Abstract

Extended sexuality (ES) refers to non-oestrus sexuality. In humans, ES is posited to enable women to secure non-genetic resources from males (Rodríguez-Gironés & Enquist, 2001) and to be regulated by progesterone as it is highest during the non-fertile luteal phase of the menstrual cycle (Grebe, Gangestad, Garver-Apgar, & Thornhill, 2013). Research on the hormonal regulation of women's sexuality has tended to view ES as an extension of fertile-phase sexuality, rather than being distinct. This study was a replication of Grøntvedt, Grebe, Kennair, and Gangestad (2016) of women on hormonal contraceptives as exogenous progesterone levels mimic those of the luteal phase. We used a longitudinal design consisting of two surveys with two months interval and ran linear mixed model analyses on women using hormonal contraceptives in committed relationships who were neither pregnant, breastfeeding nor having recently given birth (*n* = 409 observations from 271 women). In addition to the original investment measure of Loyalty and Faithfulness (LF), analyses were run with three other relationship investment measures: Partner-Specific Investment Inventory (PSII) (Ellis, 1998), Perceived Relationship Quality Components (PRQC) (Fletcher, Simpson, & Thomas, 2000) and the Attachment Features and Function Scale (Tancredy & Fraley, 2006). We hypothesised that higher levels of progestin in combination with high investment would lead to a higher frequency of sex. We found support for this hypothesis with the PSII, PRQC and attachment bond, but we did not replicate these observations with LF. We also hypothesised that sexual frequency would increase partner investment over time which we examined with a post-test regression on *n* = 140 respondents. Sexual frequency did not significantly predict partner investment levels over time, but rather earlier partner investment and changes in women's investment did. Our findings shed light on the use of synthetic progestins in behavioural research as well as which investment types are relevant to extended sexuality.

Keywords: extended sexuality, male-assistance hypothesis, progesterone, hormonal contraceptives, synthetic progestins, relationship investment

Sammendrag

Utvidet seksualitet (US) refererer til all seksualitet som oppstår utenfor den fruktbare fasen. Hos mennesker tenkes det at US muliggjør det for kvinner å sikre ikke-genetiske ressurser fra menn (Rodríguez-Gironés & Enquist, 2001) og er regulert av progesteron, da det er høyest i den ikke-fruktbare lutealfasen av menstruasjonssyklusen (Grebe et al., 2013). Forskning på hormonell regulering av kvinners seksualitet har hatt en tendens til å se på US som en forlengelse av seksualitet i den fruktbare fasen, snarere enn sin egen distinkte type. Denne studien var en replikasjon av Grøntvedt et al. (2016) på kvinner på hormonelle prevensjonsmidler da eksogene progesteronnivåer etterligner de i lutealfasen. Vi brukte en langsgående design bestående av to undersøkelser gitt ut med to måneders mellomrom og gjennomførte lineære mixed modellanalyser på kvinner som bruker hormonelle prevensjonsmidler i engasjerte forhold som verken var gravide, ammende eller nylig hadde født (*n* = 409 observasjoner fra 271 kvinner). I tillegg til det opprinnelige investeringsmålet Loyalty og Faithfulness (LF), ble det kjørt analyser med tre andre forholdsinvesteringsmål: Partner-Specific Investment Inventory (PSII) (Ellis, 1998), Perceived Relationship Quality Components (PRQC) (Fletcher et al., 2000) og Attachment Features and Function Scale (Tancredy & Fraley, 2006). Vi forventet at høyere nivåer av progestin i kombinasjon med høye investeringer vil føre til en høyere frekvens av sex. Vi fant støtte for denne hypotesen med PSII, PRQC og tilknytning, men vi repliserte ikke observasjonene med LF. Vi forventet også at seksuell frekvens ville øke partnerens investering over tid, som vi undersøkte ved å utføre en post-test regresjon på *n* = 140 respondenter. Seksuell hyppighet forutså ikke partnerens investeringsnivå betydelig over tid, men snarere partnerens tidligere investering og endringer i kvinnens investering forutså det. Våre funn kaster lys over bruken av syntetisk progestin i atferdsforskning, samt hvilke investeringstyper som er relevante for utvidet seksualitet.

Nøkkelord: utvidet seksualitet, male-assistance hypothesis, progesteron, hormonelle prevensjonsmidler, syntetisk progestin, investering i forhold

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Introduction

Oestrus sexuality is the most common form of mating in the animal kingdom as sexual behaviour occurs solely when females are fertile. In these species, male mating behaviour is triggered by oestrus females through a variety of sensory cues such as pheromones (Trotier, 2011), stereotypical behaviour patterns (Pfaff, Frohlich, & Morgan, 2002) and swellings of the genitalia (Domb & Pagel, 2001; Pagel, 1994). Mating is costly: energy is redirected from looking for food to pursuing a mate and there is an increased risk of injury and illness through contact with rivals, mates, and predators (Thornhill & Gangestad, 2008). Fertilised females need to bear the energetic costs and heightened vulnerability that come with egg production, pregnancy, brooding, lactation and caring for offspring (Trivers, 1972). Despite the high costs that come with mating, the fitness benefits of reproduction makes it worthwhile.

Extended sexuality

Extended sexuality (ES) refers to sexuality that occurs outside of a female's fertile phase when females are not only sexually receptive, but also proceptive. Typically, in species which engage in ES, non-oestrus mating occurs when females are neither pregnant nor nursing. However, humans exhibit continuous extended sexuality as they are capable of having sex throughout their sexually mature lives: from adolescence when ovulation is unreliable, throughout the menstrual cycle, during pregnancy, lactation until postmenopause (Goyette & Craton, 2013). Because extended sexuality bears many of the same costs as oestrus sexuality, without reproduction; its function, characteristics, and the mechanisms which regulate it are of interest to researchers.

Mating systems refer to the sex-specific behaviours required to attract mates and the division of labour in infant care and this is typically reflected in the degree of sexual dimorphism. In species with high sexual dimorphism, males expend more effort into mating, but their reproductive effort is limited to their genetic material as their fitness is increased by fertilising as many females as possible (Alexander, Hoogland, Howard, Noonan, & Sherman, 1979; Sapolsky, 2011). This mating effort can take the form of extensive mate-guarding of a harem of females and engaging in aggressive intrasexual competition to obtain the right to mate and they do not copulate the same female frequently. Females invest little in finding and securing a mate, but are the main reproductive investors as they are the sole caretakers of resulting offspring (Alexander et al., 1979; Thornhill & Gangestad, 2008). Due to the high levels of intrasexual competition between males in more sexually dimorphic species, new dominant males may cull the infants of their predecessor so that females can enter oestrus more quickly (Palombit, 2015). However, females have evolved ways of preventing or recouping these losses in their reproductive investment such as post-conceptive mating (Vayro, Ziegler, Fedigan, & Sicotte, 2015) or by mating with multiple males so as to create paternity confusion (Hrdy, 1979). These adaptations are forms of antagonistic coevolution. Traits that increase the reproductive fitness of one sex can reduce that of the other. Therefore, the sexes must continuously evolve adaptations to counter those of the other sex. This is the mechanism by which phenotypic differences in the sexes arise (Perry & Rowe, 2015).

The selection pressures in human evolution which led to bipedalism, increased brain size as well as the transition from foraging to hunting brought about changes in the human mating system (Benshoof & Thornhill, 1979). Human infants are extremely altricial and pregnancy and nursing are calorically demanding and especially vulnerable states in women (Benshoof & Thornhill, 1979; Emera, Romero, & Wagner, 2012; Geary, 2000; Goyette & Craton, 2013). Women are dependent on the assistance of others during childrearing to assure their own and their infants' survival and as reproductive success is more dependent on women, evolutionary pressures have acted more strongly on women's biology to promote this (Trivers, 1972). Rodríguez-Gironés and Enquist (2001) propose a male-assistance hypothesis of human extended sexuality that enables women to obtain non-genetic resources from the males they sexually engage with, as parents share the greatest level of kinship with their offspring. This is proposed to stem from the female fitness-enhancing adaptation of concealed ovulation which consists in the absence of physiological and behavioural signs of oestrus in females. To mask the presence of fertility, physical sexual signals as well as the ability and propensity for sexual intercourse is extended throughout the cycle and lifetime after puberty.

Concealed ovulation is thought to have phylogenetically preceded and contributed to the appearance of monogamy in our species (Sillén-Tullberg & Moller, 1993). As women's fertility status is unknown and conception is only possible on a few days per cycle, humans must mate frequently. This is time-consuming and reduces the amount of energy men can devote to securing sexual access to other women (Rodríguez-Gironés & Enquist, 2001). If a man leaves his partner, she may engage in extrapair copulation or be victim of sexual coercion and any resulting offspring are not guaranteed to be his and resources spent on an infant that is not his own do not contribute to his fitness (Thornhill & Gangestad, 2008). It is not in women's interest for a man to mate with multiple women either, as then partner resources will be split between different women and their children, than being invested into the couple's shared children (Thornhill & Gangestad, 2008). The non-genetic resources of food, protection and childcare came at a price: men obtained paternity assurance through exclusive sexual access (Buss & Schmitt, 1993). Greater access to resources allows the woman to ovulate more frequently, reducing the interpregnancy interval resulting in more children thereby increasing the fitness of the couple (Marlowe, 2001). Having multiple children with one woman rather than multiple women entails less time spent mate-guarding and more efficient division of resources among offspring leading to better outcomes for those that adopted this strategy than those adopting a polygynous or serially monogamous mating strategy (Francesconi, Ghiglino, & Perry, 2016). Additionally, this would contribute to the development of more complex societies as there would be fewer conflicts between men due to decreases in intrasexual competition as well as foster intersexual cooperation between couples (Geary, 2000).

The increased selection pressures placed on couples to collaborate rather than engage in intersexual competition is thought to have led to the evolution of attachment and pair-bonding within couples. This type of romantic pair-bonding is therefore proposed to be an exaptation i.e. a system that originally evolved for one purpose, but then serves another. The original parent-child pair-bonding mechanisms were being applied to couples (Eastwick, 2009; Hazan & Zeifman, 1999). The degree of attachment is what would distinguish a short-term sexual partnering from a long-term one. Attachment bonds are characterised by proximity seeking, separation distress, as well as seeing the attachment figure as a source of support and a secure base (Bowlby, 1969) and parental and romantic love are underlain by the same neural networks (Carter, 1998). Ellis (1998) proposes that

such a psychological mechanism allows partners to gain access to and maintain their partner's resources. These resources can be tangible such as provisioning, protection, parental nurturance, and sexual access, or they can be symbols of future investment. The importance of these evolutionarily meaningful investments varies according to the sex of the partner and the ability of an investment to satisfy them differs according to whether it is tangible or symbolic. Sexual access would depend on previous male investment, but future male investments would also depend on how sexually accessible a woman makes herself to her partner (Benshoof & Thornhill, 1979). Monogamy would go on to be enshrined as marriage, officialising these mutual commitments between partners and is found in all human societies (Buss & Schmitt, 1993).

Extended sexuality is often presented within the context of the dual mating hypothesis. According to this theory, posited by Pillsworth and Haselton (2006), women's mating psychology varies throughout the menstrual cycle. During the fertile phase, women are more likely to be biased towards men bearing high genetic quality i.e. good genes, whereas during the non-fertile phase, women are biased towards men showing signs of being a good parent and a willingness and ability to invest in the relationship. Therefore, unless a woman's long-term partner possessed both good genes and investment potential, she would be more inclined to engage in uncommitted extrapair sex with a high genetic quality man during her fertile phase. However, this would only be worthwhile if the costs outweigh the benefits such as there being low risk of getting caught, the ability to find another long-term mate, or if the quality of attachment or investment is low (Buss, 1988; Eastwick & Finkel, 2012; Gangestad, Thornhill, & Garver-Apgar, 2005). Men of high genetic quality would have likely been predisposed to pursuing a short-term mating strategy allowing them to mate with multiple women rather than committing to a long-term bond due to their increased mating opportunities (Pillsworth & Haselton, 2006). However, because women are reliant on a long-term male partner to provide for them and their offspring, those who favoured long-term relationships had higher reproductive success regardless of whether their partner was of high genetic quality or not (Thornhill & Gangestad, 2008). The implications of the dual sexuality hypothesis are that extended sexuality is not merely a diluted expression of oestrus sexuality, but qualitatively distinct and regulated by different hormonal processes (Grebe, Thompson, & Gangestad, 2016).

Life history theory

According to life history theory, the ontogenetic stages of an organism's life are characterised by different patterns of investment in growth, reproduction and survival. Organisms cannot afford to invest in these three dimensions equally throughout every stage as resources are finite, so trade-offs must be made. These differences are reflected in specific morphological, physiological, and behavioural changes in the rate, timing, and environment in which they occur. Patterns are similar across a species, but there are also individual differences. Both factors affect fitness as these respective levels (Dillon, Adair, Wang, & Johnson, 2013; Welling & Shackelford, 2019).

Hormones regulate multiple traits simultaneously and thus influence the expression of life history traits by mediating these trade-offs in investment (Roney, 2016; Welling & Shackelford, 2019). According to the organisational-activational hypothesis, exposure to gonadal hormones at different critical or sensitive periods throughout the lifetime cause irreversible changes to the phenotype through their effect on the morphology of tissues and the nervous system and consequently their

function, as well as adjust the metabolism and neural sensitivity to steroid hormones (Arnold, 2009; Arnold & Breedlove, 1985). These organisational effects will affect how the body responds to the activational effect of circulating hormones which are reversible and allow the organism to rapidly respond to changes in the physical and social environment. Due to their effects on fitness, hormonal traits would be naturally or sexually selected for and thus, they are of interest for the fields of behavioural endocrinology and evolutionary psychology (Roney, 2015).

The menstrual cycle

The menstrual cycle is one such example of the hormonal regulation of investment. It is composed of three phases: follicular, ovulation and luteal. The follicular phase begins with the onset of menstrual bleeding and ends upon ovulation. At the beginning of the follicular phase, oestradiol and progesterone levels are low and follicle-stimulating hormone (FSH) levels secreted by the anterior pituitary gland rise prompting the release of oocyte-containing follicles. Once the follicle has matured, it secretes oestradiol provoking the thickening of endometrial mucosa and the release of the anterior pituitary luteal hormone (LH) which spikes about two days before ovulation. Oestradiol, LH and FSH reach their peak and descend sharply bringing about ovulation as the follicle ruptures releasing the oocyte. Conception is most probable in this periovulatory phase. The luteal phase begins when the empty follicle transforms into the corpus luteum which secretes progesterone and causes further thickening of the endometrium in preparation for implantation. If fertilisation does not occur, the corpus luteum disintegrates causing progesterone to fall and oestradiol levels continue their descent leading to menstruation which is the shedding of the unused endometrium (Mihm, Gangooly, & Muttukrishna, 2011; Reed & Carr, 2018).

Most mammals have an oestrus cycle rather than a menstrual cycle and this shares a similar mechanism to human follicular phase. In these species, endometrium formation only occurs if there has been implantation. Therefore, the luteal phase can be compared to the early stages of pregnancy as menstruation and parturition share a similar mechanism (Emera et al., 2012; Pavlicev & Norwitz, 2018).

Women's mating psychology and its hormonal influences

In humans, the concepts of sexual desire, arousal and motivation are often used interchangeably in the research literature, but interpretation of these terms is not as straightforward and varies according to sex. Exposure to sexual stimuli prompts an automatic vaginal response even if the scenes do not evoke subjective sexual desire in women, therefore genital blood flow is not as reliable an indicator of arousal for women as it is for men (Bossio, Suschinsky, Puts, & Chivers, 2014; Chivers, Seto, Lalumiere, Laan, & Grimbos, 2010). Women may not consciously experience spontaneous sexual desire as frequently as men, but the automatic vaginal response may allow them to be more receptive to advances by their partner or to act upon non-sexual motivations such as desires for intimacy (Bancroft & Graham, 2011). This "arousability" in contrast to arousal, would be dependent on external stimuli, and the propensity to pay attention to and act on these stimuli is hormonally moderated (Bancroft & Graham, 2011; Whalen, 1966).

Subjective sexual desire appears to be under hormonal control as research into menopausal, ovariectomised and naturally cycling women have shown it to rise with supplementation of exogenous oestradiol and testosterone or following natural increases in their endogenous variants (Bancroft & Graham, 2011; Roney & Simmons, 2013; Roney & Simmons, 2016; van Stein, Strauß, & Brenk-Franz, 2019). While testosterone seems to play a crucial role in men's arousal and desire, its role in women's remains unclear. Possible explanations are that women may vary in their sensitivity to testosterone, therefore women with a higher sensitivity may only require smaller dosages to experience desire (Bancroft & Graham, 2011; Elaut et al., 2012). In addition to this, because testosterone is aromatized into oestrogen in the brain, distinguishing whether effects on sexual desire result from testosterone or aromatisation into oestrogen are challenging (Welling & Shackelford, 2019). Additionally, the peak in testosterone in the menstrual cycle coincides with that of oestradiol, therefore hormone sampling may undermine oestradiol's effect which could lead to conclusions that women are an exception among female mammals for whom oestradiol is the primary determinant of sexual motivation and behaviour (Wallen, 2013).

In a study of daily salivary hormone assessments of naturally cycling women, the greatest variation in desire was observed within-women within-cycle. Oestradiol levels from two days earlier had a significant positive effect on sexual desire whereas, progesterone from hours before consistently lead to decreased sexual desire (Roney & Simmons, 2013). Cycles characterised by higher levels of oestradiol and consequently progesterone, are more fertile, yet, no within-women between-cycle effects were found indicating that the midcycle peak in desire in higher oestradiol cycles is not higher than in less fertile cycles. As with testosterone, it is possible that that the necessary amount of oestradiol required for female sexuality is low or sensitivity may vary, and above threshold levels could be inconsequential (Sanders & Bancroft, 1982).

While progesterone has been consistently associated to decreased levels of sexual desire (Arslan, Schilling, Gerlach, & Penke, 2018; Jones et al., 2018; Roney & Simmons, 2013; Shirazi et al., 2019) and, to decreases in sexual behaviour in primates (Hill, 1988), sexual behaviour in humans seems to be equally spread throughout women's menstrual cycle indicating that motives other than desire lead to sex and are more important in determining overall sexual frequency (Brewis & Meyer, 2005; Caruso et al., 2014; Hill, 1988; Sheldon, Cooper, Geary, Hoard, & Desoto, 2006).

Motives for sex vary greatly and are influenced by, amongst others, sex, culture, sexual strategy, life stage, relationship quality and attachment. Sociosexuality refers to individual differences in willingness to engage in sexual relations without closeness, commitment, and other indicators of emotional bonding (Simpson & Gangestad, 1992). In individuals with a more unrestricted sociosexuality, the relationship between investment and sexual frequency is not as pronounced as in those with a restricted sociosexuality who tend to seek out partners exhibiting a greater willingness to invest in a relationship as well as exhibiting more of these traits themselves and use sex as a means to increase their bond (Sheldon et al., 2006). Men across cultures with varying levels of egalitarianism between sexes typically have a more unrestricted sociosexuality (Kennair, Grøntvedt, Mehmetoglu, Perilloux, & Buss, 2015). Individuals with an unrestricted sociosexuality are more likely to endorse pleasure or mood-enhancing motives (Grøntvedt, Kennair, & Mehmetoglu, 2015; Meston & Buss, 2007; Schmitt, 2005; Sheldon et al., 2006; Simpson & Gangestad, 1991), conversely, motives for sex in long-term relationships are similar for both sexes (Kennair et al., 2015). Women with a restricted sociosexuality endorsed pleasure motives mostly within the confines of a committed

relationship (Carroll, Volk, & Hyde, 1985) and facility to attain orgasm with a partner does factor into partner choice (Coria-Avila, Herrera-Covarrubias, Ismail, & Pfaus, 2016; Pfaus, Quintana, Mac Cionnaith, & Parada, 2016). Nonetheless, women are more likely to achieve orgasm through masturbation than through coitus alone (Bancroft & Graham, 2011; Pfaus et al., 2016). Propensity towards orgasm also has a hereditary basis, but whether it enhances fertility or is a by-product of shared ontogeny with males is still unclear (Baker & Bellis, 1993; Pavličev & Wagner, 2016; Puts, Dawood, & Welling, 2012). Frequency of masturbation may therefore be a more suitable indicator of the traditional understanding of sexual desire as desire for pleasure (Bancroft & Graham, 2011; Lawrance & Byers, 1995; van Stein et al., 2019) and the midcycle peaks in oestradiol and testosterone have been found to covary positively with masturbation frequency (Jones et al., 2018; Sheldon et al., 2006; Van Goozen, Wiegant, Endert, Helmond, & Van de Poll, 1997).

Hormones affect sexual partner preference. Oestradiol has been shown to positively covary with women's preferences for men exhibiting traits and behaviour indicative of higher testosterone levels as well as extrapair desire (Arslan et al., 2018; Grebe et al., 2016; Larson, Pillsworth, Haselton, & Engelhardt, 2012; Shirazi et al., 2019) however there is still an ongoing debate on which traits are signals of good genes and which potential confounding variables should be considered and whether potential shifts can be considered adaptations (for review and discussion see Gildersleeve, Haselton, and Fales (2014), Wood, Kressel, Joshi, and Louie (2014) and Havliček, Cobey, Barrett, Klapilová, and Roberts (2015)).

On the other hand, elevated progesterone predicts women's preferences for indicators of good health, safety, their in-group, partner, facial femininity and parental investment potential (Fessler & Navarrete, 2003; Jones et al., 2005; Navarrete, Fessler, & Eng, 2007). It is also negatively associated with extrapair desire (Roney & Simmons, 2016). Progesterone rises under stressful circumstances in rats and moderates social affiliation in human and animal models (Maner, Miller, Schmidt, & Eckel, 2010; Miller, 2011). In a mood induction study where men and women had to recall an experience of rejection, progesterone levels at post-test were lower (Maner et al., 2010), however in a salivary analysis study of naturally cycling women progesterone covaried positively with between and within-women levels of attachment anxiety (Reynolds et al., 2018). The progesterone-linked increased attention to threats of social rejection and in-group preferences could influence motives for sex. Sexual desire, stemming from mating behaviour, has different social, behavioural and neurochemical underpinnings to pair-bonded love (Diamond, 2004). Women with an anxious attachment use sex to evoke increased caregiving or attachment from their partner and may feel pressured to have sex to retain a partner (Gentzler & Kerns, 2004; Redlick & Vangelisti, 2018; Schachner & Shaver, 2004). Female sexual proceptivity in reaction to a partner's lack of investment was not found to correlate with her self-reported sexual desire (Grebe et al., 2013). Therefore, progesterone-mediated sex may be more about strengthening the pair-bond. Because progesterone levels in the luteal phase resemble those in pregnancy, Maner and Miller (2014) propose that social affiliation may be especially important to pregnant women who are reliant on those around them for their own and their offsprings' survival. When evaluating sexual satisfaction in partnered women, they tend to rate the physical aspects of sexuality as more costly, and the emotional aspects as more rewarding, in contrast to men (Lawrance & Byers, 1995). This suggests that relational context, such as feeling desired by a valuable and responsive partner (Bancroft & Graham, 2011; Birnbaum et al., 2016) and

psychological cues may play a bigger role in their sexual satisfaction (Lawrance & Byers, 1995) and this may play an even greater role in high progesterone states.

How hormonal contraception affects the menstrual cycle

The most popular form of hormonal birth control are oral contraceptive pills used by an estimated 100 million worldwide (Welling & Shackelford, 2019). Typically, pills are taken for 21 consecutive days followed by seven days where consumers either take a placebo pill or no pill at all. During these seven days, women experience withdrawal bleeding which resembles menstruation in that progesterone and oestradiol levels are at their lowest throughout the cycle, however no endometrium is shed during this time. Some women prefer to start a new round of pills rather than experience withdrawal bleeding. Aside from oral contraceptives, there are also hormonal intrauterine devices (IUDs), vaginal rings, patches, injections and implants.

Hormonal contraceptives (HC) act on the reproductive system through the negative feedback loop. The release of exogenous progesterone and, in combined contraceptives, ethinyl oestradiol (EE) inhibit the release of gonadotropin-releasing hormone from the hypothalamus, thereby preventing the pituitary gland from releasing FSH and LH and consequently halting the release and maturation of follicles. The exogenous hormones suppress endogenous hormone production and hormone levels remain flat rather than peaking and falling as in natural cycles thus curtailing the required hormonal sequence for endometrium development. The hypothalamic-pituitary-ovarian axis is temporarily reactivated during the withdrawal week, but not long enough to prompt ovulation (Welling & Shackelford, 2019). While most brands of oral contraception are monophasic i.e. the levels of progestin and EE are constant throughout the 21 days, some brands are multiphasic and attempt to mimic the menstrual cycle, therefore these pills vary in dosage throughout the month. The daily dose in IUDs and implants is not constant with dosages being highest within the months after insertion and decreasing over time.

It is assumed that because synthetic hormones act on the central nervous system to inhibit ovulation in the same manner as endogenous hormones, then they should also affect other CNS functions such as women's psychology (Grøntvedt et al., 2016), however it is still unknown to which extent they can be considered equivalent to endogenous hormones (Welling, 2013).

HCs vary in the hormones they contain and administration types and this has consequences on their effects. Older generation progestins such as desogestrel and levonorgestrel have an androgenic effect in studies of rats (Stanczyk, 2003). These same progestins have also been found to suppress endogenous testosterone function. Recent progestins such as drospirenone are antiandrogenic and bind specifically to the progesterone receptor. However, how this androgenicity affects women requires further investigation (Davis, Davison, Donath, & Bell, 2005; Mitchell & Welling, 2020; Stanczyk, 2003). The route of administration also determines the strength of hormonal contraceptives. Because oral contraceptives are subject to first pass metabolism i.e. that most of the hormonal dosage is rapidly transformed into inactive compounds into the liver before being released into the blood (Bhosle, Altit, Autmizguine, & Chemtob, 2017), a greater dosage is required to compensate for this (Stanczyk, 2003).

While the binding affinity of endogenous progesterone may be greater than that of synthetic progestins, the latter are more potent (Welling & Shackelford, 2019), because of this, concentrations of synthetic progestin found in HC is much lower than that of endogenous progesterone in the luteal phase and dosages are typically double or triple the known amount required to inhibit ovulation (Grøntvedt et al., 2016; Stanczyk, 2003). However, when natural progesterones were administered by vaginal suppository, dosages equivalent to twice the amount required to inhibit ovulation replicated mid-luteal conditions with serum progesterone levels being between 7 and 10ng/ml (von Eye Corleta, Capp, & Ferreira, 2004), well within the normal range of 2 to 25ng/ml for the luteal phase ("Progesterone," n.d.), therefore concentrations in HCs are equivalent to those found in the luteal phase.

So-called “mini pills” containing smaller amounts of progestin than traditional oral contraceptives and hormonal IUDs typically secrete a daily dose of progestin that is below the amount required to inhibit ovulation. They are contraceptive in that they cause the thickening of cervical mucus which impedes sperm mobility, the physical presence of an IUD also acts as a barrier and the hormonal disruption of endometrium development hinders implantation (Felleskatalogen, 2018, 2019a, 2019b, 2019c; Rice, Killick, Dieben, & Bennink, 1999). However, because they do not reliably inhibit ovulation, the necessary LH surge can occur leading to follicle release which subsequently causes rising serum progesterone levels. In a study comparing, progesterone only pills containing a daily dosage of 75 mcg desogestrel to a mini-pill with 30 mcg levonorgestrel, ovulation was observed in only one out of the 59 cycles observed in participants taking the desogestrel pills, in contrast to sixteen out of the fifty-seven cycles observed in those using the levonorgestrel “mini pill”. Follicle rupture led to serum progesterone levels between 10 to 30ng/ml which are high, albeit normal levels for the luteal phase and consistent with normal levels observed in the first trimester of pregnancy where levels range between 10 and 44ng/ml ("Progesterone," n.d.; Rice et al., 1999).

The effects of hormonal contraceptives on women’s mating psychology

The psychobehavioural effects of hormonal contraceptives have only recently begun to be investigated as attention was mainly focused on their contraceptive reliability and physical safety issues (Mitchell & Welling, 2020). HC use has been associated to depression diagnosis and antidepressant use in young women, though these effects varied by progestin and administration type (Skovlund, Mørch, Kessing, & Lidegaard, 2016). Monophasic oral contraceptives are often prescribed against premenstrual dysphoric disorder (PMDD) (Rubinow & Schmidt, 2006; Schmidt, Nieman, Danaceau, Adams, & Rubinow, 1998). Women with hormonally related mood disorders such as PMDD are sensitive to the rate of change in hormone levels throughout the cycle despite having average hormone levels and normal cyclicity in their cycles which explains the benefits of the stabilising effects of monophasic HC (Rubinow & Schmidt, 2006; Schmidt et al., 1998). Nonetheless, the fact that mood and sexual desire in some women are unaffected by hormonal contraceptives or natural cycle shifts suggests individual differences are at play (Boozalis, Tutlam, Robbins, & Peipert, 2016; Burrows, Basha, & Goldstein, 2012; Elaut et al., 2012; Hendrick, Altshuler, & Burt, 1996; Mitchell & Welling, 2020). Sexual desire as well as mood may also be negatively affected by the tendency of HCs to downregulate secretion of endogenous oestradiol and testosterone (Burrows et

al., 2012; Elaut et al., 2012). These effects on women's mood and sexual desire can be mutually reinforcing (Basson et al., 2003).

Mate preferences also appear to be affected by HC use. Women using HC did not report elevated preferences for men's facial and vocal masculinity in contrast to naturally cycling women and show increased preference for signs of health (Jones et al., 2005; Little, Jones, & Burriss, 2007; Penton-Voak et al., 1999). This suggests that the synthetic progestins in HC produce similar results to endogenous progesterone, however Maner and Miller (2014) did not observe increased attention to social stimuli in HC users. Additionally, while some studies observed that HC users reported higher amount of sexual partners (Little, Jones, Penton-Voak, Burt, & Perrett, 2002) and increased interest in short-term sexual encounters (Guillermo, Manlove, Gray, Zava, & Marrs, 2010), others report fewer one-night stands, affairs as well as greater in-pair sexual activity and satisfaction with partner provisioning (Klapilová et al., 2014) and eliminates midcycle interest in extrapair men (Alvergne & Lummaa, 2010; Arslan et al., 2018). However, research on other primates suggest that how HC affects sexual behaviour depends on the preexisting social and sexual conditions (Nadler, 1977; Nadler, Dahl, Gould, & Collins, 1993; Wallen, 1982). These mixed results of behavioural studies on the effects of HCs indicate either a lack of control for possible confounding variables or can be account for by the differences in synthetic hormones and administration types.

Methodological issues in hormone research

Research on the hormonal regulation of women's mating psychology has been characterised with multiple methodological challenges leading to conflicting results and making interpretation challenging (Wood et al., 2014). Because there is considerable between-women and between-cycle variability in the length of menstrual cycles, identifying ovulation in a practical and cost-effective manner has been challenging. Many studies have used either forward or backward counting methods or both. These are based on the reported day of previous or expected upcoming menses onset and typically assume participants have a 28-day cycle (Shimoda, Campbell, & Barton, 2018). Additionally, there are inconsistencies in the delimitation of the fertile period with ranges being between 5 to 11 days (Harris, Chabot, & Mickes, 2013). To remedy this, direct assessment of hormones is preferred. Salivary assays of oestrogen and progesterone may not be suitable for cycle phase estimation due to the low concentration of these hormones in saliva (Schultheiss et al., 2019). Daily LH surge tests are the cheapest, most convenient and reliable of the hormonal assays (Shimoda et al., 2018), only to be surpassed by the most accurate, but invasive and costly transvaginal ultrasonography (Cobey, Klipping, & Buunk, 2013).

Earlier studies tended to use a between-women design comparing women who were currently fertile to those who were not or a between-women design with a measure from the fertile and infertile phase (Gangestad et al., 2016; Shimoda et al., 2018). Repeated measures designs are advantageous as they allow for reduction of noise, smaller samples are required to achieve power and more measures per participant increase measurement reliability as values are aggregated across the menstrual cycle (Gangestad et al., 2016). Due to individual differences in sensitivity to hormones, within-women designs and can allow for smaller sample sizes (Gangestad et al., 2016).

Shimoda et al. (2018) also point out that the classification of fertile and non-fertile lacks measurement sensitivity as the hormonal profile of the early luteal phase differs considerably from the that of the mid-luteal phase and this premenstrual phase is associated with psychological changes which may affect results. This also applies to analyses which lump the entire follicular phase into the fertile category.

The current study

The current thesis aims to investigate whether levels of synthetic progestin in coupled Norwegian women of reproductive age using hormonal contraception moderates the relationship between their own relationship investment and their frequency of sexual intercourse. It builds upon the work of Grebe et al. (2013) and Grøntvedt et al. (2016) who examined the characterising features of non-fertile sexuality.

In their study of 50 heterosexual couples with naturally-cycling women, Grebe et al. (2013) observed that women were more likely to be sexually proceptive during the luteal phase, as opposed to the fertile phase, when their own self-reports of investment were higher than their male partners' self-reports of investment as measured through an adapted version of the Partner Specific Investment Inventory (PSII) (Ellis, 1998).

Grøntvedt et al. (2016) conceptually replicated these results in cross-sectional and longitudinal studies in coupled women using hormonal contraception. Women rated themselves and their partners on the Mate Value Inventory's (MVI) items of loyalty and faithfulness (Kirsner, Figueredo, & Jacobs, 2003) and reported their sexual frequency during the last two and seven days. The composite variable of loyalty and faithfulness (LF) was found to have a non-significant positive main effect on sexual frequency, but the level of synthetic progestin interacted significantly with women's LF. In addition, EE moderated the effect of LF in a way that was both negative and significant. These patterns were found in both the cross-sectional and longitudinal versions of the study supporting oestradiol's role in women's tendency towards uncommitted extrapair desire during oestrus sexuality.

These studies found support for ES being not simply a default state against which fertile sexuality is contrasted, but that it fosters relationship investment and is regulated by progesterone. The MVI is a measure of a person's mate value, therefore LF is indicative of a partner's ability to invest in a relationship, whereas the PSII measures "unbankable" inputs i.e. sunk costs into a relationship. Once such investments have been made into a relationship with a specific partner, they cannot be reinvested in a subsequent relationship and occur at the expense of another possible investment (Ellis, 1998). Investment in the current relationship is of greater relevance to the male-assistance hypothesis of human extended sexuality. In line with Grøntvedt et al. (2016), we consider women's perceptions of their partners' investment as more relevant to their sexual behaviour than their partners' self-ratings.

In addition to replicating the observations made by Grøntvedt et al. (2016), we will examine whether this interaction can be found with other current relationship investment measures in order to better isolate which aspects of investment contribute to extended sexuality. Along with LF and the PSII, we will use the Perceived Relationship Quality Components, a measure of relationship quality (Fletcher

et al., 2000), and the Attachment Features and Function scale, a measure of pair-bond strength (Tancredy & Fraley, 2006). We are also interested in investigating whether extended sexuality leads to increased investment from the partner over time.

We make the following hypotheses:

H1: In line with the previous research and based on the explanation of hormonal influence on extended sexuality, we expect to see progestin moderate the relationship between female investment and sexual frequency. We are explorative in which specific aspects of relationship investment, quality and perceived bond strength are most influential in this relationship.

H2: We expect that sexual frequency will predict greater partner investment when repeating measures.

Methods

Design and participants

The research questions were examined using a longitudinal design composed of two surveys administered two months apart. We used a convenience sample primarily composed of students at the Norwegian University of Science and Technology (NTNU) in Trondheim, Norway. A total of 555 respondents filled out the first round of the survey of which 503 were students. The mean age of respondents was 22.94 years (*SD* = 3.74, *Median* = 22, range = 18 – 42). 343 reported being in committed long-term relationships. Inconsistent responding regarding HC use was carefully checked and resolved leaving us with 434 participants currently using hormonal contraception of which 219 were using oral contraceptives and 215 were using non-oral contraceptives. Four respondents reported being pregnant, eight were breastfeeding and one had given birth within the last three months. 341 respondents filled out the second survey of which 213 were in long term relationships and 194 were in the same relationship as during the first round of the survey. 269 were currently using HC, of which 126 were using oral HC and 136 were using non-oral HC. 28 respondents reported changing birth control type between both sessions of the survey. One respondent was pregnant, two were breastfeeding and one had given birth in the last three months.

Procedure

Recruitment of participants occurred from mid-September until the end of October through presentations before lectures, posters and flyers spread around the various campuses at NTNU and businesses in Trondheim, as well as over social media and through word of mouth. The project was presented as a study on "female sexuality, relationships and hormones" and potential participants were encouraged to contribute to increasing knowledge on female sexuality and how hormonal contraceptives affect women's psychology as well as being informed of the possibility of winning one of two tablet computers. The survey was online enabling participants to respond in private and at their leisure, and they were informed that it would take ca. 25 minutes to fill out. The initial data-collection took place during the same period as recruitment and respondents had the option of entering their e-mail address if they wanted to participate in the second round of the survey two months later and consequently, be eligible to win a tablet. To assure that almost two months had elapsed between response-times, respondents were sent e-mails informing them of the release of the second survey in waves such that those who responded in mid-September were invited to participate in the second round in mid-November and each week, a new wave of respondents received an invitation. The last wave received their invitation before the Christmas break. Respondents who did not complete the second round upon invitation were sent a reminder three and seven days after the initial invitation. On both rounds of the survey, participants who did not check off that they were either in a long-term committed relationship, married or cohabiting were not able to access the parts of the survey pertaining to partner MVI, as well as questions on both partners' PSII, PRQC, bond and conflict. The presentation page and consent form at the beginning of rounds 1 and 2 of the survey can be found in Appendix A.

Measurements

Relationship investment indicators

While the survey was in Norwegian, the questions from the different investment measures were originally in English. To assure that the translations would reflect the same constructs, they were translated to Norwegian, then backtranslated into English and refined if necessary, to better reflect the constructs of interest. We measured both women's own self-ratings of investment as well as how they perceived their partners' investment (Appendix B).

Loyalty and Faithfulness

Loyalty and faithfulness (LF) is a composite measure of the two loyalty and faithfulness items of the Mate Value Inventory developed by Kirsner et al. (2003). Respondents were asked to rate themselves and their partner on a 7-point Likert scale ranging from ''strongly disagree'' to ''strongly agree''. The values on both items were added and then converted into z-scores. Correlations between women's self-reported LF at Timepoint 1 (T1) and Timepoint 2 (T2) are r = .72 and for women's rating of their partner's LF, r = .86. The measure is moderate to highly reliable for both women and their partners at both timepoints (women's LF at T1: α = .70, women's LF at T2, α = .74; partner's LF at T1: α = .76, partner's LF at T2: α = .76).

Partner Specific Investment Inventory

The Partner Specific Investment Inventory (PSII) (Ellis, 1998) typically requires that respondents rate their partners, but not themselves, as was done in Grebe et al. (2013). We administered an abridged and modified version of the PSII in our survey using a total of twelve items from both the behaviour and descriptive statements sections and adapted them so respondents could rate the frequency of behaviours on a 5-point Likert scale from 0 (Never) to 4 (Very Often). The rationale behind this was that a shorter PSII would encourage more respondents to complete the survey. These twelve items reflect six of the ten investment types because these items were found to correlate most highly with each other. Three items had to be reversed to match that of the others. We then summed the scores. Because the scale was such that the higher the score, the lower the investment, we then reversed the total score such than it would be like the other relationship involvement indicators in that higher values are associated to greater investment. The scale scores were then z-scored for the analysis. The correlation between women's self-ratings on PSII at both timepoints is r = .79 and partner ratings, r = .82. Internal consistency was good (women's PSII at T1: α = .69, women's PSII at T2: α = .67; partner's PSII at T1: α = .76, partner's PSII at T2: α = .81).

Perceived Relationship Quality Components

The Perceived Relationship Quality Components developed by Fletcher et al. (2000) measures six constructs traditionally used in the relationship quality literature which underlie a global perception of self-reported relationship quality. The full PRQC Inventory consists in three redundant items for each component, though, the authors recommend using only the highest loading item for each construct totalling six items. Participants were requested to rate their partner or relationship on a 7-point Likert scale from 1 (Not at all) to 7 (Extremely). We asked participants to rate their own relationship as well as how they believe their partner perceives their relationship on five of the six components. The item for trust was excluded due to translation issues. The scores of all items for

women and their partners were summed and transformed into z-scores. The correlation for women's PRQC at T1 and T2 is r = .76 and for partners' PRQC, r = .74. Reliability was high (women's PRQC at T1: α = .83, women's PRQC at T2: α = .87; partner's PRQC at T1: α = .82, men's PRQC at T2: α = .84).

Bond Strength

We assessed the strength of the attachment bond by using an adaptation of the Attachment Features and Function scale (Tancredy & Fraley, 2006). The scale was originally created to measure attachment in twins, but has been modified for couples. Respondents had to rate themselves and their partner on four statements each pertaining to the four dimensions of attachment on a 7-point Likert scale from 1 (Totally disagree) to 7 (Totally agree). Items were added together and then z-scored. The correlation for women's bond strength for T1 and T2 is r = .78, partners' bond, r = .76. Reliability was moderate to high (women's bond at T1: α = .77, at T2: α = .83; partner's bond at T1: α = .75, T2: α = .74).

Sociosexuality Index

Because positive correlations between women's unrestricted SOI and sexual frequency as well as hormonal contraception use have been found (Simpson & Gangestad, 1992; Welling, 2013), we have decided to control for this factor as it may play a role even when unrestricted individuals are in a long-term relationship. We used the Revised Sociosexuality Inventory (SOI-R) created by Penke and Asendorpf (2008) which covers three dimensions: behaviour, attitude and desire. However, we calculated the sum of the three subcategories together and then z-scored the values. A higher score is indicative of a more unrestricted sociosexuality. Internal consistency was high, at T1, α = .85 and at T2, α = .85. The correlation between T1 and T2: r = .92 indicating that this index was stable over the investigated timeframe.

Relationship length

Frequency of sexual intercourse has been shown to decrease the longer a couple has been together despite investment typically being higher the longer a pair has been together (Call, Sprecher, & Schwartz, 1995; Diamond, 2004; Ellis, 1998). To control for this, we asked respondents in committed relationships to report how long in months and years they had been in the current relationship and converted the total length into months and z-scored.

Frequency of sexual behaviours

Respondents were asked to report how often they engaged in masturbation and sexual intercourse during the last two and seven days. Because it is easier to recall behaviour from the last two days, we double-weighted these days by adding the total amount of masturbation or sex during the last two days to the total amount during the last seven days equating to the total frequency during nine days. This variable was computed in the same manner as in Grøntvedt et al. (2016).

Days unable

Respondents were asked to report how many days during the last two and seven days they were unable to have sex. The variable was computed by double-weighing the last two days; therefore, we added the amount of days partners were unable to have sex during the last two and seven days as was done by Grøntvedt et al. (2016).

Hormonal effects

Respondents could select their brand of hormonal contraception from a list of brands recommended by the Norwegian Medicines Agency (Statens Legemiddelverk, 2016), but participants could also write down their brand if it wasn't featured on the list. Hormone dosages and progestin types were found on the Norwegian Directory of Medicines "Felleskatalogen" (Felleskatalogen, n.d.). Because the dosage of multiphasic contraceptive pills varies by day, we chose to exclude these from our analyses as only two respondents reported using them and it was not possible to know the dosage during the last seven days.

Respondents using implants and IUDs were asked to report in the second survey how long they had been using the current implant or IUD as they secrete varying hormone levels over time. If they reported to have been using the device or implant for over 3 months, we subtracted two months such that we could know more specifically what the dosage was when they completed the survey at T1. In those that reported using an IUD or implant, but did not complete the second survey or in cases where it was there was no information on hormone secretion variations over time, we used the average daily dose as specified by Felleskatalogen (n.d.).

Synthetic progestins vary in potency and therefore, the minimum dosages required to inhibit ovulation differ by type. We used the same method as Grøntvedt et al. (2016) to put dosages on a common scale accounting for potency. This was done by first considering the ratio between the actual daily dose and the dose required to inhibit ovulation. Then, values were assigned a dosage on the same scale as levonorgestrel for convenience as it was the most used, therefore if the dosage was equivalent to two times the daily dose required to inhibit ovulation, then we assigned the equivalent dose for levonorgestrel. However, we only had the necessary data to calculate adjusted dosages for levonorgestrel, desogestrel, etonogestrel, drospirenone, norethisterone, norelgestromin and cyproterone acetate, but not for HCs using dienogest, medroxyprogesterone acetate and nomegestrol acetate.

Additionally, synthetic progestins vary in their androgenetic effects from being moderately to anti-androgenetic. The adjusted daily dosages were multiplied by weights (+3, +1, -1 and -3) where androgenetic effects had positive weights (Grøntvedt et al., 2016), however androgenicity was unknown for norethisterone, norelgestromin, but these consisted of only three observations (Table 1). Because every combined oral contraceptive used in our sample contained ethinyl oestradiol rather than other forms of oestrogen, no transformations were required for this hormone. The adjusted dosages of progestin, dosages of ethinyl oestradiol and androgenicity were then z-scored.

Table 1

Types of progestins used; typical and adjusted daily dosages, androgenicity weights

Type	Typical dosages	Adjusted dosages	Androgenicity weight
Levonorgestrel	20/100/150	20/100/150	+3
Desogestrel/Etonogestrel	40/75/120/150	40/75/120/150	+1
Norelgestromin	150	150	Unknown
Norethisterone	500/1000	75/150	Unknown
Drospirenone	3000	150	-1
Cyproterone acetate	2000	150	-3

Note. Typical and adjusted dosages are in mcg. Adapted from Table 2 in Grøntvedt et al. (2016).

Data Analysis

Analyses were done using Stata 16. H1 was investigated using linear mixed models analyses. The dependent variable of sexual frequency (*M* = 3.16, *SD* = 3.15, *Median* = 3, range = 0 – 20) was not normally distributed with skewness of 1.63 and kurtosis of 7.29. Use of linear mixed models with non-normal data tends to be robust and is frequent in the behavioural sciences (Arnau, Bono, Blanca, & Bendayan, 2012) and it was also used in Grøntvedt et al. (2016). Mixed models replace missing data with the mean value for that variable so as to maintain power (Grace-Martin, n.d.). In respondents who participated in both rounds of the survey, variable values were aggregated to form an individual mean (Gangestad et al., 2016). To control for within-individual differences not otherwise specified by the fixed effects, participant ID was specified as a random effects parameter. The model was fit using Restricted Maximum Likelihood which accounts for the best fit of the fixed effect parameters i.e. the independent variables, and then excludes these to calculate the variance of the random effects in the model (Oehlert, 2011). This was paired with the conservative Satterthwaite approximation for degrees of freedom to generate p-values (Luke, 2017).

Respondents were excluded from the sample if they reported being pregnant, breastfeeding, having recently given birth, were not in a committed long-term relationship and if their partner was not a man. The final sample consisted of *n* = 409 observations from 271 individual respondents. Because 52,86% of observations come from women not using oral HC, we decided not to exclude observations based on whether or not the respondent had experienced her period or withdrawal bleeding during the last seven days in contrast to Grøntvedt et al. (2016). This was done so as not to reduce sample size and women on non-oral HC are continuously exposed to exogenous hormones even when bleeding.

H2 was investigated using a post-test regression. To analyse change over time, the same exclusion criteria were applied as in the analysis of H1, however respondents also had to have participated in both rounds of the survey, be with the same partner and have been using HCs at both time points so that sexual behaviour could qualify as progestin-regulated extended sexuality. This resulted in *n* = 140 respondents.

This regression was computed for all four investment scales with the dependent variable being the partner's score at T2 on the investment scale in question. This is a type of multiple regression where

the pre-test value i.e. the partner's score on the relevant investment scale at T1, is controlled for by adding it as a covariate. The other predictors in our model were the difference in sexual frequency between T2 and T1, the difference in days partners were unable to have sex between T2 and T1, the difference in women's investment on the same scale between T2 and T1 and the total relationship duration in months at T2. None of the variables were z-scored. Because of possible multicollinearity between the pre-test covariate and other covariates, correlations were computed between the pre-test variable and other covariates in the model to prevent Type 1 errors as recommended by Farmus, Arpin-Cribbie, and Cribbie (2019). All correlations were below r= .20.

Ethics

The study was submitted for approval from the regional ethical committee of Trondheim (REK) and the Norwegian Centre of Research Data (NSD) in April 2019. A preregistration was submitted to the Centre for Open Science (OSF) in August 2019. In January, the e-mails of those who completed the second round of the survey were included in a draw for the tablets and they were awarded to the winners. On March 1, 2020, all e-mails were deleted, and confirmation of deletion was sent to NSD.

Results

Between-women analyses

We first ran our between-women analyses with the different measures of investment (Tables 2 – 5). Relationship length and days unable had significant and similar negative main effects on sexual frequency across the four models. LF, PSII and PRQC had significant main effects, but not bond strength. Ethinyl oestradiol (EE), progestin (P) and androgenicity (A) had no significant main effect in all models. No significant interaction effects between hormones and LF were observed, therefore, we did not replicate the negative interaction between EE and LF as well as the interaction between P and LF observed in Grøntvedt et al. (2016). EE and P interacted significantly with PSII. EE had a significant negative interaction with PRQC, but P had a nearly significant interaction with PRQC ($p < 0.055$). EE and P interacted significantly with bond strength. No interactions with androgenicity were observed across the models. These results support H1, in that progestin moderates the effect of current relationship involvement on sexual frequency. Similar to what was observed in Grøntvedt et al. (2016), EE and PSII, PRQC and bond strength interacted negatively. While insignificant, the general tendency observed among the models is that EE as a main effect predicts sexual frequency while P does not, but within the context of interactions with investment, they have opposite effects.

Table 2

Between-women effects on sexual frequency: Loyalty/Faithfulness model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-10.17**	**397.68**	**0.000**	**[-.49, -.33]**
Relationship length	**-1.02**	**-4.94**	**291.63**	**0.000**	**[-1.43, -.61]**
Loyalty/Faithfulness (LF)	**.46**	**2.10**	**378.85**	**0.036**	**[.03, .89]**
Ethinyl Oestradiol (EE)	.54	1.17	366.62	0.242	[-.36, 1.44]
Progestin (P)	-.44	-1.09	306.29	0.277	[-1.24, .35]
Androgenicity (A)	-.34	-1.19	388.14	0.235	[-.89, .22]
Interaction effects					
EE x LF	-.57	-0.93	380.97	0.345	[-1.77, .63]
P x LF	.05	0.08	362.91	0.933	[-1.02, 1.12]
A x LF	.09	0.21	398.92	0.837	[-.73, .90]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$

Table 3

Between-women effects on sexual frequency: Partner-Specific Investment Inventory model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-10.48**	**396.56**	**0.000**	**[-.50, -.34]**
Relationship length	**-.94**	**-4.61**	**295.80**	**0.000**	**[-1.33, -.53]**
PSII	**.72**	**4.86**	**316.93**	**0.000**	**[.42, 1.00]**
Ethinyl Oestradiol (EE)	.51	1.23	315.55	0.218	[-.30, 1.31]
Progestin (P)	-.49	-1.25	298.97	0.211	[-1.25, .27]
Androgenicity (A)	-.27	-1.41	298.95	0.161	[-.65, .10]
Interaction effects					
EE x PSII	**-1.02**	**-2.42**	**322.81**	**0.016**	**[-1.85, -.18]**
P x PSII	**.95**	**2.32**	**314.40**	**0.021**	**[.14, 1.75]**
A x PSII	-.09	-0.54	376.08	0.590	[-.43, .24]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$

Table 4

Between-women effects on sexual frequency: Perceived Relationship Quality Components model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.41**	**-10.18**	**396.47**	**0.000**	**[-.49, -.33]**
Relationship length	**-1.03**	**-5.03**	**291.30**	**0.000**	**[-1.42, -.62]**
PRQC	**.58**	**3.87**	**317.10**	**0.000**	**[.28, .87]**
Ethinyl Oestradiol (EE)	.38	0.91	317.37	0.365	[-.44, 1.19]
Progestin (P)	-.39	-0.99	301.06	0.324	[-1.15, .38]
Androgenicity (A)	-026	-1.34	307.70	0.182	[-.65, .12]
Interaction effects					
EE x PRQC	**-.93**	**-2.39**	**383.14**	**0.017**	**[-1.69, -.16]**
P x PRQC	*.74*	*1.92*	*338.65*	*0.055*	*[-.01, 1.49]*
A x PRQC	-.03	-0.19	385.58	0.851	[-.39, .32]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$, italics: $p < .10$

Table 5

Between-women effects on sexual frequency: Bond strength model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-10.25**	**397.80**	**0.000**	**[-.49, -.33]**
Relationship length	**-1.06**	**-5.15**	**290.77**	**0.000**	**[-1.46, -.65]**
Bond strength	**.56**	**3.57**	**317.06**	**0.000**	**[.25, .87]**
Ethinyl Oestradiol (EE)	.55	1.32	320.11	0.188	[-.27, 1.38]
Progestin (P)	-.49	-1.26	306.50	0.209	[-1.28, .28]
Androgenicity (A)	-.32	-1.60	302.01	0.111	[-.71, .07]
Interaction effects					
EE x Bond	**-.99**	**-2.10**	**359.13**	**0.037**	**[-1.92, -.06]**
P x Bond	**.82**	**2.00**	**329.73**	**0.046**	**[.01, 1.61]**
A x Bond	-.13	-0.64	388.28	0.524	[-.53, .27]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$

Within-women analyses

To calculate within-women differences, we created a variable representing each women's average level of investment and another variable representing the difference from this mean for both timepoints. Respondents with observations for only one session were excluded, leaving us with n = 303 observations taken from 166 different women. Afterwards, we z-scored the variables for mean and difference and ran our analyses while controlling for ID by coding it as a random effect. Days unable and relationship length had significant main effects across all models. See

The within-women differences from the mean for each scale did not have any significant main effects, nor interaction effects, therefore no within-women effects were observed. All four mean investment scales had a significant main effect. Mean PSII, PRQC and bond strength interacted significantly with EE and P as was observed in the between women analyses. See Tables 6 – 9.

Table 6

Within-women effects on sexual frequency: Loyalty/Faithfulness model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-8.45**	**288.86**	**0.000**	**[-.51, -.32]**
Relationship length	**-1.54**	**-4.63**	**154.33**	**0.000**	**[-2.19, -.88]**
Mean LF	**.76**	**2.72**	**183.30**	**0.007**	**[.21, 1.31]**
Difference from mean LF	-.20	-1.36	142.94	0.177	[-.48, .09]
Ethinyl Oestradiol (EE)	-.58	-1.06	243.45	0.289	[-1.65, .49]
Progestin (P)	.11	0.22	196.34	0.827	[-.84, 1.05]
Androgenicity (A)	.35	1.02	261.09	0.306	[-.32, 1.02]
Interaction effects					
EE x Mean LF	-.29	-0.40	220.73	0.693	[-1.73, 1.15]
P x Mean LF	.71	1.16	187.41	0.247	[-.49, 1.90]
A x Mean LF	*-1.05*	*-1.74*	*241.66*	*0.083*	*[-2.25, .13]*
EE x Difference from mean	-.29	-0.63	158.97	0.527	[-1.20, .61]
P x Difference from mean LF	-.23	-0.60	144.41	0.550	[-1.00, .53]
A x Difference from mean LF	.55	1.64	175.92	0.102	[-.11, 1.21]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.

All values are z-scored except “Days unable”.

Bold: $p < 0.05$, italics: $p < .10$

Table 7

Within-women effects on sexual frequency: Partner-Specific Investment Inventory model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.43**	**-8.96**	**288.07**	**0.000**	**[-.52, -.33]**
Relationship length	**-1.34**	**-4.28**	**153.24**	**0.000**	**[-1.95, -.71]**
Mean PSII	**.91**	**4.69**	**157.25**	**0.000**	**[.52, 1.29]**
Difference from mean PSII	.07	0.51	156.62	0.608	[-.20, .34]
Ethinyl Oestradiol (EE)	-.33	-0.65	220.78	0.518	[-1.31, .66]
Progestin (P)	.22	0.46	199.28	0.643	[-.69, 1.12]
Androgenicity (A)	.02	0.09	207.26	0.930	[-.51, .55]
Interaction effects					
EE x Mean PSII	**-1.46**	**-2.76**	**198.69**	**0.006**	**[-2.49, -.41]**
P x Mean PSII	**1.58**	**3.16**	**183.37**	**0.002**	**[.59, 2.56]**
A x Mean PSII	*-.41*	*-1.66*	*215.38*	*0.099*	*[-.90, .07]*
EE x Difference from mean PSII	.26	0.53	185.80	0.597	[-.71, 1.24]
P x Difference from mean PSII	-.27	-0.63	172.38	0.528	[-1.10, .56]
A x Difference from mean PSII	.15	0.61	235.98	0.540	[-.33, .62]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.

All values are z-scored except “Days unable”.

Bold: $p < 0.05$, italics: $p < .10$

Table 8

Within-women effects on sexual frequency: Perceived Relationship Quality Components model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-8.66**	**288.78**	**0.000**	**[-.51, -.32]**
Relationship length	**-1.41**	**-4.41**	**153.82**	**0.000**	**[-2.03, -.77]**
Mean PRQC	**.68**	**3.39**	**154.87**	**0.001**	**[.28, 1.07]**
Difference from mean PRQC	-.14	-1.02	146.62	0.308	[-.42, .13]
Ethinyl Oestradiol (EE)	-.38	-0.75	208.18	0.452	[-1.38, .61]
Progestin (P)	.25	0.52	199.83	0.604	[-.69, 1.18]
Androgenicity (A)	.03	0.10	193.34	0.917	[-.49, .54]
Interaction effects					
EE x Mean PRQC	**-1.22**	**-2.66**	**251.33**	**0.008**	**[-2.13, -.31]**
P x Mean PRQC	**1.11**	**2.30**	**187.32**	**0.022**	**[.15, 2.06]**
A x Mean PRQC	-.21	-0.88	248.40	0.381	[-.68, .26]
EE x Difference from mean PRQC	.23	0.54	145.26	0.588	[-.59, 1.04]
P x Difference from mean PRQC	-.15	-0.39	148.29	0.697	[-.88, .59]
A x Difference from mean PRQC	-.15	-0.62	148.66	0.535	[-.61, .31]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.

All values are z-scored except “Days unable”.

Bold: $p < 0.05$

Table 9

Within-women effects on sexual frequency: Bond strength model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-8.53**	**288.75**	**0.000**	**[-.51, -.32]**
Relationship length	**-1.52**	**-4.63**	**153.79**	**0.000**	**[-2.17, -.87]**
Mean bond strength	**.62**	**3.14**	**153.88**	**0.002**	**[.23, 1.01]**
Difference from mean bond	.14	0.98	164.76	0.331	[-.14, .41]
Ethinyl Oestradiol (EE)	-.03	-0.05	205.55	0.961	[-1.06, 1.01]
Progestin (P)	.01	0.03	201.48	0.976	[-.95, .98]
Androgenicity (A)	-.10	-0.38	187.62	0.703	[-.64, .43]
Interaction effects					
EE x Mean bond	**-1.59**	**-2.65**	**188.36**	**0.009**	**[-2.76, -.40]**
P x Mean bond	**1.35**	**2.73**	**181.63**	**0.007**	**[.37, 2.33]**
A x Mean bond	.07	0.25	167.53	0.803	[-.50, .64]
EE x Difference from mean bond	-.08	-0.19	192.16	0.846	[-.90, .74]
P x Difference from mean bond	.10	0.25	181.78	0.806	[-.67, .87]
A x Difference from mean bond	-.10	-0.66	149.24	0.512	[-.38, .19]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$

Session effects

Sessions were coded as -.5 for T1 and .5 for T2 so that the difference between sessions would equal 1 to facilitate interpretation. Main effects of session on sexual frequency were observed in the LF and bond strength model. Sessions were not found to interact significantly with hormonal effects, neither were they found to significantly influence the interaction effects between hormones and relationship involvement as all significant main effects of investment and interactions between hormones and investment observed in the between-subjects analyses remained significant. Days unable and relationship length had significant main effects across all models. See Tables 10 – 13.

Table 10

Session effects on sexual frequency: Loyalty/Faithfulness model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.43**	**-10.25**	**391.61**	**0.000**	**[-.50, -.34]**
Relationship length	**-1.06**	**-5.04**	**298.52**	**0.000**	**[-1.46, -.64]**
Loyalty/Faithfulness (LF)	**.46**	**2.06**	**377.58**	**0.040**	**[.02, .90]**
Session effect (SE)	**-.49**	**-2.07**	**193.03**	**0.040**	**[-.96, -.02]**
Ethinyl Oestradiol (EE)	.44	0.93	375.01	0.354	[-.49, 1.38]
Progestin (P)	-.37	-0.90	313.13	0.371	[-1.18, .44]
Androgenicity (A)	-.34	-1.08	391.92	0.281	[-.95, .27]
Interaction effects					
EE x LF	-.61	-0.96	384.19	0.337	[-1.84, .63]
P x LF	.05	0.09	361.07	0.927	[-1.02, 1.12]
A x LF	.10	0.21	380.78	0.832	[-.81, 1.01]
SE x EE	-1.05	-1.37	213.10	0.173	[-2.55, .46]
SE x P	.95	1.50	200.78	0.135	[-.29, 2.20]
SE x A	.04	0.07	212.81	0.941	[-.92, .99]
SE x LF x EE	1.39	1.33	223.96	0.185	[-.67, 3.45]
SE x LF x P	*-1.54*	*-1.77*	*216.78*	*0.079*	*[-3.25, .17]*
SE x LF x A	-.17	-0.24	232.97	0.810	[-1.59, 1.24]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.

All values are z-scored except “Days unable”.

Bold: $p < 0.05$, italics: $p < .10$

Table 11

Session effects on sexual frequency: Partner-Specific Investment Inventory model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-10.47**	**390.90**	**0.000**	**[-.50, -.34]**
Relationship length	**-.94**	**-4.56**	**299.23**	**0.000**	**[-1.34, -.53]**
PSII	**.70**	**4.65**	**321.70**	**0.000**	**[.40, .99]**
Session effect (SE)	*-.46*	*-1.92*	*196.72*	*0.057*	*[-.92, ,01]*
Ethinyl Oestradiol (EE)	.48	1.08	349.68	0.279	[-.38, 1.34]
Progestin (P)	-.40	-1.02	308.29	0.308	[-1.18, .37]
Androgenicity (A)	-.36	-1.50	380.49	0.136	[-.84, .11]
Interaction effects					
EE x PSII	**-1.06**	**-2.40**	**341.58**	**0.017**	**[-1.93, -.19]**
P x PSII	**.91**	**2.21**	**318.96**	**0.028**	**[.10, 1.72]**
A x PSII	-.04	-0.20	391.53	0.843	[-.48, .39]
SE x EE	-.70	-0.96	223.89	0.337	[-2.12, .72]
SE x P	.70	1.13	209.60	0.259	[-.52, 1.92]
SE x A	-.11	-0.27	231.65	0.789	[-.93, .71]
SE x PSII x EE	-.40	-0.56	215.43	0.575	[-1.82, 1.01]
SE x PSII x P	-.06	-0.09	207.21	0.927	[-1.32, 1.20]
SE x PSII x A	.30	0.77	240.49	0.440	[-.46, 1.06]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.

All values are z-scored except “Days unable”.

Bold: $p < 0.05$, italics: $p < 0.10$

Table 12

Session effects on sexual frequency: Perceived Relationship Quality Components model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.41**	**-10.17**	**390.61**	**0.000**	**[-.49, -.33]**
Relationship length	**-1.02**	**-4.96**	**296.88**	**0.000**	**[-1.42, -.61]**
PRQC	**.58**	**3.79**	**321.75**	**0.000**	**[.27, .88]**
Session effect (SE)	*-.43*	*-1.80*	*197.40*	*0.073*	*[-.89, .04]*
Ethinyl Oestradiol (EE)	.28	0.64	334.61	0.524	[-.57, 1.13]
Progestin (P)	-.29	-0.73	309.38	0.468	[-1.07, .49]
Androgenicity (A)	-.29	-1.30	355.51	0.194	[-.73, .14]
Interaction effects					
EE x PRQC	**-.97**	**-2.37**	**381.55**	**0.018**	**[-1.77, -.16]**
P x PRQC	*-.76*	*1.95*	*342.67*	*0.052*	*[-.00, 1.51]*
A x PRQC	.01	0.03	391.94	0.973	[-.42, .43]
SE x EE	-.86	-1.22	224.34	0.223	[-2.25, .52]
SE x P	.84	1.31	215.60	0.191	[-.41, 2.08]
SE x A	-.10	-0.27	225.47	0.784	[-.84, .64]
SE x PRQC x EE	-.42	-0.60	226.71	0.551	[-1.82, .97]
SE x PRQC x P	.21	0.33	220.54	0.739	[-1.04, 1.46]
SE x PRQC x A	.20	0.52	242.73	0.602	[-.55, .96]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.

All values are z-scored except “Days unable”.

Bold: $p < 0.05$, italics: $p < .10$

Table 13

Session effects on sexual frequency: Bond strength model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-10.23**	**391.63**	**0.000**	**[-.49, -.33]**
Relationship length	**-1.07**	**-5.13**	**296.52**	**0.000**	**[-1.48, -.65]**
Bond	**.54**	**3.42**	**311.33**	**0.001**	**[.23, .86]**
Session effect (SE)	**-.50**	**-2.17**	**194.63**	**0.031**	**[-.96, -.04]**
Ethinyl Oestradiol (EE)	.47	1.07	338.35	0.287	[-.40, 1.35]
Progestin (P)	-.44	-1.11	316.80	0.269	[-1.24, .34]
Androgenicity (A)	-.31	-1.42	333.05	0.156	[-.75, .12]
Interaction effects					
EE x Bond	*-1.00*	*-1.96*	*345.24*	*0.051*	*[-2.02, .00]*
P x Bond	**-.84**	**1.99**	**325.76**	**0.048**	**[.00, 1.66]**
A x Bond	-.14	-0.64	361.35	0.522	[-.58, .29]
SE x EE	-.58	-0.83	222.45	0.407	[-1.97, .80]
SE x P	.47	0.75	209.92	0.454	[-.77, 1.71]
SE x A	.01	0.04	239.16	0.971	[-.69, .72]
SE x Bond x EE	-.04	0.06	195.40	0.956	[-1.47, 1.56]
SE x Bond x P	-.34	-0.55	192.58	0.585	[-1.57, .89]
SE x Bond x A	.09	0.27	195.36	0.790	[-.59, .78]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$, italics: $p < 0.10$

Correlations between investment measures

As we did not replicate the interaction between progestin and LF, but found effects for the other measures, we ran pair-wise correlations between the aggregated measures (Table 14). Correlations between the LF and the other measures were moderate, however PSII, PRQC and bond correlated highly with each other.

Table 14

Correlation matrix for the four investment measures for women and partners

Women's self-ratings of investment				
	LF	PSII	PRQC	Bond
LF	1.00			
PSII	.38	1.00		
PRQC	.45	.61	1.00	
Bond	.36	.53	.65	1.00
Perceived partner ratings of investment				
	LF	PSII	PRQC	Bond
LF	1.00			
PSII	.47	1.00		
PRQC	.40	.66	1.00	
Bond	.31	.55	.60	1.00

Note. Correlation values have been rounded to the second decimal place. All correlations are significant ($p < 0.05$).

Control for other variables

Administration type

We controlled for whether HCs were administered orally or non-orally to account for first pass metabolism effects (Tables C1 – C8). To ease interpretation, we ran separate between-women analyses for oral and non-oral contraceptive users, therefore, for the oral group, n = 195 observations of 135 women, and for the non-oral group, n = 214 observations of 144 women. Because sample sizes were smaller, this may affect power.

In the LF model, no significant main effects of LF or interaction effects were observed in both the oral and non-oral groups. However, in the non-oral condition, androgenicity did have a main effect, b = 6.76, t(131.92) = 1.99 $p < 0.048$ as well as the interaction between androgenicity and LF, b = 9.21, t(177.77) = 2.28 $p < 0.024$.

In the PSII model, no significant main effects of PSII or interaction effects were observed for both conditions. However, in the oral condition, the EE x PSII interaction was nearly significant, b = -1.15, t(156.05) = -1.80 $p < 0.074$. In the non-oral condition, the main effect of PSII was nearly significant, b = 2.07, t(190.41) = 1.78 $p < 0.077$ as well as that of androgenicity, b = 6.19, t(135.88) = 1.82 $p < 0.071$.

In the PRQC model, no significant main effects of PSII or interaction effects were observed for both conditions. However, in the oral condition, the EE x PRQC interaction was nearly significant, b = -1.15, t(159.14) = -1.89 $p < 0.060$. In the non-oral condition, progestin had a nearly significant effect, b = -1.05, t(143.47) = -1.12 $p < 0.069$, as well as the A x PRQC interaction, b = 6.29, t(191.72) = 1.70
$p < 0.091$.

In the non-oral HC analyses, bond had a nearly significant main effect, b = 2.62, t(194.78) = 1.90 p < 0.058. Otherwise, no other effects were observed in either condition.

Sociosexuality Index

We controlled for the effects of sociosexuality and checked for interactions between SOI and hormones (Tables C9 – C12).

The significant main effect of LF that was present in the between-women model lost significance after controlling for SOI. Interactions between hormones and LF remained insignificant. The main effects of PSII as well as the interactions between EE and P with PSII remained significant: for PSII, b = .74, t(321.08) = 4.87 p < 0.000, for EE x P, b = -1.10, t(325.21) = -2.52 p < 0.012 and for P x PSII, b = 1.02, t(325.15) = 2.41 p < 0.016. The main effect of PRQC and its interaction with EE remained significant. After controlling for SOI, the P x PRQC interaction was now significant: for PRQC, b = .58, t(321.33) = 3.74 p < 0.000, for EE x PRQC, b = -1.13, t(361.52) = -2.69 p < 0.007 and for P x PRQC, b = .83, t(335.07) = 2.08 p < 0.038. The main effect of bond remained significant, b = .56, t(317.12) = 3.52 p < 0.000. The interactions between EE and P with bond remained significant: for EE x bond, b = -1.01, t(359.03) = -2.11 p < 0.036 and for P x PRQC, b = .83, t(333.15) = 1.98 p < 0.048.

Partner investment

We controlled for main effects of perceived partner involvement as well as its interaction effects with hormones by adding them to their respective models (Tables C13 – C16).

Main effects of women's investment remained significant across all models: LF, b = .45, t(384.44) = 1.91 p < 0.057; PSII, b = .59, t(355.39) = 3.21 p < 0.001; PRQC, b = .64, t(359.02) = 2.66 p < 0.008; bond, b = .61, t(355.54) = 3.04 p < 0.003. In the LF, PSII and PRQC models, no interaction effects were observed for either women or partner investment, however progestin appears to moderate the relationship with perceived partner bond and sexual frequency, b = .90, t(384.37) = 1.83 p < 0.068.

The significant main effects of women's investment are in line with the conclusion made by Grøntvedt et al. (2016) that women's investment appears to be the greater predictor of sexual frequency than that partner investment. Women's self-ratings and their ratings of their partners' investment correlate highly on all scales, but LF. The aggregated correlations between couples are: on LF, r = .30; on PSII, r = .62; on PRQC, r = .73 and for bond, r = .66.

Robustness checks

Masturbation

As extended sexuality pertains to potentially conceptive sex, we ran a robustness check to control whether the predictors and interaction effects used in our between-women analyses on sexual frequency would differ when applied to masturbation frequency (n = 395 observations from 268 respondents). The dependent variable of masturbation frequency (M = 1.54, SD = 2.26, *Median* = 1, range = 0 – 20) was not normally distributed with skewness of 2.86 and kurtosis of 16.47. In all four models, only Days unable had a significant effect on masturbation frequency. No main effects of

investment or interactions between hormones and investment were observed. Masturbation frequency is not a function of relationship investment. See Tables C17 – C20.

Withdrawal bleeding and menstruation

Respondents were asked whether they were currently experiencing withdrawal bleeding as well as whether they had experienced their period in the last 7 days. There were inconsistencies in responding, therefore, we excluded observations based on whether participants reported positively to one or both questions resulting in n = 189 observations from 153 women (Tables C21 – C24).

The main effect of LF remained significant, b= .64 t(169.38)= 2.21 $p < 0.029$, however interactions remained insignificant. The main effect of PSII and its interactions with EE and P remained significant. For PSII, b= .80 t(154.97)= 3.77 $p < 0.000$, for EE x PSII, b= -1.77 t(156.42)= -3.02 $p < 0.003$ and for P x PSII, b= 1.79 t(158.54)= 3.17 $p < 0.002$. The main effect of PRQC and EE x PRQC interaction remained significant. For PRQC, b= .74 t(163.99)= 3.74 $p < 0.000$ and EE x PRQC, b= -1.64 t(141.68)= -3.12 $p < 0.002$. The interaction between P and PRQC became significant, b= 1.35 t(148.11)= 2.67 $p < 0.008$. The main effect of bond strength, b= .84 t(159.05)= 3.46 $p < 0.001$, EE x bond, b= -2.02 t(151.20)= -2.77 $p < 0.006$ and P x bond, b= 1.75 t(146.13)= 2.95 $p < 0.004$.

In comparison to the analyses without this exclusion (Tables 2 – 5), the observed effects in this analysis are stronger than when women not currently exposed to exogenous hormones were included.

Effects over time

The relationship involvement score at T1 for partners on all scales was significant. The change in women's involvement between T1 and T2 was significant in the PSII, PRQC and bond strength models, but not in the LF model. The results indicate that a partner's previous investment and changes in the female partner's investment predict the male partner's future investment. In all models, changes in sexual frequency did not predict a change over time in partner investment, thus refuting H2. See Tables 15 – 18.

Table 15

Post-test regression of change over time in partner's loyalty/faithfulness

Model	*F*	*df*	*p*	*Adj. R* ^{2}
Loyalty/Faithfulness model	98.80	5, 134	0.000	0.7787
Variables	B	*t*	*p*	*CI 95%*
Difference in sexual frequency	.01	0.70	0.487	[-.01, .02]
Difference in days unable	-.002	-0.30	0.768	[-.02, .01]
Total relationship length at T2	*-.002*	*-1.69*	*0.094*	*[-.00, .00]*
Difference in woman's LF	.06	1.22	0.223	[-.03, .16]
Partner's LF at T1	**.89**	**21.89**	**0.000**	**[.80, .96]**

Note. Coefficient values have been rounded to the second decimal place. Confidence interval values have been truncated to the second decimal place.

Bold: $p < 0.05$, italics: $p < 0.10$

Table 16

Post-test regression of change over time in partner's Partner Specific Investment Inventory score

Model	*F*	*df*	*p*	*Adj. R* ^{2}
PSII Model	72.31	5, 134	0.000	0.7195
Variables	B	*t*	*p*	*CI 95%*
Difference in sexual frequency	.01	0.91	0.366	[-.00, .02]
Difference in days unable	-.01	-0.72	0.473	[-.01, .00]
Total relationship length at T2	*-.002*	*-1.95*	*0.053*	*[-00, .00]*
Difference in woman's PSII	**.42**	**4.27**	**0.000**	**[.22, .60]**
Partner's PSII at T1	**.89**	**18.39**	**0.000**	**[.79, .98]**

Note. Coefficient values have been rounded to the second decimal place. Confidence interval values have been truncated to the second decimal place.

Bold: $p < 0.05$, italics: $p < 0.10$

Table 17

Post-test regression of change over time in partner's Perceived Relationship Quality Component score

Model	*F*	*df*	*p*	*Adj. R* ²
PRQC model	61.56	5, 134	0.000	0.6854
Variables	B	*t*	*p*	*CI 95%*
Difference in sexual frequency	-.001	-0.13	0.895	[-.02, .02]
Difference in days unable	-.01	-0.42	0.678	[-.03, .01]
Total relationship length at T2	*-.003*	*-1,93*	*0.055*	*[-.00, .00]*
Difference in woman's PRQC	**.52**	**6.09**	**0.000**	**[.35, .69]**
Partner's PRQC at T1	**.87**	**16.78**	**0.000**	**[.76, .97]**

Note. Coefficient values have been rounded to the second decimal place. Confidence interval values have been truncated to the second decimal place.

Bold: $p < 0.05$, italics: $p < 0.10$

Table 18

Post-test regression of change over time in partner's bond strength

Model	*F*	*df*	*p*	*Adj. R* ²
Bond strength model	45.80	5, 134	0.000	0.6171
Variables	B	*t*	*p*	*CI 95%*
Difference in sexual frequency	-.001	-0.23	0.816	[-.03, .02]
Difference in days unable	.003	0.26	0.798	[-.02, .03]
Total relationship length at T2	-.002	-1.55	0.123	[-.00, .00]
Difference in woman's bond	**.44**	**5.70**	**0.000**	**[.28, .59]**
Partner's bond at T1	**.74**	**13.74**	**0.000**	**[.63, .84]**

Note. Coefficient values have been rounded to the second decimal place. Confidence interval values have been truncated to the second decimal place.

Bold: $p < 0.05$

Discussion

The results from our between-women analyses support our hypothesis that synthetic progestins moderate the relationship between PSII, PRQC and bond strength with sexual frequency such that high levels of progestin paired with high investment should lead to greater sexual frequency, whereas high progestin and low investment should lead to a decrease in frequency. We did not replicate the interaction effect between P and LF observed by Grøntvedt et al. (2016). They found this interaction in the longitudinal version of their study as well as in their combined linear mixed model analyses. While LF is not a measure of current relationship investment, individuals high in LF should be more inclined to invest in their relationships, therefore a significant interaction between P and LF could be more easily when observing couples over a longer period of time.

The PSII had the strongest effects of all three models and captures a diversity of both concrete and symbolic evolutionarily meaningful investment units such as giving of time, being nurturing, honest, social attention, sexual proceptivity and having a future-oriented outlook on the relationship (Ellis, 1998). Nonetheless, these three scales correlated highly with one another for both men and women indicating that they capture overlapping concepts.

The negative interactions of EE with PSII, PRQC and bond strength indicate that higher dosages of oestradiol and low levels of investment yield greater sexual frequency, whereas high EE and high investment reduced sexual frequency, in line with predictions made by the dual-mating hypothesis that women should experience heightened interest for uncommitted sexual encounters midcycle.

The absence of within-women differences can perhaps be attributed to the fact that we only took two measured two months apart. This was likely too short to observe changes in HC use and investment, additionally since our sample was composed of mainly students during the semester, life situations were maintained stable. Within-women analyses may be more important in studies of naturally cycling women due to the frequent hormonal fluctuations.

The current study controlled for the differential effects that oral contraceptives may have in contrast to non-oral HC due to differences in hormone metabolism as well as how they differ in androgenetic effects. When running the analyses on observations of oral HC, all previously observed main effects and interaction effects became insignificant. When running the analyses on the sample taking non-oral HC, androgenetic main effects and interactions reached significance or near significance. Most oral contraceptives contain EE, however among the non-oral contraceptives in our sample, only the vaginal rings: NuvaRing and Ornibel, and the Evra patch contain EE and these consisted of only 9 of the total non-oral HC observations (n = 214). Additionally, every IUD in our sample contained levonorgestrel which is the progestin with the greatest androgenetic effects; it represented 128 observations causing androgenetic effects to have been overrepresented in this sample. Significant and near significant main effects of androgenicity were also observed in our analyses excluding those experiencing withdrawal bleeding or periods. Withdrawal bleeding only occurs in women during their placebo pill or pill-free week when taking oral contraception, or when they remove their vaginal ring or patch in the case of non-oral HC. Therefore, levonorgestrel in IUDs were also overrepresented in this sample. While the androgenetic effects of synthetic progestin are unknown (Stanczyk, 2003), they appeared to have a strong moderating effect such that women on an higher dose of an androgenetic progestin with high levels of investment should have higher sexual

frequency. This may be indicative of the role of androgens on women's sexual desire. While increases in testosterone typically coincide with those of oestradiol, main effects of androgenicity and the positive moderating effect of androgenicity on investment indicate that androgens may act independently from oestradiol on desire in the same manner that testosterone supplementation without oestradiol is used to treat loss of desire in post-menopausal women (Bancroft & Graham, 2011).

Despite these issues with mode of administration, we observed a dose-dependent effect of progestin which potentiated the relationships between investment and sexual frequency and these interactions were not observed when masturbation frequency was the dependent variable. Main effects of EE and androgenicity as well as interaction of these with investment did not appear even if masturbation has been associated to the midcycle peak in oestradiol and testosterone (Van Goozen et al., 1997).

Additionally, when excluding based on withdrawal bleeding and period, all main effects and interaction effects remained significant and coefficients increased suggesting that hormonal effects were even more pronounced during recent exogenous hormone exposure than when women not currently ingesting hormones were included indicating that the latency between hormone exposure and behaviour lasts between days to hours resembling the latency of endogenous hormones (Roney & Simmons, 2013).

Sociosexuality did not appear to influence sexual frequency or interact with hormones. Our sample included only committed couples who may have had a more restricted sociosexual orientation to begin with or changed after meeting their partner, in concordance with earlier observations made by Grøntvedt et al. (2016) and Ellis (1998).

In the second hypothesis, we predicted that if the function of extended sexuality is to promote increases in partner involvement through sexual activity, then partner investment should be predicted by sexual frequency. Contrary to our predictions, partner investment at T2 was predicted by partner investment at T1 and changes in women's investment between sessions. Sexual frequency did not contribute to increased investment. The two-month interval and two measurement sessions may not have been sufficient to capture patterns of change in investment and sexual activity. When we controlled for partner investment in the mixed model analysis, women's investment on all four scales maintained its main effect on sexual frequency implying that changes in women's relationship satisfaction should be reflected in sexual frequency over time. As relationships progress, women become the primary initiators of sex (Grøntvedt, Kennair, & Bendixen, 2020), so over the course of a relationship, changes in sexual frequency may affect partner investment while also controlling for the fact that sexual frequency over time tends to decline regardless of investment status (Diamond, 2004). On the other hand, the effects of sexual frequency on partner involvement should be greatest and most critical in the early stages of a relationships. If male investments are supposed to secure sexual access to a woman, then a lack in sexual access should lead to fewer male investments, however a lack in male investments should also lead to lowered sexual access. From a historical perspective, women are most vulnerable to declining male investments and should be especially sensitive to changes in investment in the early stages of a pair-bond to avoid pregnancy with an uncommitted male (Benshoof & Thornhill, 1979). Therefore, a lack in female investment may not necessarily a sign of her own unwillingness to invest, but a reaction to declines in male investment. Whether a woman responds to declining male

investment by being sexually withholding or proceptive, as was observed by Grebe et al. (2013), may be a function of attachment type rather than her own attachment bond or how she perceives that of her partner's in the current relationship. The high correlations between women and their perceptions of their partners' investment suggest that investment between partners may be mutually reinforcing as investments become safer and sex reflects overall relationship quality (Lawrance & Byers, 1995; Sprecher, 2002). Therefore, one partner's investment can be indicative of the couple's investment as a whole, and our results are in line with those of Jones et al. (2005) in that progesterone predisposes women towards commitment cues and this responsive relational climate contributes to women's sexual arousal (Birnbaum et al., 2016; Lawrance & Byers, 1995).

The hormonal regulation of women's sexuality is subject to debate. Studies have found in-pair and extra-pair preferences at midcycle regardless of the mate value of the primary partner (Arslan et al., 2018; Roney & Simmons, 2016; Shimoda et al., 2018), implying that extended sexuality would indeed be a continuation of oestrus sexuality with heightened desire at midcycle, but changes in mate preference or motivation would be decoupled from hormonal effects altogether. Others argue that, while they exist, hormonal effects are obscured by environmental circumstances (Hill, 1988). In species in which females are the primary sexual initiators, cyclical effects tend to be more prominent, but in those where copulation results from female receptivity to male advances, these effects are less apparent. In naturalistic environments, male orangutans typically sexually coerce females, but kept in a captive environment where females had to travel through an enclosure that was too small for the males, females crawled over to mate during their midcycle (Nadler, 1977). Additionally, in rhesus monkeys kept in pairs, when a female was given a progestin-based contraceptive, females received consistent sexual attention, but in a naturalistic environment with multiple naturally cycling and contraceptive-using females, males mated more frequently and with females experiencing their follicular and periovulatory phases (Wallen, 1982). Therefore, Hill (1988) argues that human monogamy maintains couples in close proximity. In single women and in societies where female proceptivity is normalised cyclicity can be observed more readily (Caruso et al., 2014; Hill, 1988). However, Norwegian women enjoy some of the highest levels of gender equality and sexual freedom in the world (Grøntvedt et al., 2015; Kennair, Schmitt, Fjeldavli, & Harlem, 2009) and the use of contraception, whether hormonal or not, enable women to act even more freely upon their sexual motivations. Therefore, extended sexuality trends in sexual behaviour among Norwegian women can be assumed to stem from their own intrinsic motivations.

A challenge in evolutionary psychology is attempting to adapt evolutionarily relevant concepts and situations to modern times. Women from modern hunter-gatherer tribes experience about 100 ovulatory cycles in their lifetime, in contrast to western women who experience ca. 400. Tribal women spend more time being pregnant and lactating and lack the nutrition to reliably produce ovarian hormones (Strassmann, 1997). Additionally, the hormonal levels experienced during the menstrual cycle in which extended sexuality appear may not be the same as those today due to environmental pressures (Wardecker, Smith, Edelstein, & Loving, 2015). The sexual frequency of respondents in this study was comparable to those obtained in other studies of sexual frequency in young western couples and sexual frequency has been showing a steady decline over the last three decades (Call et al., 1995; Træen, Martinussen, Öberg, & Kavli, 2007; Twenge, Sherman, & Wells, 2017). Multiple factors may be responsible for this, but this may have consequences for research on extended sexuality. Intimacy in couples can also be displayed through non-sexual care-taking

behaviours (Shimoda et al., 2018), therefore these behaviours may be fulfilling the same investment roles as sexual activity.

Because human extended sexuality can be characterised by different hormonal profiles ranging from adolescence to menopause, there may also be multiple types of extended sexuality. Pleasure and mood were greater reported predictors of desire and motivation in older women, while younger women's arousal was more dependent on partner-related themes such as men's grooming or personality possibly reflecting the lifespan changes in hormonal status (Graham, Sanders, Milhausen, & McBride, 2004; Meston & Buss, 2007). Therefore, menopausal extended sexuality may be a by-product of oestrus sexuality, whereas a luteal phase extended sexuality focused on maintaining bonds and continued investment is likely to be more important to reproductive age women.

Strengths and Limitations

The current study builds upon the work done by Grøntvedt et al. (2016) by examining and comparing different measures of current relationship investment in women using hormonal contraception. Because hormone levels during the menstrual cycle are sensitive to changes in a woman's environment (Fenster et al., 1999), establishing direction of causality can be challenging. As hormonal contraceptives inhibit endogenous cycle fluctuations and eliminate between-cycle differences in absolute hormonal levels, they allow us to study the *a priori* effects of hormones. We found significant and opposite interaction effects of progestins and ethinyl oestradiol with investment indicative of qualitative differences between fertile and non-fertile sexuality as well as being regulated by different hormones.

Studying women taking HCs allows for simple and cheap control of hormonal levels, but it is not a panacea. While earlier research on the effects of hormonal contraceptives have treated them as a homogenous group, distinguishing HC by administration type and androgenicity may not be sufficient. Different progestins appear to bind differently on the multiple progesterone receptors as well as other receptors and have varying pharmacokinetics, bioavailability and metabolism which have been shown to have differential effects on women's affect, cognition and behaviour (see reviews by Mitchell and Welling (2020) and Stanczyk (2003)). Therefore, synthetic progestins are not necessarily equivalent to endogenous progesterone or other types of synthetic progestins in their effects on women's psychology and future research should attempt to divide HC-users according to progestin type and aim to identify which synthetic progestin best mimics the psychological effects associated to natural progesterone.

In the same line of thinking, HCs tend to flatten hormone levels throughout the cycle. The cyclical effects of hormonal fluctuations on sexual desire and mood appear to have more important effects than absolute levels (Roney & Simmons, 2013; Rubinow & Schmidt, 2006). While oestrogen and progesterone have been documented to have opposing effects on sexual desire in naturally cycling women, peak levels of oestradiol and progestin occur in separate phases of the menstrual cycle, whereas in combined oral contraceptives, their peaks coincide. Additionally, our findings in the non-oral HC group indicates that the androgenicity of progestins may have a novel effect on women's psychology not otherwise observed in the luteal phase. The implications of these effects on sexual behaviour remain to be resolved, though our results suggest that EE and progestins have differing effects on women's sexual behaviour even when presented together.

Sexual and masturbation frequency was non-normally distributed. We nonetheless opted to use a linear mixed model analysis as was used in Grøntvedt et al. (2016) and as appears to be commonly used in the behavioural sciences (Arnau et al., 2012). Due to the risk of a flooring effect as many respondents did not report having sex or masturbating within the last 7 days, a Poisson mixed effect model could be a suitable alternative.

Conclusion

How women feel about their relationship and partner matters to women when engaging in sexual intercourse, however this becomes even more important to them in high-progesterone states such as in the luteal phase or on hormonal contraceptives. Our results have shown that synthetic progestins do enhance the effects of women's current relationship investment on sexual frequency, lending credence to the male-assistance hypothesis (Rodríguez-Gironés & Enquist, 2001) and the progesterone-regulation of ES (Grebe et al., 2013). As the luteal phase is characterised by progesterone levels that are similar to those observed in early pregnancy, it logically ensues that this hormone would be involved in the maintenance of the necessary pair-bonds required for the survival of mother and child. Progesterone has been associated with lowered levels of sexual desire, and the differential predictive effects of our independent variables on sexual frequency and masturbation indicate that sexual behaviour was not motivated by the same factors as masturbation which is typically linked to a desire for pleasure. These progestogenic effects were even stronger in women exposed to hormones during the week preceding the survey. We did not however observe longitudinal effects of sexual frequency on women's perceptions of their partner's investment. The current study, as well those by Grebe et al. (2013) and Grøntvedt et al. (2016) have shown promising results in our understanding of women's extended sexuality. We propose that future research into extended sexuality should control for women's attachment style as this may affect how they respond to threats to their relationship and caution should be taken when comparing exogenous and endogenous hormones as their effects may not be equivalent.

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Appendices

Appendix A: Consent forms for both rounds of the survey

Appendix B: Items for the relationship investment measures in Norwegian and English for women's and perceived partner investment

Appendix C: Tables for the control of other variables in the mixed model analysis of Hypothesis 1

Appendix A

Bruk av hormonell prevensjon, forholdstilfredshet og frekvens av seksuelt samleie

Formålet med denne spørreundersøkelsen er å få mer kunnskap om sammenhengen mellom bruk av hormonell prevensjon, kvaliteter ved parforhold, personlighetstrekk og seksualitet. Undersøkelsen er et samarbeid mellom norske og amerikanske forskere. Spørsmålene i spørreskjema handler om deg, om din opplevelse av ditt parforhold, prevensjonsbruk, personlighet og symptomer på depresjon, og seksuell atferd. Vennligst svar så ærlig som mulig, selv om noen av spørsmålene kan virke nokså nærgående. Resultatene fra undersøkelsen vil bli presentert som vitenskapelige artikler i tidsskrift.

Det er ingen kjent risiko knyttet til å delta. Det er frivillig å delta, og du står fritt til å svare på de spørsmålene du selv ønsker. Du kan når som helst trekke deg fra undersøkelsen uten at det vil få noen konsekvenser for deg. Det tar ca. 25 minutter å svare på spørreskjemaet.

Når du har fullført denne delen av spørreskjema blir du sport om å oppgi e-postadressen din. I løpet av 2 måneder vil vi kontakte deg igjen for å be deg om å svare på et nytt spørreskjema som er omtrent like langt som dette. Alle som gjennomfører begge spørreskjemaene er med i trekningen av 2 stk nettbrett. Vinnerne kontaktes pr e-post, og deretter slettes e-postene fra databasen.

Bortsett fra e-postadressen registreres ingen personidentifiserende opplysninger. E-postadressen slettes ved overføring av data for statistiske analyser 2 uker etter datainnsamlingens slutt (senest 01.03.2020). Så lenge du kan identifiseres i datamaterialet, har du rett til:

- innsyn i hvilke personopplysninger som er registrert om deg,
- å få rettet personopplysninger om deg,
- å få slettet personopplysninger om deg,
- å få utlevert en kopi av dine personopplysninger (dataportabilitet), og
- å sende klage til personvernombudet eller Datatilsynet om behandlingen av dine personopplysninger.

Hva gir oss rett til å behandle personopplysninger om deg? Vi behandler opplysninger om deg basert på ditt samtykke.

NTNU er behandlingsansvarlig for undersøkelsen, og NTNUs personvernombud er Thomas Helgesen (tlf. 930 79 038).

Har du spørsmål om undersøkelsen, kontakter du postdok Trond Viggo Grøntvedt, tlf. 975 08 084 eller professor/psykologspesialist Leif Edward Ottesen Kennair, tlf. 73 59 19 56 ved institutt for psykologi ved NTNU.

Takk for at du er villig til å delta i undersøkelsen!

Trond Viggo Grøntvedt, postdoktor, NTNU
Leif Edward Ottesen Kennair, professor, NTNU
Mons Bendixen, førsteamanuensis, NTNU
Tiffany Lussier, masterstudent, NTNU

Runde 2 for Bruk av hormonell prevensjon, forholdstilfredshet og frekvens av seksuelt samleie

Takk for at du oppga e-postadresse i forrige runde. Du mottar nå et nytt spørreskjema som vi ber deg om å besvare. Vær oppmerksom på at spørsmålene er ganske like de du fikk forrige gang, men vi ber deg lese nøre gjennom spørsmålene.

Formålet med denne spørreundersøkelsen er å få mer kunnskap om sammenhengen mellom bruk av hormonell prevensjon, kvaliteter ved parforhold, personlighetstrekk og seksualitet. Undersøkelsen er et samarbeid mellom norske og amerikanske forskere. Spørsmålene i spørreskjema handler om deg, om din opplevelse av ditt parforhold, prevensjonsbruk, personlighet og symptomer på depresjon, og seksuell atferd. Vennligst svar så ærlig som mulig, selv om noen av spørsmålene kan virke nokså nærgående. Resultatene fra undersøkelsen vil bli presentert som vitenskapelige artikler i tidsskrift.

Det er ingen kjent risiko knyttet til å delta. Det er frivillig å delta, og du står fritt til å svare på de spørsmålene du selv ønsker. Du kan når som helst trekke deg fra undersøkelsen uten at det vil få noen konsekvenser for deg. Det tar ca. 25 minutter å svare på spørreskjemaet.

Bortsett fra e-postadressen registreres ingen personidentifiserende opplysninger. E-postadressen slettes ved overføring av data for statistiske analyser 2 uker etter datainnsamlingens slutt (senest 01.03.2020). Så lenge du kan identifiseres i datamaterialet, har du rett til:

- innsyn i hvilke personopplysninger som er registrert om deg,
- å få rettet personopplysninger om deg,
- å få slettet personopplysninger om deg,
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Tiffany Lussier, masterstudent, NTNU

Appendix B

Loyalty/Faithfulness

Beskriv deg selv så godt som mulig ved å krysse av på skalaen fra 1 til 7 for hvert av de 17 adjektivene/utsagnene nedenfor:

Beskriv din partner så godt som mulig ved å krysse av på skalaen fra 1 til 7 for hvert av de 17 adjektivene/utsagnene nedenfor:

1. Veldig uenig
2. Uenig
3. Litt uenig
4. Nøytral
5. Litt enig
6. Enig
7. Veldig enig

Norsk	**English**
Trofast mot partner	Faithful
Lojal	Loyal

Note. This table only includes the two items of the Mate Value Inventory (Kirsner et al., 2003) relevant for the Loyalty/Faithfulness measure of relationship involvement.

Partner Specific Investment Inventory

Beskriv på en skala fra 1 til 5, din atferd overfor din partner:

Beskriv, på en skala fra 1 til 5, partnerens atferd overfor deg:

1. Aldri
2. Sjelden
3. Noen ganger
4. Ganskje ofte
5. Veldig ofte

Women's PSII

Norwegian	**English**
Jeg deler mine følelser med min partner.	I share my feelings with my partner.
Jeg oppfører meg frekt mot min partner.	I am rude toward my partner.
Med min partner er jeg en villig og entusiastisk seksualpartner.	With my partner, I am a willing and enthusiastic sexual partner.
Jeg lyver til min partner om viktige ting.	I lie to my partner about important things.
Jeg trøster min partner når han er fortvilet.	I comfort my partner when he is distressed.
Jeg prøve å vri meg ut av små løgner jeg forteller min partner.	I tell my partner little lies and then try to wiggle out of them.
Jeg foretrekker å tilbringe fritiden min med mine venner heller enn med min partner.	I prefer to spend my free time with my friends rather than with my partner.
Når jeg prater om min fremtid er min partner alltid inkludert.	When I talk about my future, my partner is always in it.
Jeg har liksom ikke tid til min partner.	I cannot seem to find time for my partner.
Jeg er ikke seksuelt responsiv ovenfor min partner.	I am not sexually responsive toward my partner.
Jeg stoler ikke på min partner.	I don't trust my partner.
Jeg ønsker ikke å diskutere fremtiden min med min partner.	I won't discuss my future with my partner.

Perceived Partner PSII

Norwegian	**English**
Partneren min deler sine følelser med meg.	My partner shares his feelings with me.
Partneren min oppfører seg frekt mot meg.	My partner is rude toward me.
Med meg er min partner en villig og entusiastisk seksualpartner.	My partner is a willing and enthusiastic sexual partner with me.
Partneren min lyver til meg om viktige ting.	My partner lies to me about important things.
Partneren min trøster meg når jeg er fortvilet.	My partner comforts me when I am distressed.
Partneren min prøver å vri seg ut av små løgner han forteller meg.	My partner tells me little lies and then tries to wiggle out of them.
Partneren min foretrekker å tilbringe sin fritid med sine venner heller enn med meg.	My partner prefers to spend his free time with his friends rather than with me.
Når min partner prater om sin fremtid er jeg alltid inkludert.	When my partner talks about the future, I am always in it.
Partneren min har liksom ikke tid til meg.	My partner cannot seem to find time for me.
Partneren min er ikke seksuelt responsiv ovenfor meg.	My partner is not sexually responsive toward me.
Partneren min stoler ikke på meg.	My partner doesn't trust me.
Partneren min ønsker ikke å diskutere fremtiden sin med meg.	My partner won't discuss his future with me.

Perceived Relationship Quality Components

Beskriv din oppfatning av ditt parforhold på en skala fra 1 til 7:

Beskriv hvordan du syns partneren din oppfatter deres parforhold på en skala fra 1 til 7:

1. Ikke i det hele tatt
2. Veldig lite
3. Lite
4. Nøytral
5. Mye
6. Veldig mye
7. Ekstremt

Women's PRQC

Norsk	**English**
Hvor tilfreds er du med parforholdet ditt?	How satisfied are you with your relationship?
Hvor forpliktet er du i ditt parforhold?	How committed are you to your relationship?
Hvor nært knyttet er du og partneren din?	How intimate is your relationship?
Hvor lidenskapelig er du i ditt parforhold?	How passionate is your relationship?
Hvor mye elsker du partneren din?	How much do you love your partner

Perceived Partner PRQC

Norsk	**English**
Hvor tilfreds er din partner med deres parforhold?	How satisfied is your partner with your relationship?
Hvor forpliktet er din partner i deres parforhold?	How committed is your partner to your relationship?
Hvor nært knyttet er din partner til deg?	How intimate is your partner to you?
Hvor lidenskapelig er din partner i deres parforhold?	How passionate is your partner in your relationship?
Hvor mye elsker din partner deg?	How much do you love your partner?

Bond Strength

Instruksjoner: reflektere over ditt nåværende parforhold og angi på en skala fra 1 til 7 hvor enig du er i påstanden.

1. Helt uenig
2. Veldig uenig
3. Uenig
4. Nøytral
5. Enig
6. Veldig enig
7. Helt enig

Women's bond strength

Norwegian	**English**
Min partner er den personen jeg vil gå til for å få meg til å føle meg bedre når noe vondt har hendt meg eller jeg føler meg opprørt.	My partner is the person that I would want to go to, to help me feel better when something bad happens to me or I feel upset.
Partneren min er den første personen jeg tenker på når jeg har et problem.	My partner is the first person I think of when I have a problem.
Min partner er en person som jeg ikke liker å være borte fra.	My partner is a person whom I do not like to be away from.
Min partner er den personen jeg faktisk kan stole på at alltid er der for meg og bryr seg om meg uansett hva som skjer.	My partner is the person that I would actually count on to always be there for me and care about me no matter what.

Partner's perceived bond strength

Norwegian	**English**
Jeg er den personen min partner vil gå til for å få seg til å føle seg bedre når noe vondt har hendt han/hun eller han/hun føler seg opprørt.	I am the person that my partner would want to go to, to help him feel better when something bad happens to him or he feels upset.
Jeg er den første personen min partner tenker på når han/hun har et problem.	I am the first person my partner thinks of when he has a problem.
Jeg er en person som min partner ikke liker å være borte fra.	I am a person whom my partner does not like to be away from.
Jeg er den personen min partner faktisk kan stole på at alltid er der for ham/henne og bryr seg om ham/henne uansett hva som skjer.	I am the person that my partner would actually count on to always be there for him and care about him no matter what.

APPENDIX C

Administration type

Oral contraceptives

Table C1

Between-women effects on sexual frequency: Loyalty/Faithfulness model for oral HC

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.37**	**-5.97**	**183.57**	**0.000**	**[-.49, -.24]**
Relationship length	**-.71**	**-2.46**	**142.11**	**0.015**	**[-1.28, -.14]**
LF	.15	0.19	134.41	0.850	[-1.38, 1.67]
Ethinyl Oestradiol (EE)	.56	0.77	142.38	0.441	[-.86, 1.98]
Progestin (P)	-.62	-0.60	120.91	0.549	[-2.67, 1.42]
Androgenicity (A)	-.32	-1.07	178.01	0.288	[-.92, .27]
Interaction effects					
EE x LF	-.57	-0.53	144.72	0.594	[-2.67, 1.53]
P x LF	.43	0.27	136.04	0.785	[-2.57, 3.52]
A x LF	-.01	-0.01	184.81	0.990	[-.86, .85]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except “Days unable”.
Bold: $p < 0.05$

Table C2

Between-women effects on sexual frequency: Partner-Specific Investment Inventory model for oral HC

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.37**	**-6.20**	**182.07**	**0.000**	**[-.49, -.25]**
Relationship length	**-.62**	**-2.19**	**141.85**	**0.030**	**[-1.17, -.05]**
PSII	.58	1.29	141.37	0.200	[-.31, 1.47]
Ethinyl Oestradiol (EE)	.51	0.85	132.34	0.394	[-.66, 1.68]
Progestin (P)	-.56	-0.64	123.34	0.526	[-2.30, 1.18]
Androgenicity (A)	-.30	-1.45	131.78	0.150	[-.69, .10]
Interaction effects					
EE x PSII	*-1.15*	*-1.80*	*156.05*	*0.074*	*[-2.41, .11]*
P x PSII	1.18	1.28	157.41	0.204	[-.64, 3.01]
A x PSII	-.09	-0.50	151.35	0.616	[-.45, .27]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$, italics: $p < .10$

Table C3

Between-women effects on sexual frequency: Perceived Relationship Quality Components model for oral HC

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.36**	**-5.99**	**181.67**	**0.000**	**[-.48, -.24]**
Relationship length	**-.72**	**-2.59**	**140.78**	**0.011**	**[-1,27, -.17]**
PRQC	.51	1.14	134.66	0.256	[-.37, 1.39]
Ethinyl Oestradiol (EE)	.41	0.68	134.14	0.496	[-.77, 1.58]
Progestin (P)	-.56	-0.63	123.11	0.142	[-2.29, 1.18]
Androgenicity (A)	-.30	-1.48	135.96	0.527	[-.70, .10]
Interaction effects					
EE x PRQC	*-1.15*	*-1.89*	*159.14*	*0.060*	*[-2.35, .05]*
P x PRQC	1.06	1.19	138.62	0.237	[-.70, 2.81]
A x PRQC	-.06	-0.29	176.71	0.772	[-.43, .32]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$, italics: $p < .10$

Table C4

Between-women effects on sexual frequency: Bond strength model for oral HC

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.37**	**-5.99**	**183.22**	**0.000**	**[-.48, -.24]**
Relationship length	**-.77**	**-2.70**	**158.09**	**0.008**	**[-1.34, -.20]**
Bond	.42	0.97	169.95	0.335	[-.43, 1.27]
Ethinyl Oestradiol (EE)	.44	0.71	135.78	0.479	[-.78, 1.67]
Progestin (P)	-.33	-0.36	128.60	0.719	[-2.16, 1.49]
Androgenicity (A)	-.32	-1.51	133.86	0.133	[-.73, .09]
Interaction effects					
EE x Bond	-.78	-1.18	168.15	0.240	[-2.08, .52]
P x Bond	.73	1.84	159.92	0.401	[-.98, 2.44]
A x Bond	-.20	-0.84	184.84	0.404	[-.67, .27]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$

Non-oral contraceptives

Table C5

Between-women effects on sexual frequency: Loyalty/Faithfulness model for non-oral HC

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.44**	**-8.11**	**203.16**	**0.000**	**[-.55, -.33]**
Relationship length	**-1.56**	**-5.21**	**141.41**	**0.000**	**[-2.17, -.97]**
LF	2.40	0.94	172.28	0.349	[-2.63, 7.43]
Ethinyl Oestradiol (EE)	-1.65	-0.75	194.95	0.457	[-6.01, 2.71]
Progestin (P)	*-1.64*	*-1.70*	*143.70*	*0.091*	*[-3.55, .26]*
Androgenicity (A)	**6.76**	**1.99**	**131.92**	**0.048**	**[.05, 13.46]**
Interaction effects					
EE x LF	-4.93	-1.39	184.62	0.166	[-11.92, 2.06]
P x LF	.55	0.41	199.58	0.683	[-2.11, 3.21]
A x LF	**9.21**	**2.28**	**177.77**	**0.024**	*[1.22, 17.18]*

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$, italics: $p < .10$

Table C6

Between-women effects on sexual frequency: Partner-Specific Investment Inventory model for non-oral HC

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.45**	**-8.26**	**203.39**	**0.000**	**[-.56, -.34]**
Relationship length	**-1.45**	**-4.74**	**139.37**	**0.000**	**[-2.04, -.84]**
PSII	*2.07*	*1.78*	*190.41*	*0.077*	*[-.22, 4.37]*
Ethinyl Oestradiol (EE)	-1.59	-0.86	159.67	0.392	[-5.23, 2.06]
Progestin (P)	-1.14	-1.26	137.48	0.211	[-2.93, .65]
Androgenicity (A)	*6.19*	*1.82*	*135.88*	*0.071*	*[-.54, 12.94]*
Interaction effects					
EE x PSII	-1.92	-1.10	184.42	0.272	[-5.36, 1.51]
P x PSII	.01	0.01	172.03	0.990	[-1.85, 1.87]
A x PSII	*5.60*	*1.71*	*160.83*	*0.090*	*[-.88, 12.07]*

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$, italics: $p < .10$

Table C7

Between-women effects on sexual frequency: Perceived Relationship Quality Components model for non-oral HC

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.46**	**-8.30**	**203.91**	**0.000**	**[-.57, -.35]**
Relationship length	**-1.54**	**-4.84**	**139.86**	**0.000**	**[-2.16, -.90]**
PRQC	1.88	1.05	200.88	0.297	[-1.66, 5.44]
Ethinyl Oestradiol (EE)	-2.33	-1.16	164.92	0.249	[-6.30, 1.64]
Progestin (P)	*-1.05*	*-1.12*	*143.47*	*0.069*	*[-2.89, .80]*
Androgenicity (A)	6.40	1.83	136.52	0.266	[-6.30, 1.64]
Interaction effects					
EE x PRQC	-1.26	-0.52	200.48	0.607	[-6.88, 3.56]
P x PRQC	-1.06	-0.99	196.77	0.325	[-3.18, 1.06]
A x PRQC	*6.29*	*1.70*	*191.72*	*0.091*	*[-1.02, 13.60]*

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$, italics: $p < .10$

Table C8

Between-women effects on sexual frequency: Bond strength model for non-oral HC

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-8.46**	**203.65**	**0.000**	**[-.56, -.35]**
Relationship length	**-1.55**	**-5.00**	**139.14**	**0.000**	**[-2.16, -.93]**
Bond	*2.62*	*1.90*	*194.78*	*0.058*	*[-.09, 5.34]*
Ethinyl Oestradiol (EE)	-1.36	-.70	165.26	0.485	[-5.20, 2.48]
Progestin (P)	-.94	-1.02	139.20	0.310	[-2.76, .88]
Androgenicity (A)	4.97	1.40	135.56	0.164	[-2.04, 11.97]
Interaction effects					
EE x Bond	-3.16	-1.62	186.90	0.107	[-7.00, .68]
P x Bond	-.18	0.15	186.10	0.878	[-2.49, 2.13]
A x Bond	*8.55*	*2.13*	*185.15*	*0.034*	*[.64, 16.45]*

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$, italics: $p < .10$

Sociosexuality Index

Table C9

Between-women effects on sexual frequency: Control for SOI in the Loyalty/Faithfulness model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-10.18**	**393.95**	**0.000**	**[-.50, -.33]**
Relationship length	**-.97**	**-4.59**	**294.91**	**0.000**	**[-1.38, -.55]**
LF	.37	1.54	383.44	0.123	[-.10, .84]
SOI	.02	0.17	301.48	0.867	[-.29, .35]
Ethinyl Oestradiol (EE)	.50	1.07	361.30	0.284	[-.41, 1.41]
Progestin (P)	-.37	-0.90	312.56	0.368	[-1.19, .44]
Androgenicity (A)	-.35	-1.24	381.99	0.214	[-.91, .20]
Interaction effects					
EE x LF	-.97	-1.40	375.60	0.162	[-2.32, .39]
P x LF	.01	0.02	363.81	0.986	[-1.09, 1.11]
A x LF	.53	1.01	393.77	0.315	[-.50, 1.56]
EE x SOI	-.27	-0.57	371.58	0.567	[-1.19, .65]
P x SOI	-.03	-0.06	347.04	0.953	[-.89, .84]
A x SOI	.34	1.43	368.12	0.152	[-.12, .80]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except “Days unable”.
Bold: $p < 0.05$

Table C10

Between-women effects on sexual frequency: Control for SOI in the Partner-Specific Investment Inventory model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-10.45**	**392.33**	**0.000**	**[-.50, -.34]**
Relationship length	**-.90**	**-4.37**	**297.62**	**0.000**	**[-1.30, -.49]**
PSII	**.74**	**4.87**	**321.08**	**0.000**	**[.44, 1.04]**
SOI	.14	0.87	293.87	0.386	[-.17, .45]
Ethinyl Oestradiol (EE)	.48	1.10	337.52	0.271	[-.37, 1.32]
Progestin (P)	-.51	-1.29	300.15	0.200	[-1.27, .26]
Androgenicity (A)	-.21	-0.93	338.32	0.353	[-.66, .23]
Interaction effects					
EE x PSII	**-1.10**	**-2.52**	**325.21**	**0.012**	**[-1.96, -.24]**
P x PSII	**1.02**	**2.41**	**325.15**	**0.016**	**[.18, 1.85]**
A x PSII	-.05	-0.27	381.71	0.786	[-.41, .31]
EE x SOI	-.29	-0.65	381.01	0.514	[-1.16, .58]
P x SOI	.27	0.62	353.48	0.539	[-.59, 1.13]
A x SOI	.15	0.71	376.19	0.478	[-.25, .54]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except “Days unable”.
Bold: $p < 0.05$

Table C11

Between-women effects on sexual frequency: Control for SOI in the Perceived Relationship Quality Components model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-10.21**	**392.27**	**0.000**	**[-.49, -.33]**
Relationship length	**-.98**	**-4.74**	**293.22**	**0.000**	**[-1.38, -.57]**
PRQC	**.58**	**3.74**	**321.33**	**0.000**	**[.27, .87]**
SOI	.07	0.47	291.83	0.642	[-.24, .38]
Ethinyl Oestradiol (EE)	.23	0.54	330.75	0.592	[-.62, 1.09]
Progestin (P)	-.38	-0.95	301.42	0.341	[-1.15, .40]
Androgenicity (A)	-.12	-0.52	335.07	0.602	[-.57, .33]
Interaction effects					
EE x PRQC	**-1.13**	**-2.69**	**361.52**	**0.007**	**[-1.95, -.30]**
P x PRQC	**.83**	**2.08**	**335.07**	**0.038**	**[.04, 1.61]**
A x PRQC	.09	0.45	364.71	0.652	[-.31, .50]
EE x SOI	-.45	-0.98	356.98	0.329	[-1.36, .45]
P x SOI	.26	0.59	339.46	0.553	[-.60, 1.13]
A x SOI	.27	1.25	342.54	0.211	[-.15, .70]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.

All values are z-scored except “Days unable”.

Bold: $p < 0.05$

Table C12

Between-women effects on sexual frequency: Control for SOI in the bond strength model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-10.20**	**393.79**	**0.000**	**[-.49, -.33]**
Relationship length	**-1.04**	**-4.96**	**294.48**	**0.000**	**[-1.45, -.62]**
Bond	**.56**	**3.52**	**317.12**	**0.000**	**[.24, .87]**
SOI	.04	0.27	292.05	0.784	[-.27, .36]
Ethinyl Oestradiol (EE)	.51	1.17	341.90	0.244	[-.35, 1.39]
Progestin (P)	-.50	-1.25	307.27	0.213	[-1.29, .29]
Androgenicity (A)	-.27	-1.18	353.44	0.239	[-.72, .18]
Interaction effects					
EE x Bond	**-1.01**	**-2.11**	**359.03**	**0.036**	**[-1.96, -.06]**
P x Bond	**.83**	**1.98**	**333.15**	**0.048**	**[.00, 1.64]**
A x Bond	-.10	-0.46	382.19	0.649	[-.51, .32]
EE x SOI	-.09	-0.20	382.64	0.843	[-.96, .78]
P x SOI	.06	0.13	352.30	0.897	[-.80, .92]
A x SOI	.11	0.57	362.50	0.571	[-.28, .50]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.

All values are z-scored except “Days unable”.

Bold: $p < 0.05$

Partner Investment

Table C13

Between-women effects on sexual frequency: Controlling for partner's LF

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-10.09**	**393.97**	**0.000**	**[-.50, -.33]**
Relationship length	**-1.02**	**-4.90**	**287.16**	**0.000**	**[-1.43, -.61]**
Partner's LF	.05	0.28	334.63	0.777	[-.29, .40]
Woman's LF	*.45*	*1.91*	*384.44*	*0.057*	*[-.01, .92]*
Ethinyl Oestradiol (EE)	.55	1.15	358.21	0.251	[-.39, 1.51]
Progestin (P)	-.45	-1.08	317.09	0.280	[-1.29, .37]
Androgenicity (A)	-.34	-1.14	371.29	0.240	[-.92, .24]
Interaction effects					
EE x Partner's LF	.08	0.24	308.21	0.813	[-.63, .81]
P x Partner's LF	-.05	-0.15	325.62	0.879	[-.72, .63]
A x Partner's LF	.04	0.15	324.14	0.884	[-.50, .58]
EE x Woman's LF	-.62	-0.89	381.53	0.373	[-1.98, .74]
P x Woman's LF	.09	0.14	380.67	0.885	[-1.09, 1.27]
A x Woman's LF	.09	0.19	388.71	0.853	[-.82, 1.00]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$, italics: $p < 0.10$

Table C14

Between-women effects on sexual frequency: Controlling for partner's PSII

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-10.46**	**393.24**	**0.000**	**[-.50, -.34]**
Relationship length	**-.92**	**-4.52**	**293.27**	**0.000**	**[-1.32, -.52]**
Partner's PSII	.25	1.36	343.37	0.175	[-.11, .60]
Woman's PSII	**.59**	**3.21**	**355.39**	**0.001**	**[.22, .95]**
Ethinyl Oestradiol (EE)	.35	0.84	303.15	0.402	[-.47, 1.17]
Progestin (P)	-.38	-0.98	293.02	0.330	[-1.15, .38]
Androgenicity (A)	-.24	-1.20	287.13	0.231	[-.62, .15]
Interaction effects					
EE x Partner's PSII	-.58	-1.24	394.78	0.215	[-1.49, .33]
P x Partner's PSII	.13	0.31	371.70	0.760	[-.69, .95]
A x Partner's PSII	.16	0.57	386.17	0.572	[-.40, .72]
EE x Woman's PSII	-.50	-0.90	370.67	0.371	[-1.58, .59]
P x Woman's PSII	.75	1.51	351.36	0.132	[-.22, 1.72]
A x Woman's PSII	-.26	-0.91	394.48	0.362	[-.83, .30]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.

All values are z-scored except "Days unable".

Bold: $p < 0.05$

Table C15

Between-women effects on sexual frequency: Controlling for partner's PRQC

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.41**	**-10.00**	**392.00**	**0.000**	**[-.48, -.32]**
Relationship length	**-1.06**	**-5.14**	**286.41**	**0.000**	**[-1.46, -.65]**
Partner's PRQC	-.14	-0.64	368.74	0.523	[-.58, .29]
Woman's PRQC	**.64**	**2.66**	**359.02**	**0.008**	**[.16, 1.11]**
Ethinyl Oestradiol (EE)	.42	1.01	311.17	0.311	[-.39, 1.24]
Progestin (P)	-.40	-1.01	297.16	0.313	[-1.16, .37]
Androgenicity (A)	-.31	-1.53	297.71	0.127	[-.69, .08]
Interaction effects					
EE x Partner's PRQC	.04	0.07	377.26	0.947	[-1.21, 1.29]
P x Partner's PRQC	.48	0.86	370.25	0.392	[-.61, 1.56]
A x Partner's PRQC	-.41	-0.90	354.42	0.370	[-1.29, .48]
EE x Woman's PRQC	-1.10	-1.50	346.44	0.134	[-2.53, .33]
P x Woman's PRQC	.42	0.69	345.65	0.492	[-.77, 1.61]
A x Woman's PRQC	.39	0.79	344.15	0.432	[-.58, 1.36]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.

Bold: $p < 0.05$

Table C16

Between-women effects on sexual frequency: Controlling for partner's bond strength

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-10.19**	**393.98**	**0.000**	**[-.49, -.33]**
Relationship length	**-1.05**	**-5.08**	**288.79**	**0.000**	**[-1.46, -.64]**
Partner's bond	-.09	-0.48	383.14	0.633	[-.45, .27]
Woman's bond	**.61**	**3.04**	**355.54**	**0.003**	**[.21, 1.00]**
Ethinyl Oestradiol (EE)	.59	1.38	321.05	0.168	[-.25, 1.43]
Progestin (P)	-.54	-1.36	304.62	0.176	[-1.32, .24]
Androgenicity (A)	-.31	-1.47	288.85	0.143	[-.72, .10]
Interaction effects					
EE x Partner's bond	-.83	-1.60	367.89	0.110	[-1.85, .19]
P x Partner's bond	*.90*	*1.83*	*384.37*	*0.068*	*[-.06, 1.87]*
A x Partner's bond	.03	0.11	360.64	0.909	[-.49, .55]
EE x Woman's bond	-.55	-0.98	340.46	0.328	[-1.66, .55]
P x Woman's bond	.35	0.72	326.86	0.470	[-.60, 1.31]
A x Woman's bond	-.13	-0.48	394.96	0.632	[-.67, .40]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.

All values are z-scored except "Days unable".

Bold: $p < 0.05$, italics: $p < 0.10$

Masturbation

Table C17

Between-women effects on masturbation frequency: Loyalty/Faithfulness model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**.08**	**2.43**	**377.48**	**0.016**	**[.01, .14]**
Relationship length	.10	0.56	284.79	0.578	[-.24, .44]
LF	-.06	-0.37	383.64	0.713	[-.41, .28]
Ethinyl Oestradiol (EE)	.07	0.19	376.81	0.848	[-.66. .80]
Progestin (P)	-.35	-1.05	321.93	0.295	[-1.01, .31]
Androgenicity (A)	.26	1.16	384.74	0.246	[-.18, .70]
Interaction effects					
EE x LF	.47	0.96	384.60	0.337	[-.49, 1.43]
P x LF	-.11	-0.24	378.62	0.810	[-.97, .75]
A x LF	-.28	-0.85	376.38	0.398	[-.92, .36]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$

Table C18

Between-women effects on masturbation frequency: Partner-Specific Investment Inventory model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**.08**	**2.50**	**376.60**	**0.013**	**[.01, .14]**
Relationship length	.09	0.51	283.63	0.610	[-.25, .43]
PSII	-.11	-0.92	338.95	0.357	[-.36, .13]
Ethinyl Oestradiol (EE)	.19	0.55	338.83	0.582	[-.49, .88]
Progestin (P)	-.35	-1.05	315.03	0.293	[-1.00, .30]
Androgenicity (A)	.12	0.70	313.53	0.485	[-.21, .45]
Interaction effects					
EE x PSII	-.20	-0.56	348.25	0.577	[-.90, .50]
P x PSII	.14	0.40	342.25	0.689	[-.54, .82]
A x PSII	-.06	-0.41	345.64	0.681	[.86, 1.53]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$

Table C19

Between-women effects on masturbation frequency: Perceived Relationship Quality Components model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**.08**	**2.44**	**374.92**	**0.015**	**[.01, .14]**
Relationship length	.12	0.70	284.13	0.486	[-.22, .46]
PRQC	-.17	-1.37	330.84	0.170	[-.42, .07]
Ethinyl Oestradiol (EE)	.17	0.47	337.18	0.638	[-.52, .85]
Progestin (P)	-.33	-1.01	315.14	0.315	[-.98, .31]
Androgenicity (A)	.12	0.69	314.73	0.494	[-.21, .45]
Interaction effects					
EE x PRQC	-.20	-0.62	384.74	0.536	[-.81, .42]
P x PRQC	.22	0.69	352.61	0.492	[-.41, .85]
A x PRQC	-.16	-1.06	383.23	0.291	[-.45, .13]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$

Table C20

Between-women effects on masturbation frequency: Bond strength model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**.08**	**2.48**	**377.42**	**0.013**	**[.01, .14]**
Relationship length	.10	0.57	284.41	0.569	[-.24, .44]
Bond	-.10	-0.74	343.51	0.460	[-.35, .16]
Ethinyl Oestradiol (EE)	.18	0.50	340.51	0.616	[-.51, .86]
Progestin (P)	-.32	-0.97	317.59	0.332	[-.97, .33]
Androgenicity (A)	.13	0.74	313.90	0.460	[-.21, .46]
Interaction effects					
EE x Bond	.16	0.41	357.46	0.684	[-.62, .95]
P x Bond	-.00	-0.01	349.07	0.994	[-.67, .66]
A x Bond	-.14	-0.76	353.01	0.450	[-.50, .22]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$

Withdrawal bleeding and period

Table C21

Between-women effects, withdrawal bleeding and period excluded: Loyalty/Faithfulness model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.41**	**-6.80**	**178.95**	**0.000**	**[-.52, -.28]**
Relationship length	**-.72**	**-2.49**	**152.97**	**0.014**	**[-1.29, -.14]**
LF	**.64**	**2.21**	**169.38**	**0.029**	**[.06, 1.20]**
Ethinyl Oestradiol (EE)	.31	0.49	156.03	0.625	[-.94, 1.57]
Progestin (P)	-.34	-0.61	156.43	0.544	[-1.47, .77]
Androgenicity (A)	-.29	-0.87	148.41	0.387	[-.96, .37]
Interaction effects					
EE x LF	-.32	-0.42	173.02	0.671	[-1.84, 1.19]
P x LF	.39	0.57	176.40	0.569	[-.95, 1,72]
A x LF	-.53	-1.00	158.72	0.320	[-1.57, .51]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$

Table C22

Between-women effects, withdrawal bleeding and period excluded: Partner-Specific Investment Inventory model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.42**	**-7.32**	**178.99**	**0.000**	**[-.53, -.30]**
Relationship length	**-.68**	**-2.41**	**152.85**	**0.017**	[-1.24, -.12]
PSII	**.80**	**3.77**	**154.97**	**0.000**	[.38, 1.22]
Ethinyl Oestradiol (EE)	.49	0.84	151.04	0.404	[-.67, 1.65]
Progestin (P)	-.35	-0.66	153.48	0.509	[-1.40, .70]
Androgenicity (A)	*-.50*	*-1.77*	*143.79*	*0.078*	[-1.06, .05]
Interaction effects					
EE x PSII	**-1.77**	**-3.02**	**156.42**	**0.003**	[-2.92, -.61]
P x PSII	**1.79**	**3.17**	**158.54**	**0.002**	[.67, 2.90]
A x PSII	-.22	-0.89	145.45	0.372	[-.72, .27]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$, italics: $p < .10$

Table C23

Between-women effects, withdrawal bleeding and period excluded: Perceived Relationship Quality Components model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.41**	**-7.03**	**178.91**	**0.000**	**[-.51, -.29]**
Relationship length	**-.85**	**-3.02**	**153.38**	**0.003**	**[-1.40, -.29]**
PRQC	**.74**	**3.74**	**163.99**	**0.000**	**[.34, 1.13]**
Ethinyl Oestradiol (EE)	.53	0.89	153.35	0.372	[-.63, 1.69]
Progestin (P)	-.40	-0.75	155.89	0.455	[-1.46, .66]
Androgenicity (A)	*-.49*	*-1.73*	*142.10*	*0.085*	*[-1.05, .06]*
Interaction effects					
EE x PRQC	**-1.64**	**-3.12**	**141.68**	**0.002**	**[-2.68, .60]**
P x PRQC	**1.35**	**2.67**	**148.11**	**0.008**	**[.35, 2.35]**
A x PRQC	.04	0.17	162.67	0.864	[-.40, .48]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$, italics: $p < .10$

Table C24

Between-women effects, withdrawal bleeding and period excluded: Bond strength model

Main effects	B	*t*	*df*	*p*	*CI 95%*
Days unable	**-.40**	**-6.97**	**178.99**	**0.000**	**[-.51, -28]**
Relationship length	**-.96**	**-3.40**	**153.38**	**0.001**	**[-1.51, -.40]**
Bond	**.84**	**3.46**	**159.05**	**0.001**	**[.36, 1.32]**
Ethinyl Oestradiol (EE)	.95	1.52	141.68	0.132	[-.28, 2.19]
Progestin (P)	-.66	-1.19	145.75	0.234	[-1.76, .43]
Androgenicity (A)	**-.59**	**-2.04**	**142.67**	**0.043**	**[-1.17, -.01]**
Interaction effects					
EE x Bond	**-2.02**	**-2.77**	**151.20**	**0.006**	**[-3.47, -.58]**
P x Bond	**1.75**	**2.95**	**146.13**	**0.004**	**[.57, 2.93]**
A x Bond	-.06	-0.20	162.67	0.842	[-.65, .53]

Note. Coefficient values have been rounded to the second decimal place. Degrees of freedom and confidence interval values have been truncated to the second decimal place.
All values are z-scored except "Days unable".
Bold: $p < 0.05$