**Hormonal Contraceptive Use and Cycle Phase Effects on Competitive Persistence:
Progesterone as a Mediating Mechanism**

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Abstract

Objectives: Increasing evidence suggests that hormonal contraceptive (HC) use adversely affects psychological functioning and competitiveness. HCs expose women to consistently high doses of synthetic progesterone, a hormone that fluctuates across the natural menstrual cycle. We propose to test group differences in competitive persistence between HC users, naturally cycling (NC) women in their low progesterone phase, and NC women in their high progesterone phase. Additionally, we will test salivary progesterone among NC women as a mediator of phase effects on competitiveness.

Method: Pre-screening will identify HC users, and naturally cycling women at the two cycle phases. Participants will provide a sample of saliva and complete two behavioral competitiveness tasks. Covariates will also be collected.

Predicted Results: We predict that follicular phase NC women will demonstrate longer competitive persistence compared to luteal phase NC women and HC users. Progesterone will positively mediate the effect of cycle phase on competitive persistence among NC women.

Conclusions: This research will address the effects of HC use, cycle phase, and progesterone levels on competitive behavior in women. The outcome may be an artifact of female reproductive physiology that is likely adaptive, but potentially detrimental to women's social and occupational advancement, raising concerns about the social-psychological risks of HC use.

Keywords: hormonal contraceptives; competitive behavior; persistence; progesterone; menstrual cycle

Hormonal Contraceptives and psychological functioning

As one of the most commonly used prescription drugs among reproductive age women, the oral contraceptive pill is widely available and highly popular (Brynhildsen, 2014; Tinker et al., 2015; United Nations, 2019). Although originally prescribed predominantly for the prevention of pregnancy, hormonal based forms of contraception (HCs) are now commonly prescribed for menstrual related ‘problems’ such as cycle irregularity, heavy cramping and menstrual flow, and teenage acne (Prior, 2016). Recent estimates suggest that three-quarters of women in the United States have used the oral contraceptive pill at some point in their lifetime, with the majority of usage beginning in adolescence (Anderl, Li, & Chen, 2019; Daniels et al., 2013)¹. The United Nations estimates that, worldwide, approximately 25% of women of reproductive age (36% among those who are unmarried) use a hormonal-based contraceptive, either pill or injection (rates vary by world region, United Nations, 2019)². In our own research, 30-50% of our university-based sample of mostly white women aged 18–21 years reported using oral contraceptives at the time of testing, with less than 10% using other hormonally-based contraceptive methods (e.g., patch, implant), and around 10% using an intrauterine device (Casto et al., 2020).

HCs vary in their route or mode of delivery (e.g., pill, patch, injection, implant). These different categories of HCs, and brands associated with each, also vary depending on factors such as whether they deliver a single synthetic class of hormone or a combination; the specific

¹ Population based sampling in the US from 2011-2013 estimated that 16% of women aged 15-44 were currently using the pill, with the prevalence rate being higher among women aged 15-24 (22.4%), non-Hispanic white women (19.0%), women with a bachelor’s degree or higher (21.5%), and women aged 15-19 who were sexually active (35-54%) (Daniels et al., 2014; Lindberg et al., 2016; Martinez & Abma, 2015). More recent sampling (2015-2017) suggests that around one in five women of reproductive age under 30 in the US were currently using oral contraceptive pills (Daniels & Abma, 2018).

² According to UN data, the prevalence of HCs, both pill and injection, is lower among Asian women than other regions of the world. Although baseline rates of contraceptive use are relatively low among Sub-Saharan African populations, injection-based HCs are most common.

type of hormone within each class; dose of each hormone; variability of dose (monophasic or multiphasic); and the number of hormone free days (Hall & Trussell, 2012). The most common form of hormonal contraception – the combined oral contraceptive pill – typically contains a mixture of synthetic estrogens (e.g., ethyl estradiol) and progesterone (known as progestins, e.g., norgestimate; Melo & Creinin, 2016). Regardless of these sources of variance, the main mechanism of action for HCs is to prevent ovulation by suppression of hypothalamic activity necessary to develop a primordial follicle (Rivera et al., 1999). That is, the synthetic estrogen and progesterone in HCs work by mimicking these same hormones normally released from the ovaries at the endpoint of the hypothalamic-pituitary-gonadal axis. Because these hormones are regulated by a negative feedback system, binding of the synthetic forms at the relevant receptor sites in the hypothalamus prevent the hormonal cascades responsible for stimulating ovulation. In doing so, HCs effectively deliver supraphysiological doses of synthetic steroid hormones to target tissues throughout the body including the brain, while suppressing nearly all secretion of their endogenous counterparts.

Endogenous estradiol and progesterone are traditionally understood to affect female sexual differentiation, sexual development, and fertility. Further, they are also fundamentally important molecules for brain processes involved in basic neural functioning (e.g., regulating gene transcription responsible for neuron survival and growth), emotion and cognition, and social and sexual behavior (for review, Del Rio et al., 2018; Jones et al., 2019). The degree to which the synthetic forms of these hormones found in HCs differ from their endogenous counterparts in their efficacy in regulating these cognitive, behavioral, and neurological processes is not well understood (e.g. Pluchino et al., 2006). Additionally, although naturally cycling women of reproductive age are exposed to differing levels of these hormones throughout

the cycle, women taking HCs are often exposed to relatively high levels of synthetic estrogen and progestin throughout the entire cycle. The specific physiological consequence of this more consistent hormonal exposure is not known.

Despite a limited understanding of the physiological effects of synthetic estrogens and progesterone, there is mounting evidence that HC users demonstrate altered psychological functioning. Indeed, initial evidence indicates that HC users display reduced fear extinction, dysregulated social reward mechanisms, and increased emotional reactivity to aversive stimuli (for review, Montoya & Bos, 2017). Compared with non-users, oral contraceptive users show substantial alterations in hormonal levels, genetic factors, and neural structures indicative of chronic stress (Hertel et al., 2017) as well as altered stress reactivity and working memory capacity (Sharma et al., 2020). Perhaps related, HC exposure and long-term use during adolescence correlates with increased depression and mood disorders (e.g., Anderl et al., 2020; for review Del Rio et al., 2018, but c.f. Robakis et al., 2019). Oral contraceptive users have also demonstrated reduced accuracy on mental rotation tasks compared to non-users (Griksiene et al., 2018) and have been shown to spend less time solving challenging cognitive tasks – a sign of reduced perseverance (Bradshaw et al., 2020). In the arena of mating psychology, HC use may disrupt sexual desire and mate preferences, as well as negatively affect relationship satisfaction (Malmborg et al., 2016; Mark et al., 2016; for review, Burrows et al., 2012; Hahn, & Cobey, 2019; Welling et al., 2012). Although this research is still in its relative infancy, it suggests a broad range of adverse effects of HCs on social and cognitive behavior, and on the psychological well-being of women.

Hormonal contraceptives and competitive behavior

Competition is “a social interaction in which access to something valued is contested between individuals and groups” (Casto & Edwards, 2016, p. 21). Competitive encounters can be formal, occurring in a set timeframe where social status is conferred through the rule-based designation of winner or loser (e.g., a sporting outcome). Competition can also be more subtly present in everyday interactions between individuals jockeying for limited resources and social power. According to Social Comparison Theory, the drive to compete is derived from the basic human need to reduce uncertainty between one’s own performance and the performance of others in order to maintain superior relative position (Festinger, 1954; Garcia et al., 2013). Because comparison to others through competition is how relative social status is determined, individual differences in the expression of competitive effort may be a direct predictor of the motivation to attain or maintain relative social status (Casto & Mehta, 2019). Thus, competitive behavior, although considered socially antagonistic in the context of intragroup cohesion, is adaptive for an individual’s attainment of social status and resulting access to limited resources. As a result, competitive behavior aids in occupational and financial advancement among modern humans.

An initial set of studies suggests that hormonal contraceptive users display significantly lower competitive behavior than naturally cycling women. For example, studies employing economic tasks have shown that hormonal contraceptive users make disadvantageous competitive bidding decisions compared to non-users (Pearson and Schipper, 2013), and are less likely to self-select into a competitive than non-competitive tournament scheme (Buser, 2012). Self-reported competitive behavior directed towards other women, presumably for access to desired mates (e.g., intrasexual competitiveness), has also been found to be lower among users compared to non-users (Cobey et al., 2013). In a recent study of competitive persistence, Casto et

al. (2020) found that women using HCs more broadly (most were oral contraceptive users) had significantly lower task performance than naturally cycling women. Although this finding was not predicted *a priori*, it is consistent with recent research showing that HC usage reduces persistence in non-competitive cognitive tasks (Bradshaw et al., 2020; Griksiene et al., 2018).

The laboratory task in Casto et al. (2020) involved participants holding up a small weight at arm's length and shoulder height for time in competition against one or more participants. Performance in this task showed a strong and consistent positive relationship with individual differences in self-reported trait competitiveness, dominance motivation, and task-specific motivation and effort (and no relation to physical characteristics related to body size and strength). Competitive endurance is just one of the ways in which motivation to dominate or attain social status over others is materialized behaviorally, as those who succeed in social contests must display superior energy, effort, mental or physical strength, will-power, and endurance over their opponent. Thus, the competitive will task is a relatively simple and face valid application for revealing individual differences competitive motivation. The degree with which HC use affects behavior in this and other tasks of competitive behavior is an important area for future research, and is highly relevant to the advancement of women in modern society.

Progesterone as an explanatory mechanism

Previous research has shown that HC use affects neural architecture and functional connectivity in areas of the brain associated with cognition and emotion (e.g. Hertel et al., 2017; Lisofsky et al., 2016; Petersen et al., 2014; Pletzer & Kerschbaum, 2014). Relatively high levels of the synthetic progesterone—the primary active ingredient in most HCs—are thought to mediate these neurological changes (Arélin et al., 2015). Furthermore, progesterone appears to attenuate psychological motivation by inhibiting the rewarding effects of other steroid hormones

as well as addictive substances (for review, Quinones-Jenab & Jenab, 2010). The anti-androgenic properties of some progestins (e.g., Louw-du Toit et al., 2017; Sitruk-Ware & Nath, 2013) could inhibit testosterone's ability to promote competitive motivation, meaning that the behavioral effects of HCs might depend on the androgenicity of the specific progestin formula (Pletzer et al., 2015). Taken together, consistent exposure to high levels of synthetic progesterone in HCs may be responsible for altering competitive behavior among users, via complex direct and indirect interactions with neurological systems involved in motivation.

Progesterone levels in naturally cycling women vary drastically across the menstrual cycle (Choe et al., 1983; Gandara et al., 2007; Lu et al., 1999). Indeed, menstrual cycle phase and related ovarian activity is dependent on and demarcated by these hormonal shifts. The follicular phase (after menstruation and up until the six-day fertile window ending on the day of ovulation) is characterized by relative progesterone quiescence, with slow and low-rising estradiol. However, the luteal phase is marked by substantially higher progesterone (-9 to -3 days leading up to the beginning of a menstrual period), with peak progesterone superseding a lower peak of estradiol at roughly seven days prior to the next menstrual period. It is this latter hormonal pattern, characteristic of the luteal phase, which is mimicked by the hormonal cocktail contained in many HCs.

Prior research on menstrual cycle shifts in competitive behavior has focused on the *late* follicular phase, a six-day window of conception possibility usually occurring mid-cycle and marked by peak estradiol. Intrasexual competition and behavioral assertiveness peaks during this ovulatory period, presumably to support women in their acquisition of high-quality mates (e.g. Blake, Bastian, O'Dean, & Denson, 2016; Cobey et al., 2013; Durante et al., 2008; Puts, 2005). Studies examining ovulatory effects on competitive behavior outside of mating contexts have

also shown that competitive behavior in economic games increases during the fertile window (e.g., Eisenbruch and Roney, 2016; Pearson and Schipper, 2013). Whether competitive behavior differs between early- to mid-follicular phase—where progesterone is quiescent—and mid-luteal phase—where progesterone is dominant—has not been well-studied. Consonant with the prediction that progesterone directly or indirectly affects status-related and competitive motivation, high progesterone levels in naturally cycling women have been associated with lower sexual motivation (Roney & Simmons, 2016) and self-reported assertiveness (in combination with high estradiol, Blake et al., 2017). To better understand whether progesterone reduces competitive motivation, a detailed examination of differences in competitiveness between mid-follicular and mid-luteal phases, and the hormonal mechanisms underpinning any resulting effects, is warranted.

The adaptive benefit of reduced competitive behavior during the mid-luteal phase is not well understood. One important physiological function of progesterone is to maintain pregnancy: Progesterone continues to increase drastically from the mid-luteal peak in pregnant women but plummets following this mid-luteal point in non-pregnant women. There is evidence to suggest that women in this phase of high progesterone are sensitive to health cues in others and take less health risks (e.g., Conway et al., 2007; Jones et al., 2005; for review, Welling & Burris, 2019). This set of cognitive and behavioral changes would be adaptive in terms of protecting a new and particularly fragile pregnancy, should conception have occurred at ovulation. Likewise, competitive exertion or antagonistic social interaction could be risky for physical health and for the maintenance of social ties, factors that may influence the survivability of a newly developing fetus. For this reason, women may demonstrate lower competitiveness during periods of progesterone dominance.

The view that progesterone drives motivational shifts away from higher risk activity and towards health preservation is supported by evidence that phases of high progesterone are associated with increased food intake, but reduced sexual activity (Roney & Simmons, 2017). Additionally, high progesterone in naturally cycling women has been shown to associate with a paradoxical set of social cognitions: heightened sensitivity to social stimuli and attachment anxiety/insecurity, but also increased pro-social motivation and reduced feelings of social ostracism (Lobmaier et al., 2019; Maner & Miller, 2014; Reynolds et al., 2018; Wirth & Schultheiss, 2006; for review, Gangestad & Grebe, 2017). The overall picture is that high progesterone during the mid-luteal phase may functionally increase motivation for socially affiliative interactions with others (helpful for mate retention and social bonding that is beneficial for maintaining pregnancy), but—as a necessary tradeoff—reduce motivation for competitive interactions. This hypothesis is premised on the notion that high-progesterone-derived detriments in competitive behavior are beneficial to the goal of reproduction in pregnant women. However, for non-pregnant and modern women generally, the cost of reduced competitive motivation during the brief luteal window could be problematic for status attainment in contexts where competitive behavior is required for success (e.g., performing in a debate or an athletic arena, applying for a competitive job opportunity or promotion).

Proposed Study

We propose a registered report to attempt to replicate the Casto et al. (2020) finding that HC users show lower competitive persistence than non-users. Further, we propose to extend this research in several ways. First, because individual differences in levels of progesterone shift across the menstrual cycle, we test differences in competitive persistence between naturally cycling women in their mid-luteal compared to mid-follicular phase. Second, by sampling

salivary progesterone among naturally cycling women, we aim to test the role of progesterone in explaining the effect of menstrual-cycle phase on competitive persistence. Third, in addition to measuring competitive persistence in the competitive will task (weight-holding), our proposed design includes an additional task of competitive persistence in a cognitive task (time spent attempting to solve an unsolvable anagram), allowing us to determine the task specificity of this effect.

Hypotheses

- Naturally cycling women in their mid-follicular phase (NC-MF) will demonstrate higher competitive persistence than women using hormonal contraceptives (HC users) and naturally cycling women in their mid-luteal phase (NC-ML). Predicted means are shown in Figure 1.

Figure 1. Predicted main effect: NC women in their mid-follicular phase will out-perform both NC women in their mid-luteal phase and women using HCs (data are estimated from Casto et al., 2020 and only for the competitive will task, described below).

- Among naturally cycling women, progesterone levels will mediate the relationship between cycle phase and competitive persistence, with higher progesterone predicting lower competitive persistence (Figure 2).

Figure 2. Predicted mediation model in which progesterone exerts an indirect effect on the relationship between cycle phase (NC-ML and NC-MF) and competitive persistence (this prediction was facilitated by the progesterone-based mediation model recently published by Lobmaier et al., 2019).

Proposed Method

Cycle phase determination

There are a variety of methods, of varying validity, employed to estimate women’s cycle phase (Blake et al., 2016; Gangestad et al., 2016). Due to the statistical noise created by making these inferences, the importance of the choosing the most valid, yet attainable method within the parameters of the study goals and resources is paramount to the validity and replicability of the research findings. Much of the prior research dealing the effects of cycle phase on women’s behavior has focused on the window of high fertility, a relatively narrow (~6-day) part of the cycle. The present research is *not* premised on the notion that fertility influences the outcomes of interest, but rather focuses on variation in progesterone occurring outside of the fertile window. We aim to test a broader understanding of the menstrual cycle, beyond the brief period of fertility, in which the overall endocrine experience of women is marked by a drastic shift from progesterone quiescence to progesterone dominance.

In our study design we aim to capture the broader windows of mid-follicular and mid-luteal phases, rather than the precise period of complete progesterone trough or peak association with each phase respectively. This prediction is in line with the theoretical understanding that hormones—particularly those involved in reproductive behavior—coordinate behavior in a permissive or stepwise fashion in which behavior is expressed once levels of the hormone reach a particular threshold, but further increases do little to alter the degree of behavioural expression (e.g., Hau & Goymann, 2015) – an on/off switch as opposed to a dimmer. This approach of broadly categorizing the windows of interest, combined with direct measurement of progesterone, reduces the noise typically associated with correctly estimating cycle phase compared with studies wishing to capture fertility or peak progesterone. Thus, cycle phase will be estimated using the forward counting method, with supplemental instruction for making more accurate estimates (discussed below), in combination with the backward counting follow-up confirmation of next menstrual onset.

To determine phase ranges, we consulted an analysis of 612,613 menstrual cycles from 124,648 women, which indicated that the average peri-ovulatory period begins on Day 11 and ends on Day 16 (Bull et al., 2019). To account for significant between- and within-women variability in the timing and length of the fertile window, follicular phase women will be recruited to attend laboratory sessions from the cessation of menstruation to Day 8 of their cycle. Although roughly 6% of women may be in the beginning of their fertile window on Day 8 (Blake et al., 2016), it is extremely unlikely that any woman would have *ovulated* by this time (as ovulation occurs at the end of the fertile window). Thus, our confidence that this window is marked by progesterone quiescence is high. Luteal phase women will be recruited to attend laboratory sessions -8 to -4 days before their predicted next menstrual cycle onset. Bull et al.

(2019) found that the average luteal phase lasted 12.9 days, and our more conservative 4-day recruitment window is designed to capture women when progesterone is dominant, but as stated earlier, not necessarily peaked. Again, it is highly unlikely that women would experience low progesterone (relative to follicular phase levels) during this window, raising confidence in the validity of this cycle phase estimation pattern (e.g., see drastic progesterone level comparisons between ML and MF women in Sharma et al., 2020 using the forward counting method and broader day ranges for these phase windows). In summary, our primary aim in recruiting women in these phase windows is to ensure *sufficient variability* between groups in progesterone exposure, rather than capture peak progesterone, per se.

Estimating sample size for adequate statistical power

Previous investigations of competitive endurance between HC users and NC women yielded an average effect size of $d = .35$ (Casto et al., 2020), yet this effect size is likely underestimated due to unexplained variance in menstrual cycle phase variation among NC women (see Gangestad et al., 2016). An accurate estimation of follicular and luteal phases would allow recruitment of women with naturally low and high endogenous progesterone, respectively, thus increasing the expected effect size. We assume the design in Casto et al. (2020) compromised measurement validity by around 0.20, and thus estimate the size of the true effect to be 20% larger than reported (thus, $d = .42$). Given our own phase estimation is exactly that—an estimation—we calculate our sample size for the effect of $d = .40$ ($f = .20$) on the difference in competitive endurance between follicular phase women and the other two groups (HC users and NC-ML).

A balanced one-way analysis of variance power calculation with three groups indicates that 82 participants per group (total $N = 246$) would yield sufficient statistical power ($\beta = .80$,

$\alpha = .05$) to detect an effect of $f = .20$. Assuming a sample size of 170 ($N = 85 \times 2$ for the two groups of NC women), an effect of $B = -.25$ for $X \rightarrow M$ (group effect on progesterone, estimate from Blake et al., 2017), the same effect for $M \rightarrow Y$ (progesterone on competitive endurance, equivalent to $d = .50$), and a negligible direct effect for X on Y ($B = .05$), a mediation power analysis indicated that the power to detect the indirect effect of X on Y was .821

(<https://davidakenny.shinyapps.io/MedPower/>). Thus, we will recruit 250 women (82-85 women in each group) from participant pools at New College of Florida and the University of Melbourne.

Procedure

Online pre-screening

Participants first complete an online pre-screening form, which automatically determines their eligibility for the study based on their age, menstrual cycle characteristics, HC usage, and relevant medical history (See Appendix A for the full list of questions and inclusion criteria).

Assignment to day and time of laboratory session

Hormonal contraceptive users will be assigned to come in during one of the active pill days (i.e., excluding menstruation). Naturally cycling women are randomly assigned to complete their laboratory session during cessation of menstruation to Day +8 (follicular phase) or days -4 to -8 of their next menstrual cycle. Anyone unable to attend a session during their allocated window will indicate so on the pre-screening form. Doing so will trigger a response which informs them they can re-apply for participation into the study in approximately three weeks' time, by filling out the pre-screening again. Though more onerous for the participant, this procedure ensures that assignment into MF and ML phases is based on the most current menstrual cycle data from each participant. One to two days before the assigned testing session,

each participant will receive a reminder email about the scheduled time. The email to NC women will contain additional instructions about oral health and consumption limits consistent with procedures for giving a saliva sample (Appendix B), and for HC women it will request they provide information about their contraceptive brand and dose (a corresponding health declaration form will be completed as per requirements of the UMelb IRB, Appendix B).

Laboratory procedure

Mouth rinse (NC women only). Participants rinse mouth out with water.

Overview and consent. All participants will be informed about what they will be asked to do and sign a consent form indicating their understanding.

Hand-grip strength. Hand grip strength will be measured using a standard digital hand dynamometer. This data will be used to assess the role of hand-strength on task performance in the competitive will task.

Complete questionnaires. The first set of survey items will include questions about the recency and/or quantity of activities such as exercise, teeth-brushing, caffeine consumption, etc., to assess the need for quality-control of the participant's saliva sample. Additionally, participants will give their height, weight, list of medications, and frequency of engaging in upper-body strength exercise (as a part of continued efforts to assess and validate the competitive will task). Participants will then complete the Competitiveness Orientation Measure (Newby & Klein, 2014) as well as a brief mood questionnaire for the purpose of exploratory piloting for future research (beyond the scope of the present study).

Saliva sample (NC women only). After 10 minutes has elapsed since the mouth rinse, participants will use a wet-wipe to clean their lips. Approximately 1.5ml of saliva will be collected via

passive drool into plastic vials using plastic saliva collect aids. The time it takes each participant to fill the vial will be recorded.

Competitive persistence tasks. Participants will then complete the Competitive Will Task followed by the Anagram Task. The order is fixed and not counter-balanced because impossible puzzle tasks are known to induce a state of psychological depletion and reduced effort on subsequent tasks (e.g., Hagger et al., 2010; Starcke et al., 2017). Performance in the competitive will task is the primary outcome as interest due to the goal attempting to replicate earlier work with this task. There will be a brief “break” between tasks to reduce fatigue effects.

Complete questionnaires. Following the behavioral tasks, a final set of survey items will be completed. To confirm that the menstrual phase estimation data collected at prescreening is accurate at the time of testing, participants will complete a set of questions about period timing, contraceptive use, and characteristics of menstrual experiences (Appendix C).

Email follow-up. Participants will be emailed following participation to obtain the day of next menstrual onset for back-ward counting confirmation of menstrual cycle phase window.

Saliva assay

Saliva samples will be stored at -25 to -80 Celsius, then shipped to a lab to be assayed for progesterone (determined via mass spectrometry or chemiluminescence, best available method at the time of testing). Intra- and inter-assay coefficients of variance will be reported to assess assay reliability.

Competitive Will Task

Participants will hold a 1 lb./0.5-kg weight (a common dumbbell used for resistance training) at arm’s length and shoulder height for as long as they feel they can in competition

against other participants. For this study, participants will compete individually against the unknown performance of all other participants. Performance is operationalized as the time in seconds that each participant holds her arm before quitting the contest. The top five individuals of all the participants from each academic semester will receive a cash prize in increasing magnitude based on place: 1st = \$50, 2nd = \$40, 3rd=\$30, 4th=\$20, 5th=\$10. Prize winners will be contacted at the end of the semester via email to receive their cash prize. Thus, participants will not be told how their performance time compares to others or given any feedback about their performance relative to any task metrics (e.g., average times).

This task has been previously validated as measure of competitive motivation (Casto et al., 2020) – performance in the competitive will task is positively related to individual differences in traits having to do with competitiveness and dominance motivation as well as task-specific desire to win and perceived effort. Furthermore, physical qualities associated with strength – height, weight, and BMI – were unrelated to task performance and did not explain variance in the relationship between performance and psychological traits.

Competitive Anagram Task

Participants will individually compete in an anagram task. The Anagram Persistence Task (APT, Eisenberger & Leonard, 1980), which is administered in a variety of single to multi-item formats, has been validated as a measure of test-taking effort, persistence, and motivation – “defined as the motive to achieve high standards of performance in the face of frustration (Dudley, Orvis, Lebiecki, & Cortina, 2006, cited in and for review, Gignac & Wong, 2018, p.2). In Bradshaw et al., (2020), time spent attempting the unsolvable anagrams (operationally defined as “cognitive persistence”) was greater among naturally cycling women compared to women using HCs.

For the present research, we will adopt the APT to a competitive scheme in which participants are told they can increase their rank among other participants for a cash prize by solving a single unsolvable test anagram. The original design plan was to include both solvable and unsolvable anagrams across multiple sets (as in Bradshaw et al., 2020). However, piloting of the set with a 4-min time limit for each anagram, used in this prior research, led to a majority of participants reaching the time threshold (ceiling effect). Thus, we used a single unsolvable “test” anagram and had a maximum time cut-off set at 12 minutes.

Participants will be told that solving the test anagram will add 1 minute to their performance time in the Competitive Will Task, and thus, their rank among participants for the top-five cash prizes. We intend this incentive scheme to activate a sense of social rank as the goal of competition rather than money, per se, to reduce complications from participants’ financial situation influencing motivation. None of the prize money is awarded during the session and thus, prize money is a rather distant reward in terms of driving competitive behavior. The amount of time spent solving the anagram will be used as a measure of competitive persistence. Prior to starting the task, the participants will be provided with the following information on the screen:

[“Next you will complete Anagrams. Anagrams are a string of letters that can be reordered to make a word. For example, OBKO would be an anagram for BOOK. Your task is to rearrange the letters to make an actual word. Let's practice a few! Feel free to use the scratch paper and pen provided to help solve the anagrams.”]

[“Now that you have practiced, we will give you a harder one. BUT this is now a part of the study. By solving this next one correctly, you will receive 1 min added to your performance time in the weight holding task. This could drastically increase your rank among the other participants. You can take as long as you want, but you are not required to solve it - doing so is for competition, to increase your rank. Click the button below to begin.”]

[“LOIMCPRIS
-- don't guess randomly, it should be a widely recognized English word
-- you will not get an incorrect/correct indicator upon submitting

-- you cannot go back once submitting”]

Competitive Orientation Measure

The Competitiveness Orientation Measure (COM, Newby & Klein, 2014) is a 37-item survey rated on a 5-point Likert scale with subscales for (1) general trait competitiveness, (2) dominant competitiveness, (3) enjoyment or affectivity associated with competitiveness and (4) intrinsic interest in competition for personal enhancement. Factor one, general competitiveness, had the most robust factor loadings in scale development and appears intrinsically face-valid with items like “I am a competitive person.” All four subscales have demonstrated high reliability and validity among large student and community-based samples (Newby & Klein, 2014). In previous research with the competitive will task, task performance significantly and positively related to all subscales for the COM (general: $r = .31, p < .001$; dominant: $r = .22, p = .002$; competitive affectivity: $r = .24, p = .002$; personal enhancement: $r = .32, p < .001$). The COM will be used in the present research the purpose of continued task validation of the Competitive Will Task and for new validity evidence for the competitive anagram task. Although we do not predict differences in the trait competitiveness between the NC-ML, NC-MF, and HC user groups, descriptive statistics for trait competitiveness across groups will be provided. Because these subscales produce relatively high inter-correlation and for simplicity in the present design, only the total mean score (“general competitiveness”) will be tested as a covariate.

Data Analysis

Post-hoc withdrawals from analyses involving progesterone

When participants complete questionnaires, we will ask NC women about adherence to pre-session instructions regarding (1) eating/drinking anything but water, consuming caffeine, smoking nicotine, chewing gum/brushing teeth within past one hour; (2) drinking alcohol within past 12 hours; (3) Oral infections/lacerations/gum bleeding within the past one day; (4) acute illness, like a cold, respiratory infection, or fever. We will also record any medication they are on and manually search whether these medications might interfere with progesterone. If any participants meet these criteria, we will conduct a robustness test by analyzing the data with these cases included then excluded. If there is a significant change in effect size in the robustness test ($\pm 10\%$), we will present the robustness test analysis only. Finally, for unknown reasons, some participants hormone levels are non-detectable in salivary analysis; these participants will be excluded from analyses involving hormone levels.

Post-hoc withdrawals from analyses for non-matching cycle phase window on day of testing

If a participant provides information about their menstrual cycle phase timing on the day of testing or in email follow-up that indicates their day of testing was outside of the planned ML or MF window, will be excluded if they 1) are more than 2 days out of range on either side of the 4-day windows and/or 2) Progesterone levels are ± 3 SDs from the mean of their ML or MF group. If a participant unexpectedly began or ceased hormonal birth control between pre-screening to their laboratory session, this participant will be excluded from analyses.

Transformations and Dealing with Outliers

Progesterone levels will be assessed for normality. If data are non-normal, raw values will be transformed to reduce skew. Levels that are more than ± 3 -SD from the mean progesterone level within each of the NC-MF and NC-ML groups in our sample will be

winsorized (Pollet & van der Meij, 2017), a method that effectively replaces outlier values with high/low values while maintaining relative position among other data points and reducing skewness (Erceg-Hurn, & Mirosevich, 2008). Competitive endurance on the two tasks, trait competitiveness, and handgrip strength will also be assessed for normality and transformed to reduce skew where necessary. Performance in the competitive will task will be centered within each set of participants (US and Australian) prior to combining the samples for analysis due to the slight difference the 1 lb vs 0.5 kg weight. All continuous predictor variables will then be standardized for supplemental robustness analyses and to provide additional data to aid interpreting of the relative size of the coefficients.

Supplemental Robustness Tests of Exploratory Covariates

Although we do not predict differences in trait competitiveness and hand-grip strength between the NC-ML, NC-MF, and HC user groups, descriptive statistics for subscale means across groups will be provided. In supplemental robustness analyses, we will test whether including these two variables as general covariates in the proposed analyses produces substantially different results (i.e., +/- 10% difference in effect sizes). We will additionally test the significance of testing country in the Competitive Will Task (as 1lb weights are unavailable in Australia, and are 10% greater in mass than 0.5kg weights). How these robustness test results compare to the main analyses will be discussed in the text.

Analysis Code and Simulated Results

Analysis code for main analyses and simulated data results are shown in Appendix D.

Data Availability

Complete data and analysis code will be posted on the Open Science Framework.

Conflict of Interest

The authors declared that they have no conflict of interest.

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Appendix A

Pre-Screening Questions and Inclusion Criteria

QUESTION	INCLUSION CRITERIA
Enter your age in years.	18-40
Are you fluent in English?	Yes
Are you assigned female at birth?	Yes
Do you have regular menstrual cycles (i.e., roughly one menstrual period per month?)	Yes
Are you physically able to hold light weights (around 11lb/1kg) in your arms?	Yes
Do you have any cancer/tumor, immune, cardiovascular, metabolic, endocrine or kidney disorder?	No
Now or within the past three months, have you used anabolic steroids?	No
Now or within the past three months, have you been pregnant or have you breastfed?	No
Do you have a history of polycystic ovary syndrome?	No
Have you taken Depot medroxyprogesterone acetate (hormonal birth control injections) within the past 12 months?	No
Now or within the past three months, have you used hormonal birth control (e.g., the contraceptive pill, nuvaring, implanon, hormonal IUD)?	Yes or No
[For HC users only] Now or within the past three months, has the type/dose/brand of HBC you've used changed?	No
[For HC users only] Are you currently using birth control that you ingest daily in the form of a pill?	Yes
[For HC pill users only] What day of the pill pack are currently on?	N/A
[For HC pill users only] How many days long is your pill pack?	24-35 days
[For HC pill users only] Many birth control pills have "non-active" pill days (the pills on these days are sometimes a different color). Do you plan to skip your next period, that is, not take these pills and start your next pill pack?	N/A
[For HC pill users only] How many of these non-active pills are there in the brand of pill that you take?	N/A
[For HC users only] For this study, we will require you to send us a photograph of your HBC prescription/contraceptive packet. Are you willing to do this as part of the study? *Note, for photographs, we are only interested in brand and dosage info, not your personally identifying info.	Yes
Do you use a menstrual cycle tracker, or period app?	Yes or No
On what date did your last period start (i.e., the first day of proper menstrual cycle bleeding, not spotting?	<35 days from current date

How confident are you that your period started on that date? [1 – not at all, 7 – very confident]	[NC women] >3
How many days do your menstrual cycles go for (e.g., 28 days)? In other words, how many days is it from the start of one period to the start of the next period?	[NC women] 25-35 days
How confident are you that your cycle lasts this long on average? [1 – not at all, 7 – very confident]	[NC women] >4
You said your menstrual cycles go for X days on average. What is the shortest and longest they go for? For example, you might have a cycle that generally lasts 28 days, but sometimes lasts 26 days, and otherwise lasts 30 days. Some period trackers automatically give this information (its called the “range” or “variance”). If you don’t know, leave this blank.	N/A

Appendix B

Reminder Email Text

This is a reminder email about your session for Study X.

[for HC users]: Don't forget to take a photograph of your hormonal birth control prescription and bring this to your session. If you forget to bring this, we cannot complete the session. You can email it to us now, or bring it with you (but don't forget it!).

[NC only]: During your session, we will take a saliva sample from you. In order to qualify for the sample, remember the following very important information:

- You cannot currently be menstruating (i.e., experiencing your menstrual period). If you think that you will be, please contact us to rebook the session or delay it by a few days.
- Within 12 hours of your session, don't drink alcohol
- Tell us if you have an oral infection/laceration/gum bleeding today or tomorrow
- Tell us if you have an acute illness/fever/cold
- Within one hour of your session, don't eat or drink anything but water, smoke nicotine, chew gum, or brush your teeth

Health Declaration – Australian Participants Only

To be email to participants in the 24-48 hours prior to their participation and also completed on arrival. Participants must answer no to all questions, as per the University of Melbourne guidelines for attending campus.

- **Do you have any of the following symptoms? Fever, cough, shortness of breath, chills, body aches, sore or scratchy throat, headache, runny nose, muscle pain, vomiting, nausea, diarrhoea, or loss of smell or taste.**
- **Have you tested positive for Covid-19 in the past 14 days?**
- **Have you been in contact with a Covid-19 case in the last 14 days?**
- **Have you been asked to self-isolate by a health official?**
- **Do you have a household member who is a close contact of a Covid-19 case?**
- **Have you travelled to any of the covid-19 hotspots within the last 14 days?**
- **Have you had a covid-19 test and are still awaiting test results?**

Appendix C

Survey Items

Are you currently on any kind of medication? If yes, please provide the name of the prescription and the dosage if you can recall it.

To confirm that the data collected at prescreening matches the estimated phases upon arrival, the following set of questions will be asked:

[If you use a menstrual cycle tracker, or period app, you may use this to help answer the following questions]

Are you currently using birth control that you ingest daily in the form of a pill?

[If Yes] What day of the pill pack are currently on?

Have you had any changes to your birth control since doing the pre-screening X days ago? Or, have you taken any additional hormonal birth control since then.

Are you currently menstruating? (i.e., do you currently have your menstrual period?)

Has your period started/stopped since you completed the pre-screening questionnaire X days ago? If so, on what date did it start?

Saliva Sampling Questions

1. Have you eaten anything or drunk anything other than plain water within the past one hour?
2. Have you smoked nicotine within the past one hour?
3. Have you chewed gum or brushed your teeth within the past one hour?
4. How many hours ago has it been since you consumed caffeine (coffee, tea, soft drink, chocolate)?
5. How long ago, in hours, has it been since you woke up today?
6. Have you experienced any other oral infections and/ or oral lacerations/gum bleeding over the past day?
7. Do you have any kind of acute illness, like a cold, respiratory infection, or fever?
8. Have you drunk alcohol within the past 12 hours?

Appendix D

Analysis Code and Simulated Data Results

All analyses will be repeated w/ CWtime replaced by APTtime (competitive persistence as measured by the anagram task)

Effects sizes in simulated data are larger than expected in the actual data

```
ONEWAY CWtime BY Group
  /STATISTICS DESCRIPTIVES
  /PLOT MEANS
  /MISSING ANALYSIS
  /POSTHOC=TUKEY ALPHA(0.05) .
```

Oneway

Descriptives

CWtime

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
UCuser	85	220.00	60.000	6.508	207.06	232.94	70	361
NC-MF	85	280.00	60.000	6.508	267.06	292.94	174	467
NC-ML	85	220.00	60.000	6.508	207.06	232.94	102	382
Total	255	240.00	66.142	4.142	231.84	248.16	70	467

ANOVA

CWtime

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	204000.000	2	102000.000	28.333	.000
Within Groups	907200.000	252	3600.000		
Total	1111200.000	254			

Post Hoc Tests

Multiple Comparisons

Dependent Variable: CWtime

Tukey HSD

(I) Group	(J) Group	Mean Difference	Std. Error	Sig.	95% Confidence Interval	
		(I-J)			Lower Bound	Upper Bound
UCuser	NC-MF	-60.000*	9.204	.000	-81.70	-38.30
	NC-ML	.000	9.204	1.000	-21.70	21.70
NC-MF	UCuser	60.000*	9.204	.000	38.30	81.70
	NC-ML	60.000*	9.204	.000	38.30	81.70
NC-ML	UCuser	.000	9.204	1.000	-21.70	21.70
	NC-MF	-60.000*	9.204	.000	-81.70	-38.30

*. The mean difference is significant at the 0.05 level.

Homogeneous Subsets

CWtime

Tukey HSD^a

Group	N	Subset for alpha = 0.05	
		1	2
UCuser	85	220.00	
NC-ML	85	220.00	
NC-MF	85		280.00
Sig.		1.000	1.000

Means for groups in homogeneous subsets are displayed.

a. Uses Harmonic Mean Sample Size = 85.000.

Means Plots

```
USE ALL.
COMPUTE filter_$=(Group > 1).
VARIABLE LABELS filter_$ 'Group > 1 (FILTER)'.
VALUE LABELS filter_$ 0 'Not Selected' 1 'Selected'.
FORMATS filter_$ (f1.0).
FILTER BY filter_$.
EXECUTE.
```

Run MATRIX procedure:

***** PROCESS Procedure for SPSS Version 3.3 *****

Written by Andrew F. Hayes, Ph.D. www.afhayes.com
Documentation available in Hayes (2018). www.guilford.com/p/hayes3

```
Model   : 4
Y       : CWtime
X       : Group
M       : Progest
```

Sample
Size: 170

OUTCOME VARIABLE:
Progest

Model Summary	R	R-sq	MSE	F	df1	df2	p
	.8336	.6948	277.7778	382.5000	1.0000	168.0000	.0000

Model		coeff	se	t	p	LLCI	ULCI
constant		-50.0000	6.5179	-7.6711	.0000	-62.8676	-37.1324
Group		50.0000	2.5565	19.5576	.0000	44.9529	55.0471

Standardized coefficients

	coeff
Group	1.6622

 OUTCOME VARIABLE:
 CWtime

Model Summary

	R	R-sq	MSE	F	df1	df2	p
	.5018	.2518	3395.2096	28.0984	2.0000	167.0000	.0000

Model

	coeff	se	t	p	LLCI	ULCI
constant	355.0000	26.4793	13.4067	.0000	302.7226	407.2774
Group	-15.0000	16.1794	-.9271	.3552	-46.9426	16.9426
Progest	-.9000	.2697	-3.3367	.0010	-1.4325	-.3675

Standardized coefficients

	coeff
Group	-.2240
Progest	-.4043

***** TOTAL EFFECT MODEL *****
 OUTCOME VARIABLE:
 CWtime

Model Summary

	R	R-sq	MSE	F	df1	df2	p
	.4493	.2019	3600.0000	42.5000	1.0000	168.0000	.0000

Model

	coeff	se	t	p	LLCI	ULCI
constant	400.0000	23.4646	17.0469	.0000	353.6765	446.3235
Group	-60.0000	9.2036	-6.5192	.0000	-78.1696	-41.8304

Standardized coefficients

	coeff
Group	-.8960

***** TOTAL, DIRECT, AND INDIRECT EFFECTS OF X ON Y *****

Total effect of X on Y

Effect	se	t	p	LLCI	ULCI	c_ps
-60.0000	9.2036	-6.5192	.0000	-78.1696	-41.8304	-.8960

Direct effect of X on Y

Effect	se	t	p	LLCI	ULCI	c'_ps
-15.0000	16.1794	-.9271	.3552	-46.9426	16.9426	-.2240

Indirect effect(s) of X on Y:

Effect	BootSE	BootLLCI	BootULCI
--------	--------	----------	----------

```

Progest      -45.0000      12.8021      -71.5504      -19.8709

Partially standardized indirect effect(s) of X on Y:
      Effect      BootSE      BootLLCI      BootULCI
Progest      -.6720       .1882      -1.0467      -.3007

***** ANALYSIS NOTES AND ERRORS *****

Level of confidence for all confidence intervals in output:
  95.0000

Number of bootstrap samples for percentile bootstrap confidence intervals:
  5000

NOTE: Standardized coefficients for dichotomous or multicategorical X are in
      partially standardized form.

----- END MATRIX -----

```

DATA SIMULATION CODE:

```

# 3 groups
# 1 group higher than the other 2
# outcome measure is seconds 100-350 normal; group 1 = 220; group 2 = 280; group 3 = 220 :: d= .4
# progesterone levels group 1 = 0; group 2 = 50; group 3 = 100
# hire progesterone = lower performance without group 1 :: .25

ProgRTcor <- -0.25
progG1mean <- NA
progG2mean <- 50
progG3mean <- 100
Dprog <- 3
progSD <- (progG3mean-progG2mean)/Dprog

rtG1mean <- 220
rtG2mean <- 280
rtG3mean <- 220
Drt <- 1
rtSD <- (rtG2mean-rtG3mean)/Drt

nPerGroup <- 85

Sigma1 <- matrix(c(1,ProgRTcor,ProgRTcor,1),2,2)
Sigma2 <- cor2cov(Sigma1, c(progSD,rtSD), discrepancy = 1e-05)
G1dat <- mvrnorm(n = nPerGroup, c(progG3mean,rtG3mean), Sigma2, empirical = TRUE)
df.g1 <- data.frame(group = 1, progesterone = NA, RT = G1dat[,2])
G2dat <- mvrnorm(n = nPerGroup, c(progG2mean,rtG2mean), Sigma2, empirical = TRUE)
df.g2 <- data.frame(group = 2, progesterone = G2dat[,1], RT = G2dat[,2])
G3dat <- mvrnorm(n = nPerGroup, c(progG3mean,rtG3mean), Sigma2, empirical = TRUE)
df.g3 <- data.frame(group = 3, progesterone = G3dat[,1], RT = G3dat[,2])

df.all <- rbind(df.g1,df.g2, df.g3)

```



```

df.all$group <- as.factor(df.all$group)

rt.means <- with(df.all, tapply(RT,group,mean))
rt.sds <- with(df.all, tapply(RT,group,sd))

rt.aov=aov(RT~group, data=df.all)
rt.tuk <- TukeyHSD(rt.aov, 'group')

prog.means <- with(df.all, tapply(progesterone,group,mean))
prog.sds <- with(df.all, tapply(progesterone,group,sd))

prog.ttest=t.test(progesterone~group, data=df.all)

sink("simulatedData.txt", append = F)
print(rt.means)
print(rt.sds)
print(anova(rt.aov))
print(rt.tuk)

print(prog.means)
print(prog.sds)
print(prog.ttest)
sink(NULL)

with(df.all[df.all$group == 2,], plot(RT ~ progesterone, ylim = c(0,500), xlim=c(0,150)))
with(df.all[df.all$group == 3,], points(RT ~ progesterone, ylim = c(0,500), xlim=c(0,150), pch=16))
dev.copy(pdf, "plottedSimData.pdf")
dev.off();

write.table(df.all, "simData.csv", col.names=T, row.names=F, quote=F, sep=",")

##' Function to convert a correlation matrix to a covariance matrix.
##'
##' The correlation matrix to convert can be either symmetric or triangular. The covariance matrix returned is always
a symmetric matrix.
##' @title Correlation Matrix to Covariance Matrix Conversion
##' @param cor.mat the correlation matrix to be converted
##' @param sd a vector that contains the standard deviations of the variables in the correlation matrix
##' @param discrepancy a neighborhood of 1, such that numbers on the main diagonal of the correlation matrix will
be considered as equal to 1 if they fall in this neighborhood
##' @export
##' @note The correlation matrix input should be a square matrix, and the length of sd should be equal to the
number of variables in the correlation matrix (i.e., the number of rows/columns). Sometimes the correlation matrix
input may not have exactly 1's on the main diagonal, due to, eg, rounding; discrepancy specifies the allowable
discrepancy so that the function still considers the input as a correlation matrix and can proceed (but the function
does not change the numbers on the main diagonal).
##' @author Ken Kelley (University of Notre Dame; (\email{KKelley@ND.Edu})) and Keke Lai (the
\code{MBESS} package), with modifications by Dustin Fife (\email{fife.dustin@gmail.com}).
cor2cov = function (cor.mat, sd, discrepancy = 0.00001)
{
  if (dim(cor.mat)[1] != dim(cor.mat)[2])
    stop("'cor.mat' should be a square matrix")
  n <- sqrt(length(cor.mat))
  if (n != length(sd))
    stop("The length of 'sd' should be the same as the number of rows of 'cor.mat'")

```

```

if (length(sd[sd > 0]) != n)
  stop("The elements in 'sd' should all be positive")
if (isSymmetric(cor.mat))
  IS.symmetric <- TRUE
else IS.symmetric <- FALSE
p <- dim(cor.mat)[1]
q <- p * (p - 1)/2
if (isTRUE(all.equal(cor.mat[lower.tri(cor.mat)], rep(0,
  q))) || isTRUE(all.equal(cor.mat[upper.tri(cor.mat)],
  rep(0, q))))
  IS.triangular <- TRUE
else IS.triangular <- FALSE
if (!IS.symmetric & !IS.triangular)
  stop("The object 'cor.mat' should be either a symmetric or a triangular matrix")
cov.mat <- diag(sd) %*% cor.mat %*% diag(sd)
colnames(cov.mat) <- rownames(cov.mat) <- colnames(cor.mat)
return(cov.mat)
}

```