause)

Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?

15. A friend thinks you are clever.

What caused your friend to think you are clever?
(Please write down one major cause)

Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?

16. A friend thinks you are sensible.

What caused your friend to think you are sensible?
(Please write down one major cause)

Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?

17. A friend refused to help you with a job.

What caused your friend to refuse to help you with the job?
(Please write down one major cause)

Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?

18. A friend thinks you are unfair.
What caused your friend to think that you are unfair?
(Please write down one major cause)

Is this:
 a. Something about you?
 b. Something about the other person or other people?
 c. Something about the situation (circumstances or chance)?

19. A friend said that he (she) dislikes you.

What caused your friend to say that he (she) dislikes you?
(Please write down one major cause)

Is this:
 a. Something about you?
 b. Something about the other person or other people?
 c. Something about the situation (circumstances or chance)?

20. A friend called to see how you were doing.

What caused your friend to call to see how you were doing?
(Please write down one major cause)

Is this:
 a. Something about you?
 b. Something about the other person or other people?
 c. Something about the situation (circumstances or chance)?

21. A friend ignored you.

What caused your friend to ignore you?
(Please write down one major cause)

Is this:
 a. Something about you?
 b. Something about the other person or other people?
 c. Something about the situation (circumstances or chance)?

22. A friend said that she (he) admires you.

What caused your friend say that she (he) admired you?
(Please write down one major cause)

Is this:
 a. Something about you?
 b. Something about the other person or other people?
 c. Something about the situation (circumstances or chance)?
23. A friend said that he (she) finds you boring.

What caused your friend to say that he (she) finds you boring?
(Please write down one major cause)

Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?

24. A friend said that she (he) resents you.

What caused your friend to say that she (he) resents you?
(Please write down one major cause)

Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?

25. A friend visited you for a friendly chat.

What caused your friend to visit you for a chat?
(Please write down one major cause)

Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?

26. A friend believes that you are honest.

What caused your friend to believe that you are honest?
(Please write down one major cause)

Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?

27. A friend betrayed the trust you had in her.

What caused your friend to betray your trust?
(Please write down one major cause)

Is this:
  a. Something about you?
  b. Something about the other person or other people?
  c. Something about the situation (circumstances or chance)?
28. A friend ordered you to leave.

What caused your friend to order you to leave?
(Please write down one major cause)

Is this:
a. Something about you?
b. Something about the other person or other people?
c. Something about the situation (circumstances or chance)?

29. A friend said that she (he) respects you.

What caused your friend to say that she (he) respects you?
(Please write down one major cause)

Is this:
a. Something about you?
b. Something about the other person or other people?
c. Something about the situation (circumstances or chance)?

30. A friend thinks you are stupid.

What caused your friend to think you are stupid?
(Please write down one major cause)

Is this:
a. Something about you?
b. Something about the other person or other people?
c. Something about the situation (circumstances or chance)?

31. A friend said that he (she) liked you.

What caused your friend to say that he (she) liked you?
(Please write down one major cause)

Is this:
a. Something about you?
b. Something about the other person or other people?
c. Something about the situation (circumstances or chance)?

32. A neighbor invited you in for a drink.

What caused your friend to invite you in for a drink?
(Please write down one major cause)

Is this:
a. Something about you?
b. Something about the other person or other people?
11 December 2014

HREC/REF: 868/2014

Prof. M Solms
Psychology
Room 2.07
PD Hahn Building
Upper Campus

Dear Prof Solms,

Project Title: NEUROPSYCHOLOGICAL MECHANISMS OF SOCIAL POWER: THE ROLE OF SPATIAL REPRESENTATION AND COVERT ACTION SIMULATION PROCESSES (PhD-candidate- D van der Westhuizen) sub-study linked to 092/2011.

Thank you for your response letter dated 28 November 2014, addressing the issues raised by the Human Research Ethics Committee (HREC).

It is a pleasure to inform you that the HREC has formally approved the above mentioned study.

Approval is granted for one year until the 30 December 2015.

Please submit a progress form, using the standardised Annual Report Form, if the study continues beyond the approval period. Please submit a Standard Closure form if the study is completed within the approval period.

We acknowledge that the following student:-Donne van der Westhuizen is also involved in this project.

Please note that the on-going ethical conduct of the study remains the responsibility of the principal investigator.

Please quote the HREC REF in all your correspondence.

Yours sincerely,

PROFESSOR M BLOCKMAN
CHAIRPERSON, HSF HUMAN ETHICS

Federal Wide Assurance Number: FWA00001637,
Institutional Review Board (IRB) number: IRB00001938

This serves to confirm that the University of Cape Town Research Ethics Committee complies to the Ethics Standards for Clinical Research with a new drug in patients, based on the Medical Research Council (MRC-SA), Food and Drug Administration (FDA-USA), International Convention on Harmonisation Good Clinical Practice (ICH GCP) and Declaration of Helsinki guidelines.

The Research Ethics Committee granting this approval is in compliance with the ICH Harmonised Tripartite Guidelines E6: Note for Guidance on Good Clinical Practice (CPMP/ICH/135/95) and FDA Code Federal Regulation Part 50, 56 and 312.
Appendix D

Informed Consent Form

PATIENT INFORMATION LEAFLET AND INFORMED CONSENT

Informed Consent Document

Instructions:
Please read through the following questions and their answers very carefully. After you have read through the document, please comment on whether you understood everything written in it, and sign where indicated.
If you have any further questions or concerns, please feel free to contact us:

Principal Investigator: Mark Solms
Department of Psychology
University of Cape Town, Upper Campus
Rondebosch, Cape Town

Tel: 021 650-3417

Why is this research being done – what is it trying to find out?
This research is being done to find out more about how testosterone affects brain, the body and behaviour.

Why are you being invited to take part?
You are being invited to take part because you have expressed an interest to participate.

Will you need to take time off work?
During a research session, we will ask you to come in to the lab on two occasions on one day, which will be four hours apart. The first session will last approximately 30 minutes and the second should last no more than 2 hours. Prior to signing up, you will be given an opportunity to select a research session that is most convenient for you.

What procedures, drugs or other treatments are involved in this research?
In this study you will be requested to take either 0.5mg of a testosterone or placebo solution under your tongue. This is a double-blind study, meaning that during the experiment, neither you nor the experimenter will know whether or not you will be receiving testosterone or placebo. You will also be requested to donate a 5ml vial of saliva, collected in a private bathroom cubicle. The saliva sample will be used to measure the natural level of testosterone in your body. We will NOT use the saliva sample to test for anything else and they will be stored in a security-controlled laboratory.

During this experiment you will be requested to fill in several online questionnaires, undergo a hand-scan and engage in a computer-based task. Specifically, we will be assessing your perception of time by asking you to judge the time of onset of particular events and self-produced actions while playing a reaction time game. This time perception task will also be performed alongside another participant but you will be seated at separate computers.

What are the risks and discomforts of taking part in this research?
The testosterone is in liquid form with cyclodextrin as a carrier. Cylodextrin carriers can lead to diarrhea in very rare cases. Testosterone can lead to adverse drug reactions such as headache and nausea but these reactions are infrequently reported. All information you provide is kept strictly confidential. Your identity will remain anonymous throughout the research.
Are there any benefits to you if you take part in this research?
You will be compensated with R200 for taking part in this study.

What happens if you do not want to take part in this research?
Nothing. It is your right to not take part in the research, or to withdraw at any time during the research with no consequence to you, whatsoever. Furthermore you may request that your data be removed confidentially from the dataset.

What happens at the end of this research?
Debriefing will take place once all data is collected. This will allow you the opportunity to learn more about the aims and objectives of the study. You will not, however, be able to find out whether you received the testosterone or the placebo.

Having read through all the questions and answers, please comment on whether you understand everything written in it, if not then please comment on what you did not understand, or any concerns that you might have:

________________________________________________________________________

Full names and surname (Please Print): ________________________________

Signature: ____________________________________________
Date: ____________________________________________

What if Something Goes Wrong?
Prof. Mark Solms, is covered under University of Cape Town no fault clause of the University of Cape Town Insurance. As per this: the University of Cape Town (UCT) undertakes that in the event of you suffering any significant deterioration in health or well-being, or from any unexpected sensitivity or toxicity, that is caused by your participation in the study, it will provide immediate medical care. UCT has appropriate insurance cover to provide prompt payment of compensation for any trial-related injury according to the guidelines outlined by the Association of the British Pharmaceutical Industry, ABPI 1991. Broadly-speaking, the ABPI guidelines recommend that the insured company (UCT), without legal commitment, should compensate you without you having to prove that UCT is at fault. An injury is considered trial-related if, and to the extent that, it is caused by study activities. You must notify the study doctor immediately of any side effects and/or injuries during the trial, whether they are research-related or other related complications.

UCT reserves the right not to provide compensation if, and to the extent that, your injury came about because you chose not to follow the instructions that you were given while you were taking part in the study. Your right in law to claim compensation for injury where you prove negligence is not affected. Copies of these guidelines are available on request.

What if you have complaints about the study? If you want any information regarding your rights as a research participant, or have complaints regarding this research, you may contact Prof. Marc
Blockman, the Chairperson of the Research Ethics Committee at the University of Cape Town. The contact information for the HREC is as follows:

Human Research Ethics Committee  
Faculty of Health Science  
E-52-54 Groote Schuur Hospital Old Main Building  
Observatory 7925  
Tel: (021) 406 6626  
Fax: (021) 406 6411  
Email: lamees.emjedi@uct.ac.za

After you have consulted your doctor or the ethics committee and they have not provided you with answers to your satisfaction, you should write to: The Registrar, South African Medicines Control Council (MCC), Department of Health, Private Bag X 828, PRETORIA 0001.