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Pubertal timing predicts adult psychosexuality: Evidence from typically developing adults and adults with isolated GnRH deficiency

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ABSTRACT

Evidence suggests that psychosexuality in humans is modulated by both organizational effects of prenatal and peripubertal sex steroid hormones, and by activational effects of circulating hormones in adulthood. Experimental work in male rodents indicates that sensitivity to androgen-driven organization of sexual motivation decreases across the pubertal window, such that earlier puberty leads to greater sex-typicality. We test this hypothesis in typically developing men (n = 231) and women (n = 648), and in men (n = 72) and women (n = 32) with isolated GnRH deficiency (IGD), in whom the precise timing of peripubertal hormone exposure can be ascertained via the age at which hormone replacement therapy (HRT) was initiated. Psychosexuality was measured with the Sexual Desire Inventory-2 (SDI-2) and Sociosexual Orientation Inventory-Revised (SOI-R). In both sexes, earlier recalled absolute pubertal timing predicted higher psychosexuality in adulthood, although the magnitude of these associations varied with psychosexuality type and group (i.e., typically developing and IGD). Results were robust when controlling for circulating steroid hormones in typically developing participants. Age of initiation of HRT in men with IGD negatively predicted SOI-R. We discuss the clinical implications of our findings for conditions in which pubertal timing is medically altered.

1. Introduction

Puberty in humans is characterized by reproductive maturation and marked by changes in internal and external physiological structures (Terasawa and Kurian, 2012; Witchel and Plant, 2013). The detectable production of three classes of steroid hormones – progestogens, androgens, and estrogens – begins at puberty with the maturation of the hypothalamic-pituitary-gonadal (HPG) axis (Ellison, 2003; Terasawa and Kurian, 2012). Although these hormones drive sexual differentiation of the gonads, genitals, and the brain before birth, they also affect sexual differentiation of the body and central nervous system at puberty (Sisk and Zehr, 2005). Emerging psychological and behavioral changes during puberty likely stem from such neural processes as synaptogenesis, synaptic pruning, axonal grown, and axonal myelination that are modulated in part by androgens (Bramen et al., 2011; Herting et al., 2014; Neufang et al., 2009) and estrogens (Herting et al., 2014; Neufang et al., 2009; Peper et al., 2009). Concomitant with reproductive maturity, gonadal hormone production, and remodeling of neural architecture, sexual arousal and desire typically increase.

Some work suggests that sexual arousal, desire, and behavior (herein, 'psychosexuality') are modulated by circulating levels of androgens in men (Edelstein et al., 2011; McIntyre et al., 2006; Puts et al., 2015; van Anders et al., 2007) and women (Caruso et al., 2014; but see Edelstein et al., 2011; Puts et al., 2015), and progestogens and estrogens in women (Grebe et al., 2016; Jones et al., 2018; Roney and Simmons, 2013, 2016; Shirazi et al., 2019a, b). However, it is also

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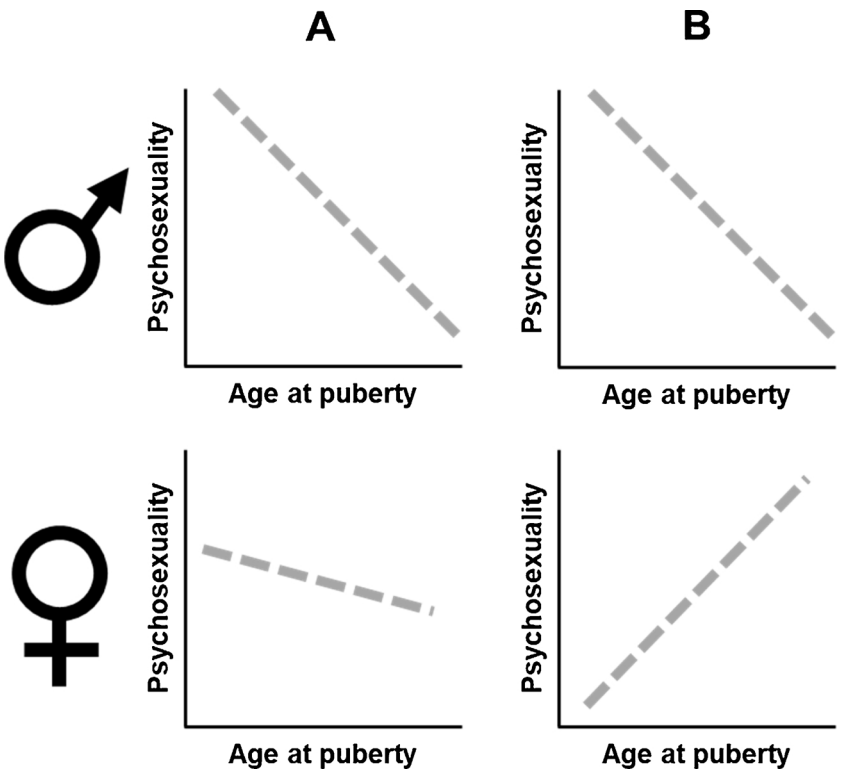

Fig. 1. Two theoretical predictions about the association between timing of puberty and psychosexuality. It has been hypothesized A) that androgens' masculinizing effect on psychology decreases in magnitude across the pubertal window, or B) that earlier sex-typical hormone exposure results in greater sex-typicality. Note that these hypotheses make similar predictions about the relationship between pubertal timing and psychosexuality in men, but they make different predictions in women.

possible that changes in psychosexuality observed across adolescence are organized by pubertal sex hormones. In this view, just as prenatal hormone action permanently alters psychology and behavior in rodents (Beatty et al., 1981; Gandelman, 1980; Isgor and Sengelaub, 1998; Matochik et al., 1994; Matsumoto et al., 2003; Sato et al., 2004) and humans (Hamann et al., 2014; Hines et al., 2004; Mueller et al., 2008; Resnick et al., 1986), peripubertal hormone action similarly exerts organizational effects that persist regardless of circulating hormone milieu in adulthood (for reviews, see Berenbaum and Beltz, 2011; Sisk and Zehr, 2005).

In hamsters, males that are castrated before puberty and administered androgens in adulthood, exhibit less sex-typical sexual behaviors (Baum, 1972; Larsson, 1967; Meek et al., 1997; Schulz et al., 2004; Sisk et al., 1992), agonistic behaviors, such as scent-marking, attacking, and chasing (De Lorme and Sisk, 2013; Schulz et al., 2006; Shrenker et al., 1985), and anxiety-related behaviors (Brown et al., 2015; Primus and Kellogg, 1989). Likewise, females that are ovariectomized before puberty and administered estrogens in adulthood exhibit less sex-typical sexual (Bakker et al., 2002), play (Smith et al., 1998), defensive (Field et al., 2004), and maternal (Kercmar et al., 2014) behaviors. Pubertal exposure to sex steroids also organizes aspects of brain morphology like androgen receptor expression (Kashon et al., 1995; Kashon and Sisk, 1994) and volume of regions implicated in sexual behavior (De Lorme et al., 2013; Schulz et al., 2009a, b). This pattern of findings suggests that pubertal hormones exert permanent, organizational effects that persist across adulthood.

Work in humans also suggests a role of organizing hormonal effects during puberty. For example, the sex difference in mental rotation abilities becomes exaggerated at puberty (reviewed in Lauer et al., 2019), and mental rotation abilities have been found to be unrelated to circulating hormones in adulthood (Herlitz et al., 2013; Puts et al., 2010), suggesting that gonadal hormones organize structures modulating mental rotation abilities during the pubertal window. Sex differences in prevalence rates for psychiatric phenotypes such as depression and anxiety (reviewed in Altemus et al., 2014) also emerge around puberty, suggesting that these phenotypes may be subject to hormone-driven organization in this developmental window.

Further, the precise *timing* of hormone exposure during the peripubertal window may also influence the degree to which the brain and behavior are permanently altered. According to this view (Schulz et al., 2009a, b; Schulz and Sisk, 2016), the peripubertal brain is decreasingly sensitive to the organizing effects of androgens across the pubertal window (decreasing sensitivity hypothesis; DSH). Alternatively, androgens could have more uniform organizing effects across puberty (constant sensitivity hypothesis; CSH). Experimental work has tested the DSH with male Syrian hamsters in which pubertal timing was manipulated to be early, on time, or late. In such studies, early pubertal timing led to greater sex-typicality in sexual behaviors (Schulz et al., 2009a, b). Comparable experimental work has not been conducted with female Syrian hamsters.

In humans, some associations between pubertal timing and psychological measures support the DSH in men. Earlier pubertal timing in men has been found to predict higher mental rotation performance (Beltz and Berenbaum, 2013; Doll et al., 2016; Shirazi et al., 2020), which shows a sex difference favoring males that increases across puberty (Geiser et al., 2008; for review, see Lauer et al., 2019). However, pubertal timing has not been found to predict mental rotations performance in women (Beltz and Berenbaum, 2013). Though androgen concentrations in both men and women rise significantly only after puberty (Albertsson-Wikland et al., 1997; Kelsey et al., 2014), adult androgen levels in men are more than an order of magnitude greater than those of women (Keevil et al., 2014), and post-pubertal rises in androgens in women are small in magnitude (Vuoksimaa et al., 2012). Thus, it is possible that peripubertal androgens have a permanent masculinizing effect that differs across the pubertal window, but that the magnitude of this effect is more subtle in women than in men (see Fig. 1a).

Similar predictions regarding relationships between pubertal timing and adult psychosexuality can also be derived from life history theory, which recognizes trade-offs inherent in the allocation of finite physiologic resources to growth, maintenance, defense, and reproduction (Wells et al., 2017). Organisms manage these trade-offs in part via their

life history strategies, which include the timing of events such as the age and relative timing of weaning, juvenile development, sexual maturation, and age of first reproduction. Individual differences in life history strategies may be driven by exposure during early life to cues to extrinsic mortality risk (Ellis et al., 2012; Walker et al., 2006; Wilson and Daly, 1997). High extrinsic mortality risk favors current reproduction over future reproduction, and hence cues to extrinsic mortality risk may promote earlier puberty and earlier age at first copulation (Friedlander et al., 2007; Lam et al., 2002). Earlier attainment of reproductive maturity is also influenced by nutritional status (Walker et al., 2006); for example, there is a secular trend of decreasing age of reproductive maturity in developed countries (Stearns et al., 2010). Individuals with better access to resources are able to undergo the somatic growth and development necessary for reproductive maturation at earlier ages. Thus, earlier sexual maturity may be attributable in some cases to cues to extrinsic morality risk favoring earlier reproduction, and in other cases to propitious reproductive circumstances associated with resource availability. In both cases, it is plausible that these factors would shift development toward the allocation of more resources toward reproduction and particularly toward mating (Del Guidice and Belsky, 2011). Indeed, men reporting earlier puberty exhibited higher phenotypic masculinization across several psychological and morphological secondary sex traits in adulthood (Doll et al., 2016), suggesting that earlier reproductive maturation positively predicts allocation of resources to mating. By extension, psychosexuality may also exhibit an association with reproductive maturity; in both men and women, earlier reproductive maturity may predict a greater emphasis on psychobehavioral phenotypes that promote mating.

We note, however, that criticisms have recently been raised regarding the application of life history theory to the study of intraspecific, as opposed to cross-specific, variation. These criticisms emphasize that the processes contributing to differences between species are fundamentally different from those contributing to differences within species (Zietsch and Sidari, 2019), and that the evidence that interspecific patterns should apply to the study of variability within species is critically lacking (Stearns and Rodrigues, 2020). More research isolating the effects of specific environmental variables hypothesized to modulate life history tempo is necessary, and such research may clarify the utility of applying patterns derived from cross-species comparisons to the study of intra-individual variation. Nonetheless, it is possible that some intraspecific patterns will resemble cross-species patterns. For the functional reasons stated above, it is in our view plausible that earlier sexual maturation will be associated with increased mating effort in humans.

It is also possible that the timing of estrogen exposure, rather than androgen exposure, during puberty plays a role in organizing sexually differentiated cognitive phenotypes and behaviors in women. Earlier pubertal timing in women predicts higher disordered eating (Fairburn et al., 1997; Graber et al., 1997; Zehr et al., 2007), a phenotype more prevalent in women than in men. Early maturing women also have a higher prevalence of mood disorders such as anxiety and depression (Angold et al., 1998; Graber et al., 1997; Kaltiala-Heino et al., 2003, 2004), both of which are significantly female-biased in their prevalence after puberty. These results suggest that whereas the timing of androgen exposure may predict sex-typicality in men, the timing of estrogen exposure may predict sex-typicality in women. If earlier peripubertal estrogen exposure positively predicts sex-typicality in women, then women reporting earlier puberty should also report lower (i.e., more 'feminine') levels of psychosexuality (see Fig. 1b).

These considerations highlight the need for further research to test the DSH in humans. Psychosexuality is a highly promising domain in which to do so: In male hamsters, earlier peri-pubertal androgen treatment led to more sex-typical sexual behavior (Schulz et al., 2009a, b). In humans, sex differences in sexual behavior and desire emerge at puberty, and increase across adolescence (Oliver and Hyde, 1993; Petersen and Hyde, 2010; see Fortenberry, 2013 for review). Relative to women, men tend to score higher on sociosexuality, or in interest in uncommitted sex (Penke and Asendorpf, 2008; Schmitt, 2005), and in trait levels of general sexual desire (reviewed in Dawson and Chivers, 2014). Clinically, women are more likely to meet diagnostic criteria for low sexual desire (Brotto, 2010a, b), whereas men are more likely to be diagnosed with hypersexuality (Långström and Hanson, 2006).

Despite the theoretical reasons for predicting associations between pubertal timing and psychosexuality, only one prior study of which we are aware has investigated these relationships. Ostovich and Sabini (2005) found that earlier pubertal timing predicted greater psychosexuality in men, but not in women (Fig. 1a), consistent with the DSH. However, the relatively modest sample size (N men = 129, N women = 148) and use of just four questions to determine pubertal timing necessitate further research.

Although an ideal study of pubertal timing and psychosexuality would require experimental manipulation of the timing of puberty, such a study would be unethical in humans. However, a human disease model can serve as a natural quasi-experiment and enable the precise measurement of the onset of pubertal hormone exposure. Isolated GnRH deficiency (IGD) is a rare disorder with an estimated prevalence of 1:30,000 male – 1:125,000 female live births, and a male-to-female ratio of about 4:1 (Boehm et al., 2015; Laitinen et al., 2011). Although many different genetic mutations have been implicated in IGD (Seminara et al., 1998; Stamou et al., 2016), the core pathophysiological cause is homogenous: individuals with IGD have either absent or nonfunctional gonadotropin-releasing hormone (GnRH) neurons in the hypothalamus (Crowley and Pitteloud, 2017; Hayes et al., 1998). In typical development, hypothalamic GnRH neurons initiate the cascade of signals to the pituitary that respond with gonadotropin release which in turn initiate gonadal sex steroid production (i.e., progestogens, androgens, and estrogens). The absence of GnRH secretion and action means an absence of endogenous gonadal hormone production in individuals with IGD. Hormone exposure in individuals with IGD does not differ from typically developing individuals during the first trimester of gestation; high levels of human chorionic gonadotropin (hCG) act to stimulate hormone production by binding to luteinizing hormone (LH) receptors (Choi and Smitz, 2014; Seminara et al., 1998). As maternal hCG drops across the second and third trimesters of gestation, gonadal hormone action declines, and the nearly-adult levels of HPG axis activity known as 'mini-puberty' that typically occur in the first few months of life are absent in individuals with IGD. Because individuals with IGD are unable to produce endogenous gonadal hormones, they require hormone replacement therapy (HRT) to initiate puberty and must remain on HRT across adulthood. By utilizing data on the timing of initiation of HRT, we are thus able to pinpoint the precise age at which pubertal hormone exposure began for this clinical group.

The present set of studies evaluates predictions derived from behavioral neuroendocrinology (the CSH and DSH) and life history theory regarding associations between pubertal timing and psychosexuality in adulthood. We examine whether the documented timing of HRT initiation predicts psychosexuality in a clinical sample of men and women with Isolated GnRH Deficiency (IGD), and whether recalled pubertal timing predicts psychosexuality in a large sample of typically developing adult men and women.

2. Method

2.1. Participants

Participants with IGD were recruited for a study on puberty, sex hormones, and psychology. Recruitment materials were posted on listservs, forums, and support groups. We also received referrals from physicians at the Reproductive Endocrine Unit at Massachusetts General Hospital and the Genetics of Puberty and Reproduction group at the National Institute of Child Health and Human Development. Men and women were required to be 18 years of age or older and fluent in

Table 1
Descriptive statistics for pubertal timing and psychosexuality measures. Values represent means and standard errors.

	Men		Women	
	Typically developing	IGD	Typically developing	IGD
Absolute pubertal timing Z	−0.34 (0.03)	1.14 (0.13) *	−0.05 (0.02)	1.99 (0.26) *
Relative pubertal timing Z	−0.28 (0.04)	1.05 (0.06) *	−0.05 (0.02)	1.55 (0.11) *
Age of HRT initiation	n/a	18.99 (0.70)	n/a	20.72 (1.47)
SOI-R	4.39 (0.10)	3.81 (0.19) *	3.36 (0.06)	2.72 (0.24) *
SDI-2	5.56 (0.07)	5.56 (0.16)	4.17 (0.05)	3.25 (0.30) *
Age at session	21.64 (0.39)	39.88 (1.69) *	20.99 (0.20)	32.94 (1.58) *

* differs from typically developing same-sex participants at $p < 0.05$.

English. Participants were compensated with $25 for survey completion. Forty-seven participants with IGD (n women = 15) were recruited through online methods, and 57 (n women = 17) were recruited through physician referrals. See Table 1 for demographic information on the sample.

Typically developing participants were recruited for a study on puberty, sex hormones, and psychology through radio, Craigslist, newspaper, and social media advertisements, as well as through a research participant pool coordinated by the Pennsylvania State University Department of Psychology. Men and women were required to be 18 years of age or older and fluent in English; women were additionally required to be not pregnant during time of sampling. Compensation was either course credit for introductory psychology courses, or monetary compensation ($20 per session). Participants with IGD received higher compensation than did typically developing participants because they completed more questionnaires (e.g., a questionnaire about the timing of HRT) than did typically developing participants.

This study was approved by the Pennsylvania State University and Massachusetts General Hospital institutional review boards.

2.2. Procedure

Participants with IGD completed questionnaires remotely and were instructed to complete the questionnaire in a single sitting. A full description of procedures for the typically developing participants has been published elsewhere (see Shirazi et al., 2019a, 2020). Briefly, laboratory sessions were scheduled between 09:00 and 12:00. Typically developing participants provided one saliva sample before completing the survey and one saliva sample after; these samples were subsequently combined to minimize the effect of pulsatile secretion of hormones. Saliva samples were not collected for participants with IGD.

After providing the initial saliva sample, typically developing participants were directed to a private workstation to fill out a series of questionnaires. Because of the present study's aims, we report on a subset of these questionnaires: the Sociosexual Orientation Inventory-Revised (SOI-R; Penke and Asendorpf, 2008), the Sexual Desire Inventory-2 (SDI-2; Spector et al., 1996), a modified Pubertal Development Scale (PDS; Petersen et al., 1988), and demographic questions related to age, sexual orientation, and for women only, current contraceptive use. These questionnaires have been uploaded as electronic supplementary materials (ESM).

2.2.1. Psychosexuality questionnaires

The SOI-R characterizes an individual's attitudes (e.g., agreement with the statement "Sex without love is OK"), behaviors (e.g., "With how many different partners have you had sexual intercourse on *one and only one* occasion?), and desires (e.g., "How often do you have fantasies about having sex with someone you are *not* in a committed romantic relationship with?") related to mating outside of a committed relationship. Responses are coded from zero (indicating lower sociosexuality) to nine (indicating higher sociosexuality), except for a single reverse-scored question. The behavior, attitude, and desire subscales each comprise three questions, and all nine questions are summed to calculate an overall SOI-R score, with higher scores corresponding to higher sociosexuality. Cronbach's α for the SOI-R in this sample was 0.88. For further information on questions and response choices for the SOI-R, the reader is referred to the ESM.

The SDI-2 characterizes an individual's general sexual desire as it relates to solitary (e.g., "How strong is your desire to engage in sexual behavior by yourself?") and dyadic (e.g., "How strong is your desire to engage in sexual behavior with a partner?") sexual desire. The solitary subscale comprises four questions, and the dyadic subscale comprises nine questions; for all questions, a higher score indicates higher sexual desire. The solitary subscale and dyadic subscales can be summed to calculate an overall SDI-2 score, with higher scores corresponding to higher general sexual desire. Cronbach's α for the SDI-2 in this sample was 0.90. For further information on questions and response choices for the SDI-2, the reader is referred to the ESM. Both typically developing participants and participants with IGD completed the SOI-R and SDI-2.

2.2.2. Pubertal timing questionnaires

The original PDS was modified so that it could be used to collect retrospective data (as in Doll et al., 2016). Participants were asked sex-specific questions about the timing of pubertal changes and provided both relative and absolute estimates of when these changes occurred. For relative estimates, participants chose from five answer choices ranging from "much earlier than my peers" to "much later than my peers," as well as an "I don't know" option. For absolute estimates, participants indicated how old they were in years and months when they experienced changes. Cronbach's α for questions on absolute timing was 0.95 for men and 0.85 for women. Both typically developing participants and participants with IGD completed out the PDS.

Participants with IGD filled out a questionnaire about HRT and reported the age when they first began an HRT treatment regimen. Age of HRT initiation was available for 64 men and 17 women. Physician- or medical-record-confirmed age of HRT initiation was available for a subset of patients, and these two measures were moderately-to-strongly (with $0.4 \geq r > 0.7$ as moderate, and $0.7 \geq r > 0.99$ as strong; see Akoglu, 2018) correlated ($r[52] = 0.78$, 95 % confidence interval [CI] = 0.64 − 0.86; in men only, $r[44] = 0.81$, 95 % CI = 0.68 − 0.89; in women only, r[8] = 0.60, 95 % CI = -0.10 − 0.90). Only typically developing participants were invited to complete a second, identical laboratory testing session between one and three months after their initial session. Two hundred thirty-one men, 369 naturally cycling (NC) women, and 279 women on hormonal contraceptives (HC) completed a first session; of these, 43 men, 108 NC women, and 71 HC women completed a second session. Sample sizes reported below vary based on the availability of all values included in models. The timing of the second session was largely dictated by participants' availability.

2.3. Data processing, treatment, and analysis

As the PDS assesses both relative and absolute pubertal timing,

composites of each were calculated by *z*-scoring and averaging all questions of relative and absolute pubertal timing, respectively. There are unique advantages and disadvantages to using measures of relative or absolute pubertal timing (see Beltz and Berenbaum, 2013 for discussion). These measures were highly correlated in typically developing men (r[207] = 0.54, 95 % confidence interval = 0.44 − 0.63) and women (r[632] = 0.66, 95 % confidence interval = 0.62 − 0.70), as well as in men with IGD (r[64] = 0.52, 95 % CI = 0.31 − 0.67) and women with IGD (r[25] = 0.77, 95 % CI = 0.56 − 0.89).

Among men with IGD, the correlation between self-reported age of HRT initiation and relative pubertal timing was r(61) = 0.31 (95 % CI = 0.07 − 0.52), and the correlation between self-reported age of HRT initiation and absolute pubertal timing was r(61) = 0.45 (95 % CI = 0.23 − 0.63). The correlation between physician-confirmed age of HRT initiation and relative pubertal timing was r(43) = 0.30 (95 % CI = 0.003 − 0.54), and the correlation between physician confirmed age of HRT initiation and absolute pubertal timing was r(43) = 0.35 (95 % CI = 0.06 − 0.58). Among women with IGD, the correlation between self-reported age of HRT initiation and relative pubertal timing was r(15) = 0.20 (95 % CI = -0.31 − 0.62), and the correlation between self-reported age of HRT initiation and absolute pubertal timing was r(13) = 0.33 (95 % CI = -0.22 − 0.72). The correlation between physician-confirmed age of HRT initiation and relative pubertal timing was r(18) = -0.36 (95 % CI = -0.70 − 0.09), and the correlation between physician confirmed age of HRT initiation and absolute pubertal timing was r (18) = 0.05 (95 % CI = -0.40 − 0.48). Because age of HRT initiation represents a precise and valid measure of the timing of exposure to sex steroids, and because both self-reported and physician-conformed ages of HRT initiation were more strongly positively correlated with absolute pubertal timing than with relative pubertal timing, we used absolute pubertal timing as the primary measure of pubertal timing in analyses that included typically developing participants and where data on HRT timing were thus unavailable for all participants. However, analyses using relative pubertal timing are reported in **ESMResults1** and **2**.

Saliva samples from typically developing participants were stored at -20° C until being shipped to the Nipissing University Biomarkers Lab (Nipissing University, North Bay, Ontario) and analyzed with commercially available enzyme immunoassay kits (DRG International). Saliva samples obtained from women were analyzed for concentrations of estradiol, progesterone, and testosterone, whereas saliva samples obtained from men were analyzed for testosterone. Sensitivities for estradiol, progesterone, and testosterone were 0.5, 3.8, and 1.9 pg/mL, respectively. Intra-assay CVs for estradiol, progesterone, and testosterone were 11 %, 14 %, and 6%, respectively, and inter-assay CVs were 10 %, 12 %, and 5%, respectively.

Hormones were then log-transformed to reduce skew and standardized separately for men and women. Hormone concentrations were included (when available) as covariates in regression models, as circulating hormone concentrations may modulate sociosexuality and general sexual desire (see Introduction).

All analyses were performed using R, and the associated data and code files have been uploaded as electronic supplementary materials (ESM). All models were run separately for men and women.

We examined relationships with pubertal timing in several ways. First, we used multilevel models to test whether absolute pubertal timing predicted overall psychosexuality, controlling for age and sexual orientation. We nested SOI-R and SDI-2 scores within subjects, and as such the dependent variable in these models considered both SOI-R and SDI-2 scores (hereafter referred to as "overall psychosexuality"). Second, we tested whether absolute pubertal timing predicted overall psychosexuality entering relevant interaction terms. These models included the following terms: absolute pubertal timing, group (i.e., typically developing and IGD), pubertal timing × psychosexuality type (i.e., SOI-R and SDI-2), pubertal timing × group, and pubertal timing × psychosexuality type × group. To test for interactions between pubertal timing and the specific type of psychosexuality considered (i.e., sociosexual desire versus general sexual desire), we created a dummy variable with SOI-R coded as 0.5 and SDI-2 coded as -0.5 (see also Shirazi et al., 2019a) to be used in the estimation of interaction terms. Although it would have been possible to add another level to this modeling structure and have scores for individual subscales nested within SOI-R and SDI-2 composites, with composites nested within participants, such models would be unwieldy, and some would require the interpretation of four-way interaction terms. Multilevel models were estimated using the *lme4* (Bates et al., 2014) and *lmerTest* packages.

Third, we performed mediation analyses, testing whether group (i.e., control and IGD) differences in psychosexuality were mediated by differences in self-reported absolute pubertal timing. Mediation was tested using R's *mediation* package.

Finally, we analyzed groups separately, using alternate measures of pubertal timing. In typically developing men, we tested whether recalled absolute pubertal timing predicted psychosexuality, while controlling for age, sexual orientation, and circulating testosterone. In typically developing women, we ran the same models controlling for contraceptive use (either NC or HC), estradiol, progesterone, estradiol × progesterone interaction, and group × hormone interactions for all hormone terms. Prior work on this sample has shown discrepant hormone-psychosexuality relationships in NC and HC women (Shirazi et al., 2019b), warranting the inclusion of group × hormone interaction terms. This same work suggests differences in psychosexuality in NC and HC women, warranting controlling for a main effect of contraceptive use. As gonadal hormone concentrations have been linked to both psychosexuality (Jones et al., 2018; Roney and Simmons, 2013; Shirazi et al., 2019a) and pubertal timing (Bishop et al., 1988), statistically controlling for steroid hormones allowed us to ensure that any pubertal timing-psychosexuality relationships were not simply artifacts of both measures' associations with hormones. For IGD participants who reported an age of HRT initiation, we tested whether log-transformed (to correct skew) age of HRT initiation predicted psychosexuality when controlling for age and sexual orientation.

3. Results

3.1. Sample characteristics

Sample characteristics can be found in Table 1. As expected, both recalled absolute and relative pubertal timing were earlier in typically developing participants than in participants with IGD. SOI-R was significantly lower in men and women with IGD relative to typically developing participants, and SDI-2 was lower in women with IGD relative to typically developing women.

3.2. Men

In a multilevel model including all men with absolute pubertal timing, age, and sexual orientation as predictors, absolute pubertal timing significantly negatively predicted psychosexuality (Fig. 2a). To test whether this finding was due to differences between typically developing men and men with IGD in pubertal timing, we ran a mediation model. When controlling for age and sexual orientation, the effect of group on psychosexuality was significantly mediated by absolute pubertal timing (Average Causal Mediation Effect [ACME] estimate = -0.30, p = 0.047), suggesting that any psychosexual group differences are due in part to differences in pubertal timing.

We next entered psychosexuality type, group, and relevant interactions to a regression model to explore whether effects of pubertal timing differed across psychosexuality types or groups. Absolute pubertal timing and group did not explain unique variance in psychosexuality scores (Table 2), likely because of their strong intercorrelation. Pubertal timing × psychosexuality type, pubertal timing × group,

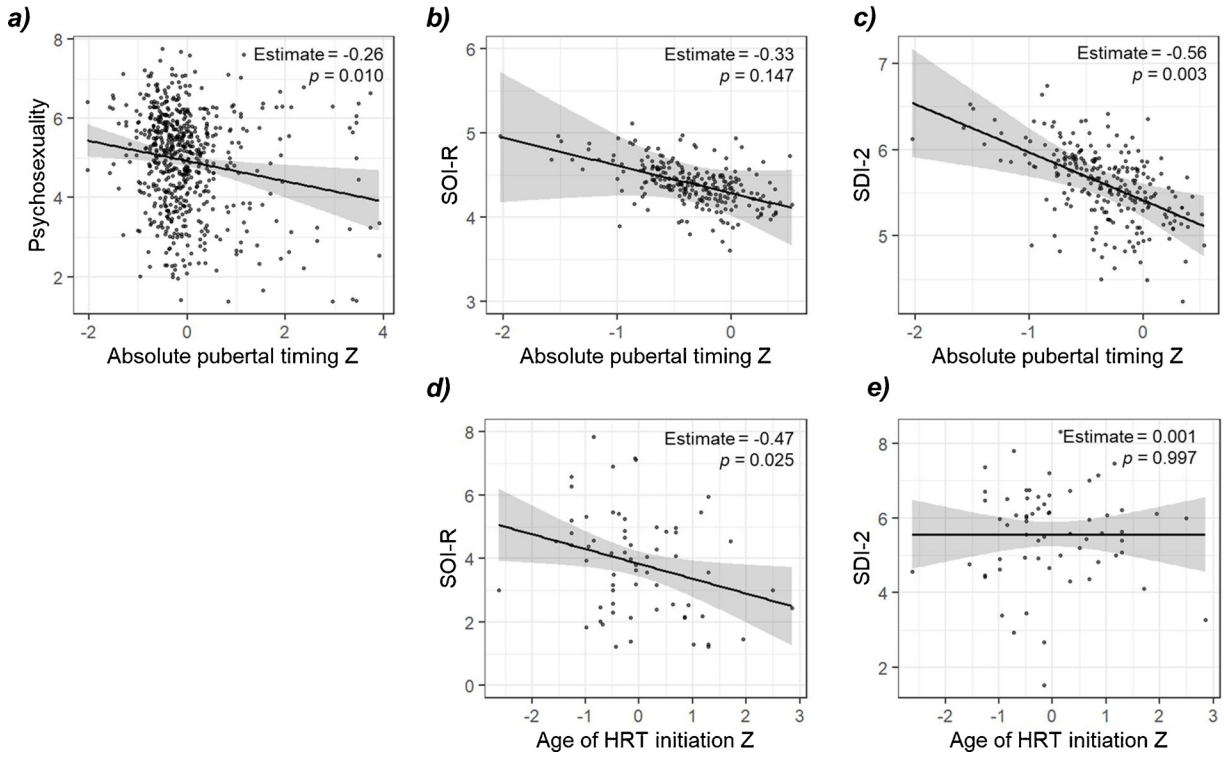

Fig. 2. Results of analyses showing relationships between (a) absolute pubertal timing and overall psychosexuality in all men, absolute pubertal timing and SOI-R (b) and SDI-2 (c) in typically developing men, and age of HRT initiation and SOI-R (d) and SDI-2 (e) in men with IGD.

Table 2
Results from full multilevel models predicting psychosexuality.

		Estimate (SE)	*p*
Men	Absolute pubertal timing	0.20 (0.23)	0.386
	Group	−0.13 (0.28)	0.635
	Absolute pubertal timing × psychosexuality type	−1.54 (0.20)	< **0.001**
	Absolute pubertal timing × group	−0.75 (0.27)	**0.007**
	Absolute pubertal timing × psychosexuality type × group	2.53 (0.24)	< **0.001**
Women	Absolute pubertal timing	0.001 (0.08)	0.998
	Group	−0.69 (0.46)	0.134
	Absolute pubertal timing × psychosexuality type	−0.23 (0.08)	**0.006**
	Absolute pubertal timing × group	−0.32 (0.22)	0.139
	Absolute pubertal timing × psychosexuality type × group	0.38 (0.15)	**0.014**

and pubertal timing × psychosexuality type × group interactions were statistically significant. To elucidate the pubertal timing × group interaction, separate regressions were run for typically developing men and men with IGD with only absolute pubertal timing, age, and sexual orientation to predict psychosexuality. Absolute pubertal timing significantly predicted psychosexuality in typically developing men (estimate = −0.57, p = 0.006), but not in men with IGD (estimate = −0.04, p = 0.768). To elucidate the pubertal timing × psychosexuality type interaction, separate regressions were run for SOI-R and SDI-2, including all men. Absolute pubertal timing predicted SOI-R (estimate = −0.29, p = 0.018), but not SDI-2 (estimate = −0.16, p = 0.104). Absolute pubertal timing did not significantly mediate group differences in SOI-R (ACME estimate = −0.31, p = 0.090) or SDI-2 (ACME estimate = −0.19, p = 0.190) separately.

Next, to elucidate the pubertal timing × psychosexuality type × group interaction, a series of separate regressions were run for typically developing men and men with IGD with absolute pubertal timing and the pubertal timing × psychosexuality type interaction as predictors, along with age and sexual orientation. This interaction was significant in both typically developing men and men with IGD, though in opposite directions (typically developing men estimate = −1.54, $p < 0.001$; men with IGD estimate = 0.99, $p < 0.001$). In typically developing men absolute pubertal timing was negatively associated with SOI-R (albeit not significantly; Fig. 2b) and SDI-2 (Fig. 2c). Among men with IGD, absolute pubertal timing was negatively associated with SOI-R (estimate = −0.21, p = 0.267), and positively with SDI-2 (estimate = 0.13, p = 0.415), though not statistically significant in either case. Models with pubertal timing × group interactions suggested that the association between absolute pubertal timing and SOI-R did not differ by group (estimate = 0.37, p = 0.250), whereas the association between absolute pubertal timing and SDI-2 did differ by group (estimate = 0.70, p = 0.003).

Finally, we analyzed data for typically developing men and men with IGD separately. In a multilevel model (with sessions and desire types nested within participants) with typically developing men only, when controlling for current age, sexual orientation, and testosterone, there was a significant effect of absolute pubertal timing on psychosexuality (estimate = −0.43, p = 0.048). In a subsequent model also including the absolute pubertal timing × psychosexuality type interaction, this interaction was significant (estimate = −1.50, $p < 0.001$). Absolute pubertal timing did not significantly predict SOI-R (estimate = −0.19, p = 0.43), but did predict SDI-2 (estimate = −0.44, p = 0.022). In a multilevel model (with desire types nested within participants) with men with IGD only, when controlling for current age and sexual orientation, the effect of age of HRT initiation was not significant (estimate = −0.23, p = 0.134). In a subsequent model also including the age of HRT initiation × psychosexuality type interaction, this interaction was significant (estimate = 0.46, p = 0.045). Follow-up regressions revealed that earlier HRT predicted elevated SOI-R (Fig. 2d), but not SDI-2 (Fig. 2e) scores.

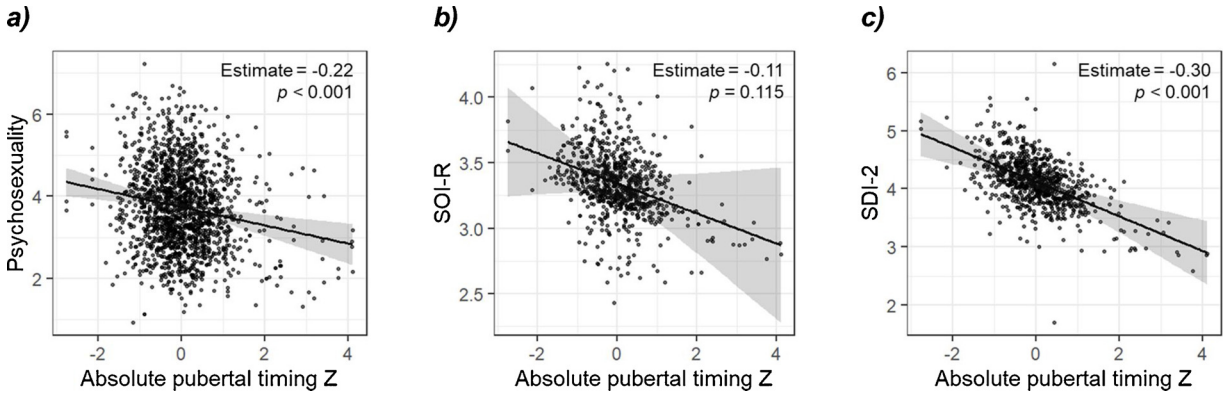

Fig. 3. Results of analyses across all women showing relationships between absolute pubertal timing and (a) overall psychosexuality, (b) SOI-R, and (c) SDI-2.

3.3. Women

In a multilevel model including all women with absolute pubertal timing, age, and sexual orientation as predictors, absolute pubertal timing significantly negatively predicted psychosexuality (Fig. 3a). We then ran a mediation model to determine whether this effect of pubertal timing was driven by group differences in pubertal timing. Controlling for age and sexual orientation, the effect of group on psychosexuality was significantly mediated by absolute pubertal timing (ACME estimate $= -0.26$, $p = 0.046$), suggesting that any psychosexual group differences are due in part to differences in pubertal timing.

We subsequently entered psychosexuality type, group, and relevant interactions to the model. Absolute pubertal timing and group did not explain unique variance in psychosexuality scores (Table 2), likely because of their strong intercorrelation. Pubertal timing × psychosexuality type and pubertal timing × psychosexuality type × group interactions were statistically significant, but pubertal timing × group was not, indicating that the negative association between pubertal timing and overall psychosexuality did not differ by group. To elucidate the pubertal timing × psychosexuality type interaction, separate regressions for SOI-R and SDI-2 were conducted across all women. Absolute pubertal timing did not significantly predict SOI-R (Fig. 3b), but significantly predicted SDI-2 (Fig. 3c). In subsequent mediation models, the mediation effects of absolute pubertal timing were not statistically significant for SOI-R (ACME estimate $= -0.07$, $p = 0.767$), but were significant for SDI-2 (ACME estimate $= -0.39$, $p = 0.010$).

To elucidate the pubertal timing × psychosexuality type × group interaction, a series of separate regressions were run for typically developing women and women with IGD with absolute pubertal timing and the pubertal timing × psychosexuality type interaction as predictors, along with age and sexual orientation. The pubertal timing × psychosexuality type interaction was significant in typically developing women (estimate $= -0.23$, $p = 0.005$), but not in women with IGD (estimate $= 0.15$, $p = 0.310$), indicating that the effect of pubertal timing differs across psychosexuality types in typically developing women only. Among typically developing women, absolute pubertal timing did not predict SOI-R (estimate $= -0.01$, $p = 0.923$), but negatively predicted SDI-2 (estimate $= -0.18$, $p = 0.022$). Models with pubertal timing × group interactions suggested that the association between absolute pubertal timing and SOI-R (estimate $= -0.22$, $p = 0.368$) and SDI-2 (estimate $= -0.09$, $p = 0.687$) did not differ by group.

Finally, we analyzed data for typically developing women and women with IGD separately. In a multilevel model (with sessions and desire types nested within participants) with typically developing women only and controlling for current age, sexual orientation, contraceptive use, estradiol, progesterone, testosterone, and contraceptive use × hormone interactions, absolute pubertal timing did not predict psychosexuality (estimate $= -0.14$, $p = 0.116$). In a subsequent model also including the absolute pubertal timing × psychosexuality type interaction, this interaction was significant (estimate $= -2.28$, $p = 0.007$). Whereas absolute pubertal timing did not predict SOI-R (estimate $= 0.01$, $p = 0.914$), it did significantly negatively predict SDI-2 (estimate $= -0.20$, $p = 0.013$). In a multilevel model (with desire types nested within participants) with women with IGD only, when controlling for current age and sexual orientation, the effect of age of HRT initiation was not statistically significant (estimate $= -0.21$, $p = 0.541$). In a subsequent model also including the absolute pubertal timing × psychosexuality type interaction, this interaction was not significant (estimate $= 0.51$, $p = 0.335$).

4. Discussion

We examined relationships between pubertal timing and adult psychosexuality to evaluate hypotheses derived from experimental behavioral neuroendocrinology and life history theory. We utilized data from men and women with IGD who fail to enter puberty spontaneously, in whom the precise timing of peripubertal hormone exposure could be ascertained through the timing of HRT initiation. We further assessed relationships between pubertal timing and psychosexuality in a large sample of typically developing men and women and were able to control for circulating steroid hormone concentrations when available, and measured pubertal timing using previously validated questionnaires.

In both sexes, there was a significant overall effect of the recalled timing of pubertal events on psychosexuality such that earlier onset of puberty predicted higher psychosexuality, and all significant or near-significant effects were in the same direction across analyses. Results were robust to the inclusion of testosterone as a covariate in typically developing men, and to the inclusion of estradiol, progesterone, contraceptive use, and relevant interactions as covariates in typically developing women.

Among men, regressions evaluating the effects of absolute pubertal timing, group, psychosexuality type (i.e., SOI-R and SDI-2), and their interactions revealed that in analyses including all men, the effect of absolute pubertal timing on psychosexuality was stronger in typically developing men than in men with IGD, and stronger on SOI-R than SDI-2. Post-hoc analyses exploring the pubertal timing × psychosexuality type × group interaction, along with separate analyses conducted in typically developing and IGD men separately, indicated that pubertal timing was related only to SOI-R in men with IGD, and only to SDI-2 in typically developing men. Individuals with IGD differ from typically developing individuals in a lack of exposure to gonadal hormones after the first trimester of gestation and hence also do not experience the early postnatal 'mini-puberty' that typically developing infants do. It is therefore possible that hormone exposure during these critical periods is necessary for some typical organizational effects of sex hormones, including possible effects on general sexual desire as measured by the

SDI-2. It is also possible that, on average, participants with IGD began HRT too late in the peripubertal window for organizational effects on the neural architecture underlying SDI-2 to be detectable in this sample.

Among women, the overall effect of absolute pubertal timing on psychosexuality did not differ between typically developing individuals and those with IGD. However, absolute pubertal timing more strongly predicted SDI-2 than SOI-R, and this interaction between pubertal timing and psychosexuality type was stronger and statistically significant only in typically developing women. As in men, it is possible that this group difference reflects a lack of gonadal hormones during a critical period following the first trimester in women with IGD, or an average age of HRT initiation that is too late for some typical organizational effects of pubertal hormones. A difference is that pubertal timing more strongly predicted SOI-R in men with IGD and more strongly predicted SDI-2 in women with IGD. One possibility is that this difference reflects the diverse influences of early testicular and ovarian hormones—for example, early ovarian hormones (progestogens and/or estrogens) may be critical to later effects of pubertal hormones on sociosexuality in women, whereas early testicular hormones (androgens) may be critical to later effects of pubertal hormones on general sexual desire in men.

In women with IGD, age of HRT initiation did not significantly predict overall psychosexuality. Because the effect was relatively large in magnitude (estimate $= -0.46$), and because the effect of absolute pubertal timing did not differ significantly across groups, the lack of statistical significance in the relationship between HRT timing and overall psychosexuality may reflect the small sample in this group and/or the restricted range pubertal timing values.

Mean SOI-R and SDI-2 scores were correlated in the present study at $r(294) = 0.47$ and $r(676) = 0.48$ for men and women, respectively, yet they are distinct psychological constructs. Our results corroborate recent work suggesting that the hormonal predictors of sociosexuality and general sexual desire differ (Jones et al., 2018; Shirazi et al., 2019b). Data on the shared and discrepant neuroanatomical regions involved in these components of psychosexuality are lacking. It is possible, for example, that brain regions heavily implicated in sociosexuality and *not* in general sexual desire are differentially affected by pubertal timing, and that differences between SOI-R-based and SDI-2-based analyses are driven by region-specific sensitivity to the organizational effects of pubertal timing.

Our data do not support the hypothesis that earlier pubertal timing predicts higher sex typicality in both sexes (Fig. 1b). This hypothesis predicts that earlier puberty will be associated with higher psychosexuality in men, and with lower psychosexuality in women, while our data generally suggest that earlier pubertal timing predicts higher psychosexuality in both sexes.

That earlier puberty tended to be associated with higher psychosexuality in both sexes is more consistent with life history theory and with the hypothesis initially proposed by Sisk and colleagues wherein earlier puberty is associated with greater phenotypic masculinization (DSH; Fig. 1a). Per the decreasing sensitivity hypothesis, the brain becomes less sensitive to the organizational (and specifically, masculinizing) effect of pubertal hormones across the pubertal window, with highest masculinity in brain morphology, psychology, and behavior in individuals who experienced puberty earliest. Life history theory-based hypotheses predict that those with earlier pubertal timing should generally allocate more resources toward mating effort, perhaps in part by developing psychological and behavioral traits that promote mating.

It is important to note that the DSH addresses causation at a different level from life history theory-derived hypotheses. Whereas the DSH speculates about the ontogenetic and proximate physiological mechanisms driving sexual differentiation of the brain and behavior, life history theory speculates about the adaptive function of mechanisms linking adult trait expression to pubertal timing. According to some iterations of life history theory, the same conditions that favor earlier mating and reproduction (and hence earlier reproductive hormone production and reproductive maturity) should favor psychobehavioral emphasis on mating in adulthood (Del Guidice and Belsky, 2011; Doll et al., 2016). This linkage between the timing of reproductive maturity and adult psychobehavioral phenotypes would need to be driven by some underlying biological process, and we view the DSH as a likely candidate. Future work may elucidate the specific neurobiological mechanisms through which earlier pubertal timing may modulate adult psychosexuality. For example, pubertal testosterone plays an organizational role in determining the volume and number of neurons within the regions of the amygdala in male Syrian hamsters (De Lorme et al., 2013). If subsequent experimental work were to show that the timing of pubertal testosterone (rather than simply its presence or absence) modulated the magnitude of such organizational effects on neural structure and function, then this could be a viable mechanism by which earlier pubertal timing drives more male-typical psychosexuality in adulthood. There is evidence in humans that amygdalar function is sensitive to androgen exposure, such that increased androgen during early development predicts higher amygdalar activity in response to sexual images (Hamann et al., 2004, 2014). Thus, it is possible that amygdalar structure and function are also modulated by pubertal timing. We present the amygdala as one example of many and urge future work to examine the effect of pubertal timing on a wide range of neural regions that have previously been implicated in mating and sexuality, such as those in the hypothalamus (reviewed in Simerly, 2002).

Tangential to the primary aim of the present study, our data may be informative to researchers retrospectively assessing pubertal timing. Though it has been established that even self-reports of current pubertal development do not correlate strongly with physical examinations of pubertal status and hormone concentrations (Shirtcliff et al., 2009), self-reports of pubertal timing remain the only way to assess pubertal timing retrospectively. Although some have asserted that relative pubertal timing is a superior measure to absolute pubertal timing (e.g., Graber et al., 1997), our findings among men and women with IGD seemingly contradict this. In both sexes, retrospective reports of absolute pubertal timing were more strongly correlated with age of HRT initiation and with physician-confirmed age of HRT initiation, which are arguably the most reliable measures of pubertal onset in the present study. The lack of tight correlations between relative pubertal timing and other measures of pubertal timing (here, absolute pubertal timing and age of HRT initiation) likely explain some of the discrepancies among analyses that utilize these different measures of pubertal timing. As interest grows in the putative organizational effects of pubertal timing, and in the effects of pubertal timing more generally, it is important to continue probing how we can best measure pubertal timing retrospectively in the absence of longitudinal data.

The present study was not without limitations. First, though several hypotheses predict links between pubertal timing and adult psychosexual phenotypes, we are unable to make claims about causality. True experiments in which pubertal timing is systematically manipulated could allow us to draw inferences about causality, but such experiments in humans are unethical. Careful study of men and women with IGD represents a naturally occurring quasi-experiment (see also Shirazi et al., 2020). Future work could focus on testing putative causality in animal models wherein pubertal timing can be experimentally manipulated (as has been done previously, discussed in Schulz and Sisk, 2016). Second, as most effect sizes in previous examinations of the effect of pubertal timing on adult psychological phenotypes are small in magnitude, it is possible that our analyses of men and women with IGD were not sufficiently powered to detect significant effects due to sample size constraints. Our examination of participants with IGD demonstrates the value of this disease model but also highlight difficulties inherent in studying rare conditions. The formation of multi-site collaborations to study IGD and psychology could yield a larger pool of participants and facilitate more powerful statistical analyses. Finally, links between pubertal timing and adult phenotypes can be investigated

using longitudinal designs. Such designs ideally would recruit participants prior to puberty and measure hormone production, physical development, and a wide range of neuropsychological phenotypes across the pubertal window and into adulthood.

Presumably links between pubertal timing and adult psychosexuality are driven by other variables, such as socioeconomic status (SES). For example, low SES has been associated with earlier spontaneous pubertal timing (Parent et al., 2003). Although it is unclear whether SES predicts psychosexuality (Szepsenwol et al., 2017), any such link would nevertheless require an underlying mechanism, such as differential CNS sensitivity to the organizing effects of sex steroids across puberty. Moreover, while low SES may predict earlier spontaneous puberty in the general population, it is likely in patients with IGD that the reverse is true, as patients with high SES may have greater access to general care and specialist services and hence initiate HRT at an earlier age. If our results reflected differences in SES, we would expect pubertal timing to relate to psychosexuality in opposite directions in typically developing participants and those with IGD, which we did not find. Nevertheless, future research should explore possible mediating and moderating roles of demographic variables, such as SES and education.

We also note that although we present life history theory-derived hypotheses and interpret our results within that theoretical context, significant criticisms have been raised about the underlying assumptions made when using life history theory to study intraspecies differences (Stearns and Rodrigues, 2020; Zietsch and Sidari, 2019). More research is necessary to understand the extent to which fundamental interspecific relationships between variables such as relative effort to mating versus parenting, the timing of reproductive maturity, extrinsic mortality, and resource availability are seen at the within-species level, which will in turn elucidate the utility of life history theory in the study of human psychological differences.

5. Clinical implications

Our findings shed light on hypotheses in behavioral neuroendocrinology that have clear implications for clinical practice. Pubertal timing is medically altered across a suite of conditions, including but not limited to IGD, constitutionally delayed puberty, and precocious puberty (Hoffman and Crowley, 1982; Nass et al., 2013). More novel application of medications to alter pubertal timing include prescribing such medications to gender nonconforming children (Vance et al., 2014). In all these cases, physicians weigh the physical and social sequelae of medically altering endocrine profiles via treatments such as HRT and GnRH antagonist “puberty blockers” (Palmert and Dunkel, 2012). However, the potential long-term effects of the timing of these treatments on cognition and behavior are often not considered. Together with other studies suggesting an effect of pubertal timing on sexually differentiated psychological traits (Beltz and Berenbaum, 2013; Doll et al., 2016; Shirazi et al., 2020), the present study suggests that potentially long-lasting effects on cognitive and behavioral phenotypes should also be considered when decisions are made regarding when to initiate HRT.

6. Conclusion

The present study finds evidence for relationships between earlier puberty and higher psychosexuality in adult men and women, and for differences in this relationship as a function of group (i.e., typically developing and IGD) and type of psychosexuality. These results clarify peripubertal organization of the brain and behavior and have clinical implications for physicians who treat conditions wherein pubertal timing is medically altered. The present research also highlights the utility of IGD as a disease model for investigating these effects as it approximates the experimental control possible in nonhuman models, and relationships with psychosexuality were generally more evident with documented HRT timing than with recalled timing of pubertal events. Nevertheless, future work should evaluate putative effects of pubertal timing on adult phenotypes using alternative approaches as well, including endocrine measures of pubertal timing in non-clinical populations and longitudinal designs.

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Declaration of Competing Interest

None.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.psyneuen.2020.104733.

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