COMMENTARY

Female Nonheterosexuality Is Associated with Both "Fast" and "Slow" Male-Typical Strategies: Implications for Evolutionary Scenarios

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The Target Article by Luoto, Rantala, and Krams (2018) is an ambitious attempt to review and synthesize the current hypotheses on the evolution of female nonheterosexuality in light of the available data on development, phylogeny, and proximate mechanisms (particularly hormonal factors). The article packs a lot of information, and I commend Luoto et al. for bringing many disparate sources of evidence to bear on this important question. The most original aspect of the article is the hypothesis that female nonheterosexuality results from selection for male-typical "fast" life history strategies (a similar argument regarding male nonheterosexuality was advanced by Xu, Norton, and Rahman, 2018). According to this hypothesis, the behavioral correlates of masculinized life history strategies include unrestricted sociosexuality, impulsivity, sensation seeking, risky sexual behavior, and a preference for physically attractive, but noninvesting partners. While exclusive homosexuality substantially reduces reproductive success, the benefits of faster strategies in male and/or female relatives who express milder variants of the same pattern may be enough to offset the fitness loss (Burri, Spector, & Rahman, 2015; Camperio Ciani, Battaglia, Cesare, Camperio Ciani, & Capiluppi, 2018).

My goal in this Commentary is to critically evaluate Luoto et al.'s (2018) life history hypothesis. While Luoto et al. make a compelling case that female nonheterosexuality is linked to physiological and behavioral masculinization, I argue that the data do not support a unique association with fast life history strategies. Rather, nonheterosexuality in women is associated with male-typical trait profiles at *both* ends of the fast–slow continuum. This empirical pattern has implications for some

of the evolutionary scenarios reviewed in the Target Article; more broadly, the case of female sexual orientation illustrates the potential pitfalls of conflating male-typical strategies with fast life histories.

Throughout the Target Article, Luoto et al. (2018) subscribe to the idea that, on average, male strategies are faster than their female counterparts because of men's higher investment in mating at the expense of parenting. (Although at times they describe this trade-off as one between reproductive and parental efforts, reproductive effort is a broader category that subsumes both mating and parenting.) This formulation of sex differences is sometimes found in the human and animal literature (e.g., Hämäläinen, Immonen, Tarka, & Schuett, 2018) and can be useful for some purposes, but is not without limitations. In particular, the allocation problems faced by males and females may be distinct enough that it becomes difficult to compare the sexes on the same axis used to describe individual differences within each sex. Consistent with upregulated mating effort, men score lower than women in agreeableness and higher in risk-taking, sensation seeking, and unrestricted sociosexuality. However, sex differences in other key life history-related traits, such as impulsivity and conscientiousness, are small and unreliable (Cross, Copping, & Campbell, 2011; Del Giudice, 2015, 2018). Also, boys tend to mature later than girls despite their higher mortality—another exception to the idea that males are faster strategists across the board (Bogin & Smith, 1996; Del Giudice, Gangestad, & Kaplan, 2015). Of note, in sexually reproducing species each offspring has one mother and one father, which constrains the scope for *average* sex differences in allocation to offspring quality versus quantity.

When addressing the issue of male- and female-typical strategies in humans, it may be more accurate to state that fast strategies involve somewhat different allocation patterns in the two sexes: while fast strategies in men are primarily characterized by increased mating effort and risky competition, fast strategies in women are more strongly linked to earlier maturation and reproduction, owing to the tighter physiological constraints on female fertility and fecundity

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(see Del Giudice, 2018). This alternative formulation allows for the fact that the male average of some mating-related traits (e.g., sociosexuality, risk-taking) is shifted toward the fast end of the continuum, without equating sex differences with overall differences in life history speed (for more discussion of sex-specific strategies, see Hämäläinen et al., 2018).

An even stronger challenge to the idea that male-typical strategies are necessarily faster comes from research on autistic-like traits, also known as the "broader autism phenotype" (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001). These traits include poor mindreading skills ("empathizing"), difficulties in communication (e.g., irony, humor, and other forms of nonliteral speech), restricted imagination, preference for routines, narrowly focused interests, and heightened attention to details and patterns, which in turn correlates with enhanced drive to reason about rule-based systems ("systemizing"; Baron-Cohen, 2003). Autistic-like traits are consistently higher in males, people in technical professions, and relatives of patients with autism spectrum disorder (ASD) (Baron-Cohen et al., 2001; French, Bertone, Hyde, & Fombonne, 2013; Ruzich et al., 2015a, b). The male-typical nature of this phenotype is underscored by the strongly sex-biased distribution of autism, which is about 4:1 overall, but reaches 10:1 for milder cases of ASD with normal intelligence and high familiality (Anney, 2013; Robinson et al., 2014). Unsurprisingly, research has found evidence that prenatal exposure to androgens contributes to the risk of autism (Auyeung & Baron-Cohen, 2013; Kosidou et al., 2016; Teatero & Netley, 2013). As my colleagues and I have shown, autistic-like traits in the nonclinical population bear the hallmarks of a male-typical variant of slow life history strategy: for example, they are associated with restricted sociosexuality, reduced sex drive, high investment in long-term romantic relationships, and low levels of impulsivity and sensation seeking (see Del Giudice, 2014, 2018; Del Giudice, Angeleri, Brizio, & Elena, 2010; Del Giudice, Klimczuk, Traficonte, & Maestripieri, 2014). Baron-Cohen (2003) famously described autism as the manifestation of an "extreme male brain," but this catchphrase disregards the remarkable variability of male strategies. At the fast end of the life history continuum, male-typical strategies include psychopathy and antisocial personality disorder (ASPD), whose behavioral profile is marked by extreme impulsivity, sexual promiscuity, and risky competition—another kind of "extreme male brain." At the slow end of the continuum, the spectrum from autistic-like traits to mild ASD reflects the existence of a male-typical strategy geared toward long-term resource allocation and indirect parental investment. (In contrast, most cases of severe ASD with intellectual disability seem to arise from rare deleterious mutations and are likely unrelated to variation in life history strategy; for extended discussion, see Del Giudice, 2018.)

As is apparent from the previous paragraph, autism is an ideal test case to discriminate between a generic masculinization account of female nonheterosexuality and Luoto et al.'s

(2018) more specific life history hypothesis. If nonheterosexuality were uniquely associated with fast strategies, it should correlate positively with impulsivity, sensation seeking, unrestricted sociosexuality, and psychopathy (as documented in the Target Article; see also Kerridge et al., 2017 for evidence of higher ASPD rates), but not (or negatively) with autistic-like traits and ASD. Conversely, a higher prevalence of autistic phenotypes in lesbian and bisexual women would be more consistent with a nonspecific pattern of masculinization cutting across the fast–slow continuum. As it turns out, the available data consistently support the latter alternative. In the nonclinical population, autistic-like traits predict increased likelihood of same-sex attraction and nonheterosexual orientation in females (Qualls, Hartmann, & Paulson, 2018). As noted in the Target Article, there is also some evidence that butches have higher systemizing scores than femmes (Zheng & Zheng, 2013). Likewise, women with ASD are much more likely to experience same-sex attractions and identify as bisexuals or lesbians compared with typically developing controls (Bejerot & Eriksson, 2014; Dewinter, De Graaf, & Begeer, 2017; George & Stokes, 2017, 2018; Gilmour, Schalomon, & Smith, 2012). Developmental data cast further doubt on the hypothesis of a specific association with fast life histories. As noted in the Target Article, findings on puberty timing in nonheterosexual females are mixed (and largely negative; see Bogaert, Friesen, & Klentrou, 2002; Grossman, Foss, & D'Augelli, 2014; Reese, Trinh, & Halpern, 2017; Savin-Williams & Ream, 2006). Luoto et al. also suggest that prenatal stress (a cue of harsher environmental conditions) should affect masculinization—and hence sexual orientation—in female offspring as a predictive adaptive response; however, the few available studies have failed to find consistent associations between prenatal stress and nonheterosexuality in women (Bailey, Willerman, & Parks, 1991; Ellis & Cole-Harding, 2001; Rahman, 2005).

To sum up, female nonheterosexuality is clearly linked to masculinized phenotypes, with a highly plausible femme–butch gradient of increasing masculinity. Of the hormonal factors reviewed in the Target Article, androgens enjoy the strongest empirical support, while the evidence is considerably more mixed for a role of estrogen and/or progesterone (and thus for the existence of "discrete" hormonal mechanisms in the development of butches vs. femmes). At the same time, the hypothesis of a specific link with fast strategies is challenged by the data on autism and by the lack of reliable associations among nonheterosexuality, puberty timing, and prenatal stress. Instead, the evidence indicates that nonheterosexual women are more likely to show male-typical fast strategies characterized by impulsivity, risk-taking, and unrestricted sociosexuality (including psychopathy and ASPD), but also male-typical slow strategies marked by elevated autistic-like traits.

What is the evolutionary relevance of these findings? As noted above, Luoto et al.'s (2018) life history hypothesis does not seem tenable, at least in its original formulation. The

hypothesis might be revised along these lines: if selection for male-typical fast traits in women (and/or their male relatives) translates into selection for higher masculinization, male-typical slow traits (including autistic-like traits) may also increase in frequency as a nonadaptive or weakly maladaptive side effect of selection for masculinization. The plausibility of this revised hypothesis hinges on the relative impact of masculinization on fast versus slow traits and the corresponding fitness costs and benefits. If, on the other hand, both fast and slow traits can be adaptive in different circumstances and/or different individuals in a population, the data reviewed here may support an extended version of the "balanced polymorphism of masculinity" hypothesis—one in which the potential adaptive benefits of masculinization include those of slow, future-oriented strategies. As discussed in the Target Article, Burri et al. (2015) found evidence of a common latent factor underlying same-sex attraction, gender nonconformity in childhood, and number of sexual partners. However, the latent factor accounted for a relatively small proportion of variance (13–22%) in the manifest behavioral traits. An intriguing possibility is that overall correlations among traits obscure the existence of more complex effects, whereby some nonheterosexual women (those engaging in slow strategies) tend to have *fewer* rather than more sexual partners. Crucially, a small number of sexual partners are disadvantageous in the context of fast, mating-oriented strategies, but can be perfectly adaptive for slow, parenting-oriented strategists (who should also be selected to have fewer, higher quality offspring; see Del Giudice et al., 2015). This complicates the interpretation of existing findings, including those on fertility in nonheterosexual women and their relatives (e.g., Camperio Ciani et al., 2018). As others have noted, contemporary fertility data are especially tricky to interpret because contraception and abortion decouple intercourse from reproduction, and indeed, there is some evidence that modern conditions boost the relative fertility of people with "slower" personality profiles (Woodley of Menie et al., 2017; see also Del Giudice, 2018). Finally, it might be interesting to consider a hypothesis that my colleagues and I advanced in a recent paper, namely, that early androgen exposure may increase susceptibility to environmental conditions in both sexes (Del Giudice et al., 2018). The hypothesis is admittedly speculative, but if it turns out to be correct, it may prove relevant to some of the issues raised in the Target Article and this commentary, from the variability of female strategies to the development of sexual fluidity.

In conclusion, Luoto et al. (2018) have made a valuable contribution to this topic, even if the life history hypothesis they advanced is problematic and should be revised or abandoned. I expect that their call for deeper consideration of proximate mechanisms, development, and phylogeny will have a salutary effect on the scientific debate. There is still much we do not understand about the origin and development of sexual orientation (Bailey et al., 2016), but research is becoming more theoretically sophisticated and empirically grounded, and the

solution of this endlessly fascinating puzzle may soon be within our reach.

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COMMENTARY

Response to Commentaries: Life History Evolution, Causal Mechanisms, and Female Sexual Orientation

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Female sexual orientation and its various species-wide and species-specific manifestations comprise a topic to which one scientific article can hardly do justice. We have nevertheless attempted to analyze the topic more broadly than any one scientific article of which we are aware. To this end, we applied Tinbergen's four questions and life history theory, the most holistic tools available to biological psychologists, to explore the evolutionary-developmental mechanisms underlying variation in female sexual orientation (Luoto, Krams, & Rantala, 2019).

A Word on Terminology

We were surprised that Figueredo, Fernandes, and Peñaherrera-Aguirre (2019) objected to the term "nonheterosexual" which we used throughout the Target Article to refer to women whose Kinsey score is nonzero (i.e., Kinsey 1–6: Luoto et al., 2019). Figueredo et al.'s principal reason to object to the term arose from the fact that few women are exclusively homosexual. We see no reason to change the terminology based on this objection: the relative proportions of Kinsey 1s, 2s, 3s, 4s, 5s, and 6s do not influence the usefulness of the term "nonheterosexual" in distinguishing between heterosexual and nonheterosexual women.

As an alternative, Figueredo et al. (2019) suggested the term "atypical sexuality," a term which we find problematic not only because it carries a stronger heteronormative undertone but also because it is too broad for the purposes of sexual orientation research. "Atypical sexuality" could, after all, refer to zoophilia, pedophilia, asexuality, and various paraphilias. None of these are directly relevant to research on female sexual orientation, which is why we advocate using the term "nonheterosexual" in contexts where only sexual orientation is concerned. "Atypical sexuality" could be used in broader conceptualizations of sexual behavior than the one which we presented, but those considerations fall outside the scope of the Target Article, the five Commentaries, and our response.

Maternal Stress and Female Sexual Orientation

As the first researchers to apply life history (LH) theory to the study of female sexual orientation, we presented evidence for the hypothesis that prenatal stress experienced by mothers could affect masculinization and sexual orientation in female offspring, potentially forming a predictive adaptive response to harsh and unpredictable life conditions (Luoto et al., 2019). Del Giudice (2019) criticized this hypothesis by noting that "the few available studies have failed to find consistent associations between prenatal stress and nonheterosexuality in women," citing three articles. A more detailed inspection of the findings of those articles, however, reveals interesting patterns and calls into question Del Giudice's criticism.

First, Ellis and Cole-Harding (2001) found that mothers of bisexual women recalled experiencing greater stress during pregnancy than mothers of either heterosexuals or homosexuals. Figure 2 in Ellis and Cole-Harding shows that the mothers of bisexual offspring recalled being exposed to much higher levels of stress during pregnancy than the mothers of heterosexual and homosexual female offspring. Yet because of a small sample size of bisexual women ($n = 48$), only the amount of

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recalled stress during the eighth month was statistically different between the groups. What is striking is the magnitude of the effect of stress recalled by mothers of bisexual offspring versus mothers of offspring of other sexual orientations, with very little overlap in confidence intervals between bisexuals and the other groups (Fig. 2, Ellis & Cole-Harding, 2001). Irrespective of *p* values, this calls for a replication using larger samples of nonheterosexual women (cf. Trafimow et al., 2018).

The second article referred to by Del Giudice is Bailey, Willerman, and Parks (1991). Bailey et al. found that both between- and within-family analyses provided support for a small maternal stress effect for nonheterosexuality in female offspring. However, Bailey et al. were sceptical about their findings because they did not have a plausible explanation for the proximate mechanism that links maternal stress to increased same-sex preferences in female offspring. Importantly, we provided evidence for that mechanistic connection, linking maternal stress to heightened bioavailability of estrogen and same-sex sexual preferences in female offspring (Luoto et al., 2019).

The third article that Del Giudice (2019) cited to support his argument about inconsistent findings between prenatal stress and nonheterosexuality in women studied fluctuating asymmetry (Rahman, 2005). The hypothesis here—unvoiced by Del Giudice but explained by Rahman (2005, p. 384)—is that "homosexuality could represent a neurodevelopmental perturbation in the human sexual orientation system" (see also Tran, Kossmeier, & Voracek, 2018, for a recent review). If developmental instability caused nonheterosexual orientation, it would, the hypothesis goes, also cause other traces of developmental instability, such as fluctuating asymmetry. Rahman (2005) found no significant differences in fluctuating asymmetry between lesbian and heterosexual women. We note that this null finding in itself does not challenge the findings reported by Bailey et al. (1991) and Ellis and Cole-Harding (2001) because fluctuating asymmetry would be an indirect and possibly only a weak marker of stress experienced by the pregnant mother. Moreover, Rahman (2005) did not include bisexual women in his analyses of fluctuating asymmetry, and it is precisely in bisexual women that Ellis and Cole-Harding (2001) reported higher recalled maternal stress during pregnancy.

A study that Del Giudice did not cite found a very large (Cohen's $d = 1.15$) difference in the composite score of fluctuating asymmetry between heterosexual and homosexual women, with homosexual women having significantly higher fluctuating asymmetry (Hall & Schaeff, 2008). Hall and Schaeff noted that Rahman's (2005) study suffered from methodological challenges highlighted in the literature.[1] In sum, a more careful analysis of the existing evidence does indicate a stronger

relationship between maternal stress and nonheterosexuality in female offspring than Del Giudice concedes. Acknowledging the possible mechanistic connection that heightened bioavailability of estrogen creates between maternal stress and female nonheterosexuality (especially bisexuality) makes the association more robust both theoretically and empirically (Luoto et al., 2019).

To our knowledge, fluctuating asymmetry has not been analyzed by comparing bisexual women with heterosexual and lesbian women. Because of the higher prenatal stress levels recalled by bisexual women's mothers (Ellis & Cole-Harding, 2001), this would be an interesting avenue for future research, especially if butch/femme differences were also included in the analyses.

Butch/Femme

Diamond and Alley (2019) questioned the emphasis that we placed on the butch/femme classification in the Target Article. They doubt whether "these subtypes represent 'natural types' especially in light of the weak empirical basis for [Luoto et al.'s] claims and their surprisingly scant attention to the well-documented historical and cultural bases—and hence social malleability—of these categories" (Diamond & Alley, 2019). Diamond and Alley cited seven articles to support this claim. A closer inspection calls into question the generalizability of those studies: One is a study of two Angelina Jolie films (Stasia, 2003), while the others are personal reports or suffer from otherwise unrigorous methodology.

Diamond and Alley (2019) argued that there is weak empirical evidence to support our thesis about the importance of butch/femme categories. This argument is astonishing. We provided broad evidence showing physiological, psychological, and behavioral differences between butch and femme women (see sections "Categories of Nonheterosexual Women" and "Psychological Characteristics of Nonheterosexual Women," as well as Table 2 in Luoto et al., 2019). It appears that the main thrust of Diamond and Alley's criticism is concerned with butch/femme as identity categories. However, this misses the important point that we made in the Target Article: that butch/femme may constitute important biological categories. These are two different things and may lead those who unscientifically (Bailey, 2019) view humans as performers of socially constructed gendered identities to misconstrue and become opposed to some of the main arguments made in the Target Article. The difference in

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Footnote 1 (continued)

women ($n = 27$). A larger study by Hall and Schaeff (2008), in contrast, did record statistically significant differences in fluctuating asymmetry scores between heterosexual ($n = 97$) and homosexual women ($n = 75$). Notably, Hall and Schaeff did not include bisexual women in their study. Miller et al. combined bisexual and homosexual women into one 'homosexual' category (Kinsey score ≥ 2) in their analyses.

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[1] Limited sample size, for example, prevented Miller, Hoffmann, and Mustanski (2008) from finding statistically significant differences in fluctuating asymmetry scores between heterosexual ($n = 41$) and homosexual

conceptualizing butch/femme as biobehavioral as opposed to identity categories appears to have caused Diamond and Alley's disagreement. The evidence we reviewed in the Target Article furnishes broad empirical support for the biobehavioral conceptualization, whereas opinions inevitably differ on how useful the butch/femme distinction is to characterize nonheterosexual women's identities. Nevertheless, it strikes us as an empirically interesting finding that women's self-identification as either butch or femme is statistically related to individual differences in a range of biobehavioral and psychological traits, as reviewed in the Target Article, and that this variation occurs in the direction predicted by life history theory (Table 2, Luoto et al., 2019).

Although we disagree with Diamond and Alley (2019) about the conceptual utility of butch/femme, we do agree with their point about the importance that should be placed on analyzing lesbians and bisexuals separately. This is indeed customary in most existing research on nonheterosexual women (if it is not, it should be), and we followed this practice by distinguishing between various degrees and kinds of female nonheterosexuality in the Target Article. Diamond and Alley pointed out that bisexual women have had higher rates of early-life abuse and adversity, early sexual behavior, adolescent pregnancy, and high-risk sexual behavior than lesbians. These are interesting findings and they serve as a useful extension to our life history analyses (Luoto et al., 2019).

In this context, it should be noted that one of our principal aims was to compare nonheterosexual women with heterosexual women. The other important contribution of the Target Article was to call more attention to the butch/femme phenotypes, because differences between these phenotypes are only occasionally considered in research on nonheterosexual women. We made the case that biologically, evolutionarily, and psychologically sophisticated research should take those categories into account, as conflating them runs the risk of not detecting significant differences between different types of nonheterosexual women (Luoto et al., 2019). Distinguishing between mostly heterosexuals, bisexuals, and lesbians should already be the modus operandi in sexual orientation research (e.g., Calzo et al., 2017; Zheng, Wen, & Zheng, 2018). We reviewed substantive evidence indicating that the butch/femme division should also be more systematically utilized in female sexual orientation research (Luoto et al., 2019).

Hallmarks of Fast Life Histories in Nonheterosexual Women

Despite providing valuable information on the link between bisexuality and fast LH strategies, Diamond and Alley (2019) misrepresented the existing findings in making their point. They noted that "it is bisexuals rather than exclusive lesbians who have been found to have the highest rates of early-life abuse and adversity," citing several studies to support this claim. However,

a closer inspection of their sources reveals findings that are inconsistent with this claim. Andersen and Blosnich (2013), for example, found higher exposure to violence among gay/lesbian participants than among bisexual participants. They also found that gay/lesbian and bisexual respondents had, respectively, nearly 1.7 and 1.6 times the rate of adverse childhood experiences compared with their heterosexual peers. Likewise, McLaughlin, Hatzenbuehler, Xuan, and Conron (2012) reported higher rates of child abuse in lesbian/gay participants than in bisexuals. Alvy, Hughes, Kristjanson, and Wilsnack (2013), also cited by Diamond and Alley, offered mixed evidence for the claim about higher early-life adversity in bisexual women. Although other studies cited by Diamond and Alley do show the mentioned patterns (replicated in a more recent study by Schwab-Reese, Currie, Mishra, & Peek-Asa, 2018), the findings are not uniform across studies. Therefore, we find it curious that Diamond and Alley would call it "a *robust* pattern of results" (emphasis added) and that they criticize our work for neglecting to mention it. To us, the clearest pattern that emerges from these findings is that nonheterosexual women ("mostly heterosexuals," bisexuals, and lesbians combined) have faced more early-life adversity and show significantly more hallmarks of fast LH strategies than heterosexual women (Friedman et al., 2011; Luoto et al., 2019; Schwab-Reese et al., 2018; Tornello, Riskind, & Patterson, 2014; Zou & Andersen, 2015).

Diamond and Alley (2019) further claimed that "exclusively lesbian women do not generally show greater sociosexuality or sex drive than exclusively heterosexual women." However, it is striking to us that this claim is not supported by the evidence that Diamond and Alley cited. Schmitt (2006) found that whereas bisexual women had the highest sociosexual behavior scores, lesbians still had significantly higher sociosexual behavior scores than heterosexual women. Semenyna, Belu, Vasey, and Honey (2018) reported higher sociosexual attitude scores in lesbians than in heterosexual women (despite their sociosexual behavior scores being similar). Lippa (2006) found that lesbians had a higher sex drive than heterosexual women, a finding replicated by Semenyna et al. (2018). Lesbians also have more than twice the number of male sex partners than heterosexual women (Tornello et al., 2014). The percentage of lesbians who have used emergency contraception (27.3%) is much higher than in heterosexual women (16.3%; Tornello et al., 2014). Lesbians' first intercourse with men occurs at a younger age (14.86, SD = 1.67) than heterosexual women's first intercourse with men (15.68, SD = 1.71; Tornello et al., 2014). We conducted a *t* test to compare these findings, and found that the age difference between lesbians' and heterosexual women's first intercourse with men was statistically significant, $t(2481) = 3.22$, $d = -.49$, $p < .002$. The difference between bisexual women (14.42, SD = 2.04) and heterosexual women was even larger, $t(2616) = 9.43$, $d = -.67$, $p < .001$. In the aggregate, these findings show how inaccurately Diamond and Alley represented the existing research. Importantly, these findings furnish additional

evidence for our argument that nonheterosexual women—that is, Kinsey 1 through 6—show multiple hallmarks of faster life history strategies when compared with heterosexual women (Luoto et al., 2019).

Although we agree with Diamond and Alley (2019) that bisexual women and lesbians should be analyzed separately, we also highlight that the evidence on similarities between lesbian women and heterosexual women is much less clear than Diamond and Alley suggest. It is ironic and counterproductive that Diamond and Alley criticize us for misrepresenting the existing findings, while in their counterargument they clearly misrepresent even the data that they cite themselves. The existing findings suggest that although lesbians are similar to heterosexual women on some psychobehavioral traits, lesbians show indicators of significantly faster LH strategies on other traits, such as sex drive and age at first intercourse with men (Lippa, 2006; Semenyna et al., 2018; Tornello et al., 2014).

Life History Strategies: Causal Mechanisms

To analyze these questions in more detail, it can be valuable to evaluate the direction of causality: Do nonheterosexual women exhibit hallmarks of fast LH strategies because of early-life adversity or are they more likely to experience early-life adversity because of fast LH strategies? What is the extent of genetic confounding underlying these findings (e.g., Hartling et al., 2019; Hartman, Widaman, & Belsky, 2015; Luoto, 2019b; Ramos, Griffin, Neiderhiser, & Reiss, 2019; Willems, Boesen, Li, Finkenauer, & Bartels, 2019)? Are early-life adversity and hallmarks of fast LH strategies a family-wide pattern or are nonheterosexual women the only ones in the family who exhibit hallmarks of fast LH strategies? In other words, to what extent does their family environment create conditions that activate or intensify the (genetic) predisposition to adopt fast LH strategies?

Current evidence suggests the existence of familial patterns of fast LH strategies that profoundly shape the developmental niche of nonheterosexual women. Zou and Andersen (2015) and Xu, Norton, and Rahman (2019), for instance, found indicators of faster LH strategies in the family environments of nonheterosexual women as opposed to heterosexual women. Nonheterosexual women report a higher number of adverse childhood events such as parental divorce rates, household alcohol or drug problems, household verbal or physical abuse, household member incarceration (Zou & Andersen, 2015), parental absence since birth, low prenatal family socioeconomic position, and poorer parent–child relationships (Xu et al., 2019). Although Diamond and Alley (2019) suggested that adverse early-life experiences might be central factors in shaping the fast LH strategies that we observed in nonheterosexual women, the Target Article provides ample evidence that this phenotypic plasticity already occurs in utero (Luoto

et al., 2019). Why else would we find clear linkages between biomarkers of prenatal androgen exposure, hallmarks of fast LH strategies, and same-sex sexual preferences in women? Nevertheless, we do not rule out the possibility that early-life adversity may further influence or fine-tune life history characteristics that were more fundamentally shaped by the more distal processes of genetic heritability and prenatal development (Hartman et al., 2015; Krams et al., 2019; Luoto, 2019b; Luoto et al., 2019; Willems et al., 2019; see also Frankenhuis, Nettle, & Dall, 2019; Nelson, 2017).

Inconsistent Support for the Extreme Male Brain Hypothesis of Autism Spectrum Disorder

The main thrust of Del Giudice's (2019) criticism of the hormonally mediated fast life history strategy hypothesis of female nonheterosexuality was based on findings on autism spectrum disorder (ASD). As Del Giudice pointed out, women with ASD are significantly more likely to experience same-sex attractions and to identify as bisexuals or lesbians compared with typically developing control women. Autistic-like traits, Del Giudice argued, are hallmarks of male-typical slow life history strategies.

This criticism does not challenge the hormonally mediated fast life history strategy hypothesis for the following reasons. First, Del Giudice (2019) did not mention that the same studies that have found that nonheterosexulity is more common in ASD women found that nonheterosexual orientation was also much more common in men with ASD than in neurotypical men (George & Stokes, 2018). Importantly, as indicated in Figueredo et al.'s (2019) Commentary, homosexual men do not appear to have faster life history strategies than heterosexual men. Homosexual men are also cognitively more feminine than heterosexual men (Xu, Norton, & Rahman, 2017). Therefore, the potential association between the extreme male brain hypothesis (Del Giudice, 2019; Greenberg, Warrier, Allison, & Baron-Cohen, 2018), male-typical slow life history strategies, ASD, and nonheterosexuality is much more complicated, or much weaker, than what Del Giudice implied.

Bejerot and Erikson (2014) found that masculinity (e.g., assertiveness, leadership, and competitiveness) was lower in women and men with ASD than in controls. Thus, the extreme male patterns of cognitive functions in autistic persons (Geary, 2018; Greenberg et al., 2018) do not seem to extend to other gender-typical traits and sexual orientation (Bejerot & Eriksson, 2014). Autistic males also show a shift toward neurotypical female brain activity in sensorimotor networks, providing further evidence against the extreme male brain hypothesis (Floris, Lai, Nath, Milham, & Di Martino, 2018).

Second, recent studies suggest that ASD is caused by gut dysbiosis, neuroinflammation (Hughes, Rose, & Ashwood,

2018; Rose et al., 2018; Zhang, Ma, Zhang, He, & Wang, 2018), maternal autoimmune response during gestation (Braunschweig et al., 2013), and/or exposure to certain environmental pollutants and industrial chemicals, including endocrine-disrupting compounds (Thongkorn et al., 2019). It therefore seems that rather than being a byproduct of an extreme male brain, or rather than comprising male-typical slow life history strategies, many traits associated with ASD are pathological side effects of neuroinflammation or maternal autoimmune response.

If ASD is caused by maternal autoimmune response or by endocrine-disrupting compounds, it may explain why homosexual orientation is more common in people with ASD, since autoimmune response and endocrine-disrupting compounds may influence prenatal hormone exposure and sexual differentiation of the brain. Immune-system-derived mast cells are known to be involved in autoimmune responses and diseases (Costela-Ruiz, Illescas-Montes, Pavon-Martinez, Ruiz, & Melguizo-Rodriguez, 2018), including autism (Theoharides, Stewart, Panagiotidou, & Melamed, 2016). They are also a primary target for the masculinizing hormone estradiol. Mast cells comprise primary mediators of brain sexual differentiation. Newborn female rats treated with a mast cell stimulator compound develop male-typical copulatory behavior with other females (Lenz et al., 2018). The neurodevelopmental link between mast cells, brain sexual differentiation, sexual behavior, and autoimmune responses constitutes a mechanism that may explain the observed associations between maternal autoimmune response, ASD, and sexual orientation. Research on the fraternal birth order effect in male homosexuality (Balthazart, 2018; Bogaert et al., 2018) could also benefit from considering this mechanistic link, seeing that mast cell inhibition decreases masculinization of sexual behavior in males (Lenz et al., 2018).

It is important to note that ASD is a highly heterogeneous disorder that is likely to have multiple etiologies and converging pathophysiological pathways (Hughes et al., 2018; Thongkorn et al., 2019). ASD should not be referred to as a male-typical variant of slow life history strategy nor as an example of extreme male brains: The totality of its phenotypic outcomes and etiological pathways are not consistent with predictions that arise from those hypotheses. Thus, Del Giudice's (2019) suggestion to revise the fast life history strategy hypothesis because of findings on ASD should be abandoned, based as it was on an inadequate assessment of ASD etiology and phenotypic outcomes. ASD cannot be used to test a generic masculinization hypothesis of female nonheterosexuality.

The Endocrinological Mechanisms of Female Sexual Orientation and Brain Sexual Differentiation

Cornil and Bakker (2019) criticized our model about the effect of prenatal hormones on the origin of the butch–femme continuum by arguing that the model is "too strongly based on specific findings reported in rodents, which are difficult to extrapolate to humans at this stage." This comment misrepresents the scientific evidence that we used to support our model. Cornil and Bakker ignore one of the strongest lines of evidence for our model: the influence of diethylstilbestrol (DES) on sexual orientation. As noted in the Target Article, DES is a synthetic estrogen which increases the probability of females to develop bisexual or homosexual preferences—an effect found in rats, rhesus macaques, and humans (Luoto et al., 2019).

There is further evidence to support our model: We did not have space to present all of it in the Target Article. Treating rhesus macaques with flutamide (a nonsteroidal anti-androgen that blocks androgen receptors) late in gestation paradoxically hypermasculinized males' behavior despite preventing full genital masculinization (Wallen, 2005). This finding is consistent with the hypothesis that estrogen masculinizes brains also in primates: Estrogen receptors, after all, would not be blocked by flutamide (which only blocks androgen receptors), while the testes will still produce testosterone that is aromatized to estrogen (Roselli, Meaker, Stormshak, & Estill, 2016; Wallen, 2005). When androgen receptors have been blocked, there is an increased bioavailability of testosterone to be aromatized to estrogen, leading to some behaviors to become more masculinized. The finding that flutamide hypermasculinizes behaviors, as it did in rhesus macaque males, has been replicated in rams. Despite having phenotypically female genitalia, caused by the elimination of androgenic actions of testosterone with flutamide, flutamide-treated rams showed more frequent mounting behavior with females than control rams (Roselli et al., 2016).

Cornil and Bakker (2019) cited a review article by Roselli and Stormshak (2009) as evidence against our endocrinological model of female sexual orientation. Cornil and Bakker write that "inhibiting aromatase during gestation failed to affect the volume of this nucleus [oSDN-POA: ovine sexually dimorphic nucleus] as well as sexual partner preference (Roselli & Stormshak, 2009), thereby not supporting a role for estrogens in the development of this nucleus and perhaps sexual orientation." This claim does not, however, stand up to closer scrutiny. The study which is cited in Roselli and Stormshak's review article administered 1,4,6-androstatriene-3,17-dione (ATD), an aromatase inhibitor, to sheep during gestation (Roselli, Schrunk, Stadelman, Resko, & Stormshak, 2006). In contrast to Cornil and Bakker's statement, Roselli et al. (2006) noted that exposure to ATD partially disrupts masculinization of adult copulatory behavior. Roselli et al. concluded that aromatization of

testosterone to estrogen is necessary for complete behavioral masculinization in sheep.

The sample size in this study was only six control males and eight ATD-treated males; the differences were statistically significant only in the number of female-directed mounts, which were lower in ATD-treated males (Roselli et al., 2006). A larger sample would have been needed to find statistically significant differences in the volume of oSDN-POA and/ or behavioral traits such as male-directed mounts. This was achieved in a more recent study that Cornil and Bakker failed to cite. Roselli et al. (2016) reported that blocking androgenic actions of testosterone in utero with flutamide in rams ($n=7$) led to phenotypically female external genitalia, hypermasculine sexual behavior, and an oSDN-POA intermediate in volume between control rams ($n=7$) and ewes ($n=6$) (significantly different from both). Thus, consistent with our model (Luoto et al., 2019), estrogen does masculinize brains and behaviors also in other species than just rats and humans.

The finding that ATD (an aromatase inhibitor) does not have a strong feminizing effect on rams (Roselli et al., 2006) does not rule out the possibility that estrogen masculinizes sexual preferences in females. At first glance at the above findings, one could conclude, as Cornil and Bakker (2019) did, that testosterone masculinizes the brain directly: This is because ATD prevents aromatization of testosterone to estrogen, but results in partially masculinized behavior despite the lack of aromatization. However, there is a mechanism that may explain why blocking aromatization of testosterone to estrogen with ATD does not prevent masculinization of the brain. In addition to estradiol, there is a second major endogenous estrogen, 5α-androstane-3β,17β-diol (3βAdiol), which is produced in immature ovaries and testes (reviewed in Sugiyama, Barros, Warner, & Gustafsson, 2010). It is an estrogenic metabolite of 5α-dihydrotestosterone (DHT). Males exposed to ATD are unable to aromatize testosterone to estrogen; however, their DHT is still able to convert to 3βAdiol that can bind to estrogen receptors and masculinize the brain. Unlike estradiol, 3βAdiol does not bind to alpha-fetoprotein and has free access to the embryonic brain (Sugiyama et al., 2010).

Cornil and Bakker (2019) also stated that there is no evidence that estradiol masculinizes INAH3 and sexual orientation in humans. They support this statement by citing findings showing that men with gene mutations in either the estradiol receptor (Smith et al., 1994) or the aromatase gene (Jones, Boon, Proietto, & Simpson, 2006) are heterosexual and have a male-typical gender identity. These findings on men do not provide evidence against estradiol's masculinizing influence on women's brains (cf. Jain, Huang, & Woolley, 2019; Luoto & Rantala, 2018; Oberlander & Woolley, 2016). It is important to note that there are two different kinds of estrogen receptors: ERα and ERβ (Sugiyama et al., 2010). Thus, a mutation in one of the receptor types does not automatically mean that the other receptor type would not function (and masculinize the brain). Since 3βAdiol is able to masculinize brains without the aromatase enzyme (Sugiyama et al., 2010), it is possible that the masculinization of males' brains occurring despite a mutation in the aromatase gene results from the effects of 3βAdiol. Based on studies on DES and flutamide reviewed above, it appears that estrogens are able to masculinize brains in several species. Nevertheless, it is important to repeat that our model involves the possibility that testosterone can directly masculinize the brain.

Cornil and Bakker (2019) were surprised that we did not consider the feminizing role that estrogen has on brain development, except in Fig. 1 of the Target Article (Luoto et al., 2019). This is a valid point. The reason why femme lesbians can be more feminine in some of their behavioral characteristics (excluding sexual preference and sexual behavior) than heterosexual women may indeed be the feminizing effect of estrogen. This is supported by findings on women who were prenatally exposed to DES. They were more feminine in their behavior than control women (Bekker, VanHeck, & Vingerhoets, 1996). More specifically, DES-exposed women had more feminine Bem Sex Role Inventory scores, they had a stronger wish for having children, and they expressed more emotionality concerning the subject than controls (Bekker et al., 1996).

It is important to note that all women are exposed to estrogen and testosterone during their development—only the doses differ. Thus, heterosexuals, bisexuals, and femme and butch lesbians do not form a single continuum; rather, neurodevelopmental mechanisms of sexual differentiation lead to a two-dimensional space in which the prevalence of (female) phenotypes in populations decreases as testosterone and estrogen exposure increase (Fig. 1). The two-dimensionality of the neurodevelopmental pathways leading to variation in female sexual orientation can partially account for the finding that not all lesbians identify themselves clearly as butch/femme, even though the majority do (reviewed in Luoto et al., 2019).

Figueredo et al. (2019) requested clarification on our model of the endocrinological mechanisms behind the femme/butch phenotypes. They asked:

> If the high levels of prenatal T to which the future 'butch' phenotypes are exposed are nonetheless converted into E within the brain by the process of aromatization (Leshner 1978), then what distinguishes this situation from the high levels of prenatal E to which the future 'femme' phenotypes are exposed? The actions of T are characterized as 'more global masculinizing effects' that produces [*sic*] 'a more masculine brain'; but how is this possible if the T is aromatized to E within the brain?

Figueredo et al. thought that there may be a typo, as otherwise this would seem to set up a paradox. It is not a typo, but it does appear that we should have explained the neuroendocrinology of sexual differentiation of the brain more deeply.

Although it was previously thought that female brains develop without the effect of sex hormones (with "female"
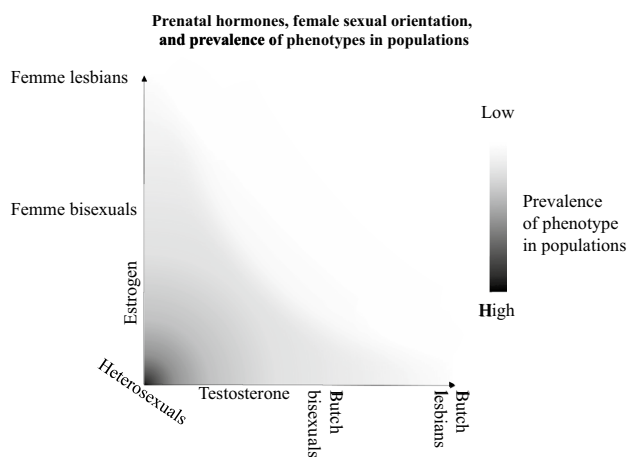
Fig. 1 A model on the influence of prenatal testosterone and estrogen exposure on the development of sexual orientation in women. Prevalence of respective phenotypes in populations is shown in the model (prevalence studies reviewed in Luoto et al., 2019)

being the "default state" of brains, and the development of a male brain requiring the influence of sex hormones: McCarthy, 2012), more recent evidence indicates that estrogens play an active role in the feminization of the brain (Bakker & Brock, 2010; Koebele & Bimonte-Nelson, 2015). Rodent studies suggest that there may be different sensitive periods for masculinization and feminization of the brain (Bakker & Brock, 2010). When masculinization occurs, estrogens (possibly those aromatized from testosterone) masculinize those parts of the brain that have estrogen receptors during that particular moment. Testosterone may directly masculinize those parts of the brain that have testosterone receptors. When feminization takes place, the location of estrogen receptors influences which parts of the brain will be feminized. Estrogen-driven feminization may even extend to puberty (Bakker & Brock, 2010). Thus, the timing of the activation of estrogen receptors, together with their location and the magnitude of the estrogen dose, is crucial for masculinization and feminization.

As noted above, there are two different estrogen receptors: ERα and ERβ (reviewed in Sugiyama et al., 2010). Locations of these receptors differ in the brain. ERα is generally expressed in the regions of the brain involved in reproductive functions, including the medial hypothalamic nucleus, the bed nucleus of stria terminalis, the ventromedial nucleus, and the posterodorsal part of the medial amygdala, while ERβ is located in hippocampus, the amygdala, and midbrains (reviewed in Sugiyama et al., 2010). 3βAdiol (an estrogen that is converted from DHT) binds more strongly to ERβ than to ERα and has similar transcriptional activity as estradiol (Sugiyama et al., 2010). The fact that butch lesbians have more masculine bodies and behavioral patterns than other women (Luoto et al., 2019) indicates that butch lesbians may have been exposed to higher 3βAdiol and testosterone levels (and aromatized estrogen levels) prenatally than

heterosexual women and femme lesbians. Thus, an alternative hypothesis to that provided in the Target Article is that in butch lesbians, the parts of the brain that have ERβ receptors and those that have ERα receptors and those that have testosterone receptors are masculinized. In femme lesbians, the hypothesis continues, only the parts of the brain that have ERα receptors become masculinized, while other parts of the brain remain in their default state, ready for the critical period of feminization. In both sexes, testosterone may masculinize parts of the brain directly (without conversion to estrogen), which may explain the higher overall masculinity of butch lesbians than femme lesbians since testosterone also induces masculinization of the body (Luoto et al., 2019; O'Shaughnessy et al., 2019).

Pleasure, Dopamine, Female Sexual Orientation, and Life History Strategies

As a final point of their Commentary, Diamond and Alley (2019) suggested that the role of pleasure should be given more consideration in research on female sexual orientation. We agree (Luoto, 2017; Luoto & Rantala, 2017), and although we did not have adequate space to discuss pleasure at length in the Target Article, we did note the salient and important pattern of nonheterosexual women being more predisposed than heterosexual women to immediate rather than postponed rewards, especially with regard to sexual behavior and substance use (Luoto et al., 2019).

Intriguingly, recent experimental work has suggested the existence of a mechanistic link between neonatal estradiol exposure, increased dopamine synthesis and transmission, and increases in female rats' likelihood of being conditioned to morphine, a drug of abuse (Bonansco et al., 2018). Research on male rats has shown that both estrogen and testosterone treatment increases dopamine content in substantia nigra–ventral tegmental area and dopamine release in nucleus accumbens (Espinosa et al., 2016). Dopaminergic neurotransmission underlies mesocorticolimbic pathway activation—dopamine is thus a central component of the "reward circuit" that is sensitive to natural rewards such as sex and food but also to drugs of abuse (Bonansco et al., 2018; see also Borland et al., 2019; Struik, Sanna, & Fattore, 2018). In the Target Article, we reviewed evidence indicating that nonheterosexual women are more likely to pursue these rewards than heterosexual women are. It is thus an intriguing possibility that increased estrogen and/or testosterone exposure in nonheterosexual women (Luoto et al., 2019) may also drive heightened dopaminergic neurotransmission in the mesocorticolimbic pathway in the same way as it does in rats (Bonansco et al., 2018; Espinosa et al., 2016; Struik et al., 2018). The existing evidence indicates that the physiological and behavioral outcomes in nonheterosexual women (Luoto et al., 2019) are consistent with that mechanistic explanation.

There is wider recognition in biomedical research on the relationship between early-life adversity, increased reward seeking, substance use, and pursuit of other highly rewarding stimuli such as high-sugar and high-fat foods (Berens, Jensen, & Nelson, 2017; Duffy, McLaughlin, & Green, 2018; Richardson et al., 2016). Other research has correspondingly linked early-life adversity with fast LH strategies, obesity (Maner, Dittmann, Meltzer, & McNulty, 2017; see also Luoto et al., 2019), and female nonheterosexuality (Xu et al., 2019). A genetic predisposition for heightened dopaminergic (and androgenic) stimulation has also been linked with early reproduction and fast LH strategies (Luoto, 2019b; Minkov & Bond, 2015, and references therein). We thus agree with Diamond and Alley's (2019) emphasis on the important relationship between early-life adversity and heightened reward seeking. Elevated reward seeking is a psychological mechanism that has the potential to explain the majority of the behavioral outcomes that we observed in nonheterosexual women (Luoto et al., 2019) and heightened dopaminergic neurotransmission in the mesocorticolimbic pathway, brought about by elevated sex hormone exposure, may be one proximate mechanism driving the psychobehavioral variation between women of different sexual orientations (cf. Struik et al., 2018).

Different Evolutionary-Developmental Mechanisms Underlying Female and Male Sexual Orientation

Figueredo et al. (2019) pointed out that although the hormonally mediated fast life history strategy hypothesis that we proposed to explain nonheterosexuality is well supported in women, it may not be generalizable to men. Sure enough: we only made the case for the hypothesis with regard to female sexual orientation (Luoto et al., 2019). To evaluate the generalizability of the hypothesis, Figueredo et al. noted that homosexual men experience higher childhood adversity than heterosexual men and that "studies of hormonal changes in utero as predictors of sexual orientation in men are abundant." Figueredo et al. would therefore predict, following the logic of our Target Article, that homosexual men are hypermasculinized.

Crucially, however, although Figueredo et al. (2019) noted the abundance of studies on prenatal hormonal changes as predictors of sexual orientation in men, they failed to mention any specificities regarding the findings of those studies. In short, the abundant studies referred to by Figueredo et al. have found no evidence of heightened exposure to androgens in the prenatal development of homosexual men—with the exception of larger penises in adulthood (Breedlove, 2017). The mechanisms and the timing of genital as opposed to neurobehavioral masculinization differ in humans (Hines, 2011; O'Shaughnessy et al., 2019; Panksepp, 1998, p. 233), and there is no evidence of heightened neurobehavioral masculinization in homosexual

men (Breedlove, 2017; Xu et al., 2017). In contrast, there is an accumulating body of evidence for the fraternal birth order effect on male but not female homosexuality (Balthazart, 2018; Blanchard, 2018). Thus, the mechanisms that cause variation in female and male sexual orientation appear to be different.

Because of fundamental differences in male and female reproductive biology, ecology, and psychology (Archer, 2019; Luoto, 2019a), there is significant reason to expect sex differences also in ultimate-level causes of nonheterosexuality. Because of these fundamental sex differences, we might expect to see the kin selection hypothesis play out differently between homosexual males (Semenyna et al., 2017; VanderLaan, Petterson, & Vasey, 2017) and homosexual females (Luoto et al., 2019). Indeed, if life history theory was used in the study of male sexual orientation, it would be unlikely to form similar patterns as in nonheterosexual females (cf. Xu et al., 2018, 2019) because of the abovementioned fundamental differences operating both at proximate and ultimate levels.

Critique of Mattison, Reynolds, and Wander (2019)

Mattison, Reynolds, and Wander (2019) noted that "Given that female nonheterosexual behavior is, by definition, nonreproductive, it is not clear to us that the specific application of life history theory here is appropriate." This comment shows Mattison et al.'s inadequate engagement with (1) evolutionary theory, (2) life history theory, and (3) the contents of the Target Article. As discussed in the Target Article, most lesbians have sex with men: Only 7% of women who classify themselves as homosexual had not engaged in intercourse with men (reviewed in Luoto et al., 2019). Findings by Tornello et al. (2014) further show that lesbians have more than twice as many male sex partners as heterosexual women; lesbians have been pregnant as often as heterosexual women; and they have used emergency contraception much more often than heterosexual women.

Even if nonheterosexual women never had sexual encounters with anyone, would it mean that life history theory cannot be applied to them? Of course it would not: Life history theory is a theory about resource allocation between competing domains, only one of which is reproduction (e.g., Krams et al., 2019; Luoto, 2019a; Luoto, Rantala, & Krams, in press). If organisms spend portions of their energy budgets in nonreproductive activities, it in no way indicates that life history theory suddenly cannot be applied to those individuals.

This point should be even more obvious when thinking about sexually antagonistic selection. As we hypothesized, it may be that sexually antagonistic selection partially explains same-sex sexual preferences in women (Luoto et al., 2019). Sexually antagonistic selection arises from the fact that the attributes favored in one sex are sometimes diametrically opposed to those favored in the other sex. Analyzing female nonheterosexuality,

it is therefore necessary to consider not only the nonheterosexual female phenotypes, but also the characteristics of the kin of nonheterosexual women (Luoto et al., 2019). When analyzed from a genetic, evolutionary point of view, it may not matter if there is a fitness impairment when masculinizing alleles appear in one sex if there is a theoretical or a manifest fitness benefit when they appear in the other sex. Thus, even exclusively homosexual women who might be theoretically unlikely to reproduce can constitute evolutionarily viable phenotypes if their male kin have a significant reproductive advantage over other men (Luoto et al., 2019).

Mattison et al. (2019) misrepresented the Target Article by claiming that we associated risky, unrestricted sexual behavior, increased impulsivity, and present orientation with fitness impairments in nonheterosexual women. This is a misreading of the Target Article: The term "fitness impairment" was associated with empirical findings on lower overall reproductive success of nonheterosexual women when compared with heterosexual women (Camperio Ciani, Battaglia, Cesare, Camperio-Ciani, & Capiluppi, 2018; Luoto et al., 2019; Sabia, Wooden, & Nguyen, 2017). The hallmarks of fast LH strategies, in contrast, were not framed as a fitness impairment as Mattison et al. claimed: Instead, they were hypothesized to form a predictive adaptive response that "may in fact be adaptive under certain ecological conditions" (Luoto et al., 2019). We find it counterproductive when Commentary authors misread the work they are commenting. This characteristic was particularly noticeable in Mattison et al.'s response. We do not have space here to discuss all of the ways in which they misrepresent the Target Article, and instead of seeing their Commentary as a valuable addition to the discussion, we urge the authors to read the Target Article with greater care.

Pair Bonding, Promiscuity, Paternity Uncertainty, and Infanticide

Mattison et al. (2019) and Diamond and Alley (2019) highlighted the importance of postnatal environmental conditions to the development of LH strategies, but they do not consider the role of genetic relatedness and heterosexual pair bonding. Based on Hamilton's (1964) rule, individuals in a traditional family unit consisting of a sexually reproducing male and a female have more reasons to cooperate with each other than genetically unrelated individuals do. Hamilton's rule posits that cooperation is favored by the degree of relatedness: while members of traditional heterosexual families increase the stability of their common environment as these families are built upon blood-ties, coalitions of nonheterosexual women generally cannot be based on shared genes of their offspring. The increased number of sexual partners of nonheterosexual women (Luoto et al., 2019) may, however, compensate for this. Interestingly, although promiscuous mating behavior is widespread in nature, the adaptive reasons for this reproductive strategy are far from

being understood (Eliassen & Jørgensen, 2014). It has been shown that extra-pair mating causes paternity uncertainty which reduces the risk of infanticide (Opie, Atkinson, Dunbar, & Shultz, 2013). This could be an additional mechanism that has driven the evolution of female nonheterosexuality as a behavioral strategy that reduces the risk of infanticide (Figueredo et al., 2019; Luoto et al., 2019).

In the Target Article, we proposed the hypothesis that same-sex sexual behavior may be an evolutionary "countermeasure" against the possibility of male infanticide (Luoto et al., 2019). While van Schaik and Kappeler (1997) found that male and female alliances in primates evolved to avoid the risk of infanticide by other males, Figueredo et al.'s (2019) phylogenetic analyses link infanticide risk with the evolution of female coalitions and alliances. Female same-sex genital interactions, in turn, coevolved with female coalitions and alliances (Figueredo et al., 2019). We commend Figueredo et al. for presenting these novel phylogenetic analyses that furnish empirical support for the infanticide avoidance hypothesis of female same-sex sexual behavior (Luoto et al., 2019).

A potential next step would be to test whether the children of those nonheterosexual women who are pair-bonded with another woman suffer less domestic violence and fall victim to infanticide less often than the children living in a household of (1) a single biological mother or (2) a biological mother pair-bonded with a new male. The infanticide avoidance hypothesis (Luoto et al., 2019) predicts lower rates of domestic violence and infanticide in the nonheterosexual female dyad than in the other two conditions. It should, however, be noted that the hypothesis is evolutionary. That is why the phylogenetic results provided by Figueredo et al. (2019) are particularly illuminating and findings may not be as clear in modern human populations.

Envoi

Recent notable research manifestos have advocated that psychological science be moored more solidly in broader theory from the life sciences rather than just accumulating individual facts that lack a connection to a broader scientific framework (Gurven, 2018; Muthukrishna & Henrich, 2019). This is an approach that we have advanced in relation to psychopathology (Rantala, Luoto, & Krams, 2017, Rantala, Luoto, Krams, & Karlsson, 2018), human sexual selection (Luoto, 2019a), and female sexual orientation (Luoto et al., 2019). By analyzing the female sexual orientation spectrum using evolutionary theory in general, and Tinbergen's four questions and life history theory in particular, we have provided a synthetic approach (Luoto et al., 2019) with a particular focus on neurodevelopmental mechanisms that, across species, have been shown to underlie the evolutionary-developmental origins of sex differences and variation in female sexual orientation.

The contents of the Commentaries reflect the strength of this approach: The majority of Commentary authors called for extensions to rather than re-evaluations of our model. The arguments for re-evaluations that were made (Cornil & Bakker, 2019; Del Giudice, 2019) we have here shown to be unfounded. As such, particularly the Commentaries by Figueredo et al. (2019) and Diamond and Alley (2019) offer helpful suggestions to adjacent areas of research that can be feasibly linked to the Target Article, such as early-life adversity, adaptive value (or lack thereof) of fast life history strategies in nonheterosexual women, the role of pleasure in motivating sexual behavior, and phylogenetic approaches to the origins of variability in female sexual orientation.

We expect the synthetic approach taken here to provide further impetus to research on the origins and present-day manifestations of the female sexual orientation spectrum. The lack of a synthetic approach integrating proximate mechanisms with ultimate causes has restricted the range and theoretical cohesion of prior research. The joint application of evolutionary theory, life history theory, and behavioral neuroscience to sexual orientation research holds significant promise for future work—both theoretically and empirically.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval The material contained in this article is a review of previously published or presented data. This article does not contain any studies with human participants or animals performed by the authors.

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