

DOES TESTOSTERONE AFFECT SEXIST ATTITUDES IN MEN? A SIMULATED
ANALYSIS

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Abstract

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Sexism continues to negatively affect the lives of women across many cultures and modern societies. Although sexism has a damaging effect on people overall, women are disproportionately affected. Previous research on the topic generally explores attitudes, culture, socioeconomic status, sexual and violent crime census data, and developmental factors, but there is a lack of research investigating potential links among biological factors, such as hormone levels, and sexist attitudes. The present study used simulated data to simulate a study investigating the relationship between endogenous testosterone, cognitive aggression, and sexist attitudes. Using the (“faux”), (“pwr2ppl”), and (“ProcessR”) packages in R open-source statistical software, I utilized means and standard deviations from current literature to simulate and analyze computer generated data for ambivalent sexism, aggression, and endogenous testosterone. A simulated analysis was employed here because the COVID-19 pandemic rendered data collection impossible. Although it is not possible to draw conclusions about any actual relationship among these variables without actual data, this paper demonstrates how performing simulated analyses can be a useful tool in teaching, preliminary research, and conserving

time and financial resources. The findings also highlight the relevance and importance of investigating the relationship between endogenous testosterone and sexism.

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Introduction

Sexism

Sexism and sexual violence are an enduring problem across many cultures and societies. Much of the research on sexism generally explores attitudes, culture, socioeconomic status, sexual and violent crime census data, and developmental factors when researching the topic. There is, however, little information available on the biological factors that may affect sexist attitudes and behavior. Recent neuroendocrinological studies have linked hormones such as testosterone and cortisol to aggression more generally via decreased activity in the orbitofrontal cortex (OFC), which is associated with decision making and executive function (Mehta & Beer, 2010). This finding suggests higher levels of testosterone may be associated with decreased reasoning ability, impulse control, and increased aggressive behavior. Indeed, studies have shown that testosterone levels are positively correlated with aggressiveness, antisocial behavior, and domination behavior in animals (Archer, 1991; Book et al., 2001; van Wingen et al., 2011), but not in humans (Sapolsky, 1997). Because sexism may influence and predict aggression that is specifically directed at women and because sexism disproportionately affects women, it is important to investigate the potential hormonal modulators of sexist attitudes/behaviors. Gordon Allport wrote, “prejudice is an antipathy based upon a faulty and inflexible generalization,” (Allport, 1954, p. 53). Sexism is prejudice directed at members of a particular sex, typically women (Glick & Fiske, 1996). Sexism is harmful to both men and women, but much of the research on the topic shows that women are

disproportionately negatively affected by it (Chapleau, Oswald, & Russell, 2007).

For women, sexism has a negative impact on just about every aspect of life including starting salaries, pay raises, physical and mental health, interpersonal relationships, family life, job promotion, and other lucrative opportunities in the workplace. Sexism in the workplace can have a negative effect on women's earning power and impede opportunities for advancement. According to a study from 2010 on the gender-wage gap, women make roughly \$.79 on the dollar compared to men in the United States, but that gap widens significantly as women approach their thirties (Bertrand, Goldin, & Katz, 2010). Having a child has a negative impact on women's salaries, but men's salaries are unaffected. Women with children make about 20% less than men with children (Kleven, Landais, & Sjøgaard, 2018).

Simply experiencing sexism can also negatively impact the physical and mental health of women. Multiple studies have shown a positive relationship between perceived experiences of sexism and self-reported physical stress-related symptoms including nausea, headaches, depression, and gastrointestinal distress (e.g., Berg, 2006; Goldenhar, Swanson, Hurrell, Ruder, & Deddens, 1998; Landrine & Klonoff, 2001; Landrine, Klonoff, Gibbs, Manning, & Lund, 1995). For example, one study found that women who experienced chronic perceptions of sexism (e.g., women being rejected for a position in favor of a male confederate for sexist reasons) had a higher stress response (measured via levels of cortisol) than women that were rejected in favor of another woman (e.g. for merit-based reasons) (Townsend, Major, Gangi, & Mendes, 2013). The influence of

sexism on rates of sexual violence is also alarming. Some studies showed that negative beliefs related to gender and interpersonal violence were found to have a direct effect on their use of violence in their relationships. (Forbes, Adams-Curtis, & White, 2004; Hald, Malamuth, & Yuen, 2010; Reitzel-Jaffe & Wolfe, 2001).

Aggression is often used to influence, control, and/or illicit certain types of social behavior by men and women. Sexual aggression (i.e., sexist attitudes and sexual violence) is used to influence or coerce behavior (often of a sexual nature) and is typically exerted by men against women (EEOC, 2016). This pattern is striking in the statistics surrounding sexual violence. Women are more likely to be sexually assaulted. Nine out of ten rape victims are women. One out of every six women has been a victim of an attempted or completed rape in her lifetime (Tjaden & Thoennes, 1998). Additionally, women in college ages 18 to 24 are three times more likely to be victims of sexual violence than women in the general public. Women of this age group are four times more likely to experience sexual violence than women of other ages (DOJ, 2014). Sexism and sexual violence negatively effects women's mental health as well. Ninety-four percent of women who are raped experience PTSD symptoms after the event, 33% contemplate suicide, and roughly 13% attempt suicide. About 70% of sexual assault victims suffer from moderate to severe distress (DOJ, 2014).

Sexism is a multifaceted construct. Ambivalent sexism is a theoretical framework that conceptualizes sexism into two sub-categories: Hostile Sexism (HS) and Benevolent Sexism (BS). Hostile sexism is antipathy toward women based on a faulty and inflexible

generalization which is consistent with Allport's (1954) definition of prejudice.

Benevolent sexism is defined as, "as a set of interrelated attitudes toward women that are sexist in terms of viewing women stereotypically and in restricted roles but that are subjectively positive in feeling tone (for the perceiver) and also tend to elicit behaviors typically categorized as prosocial (e.g., helping) or intimacy-seeking (e.g., self-disclosure)," (Glick & Fiske, 1996, p. 491). The ambivalent aspect of this theoretical framework posits that men are often unaware that they harbor and/or display sexist attitudes and behavior. For example, traditional gender roles across cultures have maintained that men should be the breadwinners in heterosexual relationships so that they may "take care" of their partners. While this may seem as though the man is being a good provider and helping his partner, it simultaneously undermines the woman's own self-efficacy and self-esteem by suggesting that women need to be taken care of because they are not capable of taking care of themselves. Because benevolent sexism is a more prevalent form of sexism, it is important to include in the theoretical framework as a contrast to hostile sexism, which lies at the other end of the spectrum of social behavior and is much easier to identify.

Many studies on sexism focus on factors such as attitudes, gender roles, culture, and social dominance (e.g., Dill & Thill, 2007; Frese, Moya, & Megías, 2004; Glick & Fiske, 1996; Russell & Trigg, 2004), resulting in a limited amount of literature on the biological influences on these attitudes/behaviors. Both neurophysiology and endocrinology play a substantial role in social attitudes and social behavior (e.g., 2008;

Arana, et al., 2003; Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003; Rudebeck, Bannerman, & Rushworth), therefore it is important that studies begin to focus on the potential role of biological factors on aspects of sexism and sexual violence.

Aggression

Early studies on the neural correlates of aggression using rodent models identified the hypothalamus as one of the key regions involved in aggression (Brucke, Kaindl, & Mayer, 1952). Another study found that stimulating this region in cats has been shown to produce aggressive behavioral responses (Folkow & Euler, 1954). The hypothalamus is a key neuroendocrine center of the brain and has a high density of steroid hormone receptors, suggesting that hormonal fluctuations are likely to influence aggressive responses. Indeed, many studies show that androgen receptor (AR) density in this region (as well as in the amygdala, hippocampus, and frontal cortex) is linked to aggression (Sarkey, Azcoitia, Garcia-Segura, Garcia-Ovejero, & DonCarlos, 2008). AR density is related to the number of receptor sites available for steroid hormones to bind to. Greater AR density is associated with increased hippocampal activation and aggression as opposed to low AR density due to the increased number of sites that steroid hormones can activate. For example, recent research using rodent models has indicated that greater AR density is related to increased aggression. Researchers injected control (i.e., normal AR density) and feminized (i.e., AR deficient mice) with a cocktail of Anabolic-Androgenic Steroids (AAS), a compound that mimics testosterone. Feminized mice showed less aggressive

behavior than control group mice, suggesting that greater AR density seen in the control mice may be linked to increased aggression (Robinson, Penatti, & Clark, 2012).

Another recent study, which found that testosterone was associated with increased aggression following social provocation, has further implicated the frontal cortex as a key neural correlate of aggression (Mehta & Beer, 2010). This study used a testosterone administration paradigm and found that the effect of testosterone on aggression was associated with decreased activation in the medial OFC; a region implicated in decision-making and impulse control. The OFC is densely connected with the hypothalamus (Hirose et al., 2016). Because the OFC is associated with decision-making and impulse control, it is likely the key modulator on the effect of hypothalamus activation on aggressive behavior. The researchers suggested that testosterone may reduce activity in the OFC via inhibition of serotonin production. Low serotonin has been previously implicated in impulsivity and aggression, including aggressive behavioral reactions to social provocation. Androgens are also known to downregulate serotonin receptor mRNA and serotonin turnover in the frontal cortex. Low serotonin therefore may lead to hypometabolism in the medial OFC. These findings suggest that testosterone may increase aggression via serotonin deficiency in the medial OFC but would likely not have as dramatic an effect as administration of endogenous testosterone or the effects of sudden environmental changes such as physical threat.

A number of additional studies on humans have also suggested that testosterone is a key modulator of social aggression. There are a large number of correlational studies

linking testosterone to various forms of social aggression (Albert, Walsh, & Jonik, 1993; Archer, 1994; Olweus, 1986). For example, Harris and colleagues found that testosterone levels were positively associated with aggressive behavior and negatively associated with prosocial behavior (Harris, Rushton, Hampson, & Jackson, 1996). A recent meta-analysis of 45 independent studies that investigated the relationship between testosterone and social aggression reported a positive, albeit moderate relationship ($r = 0.14$), between these two variables (Book, Startzyk, & Quinsey 2001). Whereas these correlational studies suggest a positive relationship between androgens and aggression, they do not speak to the potentially causal effects of androgens, such as testosterone, on aggression. This may be related to several factors such as the effect of environmental cues and the outcome a social or physical confrontation. For instance, winning a fight or sports competition can elevate testosterone levels, but the relationship between testosterone and behavior seems to be bidirectional.

Testosterone

A handful of studies report that administration of exogenous testosterone has been linked aggression or similar behaviors in men, speaking more directly to the issue of causality (Connor, Archer, Hair, & Wu, 2002; Carré, Geniole, Ortiz, Bird, Videto, & Bonin, 2017; Mehta & Beer, 2010). More specifically, one study found an increase in aggressive behavior for men with high-risk personality scores when nasally injected with testosterone gel over placebo. This effect was more pronounced in men with low cytosine-adenine-guanine (CAG) repeats in exon 1 of the androgen receptor (AR) gene.

(Geniole, et al, 2019) Another study found that administration of exogenous testosterone increased men's perception of their own physical dominance. Physical dominance can impact one's own sense of physical strength and perceived ability to successfully carry out aggressive behavior. Increased perceptions of one's own physical dominance could lead individuals to reassess their own social rank and become more aggressive toward lower ranking members of the group. This effect was significantly higher in men with low baseline testosterone (Welling, Moreau, Bird, Hansen, & Carré, 2016). Similarly, Mehta and Beer (2010) found that testosterone administration was associated with increased aggression following social provocation in men. Despite this collection of studies that show exogenous testosterone may be related to aggressive behavior, evidence from non-human primates suggests that natural fluctuations of testosterone may not predict actual aggression, (Sapolsky, 1997). When a mid-ranking, male chimpanzee is administered large amounts of exogenous testosterone, he will become more aggressive toward similar and lower ranking members, but he will not direct his aggression toward the alpha chimps (Sapolsky, 2015). With natural levels of testosterone, the chimp will resume his normal place in the pecking order, but his aggression will not be significantly exaggerated toward lower ranking members of the group, (Mehta & Beer, 2010). Given the established link between testosterone and aggression, it is possible that testosterone may influence sexism. Sexism can be considered a form of social aggression that is specifically directed towards women, by men, to coerce, intimidate, and control females and control their behavior. Investigating the role of hormones in sexist attitudes/behaviors will provide a greater understanding of sexism and sexual violence and could offer

potential insights into future treatments, therapies, and workshops that address these issues. The enduring threat that sexism and sexual violence present to women's health, safety, wage and social equality, and personal freedom demand vigorous, innovative, and more dynamic investigations into this problem. Because high levels of testosterone have been associated with higher levels of aggression (Dabbs & Morris, 1990), and sexism is a form of aggression (Glick & Fiske, 1996), examining the role of testosterone in sexist behavior and sexual violence is imperative.

The Current Study

The aim of this study was to examine the relationship between endogenous testosterone levels in men, cognitive aggression, and sexist attitudes (both hostile and benevolent). In particular, I examined whether there is 1) a relationship between testosterone and ambivalent sexism or cognitive aggression and sexism; 2) whether there is a difference in the relationship between testosterone and hostile sexism and benevolent or cognitive aggression and hostile sexism and benevolent; 3) whether cognitive aggression mediates the relationship between testosterone and sexism. For hypothesis 1, I predict that there will be a positive relationship between testosterone hostile sexism and benevolent sexism. I also predict there will be a positive relationship between cognitive aggression and hostile sexism and benevolent sexism. For hypothesis 2, I predict there will be a stronger relationship between testosterone and hostile sexism than testosterone and benevolent sexism. For hypothesis 3, I predict cognitive aggression will mediate the relationship between testosterone and sexism.

Although I had originally intended to measure salivary testosterone levels, cognitive aggression, and sexism in a sample of 171 men, data collection was not possible due to the COVID-19 pandemic. Due to the emergence of the COVID-19 virus, the seriousness of viral transmission risk, illness, and a significant mortality rate associated with this novel disease, the previously proposed method of testosterone measurement has been rendered impossible given the current state of affairs and risk such data collection could present to public health and safety. Given this predicament, it was necessary to devise a learning exercise to create a hypothetical examination of the relationship between testosterone, aggression, and sexism. Although this process would not allow me to make any real assumptions about the results, it provides some of the experience of conducting experiments and analyses. Using R Studio open-source statistical software, and means and standard deviations from current research data, I was able to construct simulated data and create a mediation model assessing these relationships. Although these findings are based on simulated data, some are significant, but have no real meaning because the data are strictly hypothetical in nature.

Method

Data Simulation

Data simulation is a useful tool in research and education. It is a research method widely used to generate hypothetical data based on means, standard deviations, and effect sizes reported by other studies, or hypothetical inputs. R open-source statistical analysis software offers a growing number of coding ‘packages’ that can generate simulated data for use in many types of research. R code is used to generate data, perform a variety of statistical analyses, and visualize the results via the use of graphs and tables. Data simulation allows researchers and students to make reasonable assumptions about the results of proposed experiments and analyses, run multiple analyses to help determine optimal sample size and parameters, observe how data behaves, make reasonable assumptions about how it may behave in future studies, and determine the best method and analysis for future research. Data simulation also forces one to consider the numbers and distributions associated with those numbers in their current study and consider which type of tests to perform as well as offers the opportunity to learn how to code, which is an increasingly valuable skill in research and the workplace.

The (“faux”) package in R generates hypothetical data from inputs for mean, standard deviation, effect size, and number of observations. Faux uses these inputs to simulate and label three sets of data within given parameters. Inputs can be used from previous research or created on the spot to observe hypothetical scenarios and analyses. Faux also allows researchers to specify correlations via four methods: a single r for all

pairs, a variable-by-variable matrix, a vars* vars length vector, and a vars*(vars-1)/2 length vector. All correlations are presented in matrix tables. The simulated data file is displayed in a list of observations for each variable.

Participants

The sample size recommended to detect an $r = 0.21$, if $\alpha = 0.05$ and power level is set at 80% was 171 (Schultheiss & Metha, 2018). For this reason, the sample size for the simulated data used below was set to $n = 171$.

Measures

Cognitive aggression would have been measured using a sentence completion task. This task involves completing a word with missing letters that can be completed to for an aggressive or non-aggressive manner (e.g., _a__le can be completed as battle or saddle). For the simulated data, the mean (4.85) and standard deviation (.86) for this task were taken from (Vandello, Bosson, Cohen, Burnaford, & Weaver, 2008), who measured cognitive aggression in a sample of $n = 41$ men.

Sexist attitudes would have been measured via questionnaire using the Ambivalent Sexism Inventory (ASI; Glick & Fiske, 1996). The ASI, across six studies on 2,250 respondents established convergent, discriminant, and predictive validity. The ASI uses two positively correlated components of sexism that represent opposite evaluative orientations toward women: Hostile Sexism and Benevolent Sexism. Items include “Feminists are making entirely reasonable demands of men,” and “Most women interpret innocent remarks or acts as being sexist.” Responses are measured using a 6-point Likert

scale ranged from 0 (disagree strongly) to 5 (agree strongly). For the simulated data, the mean (2.64) and standard deviation (0.89) for the hostile sexism subscale and mean (2.11) and standard deviation (0.85) for the benevolent sexism subscale of the ASI were taken from (Glick & Fiske, 1996). All means and standard deviations were averaged from men's scores in the study.

Hormone Sampling & Analysis

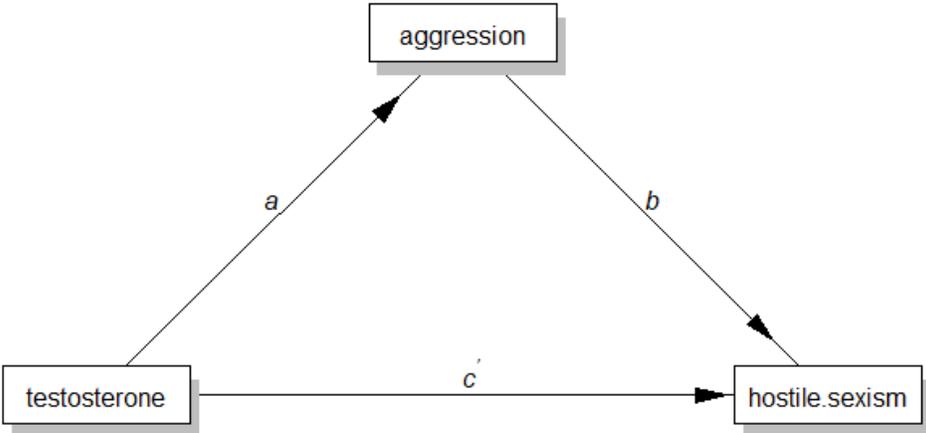
For the simulated data, the mean (177.5) and standard deviation (42.2) of salivary testosterone were taken from (Torrance, Hahn, Kandrik, DeBruine, & Jones, 2018), who collected saliva samples multiple times in a 5-week span using the passive drool technique from a sample of $n = 172$ men.

Data Analysis

All data analysis was done using R. Data simulation was done using the faux package (package = "faux", DeBruine, 2020). Mediation analyses (see Figure 1) were used to determine the relationship between endogenous testosterone levels and the two types of ambivalent sexism (hostile and benevolent subscales) with cognitive aggression (gender conforming or threatening) as the mediator. See Appendix A for the full R code used for this data simulation and analysis.

Figure 1

Mediation Model for Testosterone, Aggression, and Sexism

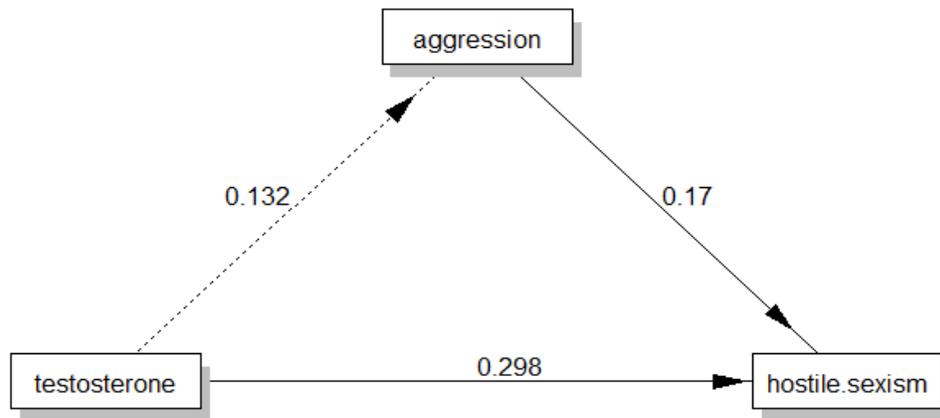


Results

Mediation analysis of simulated showed a hypothetical direct effect of testosterone on hostile sexism, ($z = 4.148$, $S.E. = 0.002$, $p < .001$), and benevolent sexism, ($Z = 4.196$, $S.E. = 0.002$, $p < .001$). There was also a hypothetical relationship between cognitive aggression and hostile sexism, ($z = 2.364$, $se = 0.07$, $p = .018$), and benevolent sexism, ($z = 2.239$, $se = 0.07$, $p = .025$). There was no hypothetical relationship between testosterone and aggression, ($z = 1.746$, $se = 0.002$, $p = .081$). There was no hypothetical indirect effect of testosterone on hostile sexism, ($z = 1.404$, $se < 0.001$, $p = .16$). See Figures 2 and 3.

Figure 2

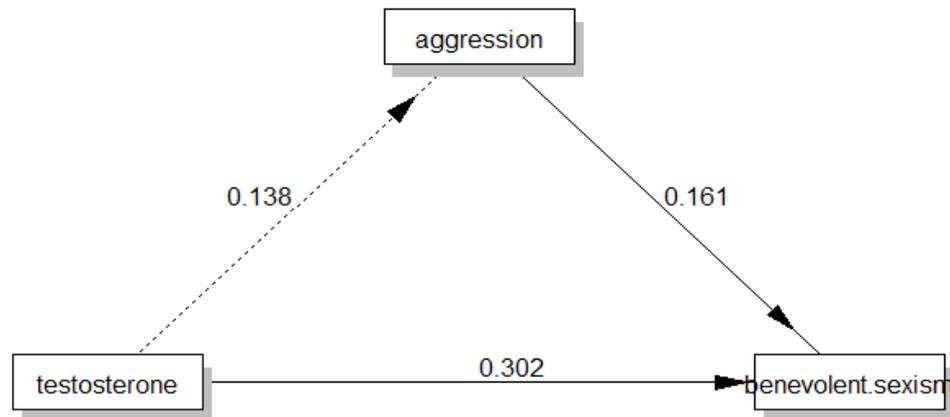
Mediation Analysis of Simulated Data for Testosterone, Aggression, and Hostile Sexism



Note. Mediation analysis of simulated data indicates a hypothetical relationship between testosterone and hostile sexism with cognitive aggression as a mediator. a) Results indicate there was no hypothetical relationship between, a, testosterone and aggression, ($z = 1.746$, $se = 0.002$, $p = .081$). b) There was a hypothetical relationship, b, between aggression and hostile sexism, ($z = 4.148$, $S.E. = 0.002$, $p < .001$). c) There was a hypothetical direct effect, c', of testosterone on hostile sexism, ($z = 2.364$, $se = 0.072$, $p = .018$). There was no hypothetical indirect effect, c, of testosterone on hostile sexism, ($z = 1.404$, $se = 0.001$, $p = .16$). Solid lines indicate a significant relationship, $p > 0.05$. Dotted lines indicate the relationship is not significant.

Figure 3

Mediation Analysis of Simulated Data for Testosterone, Aggression, and Benevolent Sexism



Note: Mediation analysis of simulated data indicates a hypothetical relationship between testosterone and benevolent sexism with aggression as a mediator. a) Results indicate there was no hypothetical relationship between, a, testosterone and aggression, ($z = 1.746$, $se = 0.002$, $p = .081$). b) There was a hypothetical relationship between, b, aggression and benevolent sexism, ($z = 2.239$, $se = 0.070$, $p = .025$). c) There was a hypothetical direct effect, c') of testosterone on benevolent sexism, ($z = 4.196$, $se = 0.002$, $p < .001$). There was no hypothetical indirect effect, c, of testosterone on hostile sexism, ($z = 1.415$, $se = 0.001$, $p = .157$) Solid lines indicate a significant relationship, $p > 0.05$. Dotted lines indicate the relationship is not significant.

Discussion

The purpose of this study was to examine whether there is 1) a relationship between testosterone and ambivalent sexism or cognitive aggression and ambivalent sexism, and 2) whether there is a difference in the relationship between testosterone and hostile sexism and benevolent or cognitive aggression and hostile sexism and benevolent, and 3) whether cognitive aggression mediates the relationship between testosterone and sexism. For hypothesis 1, I predicted that there would be a positive relationship between testosterone hostile sexism and benevolent sexism. I also predicted there would be a positive relationship between cognitive aggression and hostile sexism and benevolent sexism. For hypothesis 2, I predicted that there would be a stronger relationship between testosterone and hostile sexism than testosterone and benevolent sexism. For hypothesis 3) I predicted cognitive aggression would mediate the relationship between cognitive aggression and sexism.

Because the COVID-19 pandemic rendered actual data collection impossible, this study used simulated data to generate the analyses that would be necessary to test these hypotheses. Days before collection of biological samples and survey data for the current study was to begin, Governor Gavin Newsome imposed a stay-at-home order for all of California mandating many businesses to close or limit operations, suspend face-to-face learning in schools, wear protective personal equipment, and remain physically and socially distant from others. The impact of COVID-19 has limited the types of data that can be safely collected which restricts many branches of research. The use of data

simulation creates an alternative method for conducting hypothetical research in lieu of collecting real data when real-world threats, such as the COVID-19 pandemic and the subsequent restrictions it has imposed on the lives of people, make data collection difficult or impossible. Additionally, data simulation offers an alternative way to perform analyses that can inform researchers prior to real data collection.

The hypothetical findings for hypothesis 1 suggest that biology and cognition influence sexist attitudes. This would be significant because the result would suggest that these factors may influence other attitudes, such as racial prejudice or ageism. Additionally, if these findings came from real data findings could have some implications for hormone replacement therapy and other medical treatments that involve the use of exogenous testosterone because some studies have shown that administration of exogenous testosterone has been shown to increase aggression in human and nonhuman primates (Sapolsky, 1997; Carré, Geniole, Ortiz, Bird, Videto, & Bonin, 2017). Therefore, administration of exogenous testosterone may also influence sexist attitudes if these findings were to emerge from real data. The hypothetical relationship between cognitive aggression and sexism would be interesting because it would support previous findings on aggression and sexism, but also because the potential influence of testosterone on sexist attitudes may address some of the variance in some research (Fox & Potacki, 2015; Cross, Overall, Hammond, & Fletcher, 2017). Hypothesis 2 was not supported. If real data had shown this finding, it might suggest that testosterone may have an equal effect on hostile and benevolent sexism. Hypothesis 3 was not supported. If this

finding were based on real data, this would be interesting because the lack of a relationship between endogenous testosterone cognitive aggression supports previous research on the topic (Sapolsky, 1997).

The obvious limitation of this study is that it is based on simulated data using means and standard deviations from previous research rather than real data collection due to the risks associated with saliva collection and analysis during the current pandemic. Ideally, real data is preferable to simulated data, but the risks of contracting and transmitting COVID-19 prevents in-person surveys. Another limitation is that these simulated data come from real data in different sample populations. This violates assumptions about random samples in regression designs. The unforeseen benefit of conducting research during the COVID-19 pandemic is that it challenges researchers to find novel ways to do research to mitigate some of the problems the current and future situations may present. The use of simulated data provides an alternative as a learning tool to demonstrate how data can be analyzed. Other limitations include the potential effects of genetics that may interact with the relationship between testosterone and aggression (Carré, Geniole, Ortiz, Bird, Videto, & Bonin, 2017) and a lack of previous research on the topic.

The findings in this study indicate the need for further research in this area. It supports previous research and provides novel insights into the potential relationship between hormones and attitudes. Future research should focus on the relationship between testosterone and other attitudes, such as racial prejudice, while continuing to

examine its relationship with sexist attitudes. These findings also serve to inform the medical community and the potential effects of hormone replacement therapy and other hormone related treatments.

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Appendix

R code

```
knitr::opts_chunk$set(echo = TRUE)

library(car)

library(pwr2ppl)
library(mvnormttest)
library(MASS)
library(dplyr)

library(tidyr)
library(lavaan)

med(rxm1 = .32, rxm2 = 0, rxm3 = 0, rxm4 = 0, rxy = .21, rym1 = .32, rym2 = 0, rym3 = 0,
    rym4 = 0, rm1m2 = 0, rm1m3 = 0, rm1m4 = 0, rm2m3 = 0, rm2m4 = 0,
    rm3m4 = 0, alpha = 0.05, mvars = 1, n = 171)

library(faux)

library(processR)

set.seed(0)
dat <- rnorm_multi(
  n = 171,
  mu = c(177.5, 4.85, 2.64),
  sd = c(42.2, .86, .89),
  r = c(0.21, 0.3, 0.3),
  varnames = c("testosterone", "aggression", "hostile.sexism"),
  empirical = FALSE
)

sort(dat$testosterone)

labels=list(X="testosterone",M="aggression",Y="hostile.sexism")
statisticalDiagram(4, labels = labels)

moderator=list(name="aggression",site=list(c("testosterone","hostile.sexism")))
model=tripleEquation(X="testosterone",M="aggression",Y="hostile.sexism",moderator=moderator)
covar=list("aggression")
cat(model)
```

```
covar  
semfit=sem(model=model,data=dat)  
summary(semfit)  
estimatesTable(semfit)  
statisticalDiagram(4,labels=labels,fit=semfit,whatLabel="std")
```