Parental income inequality and children’s digit ratio (2D:4D): a ‘Trivers-Willard’ effect on prenatal androgenization?

J.T. Manning1, B. Fink2,3,*, L. Mason1 and R. Trivers4

1Applied Sports, Technology, Exercise, and Medicine (A-STEM), Swansea University, Swansea, UK, 2Biosocial Science Information, Biedermannsdorf, Austria, 3Department of Evolutionary Anthropology, University of Vienna, Austria and 4Biosocial Research Foundation, Southfield, St Elizabeth, Jamaica

*Corresponding author. Email: bfink@gwdg.de

(Received 10 November 2020; revised 12 December 2020; accepted 14 December 2020)

Abstract
Income inequality is associated positively with disease prevalence and mortality. Digit ratio (2D:4D) – a negative proxy for prenatal testosterone and a positive correlate of prenatal oestrogen – is related to several diseases. This study examined the association of income inequality (operationalized as relative parental income) and children’s 2D:4D. Participants self-measured finger lengths (2D=index finger, and 4D=ring finger) in a large online survey conducted in July 2005 (the BBC Internet Study) and reported their parents’ income. Children of parents of above-average income had low 2D:4D (high prenatal testosterone, low prenatal oestrogen) while the children of parents of below-average income had high 2D:4D (low prenatal testosterone, high prenatal oestrogen). The effects were significant in the total sample, present among Whites (the largest group in the sample), in the two largest national samples (UK and USA) and were greater for males than females. The findings suggest a Trivers-Willard effect, such that high-income women may prenatally masculinize their sons at the expense of the fitness of their daughters. Women with low income may prenatally feminize their daughters at the fitness expense of their sons. The effect could, in part, explain associations between low income, high 2D:4D (low prenatal testosterone) and some major causes of mortality such as cardiovascular disease.

Keywords: 2D:4D; Income disparity; Trivers-Willard hypothesis

Introduction
The relative lengths of the 2nd and 4th digits (digit ratio; 2D:4D) is thought to be a negative correlate of prenatal testosterone (T) and a positive correlate of prenatal oestrogen (E) (Manning et al., 1998; Manning, 2002). The evidence for this, in humans (Breedlove, 2010; Manning et al., 2013; Sadr et al., 2020) and non-human animals (Zheng & Cohn, 2011; Auger et al., 2013), has been the subject of debate over the last 20 years (see Swift-Gallant et al., 2020, in support, and McCormick & Carré, 2020, in dispute). Presently, there is a substantial and growing body of evidence that links 2D:4D and prenatal sex steroids (Manning & Fink, 2018a,b). However, the source of the sex steroids, i.e. whether from the mother and/or the fetus, remains unclear. Maternal influence on fetal 2D:4D may be inferred from factors that influence maternal fitness.

The present report considers the effect of inequality of parental income on the 2D:4D of their children. It focuses on evolutionary influences on both the mother and her children by considering the Trivers-Willard hypothesis (Trivers & Willard, 1973). This hypothesis rests on the assumption that sons from high-resource mothers have higher reproductive success than daughters from
high-resource mothers, while daughters from low-resource mothers will be more reproductively successful than sons from low-resource mothers. In the present study, the proxy for the maternal resource is parental income such that parents with high income are expected to invest in sons while those with low income will invest in daughters. Considering this through the lens of the Trivers-Willard hypothesis, it is suggested that if maternal sex steroid levels cannot be adjusted to the sex of the fetus then mothers with high income will masculinize their children in utero while mothers with low income will feminize their children.

Digit ratio is sexually dimorphic (males < females; Manning et al., 1998; Manning, 2002). Adult-typical ratios of the lengths of 2D and 4D are attained very early in fetal development (i.e. before the end of the 1st trimester; Garn et al., 1975), and the sex difference in 2D:4D is established at this stage (Malas et al., 2006; Galis et al., 2010). In humans, direct evidence for a causal relationship between 2D:4D and T:E levels at the end of the first trimester is difficult to obtain. However, sex differences in digit ratios are common among the tetrapods and may have arisen as early as the transition from aquatic to terrestrial locomotion, i.e. in the common ancestor(s) to reptiles, birds and mammals (Manning, 2002; Lofeu et al., 2017). Exogenous T and/or E have been shown to masculinize or feminize 2D:4D in non-human animals, including the larval stages of Anurans (Lofeu et al., 2017), rodents (Talarovicova et al., 2009; Auger et al., 2013; Lofeu et al., 2017) and primates (Abbott et al., 2012). A detailed consideration of the effects of sex steroids (and sex steroid blockers) on the fetus of the mouse has shown that exogenous T masculinizes 2D:4D and E feminizes 2D:4D, with these effects being restricted to a narrow developmental window (Zheng & Cohn, 2011). In humans, fetal T concentrations and T:E ratios obtained by routine amniocentesis in the 2nd trimester have shown negative relationships with children’s 2D:4D (Lutchmaya et al., 2004; Ventura et al., 2013). High (feminized) 2D:4D has been reported for traits associated with low prenatal T, i.e. Klinefelter’s Syndrome (Manning et al., 2013; Chang et al., 2015), or low sensitivity to T (i.e. androgen insensitivity, Berenbaum et al., 2009). Low (masculinized) 2D:4D has been found in children with high prenatal T, i.e. congenital adrenal hyperplasia (CAH) (see for a review Sadr et al., 2020).

Perturbations in prenatal sex steroids may be of fetal origin, e.g. in Klinefelter’s Syndrome and CAH. However, maternal sex steroids may also cross the placenta and influence fetal 2D:4D (Barona et al., 2015; Manning & Fink, 2017; Ellis et al., 2018). For example, in Titi monkeys (Plecturocebus cupreus) right-hand 2D:4D is sexually dimorphic in a human-like pattern (males < females), and high maternal T and high T:E ratio are negatively related to 2D:4D (Baxter et al., 2020). In humans, there are reports that traits correlated with maternal sex steroids are also related to children’s 2D:4D. Mothers with high waist-to-hip ratios (WHR; a trait that correlates with high T:E ratio) have children with masculinized 2D:4D (Manning et al., 1999; Manning, 2002). Maternal T levels may be elevated also by exposure to sunlight with consequences for the 2D:4D of the children. Thus, low 2D:4D has been reported in children whose mothers experienced long day lengths during the 1st trimester of their pregnancies (Szwed et al., 2017). Maternal T levels assayed in the 2nd trimester were reported to be negatively related to their children’s 2D:4D (Ventura et al., 2013; Barona et al., 2015).

The present study suggests a Trivers-Willard effect of parental income on the masculinization/feminization (as measured by 2D:4D) of their children such that high-income mothers masculinize their children in utero and low-income mothers prenatally feminize their children. Therefore, parental income should be negatively related to the 2D:4D of their children. This prediction is tested in a large online survey (the BBC Internet Study).

**Methods**

The BBC Internet Study was a multi-ethnic and multi-national survey, hosted by the BBC Science and Nature website in July 2005. It comprised around 200 questions concerning cognitive and...
behavioural tests and included information on demographics, personality, sexual behaviour and physical characteristics, such as 2D:4D (Reimers, 2007). A sample of 255,116 participants completed all study tasks. In addition to ethnicity (Asian/Asian British, Black/Black British, Black other, Chinese, Middle/Near Eastern, Mixed Ethnic, White), participants provided information about their age (integer 0 to 99 years), gender (male or female) and where they lived (the United Kingdom, then 240 other countries). The predominant ethnicity was White (reported by 84.1% of participants), and the most commonly represented nationalities were the United Kingdom (46.9%), the United States (27.7%), Canada (5.2%) and Australia (3.6%), with eleven other nations represented by >1000 participants.

Participants responded to a single question item concerning their parents’ income. The item was phrased ‘What best describes your parents’ income [while growing up],’ with response options: Group I= much lower than others (bottom 25% of the population), Group II= slightly lower than others (low 50% of the population), Group III= slightly higher than others (upper 50% of the population) and Group IV= much higher than others (top 25% of the population).

Participants self-measured 2D and 4D of their right and left hands using the methodology of Manning et al. (1998). They viewed a diagram of the hand and were instructed to measure their fingers on the ventral side of the digit from the fingertip to the most proximal crease with a conventional ruler. Measurements were reported to the nearest millimetre using dropdown menus. The 2D:4D was calculated by dividing the 2D by 4D digit lengths. In the present study, the analyses were restricted to participants 18 years and older. As in earlier reports, the tails of the 2D:4D distributions were removed by considering right and left 2D:4D within the range of $\geq 0.80$ to $\leq 1.20$.

Results

Descriptive statistics

There were 189,318 participants (99,672 males). The numbers of male and female participants in each parental income group are reported in Table 1. Mean (SD) male 2D:4D varied from 0.985 (0.051) right and 0.986 (0.049) left in Group I down to 0.982 (0.049) right and 0.983 (0.048) left for Group IV. For females, the respective variation was from 0.995 (0.052) right and 0.993 (0.050) left for income Group I down to 0.993 (0.050) right and 0.991 (0.050) left for Group IV (see Table 1 and Figure 1).

Analyses for the total sample

In males, parental income showed an effect on both right and left 2D:4D (right hand $F_{(3,109668)}=12.55$, $p<0.0001$; left hand $F_{(3,109668)}=14.86$, $p<0.0001$). Post-hoc tests (Fisher’s PLSD) indicated differences for both right and left hand 2D:4D for all pairwise comparisons between parental income groups below and above population average (2D:4D below-average income > 2D:4D above-average income) with mean differences I–III=0.002, I–IV=0.003, II–III=0.002 and II–IV= 0.002 (all $p<0.0001$). There were no differences in right or left 2D:4D for the pairwise comparisons between parental income groups below population average (I–II) or above population average (III–IV) (all $p>0.05$; Figure 1).

In females, parental income showed an effect on both right and left hand 2D:4D (right hand $F_{(3,109642)}=4.74$, $p=0.003$; left hand $F_{(3,109642)}=4.02$, $p=0.007$). These effects were weaker than for males. There were differences (Fisher’s PLSD) for right hand 2D:4D for only two pairwise comparisons between parental income groups with mean differences I–III=0.002 ($p=0.006$) and II–III=0.001 ($p=0.002$). For left hand 2D:4D there were three significant pairwise comparisons with mean differences I–III=0.002 ($p=0.003$), I–IV=0.002 ($p=0.03$) and II–III=0.001 ($p=0.02$). All significant pairwise comparisons showed offspring 2D:4D from below population average
Table 1. Means and SDs for 2D:4D by sex and hand by parental income groups for all participants, White participants, and UK and US participants

|                  | 2D:4D                        | Group I | Group II | Group III | Group IV |
|------------------|------------------------------|---------|----------|-----------|----------|
| All males        |                              | n=11,185| n=38,408 | n=51,026  | n=9053   |
| Right hand       | 0.985 (0.051)                | 0.985 (0.050) | 0.983 (0.049) | 0.982 (0.049) |
| Left hand        | 0.986 (0.049)                | 0.985 (0.048) | 0.984 (0.047) | 0.983 (0.048) |
| All females      | n=8451                       | n=33,102| n=43,200 | n=4893    |
| Right hand       | 0.995 (0.052)                | 0.994 (0.052) | 0.993 (0.050) | 0.993 (0.052) |
| Left hand        | 0.993 (0.050)                | 0.992 (0.050) | 0.991 (0.048) | 0.991 (0.050) |
| White males      | n=9290                       | n=33,128| n=43,063 | n=7445    |
| Right hand       | 0.986 (0.051)                | 0.985 (0.049) | 0.984 (0.048) | 0.983 (0.049) |
| Left hand        | 0.987 (0.048)                | 0.986 (0.047) | 0.984 (0.047) | 0.984 (0.047) |
| White females    | n=7183                       | n=28,769| n=36,604 | n=3985    |
| Right hand       | 0.996 (0.052)                | 0.995 (0.052) | 0.994 (0.050) | 0.993 (0.052) |
| Left hand        | 0.994 (0.050)                | 0.993 (0.050) | 0.992 (0.048) | 0.992 (0.050) |
| UK males         | n=5547                       | n=17,398| n=20,679 | n=3415    |
| Right hand       | 0.986 (0.050)                | 0.986 (0.048) | 0.984 (0.047) | 0.983 (0.048) |
| Left hand        | 0.987 (0.047)                | 0.987 (0.046) | 0.985 (0.045) | 0.984 (0.046) |
| UK females       | n=3953                       | n=14,227| n=16,793 | n=1636    |
| Right hand       | 0.994 (0.051)                | 0.994 (0.051) | 0.993 (0.048) | 0.993 (0.049) |
| Left hand        | 0.994 (0.048)                | 0.993 (0.048) | 0.992 (0.046) | 0.992 (0.048) |
| USA males        | n=1805                       | n=7134  | n=9440   | n=1854    |
| Right hand       | 0.987 (0.053)                | 0.986 (0.053) | 0.985 (0.052) | 0.984 (0.051) |
| Left hand        | 0.987 (0.051)                | 0.986 (0.051) | 0.985 (0.050) | 0.984 (0.050) |
| USA females      | n=1570                       | n=6521  | n=8077   | n=1129    |
| Right hand       | 0.999 (0.055)                | 0.998 (0.055) | 0.997 (0.054) | 0.995 (0.056) |
| Left hand        | 0.995 (0.053)                | 0.994 (0.053) | 0.994 (0.051) | 0.993 (0.051) |

Income groups: I=bottom 25%, II=low 50%, III=upper 50% and IV=top 25%.

Figure 1. The relationship between parental income and mean right and left hand 2D:4D in males and females for all participants.
parental income groups were greater than offspring 2D:4D from above population average parental income groups (Table 1, Figure 1).

Thus far the analyses considered a multi-ethnic sample. However, 2D:4D varies across ethnic groups. Splitting the sample by ethnicity resulted in smaller groups and a consequent reduction in power. Therefore, the following analyses considered patterns of offspring 2D:4D across parental income groups in the numerically largest ethnicity in the BBC Internet Study, i.e. Whites.

White participants

There were 169,467 White participants in the analyses. The numbers of male and female participants in each parental income group are reported in Table 1. The pattern for White participants showed high 2D:4D in income Group I and low 2D:4D in income Group IV, e.g. for the right hand, Group I males 0.986 (0.051), females 0.996 (0.052); Group IV males 0.983 (0.048), females 0.993 (0.052) (see Figure 2).

Analyses for White participants

Parental income had an effect on both the male right and left hands (right hand $F_{(3,109668)}=12.55$, $p<0.0001$; left hand $F_{(3,109668)}=14.86$, $p<0.0001$). Post-hoc tests (Fisher’s PLSD) showed similar patterns to those found in the total sample. There were differences for both right and left hand 2D:4D in four pairwise comparisons between parental income groups below and above population average (2D:4D below-average income > 2D:4D above-average income), with mean differences I–III=0.002 ($p=0.0002$) for right hand and 0.002 ($p<0.0001$) for left hand, I–IV=0.003 ($p<0.0001$) for both hands, II–III=0.002 ($p<0.0001$) for both hands and below II–IV=0.003 ($p<0.0001$) for both hands. There were no differences in right or left 2D:4D for the pairwise comparisons between parental income groups below population average, I–II or above population average III–IV (all $p>0.05$).

In White female participants, parental income showed an effect for both right and left hand 2D:4D (right hand $F_{(3,76537)}=5.14$, $p=0.002$; left hand $F_{(3,76537)}=4.01$, $p=0.007$). These effects were weaker than for males. There were differences (Fisher’s PLSD) for 2D:4D for only three pairwise comparisons between parental income groups with mean differences I–III=0.002 ($p=0.004$ and $p=0.008$ for right and left hands, respectively), II–IV=0.002 ($p=0.03$ and $p=0.02$ for right and left hands, respectively) and II–III=0.001 ($p=0.002$ and $p=0.01$ right and left, respectively). Pairwise comparisons I–II, II–IV and III–IV were not significant (all $p<0.05$). All significant pairwise comparisons showed offspring 2D:4D from below population average parental income groups were greater than offspring 2D:4D from above-average parental income groups.
**White participants in the UK and USA**

To account for the effect of nation, two-way ANOVAs were performed for males and females with the independent variables parental income and the nations in the study with the largest White representation (UK and the USA) and the dependent variable 2D:4D.

For males, there were main effects for parental income for both the right hand ($F_{(3,67264)}=9.62, p<0.0001$) and left hand ($F_{(3,67264)}=10.11, p<0.0001$) 2D:4D and no effects of nation (right hand $F_{(1,67264)}=1.89, p=0.17$; left hand $F_{(1,67264)}=0.008, p=0.93$). There were no interaction effects (all $p>0.05$). Overall, male mean 2D:4D reduced with increasing income but there were no differences between the mean 2D:4Ds of UK and USA participants (Figure 2).

For females, there was a main effect of parental income for the right hand ($F_{(3,53898)}=4.18, p=0.006$) but not the left hand ($F_{(3,53898)}=1.52, p=0.21$). Nation showed effects for both right ($F_{(1,53898)}=28.15, p<0.0001$) and left hands ($F_{(1,53898)}=5.85, p=0.02$). There were no interaction effects (all $p>0.05$). Overall, female mean 2D:4D reduced with increasing income but the effect was smaller than that seen for males. Mean 2D:4D was lower in UK participants than in participants from the USA (see Figure 2).

**Discussion**

The present study found that parental income affects children’s 2D:4D such that below-average income is related to high 2D:4D (feminization of the fetus) and above-average parental income is associated with low 2D:4D (masculinization of the fetus). These findings applied to the total study sample, the most numerous ethnic group in the study (i.e. Whites) and the most numerous national samples (UK and USA). Regarding the total sample, for male children, the effect was present for right and left 2D:4D and for all pairwise comparisons between parental income groups above and below the population average. For female children, the effect was also present on right and left hand 2D:4D and was found in pairwise comparisons between parental income groups above and below the population average. However, the female effects were weaker than the male effects, with only two significant pairwise comparisons for right hand 2D:4D and three for left hand 2D:4D.

The present findings are consistent with the Trivers-Willard hypothesis concerning maternal resources and its links to the influence of the mother’s sex steroids on fetal 2D:4D. Thus, mothers with high income will secrete elevated levels of T relative to E during the 1st trimester of their pregnancy, i.e. they will masculinize their male and female children. In contrast, women with low income will secrete low levels of T:E in the early stages of pregnancy. This hormonal milieu will feminize their male and female children. That is, high-income mothers will increase the fitness of their sons at the expense of their daughters while low-income mothers will increase the fitness of their daughters at the expense of their sons. Thus, there will be sexually antagonistic effects on the children of both high- and low-income mothers (Manning et al., 2000). There is evidence for an effect of female condition on the production of sex steroids and sexually antagonistic effects of maternal sex steroids on the developing fetus.

There is only weak support for a link between the 2D:4D of women selected at random from the population and their production of T and E (Muller et al., 2011). However, in contrast to women with low income, high-income women may benefit from high levels of nutrition. There may be associations between high income, good nutrition and the production of androgens in women. Elite women athletes with high standards of nutrition and in good condition (with high lean mass and low body fat levels) show negative relationships between their 2D:4D and the breakdown products of T (Eklund et al., 2020) or salivary levels of T (Crewther & Cook, 2019). It appears that women in good condition can, and probably do, secrete elevated levels of T, particularly if they have low 2D:4D.
The Trivers-Willard hypothesis was originally formulated in the context of maternal manipulation of the sex ratio of progeny. There is indeed evidence that masculinized women (with high WHR and/or low 2D:4D) have more sons than feminized women (low WHR and/or high 2D:4D). This may be the result of the deleterious effects of high prenatal T on female fetuses and/or the advantageous effect of high prenatal T on male fetuses (Manning et al., 1996; Singh & Zambarano, 1997; Manning & Bundred, 2001; Kim et al., 2015).

Maternal manipulation of the prenatal sex steroid environment of their children is likely to have later-life health consequences. For example, male children of low-income mothers will be feminized and more prone to several diseases. Prominent among these is likely to be the poverty influenced male-biased burden of cardiovascular diseases. A low income level has been consistently associated with cardiovascular disease, especially in high-income countries. In addition, disparities based on sex (males > females) have been shown in several studies. High 2D:4D in men has been linked to poor outcomes for cardiovascular disease such as early myocardial infarction, high blood pressure, atherosclerotic plaque development, high fibrinogen levels and markers of obesity (Manning & Bundred, 2001; Fink et al., 2006; Lu et al., 2008, 2015; Ozdogmus et al., 2010; Kyriakidis et al., 2010; Wu et al., 2013; Manning et al., 2019; Bagepally et al., 2020). Associated with all of these factors is a high level of parental poverty (Kucharska-Newton et al., 2011; Mosquera et al., 2016).

One possible limitation of the present study is that estimates of parental income in the early years of the family are dependent on their children’s recall. Inaccuracies that may result from faulty recall are likely to reduce the Trivers-Willard influence on 2D:4D. Thus, the reported effects may be conservative estimates of maternal influence on offspring 2D:4D. In order to minimize the recall effects of children’s estimates of parental income it is suggested that future studies should also include parental reports of family income.

In conclusion, inequality in parental income may be associated with the 2D:4D of their offspring. Children of parents of above-average income had low 2D:4D (high prenatal T:E) while the children of parents of below-average income had high 2D:4D (low prenatal T:E). The differences in offspring 2D:4D across income groups may arise because of maternal manipulation of sex steroids. Interpreting the findings of the present study through the lens of the Trivers-Willard hypothesis suggests that high-income mothers may masculinize their sons via increased levels of prenatal T. Male reproductive success shows higher variance than female reproductive success. Therefore, the fitness rewards from the sons of high-income mothers are likely to outweigh the deleterious effects of T on the daughters of high-income mothers. In contrast, mothers with low income are expected to feminize their children via increases in prenatal E. The fitness gain from feminized daughters is likely to outweigh the fitness loss of feminized sons. Moreover, the health costs of maternal manipulation of prenatal sex steroids may include hypertension, cardiovascular disease, high levels of fibrinogen and early myocardial infarction and could be focused on the feminized (low T, high E) sons of low-income mothers.

Funding. This research received no specific grant from any funding agency, commercial entity, or not-for-profit organization.

Conflict of Interest. The authors have no conflicts of interest to declare.

Ethical Approval. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

References

Abbott AD, Colman RJ, Tiefenthaler R, Dumesic DA and Abbott DH (2012) Early-to-mid gestation fetal testosterone increases right hand 2D:4D finger length ratio in polycystic ovary syndrome-like monkeys. PLoS One 7(8), e42372.

Auger J, Le Denmat D, Berges R, Doridot L, Salmon B, Canivenc-Lavier MC and Eustache F (2013) Environmental levels of oestrogenic and antiandrogenic compounds feminize digit ratios in male rats and their unexposed male progeny. Proceedings of the Royal Society of London B: Biological Sciences 280(1768), 20131532.
Bagepally BS, Majumder J and Kotadiya S (2020) Association between the 2d:4d and cardiovascular risk factors: body mass index, blood pressure and body fat. Early Human Development 151, 105193.

Barona M, Kothari R, Skuse D and Micali N (2015) Social communication and emotion difficulties and second to fourth digit ratio in a large community-based sample. Molecular Autism 6(1), 68.

Baxter A, Wood EK, Witczak LR, Bales KL and Higley JD (2020) Sexual dimorphism in Titi monkeys’ digit (2D:4D) ratio is associated with maternal urinary sex hormones during pregnancy. Developmental Psychobiology 62(7), 979–991.

Berenaum SA, Bryk KK, Nowak N, Quigley CA and Moffat S (2009) Fingers as a marker of prenatal androgen exposure. Endocrinology 150(11), 5119–5124.

Breedlove SM (2010) Minireview: organizational hypothesis: instances of the fingerpost. Endocrinology 151(9), 4116–4122.

Chang S, Skakkebaek A, Trolle C, Bojesen A, Hertz JM, Cohen A et al. (2015) Anthropometry in Klinefelter syndrome – multifactorial influences due to CAG length, testosterone treatment and possibly intrauterine hypogonadism. Journal of Clinical Endocrinology & Metabolism 100(3), E508–E517.

Crewther BT and Cook CJ (2019) The digit ratio (2D:4D) relationship with testosterone is moderated by physical training: evidence of prenatal organizational influences on activational patterns of adult testosterone in physically-active women. Early Human Development 131, 51–55.

Eklund E, Ekström L, Thörngren J-O, Ericsson M, Berglund B and Hirschberg AL (2020) Digit Ratio (2D:4D) and physical performance in female Olympic athletes. Frontiers in Endocrinology 11, 292.

Ellis L, Eisenmann R and Hoskin A (2018) Maternal activity during pregnancy and sexually dimorphic traits in offspring. Journal of Biosocial Science 50(1), 114–123.

Fink B, Manning JT and Neave N (2006) The 2nd–4th digit ratio (2D:4D) and neck circumference: implications for risk factors in coronary heart disease. International Journal of Obesity 30(4), 711–714.

Galis F, Ten Broek CM, Van Dongen S and Wijnands IC (2010) Sexual dimorphism in the prenatal digit ratio (2D:4D). Archives of Sexual Behavior 39(1), 57–62.

Garn SM, Burdi AR, Babler WJ and Stinson S (1975) Early prenatal attainment of adult metacarpal-phalangeal rankings and proportions. American Journal of Physical Anthropology 43, 327–332.

Kim TB, Oh JK, Kim KT, Yoon SJ and Kim SW (2015) Does the mother or father determine the offspring sex ratio? Investigating the relationship between maternal digit ratio and offspring sex ratio. PlaOs One 10(11), e0143054.

Kucharska-Newton AM, Harald K, Rosamond WD, Rose KM, Rea TD and Salomaa V (2011) Socioeconomic indicators and the risk of acute coronary heart disease events: comparison of population-based data from the United States and Finland. Annals of Epidemiology 21, 572–579.

Kyriakidis I, Papaoannidou P, Pantelidou V, Kalles V and Gemitzis K (2010) Digit ratios and relation to myocardial infarction in Greek men and women. Gender Medicine 7, 628–636.

Lofeu L, Brandt R and Kohlsdorf T (2017) Phenotypic integration mediated by hormones: associations among digit ratios, body size and testosterone during tadpole development. BMC Evolutionary Biology 17, 175.

Lu H, Huo Z, Zhang K, Wei N, Shi Z, Peng L et al. (2008) Relations between digit ratio and coronary heart disease. Acta Anatomica Sinica 39, 765–768.

Lu H, Ma Z, Zhao J and Huo Z (2015) To second to fourth digit ratio (2D:4D) and coronary heart disease. Early Human Development 91, 417–420.

Lutchmaya S, Baron-Cohen S, Raggatt P, Knickmeyer R and Manning JT (2004) 2nd to 4th digit ratios, fetal testosterone and estradiol. Early Human Development 77(1-2), 23–28.

McCormick CM and Carré JM (2020) Facing off with the phalangeal phenomenon and editorial policies: a commentary on Swift-Gallant, Johnson, Di Rita and Breedlove. Hormones and Behavior 120, 104710.

Malas MA, Dogan S, Evcil EH and Desdicoglu K (2006) Fetal development of the hand, digits and digit ratio (2D:4D). Early Human Development 82(7), 469–475.

Manning JT (2002) Digit Ratio: a Pointer to Fertility, Behavior and Health. Rutgers University Press, New Brunswick, NJ.

Manning, JT, Anderton R and Washington SMJ (1996) Women’s waists and the sex ratio of their progeny: evolutionary aspects of the ideal female body shape. Journal of Human Evolution 31(1), 41–47.

Manning JT, Barley L, Walton J, Lewis-Jones DI, Trivers RL, Singh D et al. (2000) The 2nd–4th digit ratio, sexual dimorphism, population differences, and reproductive success. evidence for sexually antagonistic genes? Evolution and Human Behavior 21(3), 163–183.

Manning JT and Bundred PE (2001) The ratio of 2nd to 4th digit length and age at first myocardial infarction in men: a link with testosterone? British Journal of Cardiology 8, 720–723.

Manning JT, Bundred PE, Kasielska-Trojan A, Smith-Straney T and Mason L (2019) Digit ratio (2D:4D), myocardial infarction and fibrinogen in men. Early Human Development 133, 18–22.

Manning JT and Fink B (2017) Are there any “direct” human studies of digit ratio (2D:4D) and measures of prenatal sex hormones? Early Human Development 113, 73–74.

Manning JT and Fink B (2018a) Digit ratio. In Shackelford TK and Weekes-Shackelford VA (eds) Encyclopedia of Evolutionary Psychological Science. Springer, Cham. doi: 10.1007/978-3-319-16999-6_3829-1.
Manning JT and Fink B (2018b) Digit ratio and personality and individual differences. In Zeigler-Hill V and Shackelford TK (eds) The SAGE Handbook of Personality and Individual Differences. SAGE Publications, Thousand Oaks, pp. 40–50.

Manning JT, Kilduff LP and Trivers R (2013) Digit ratio (2D:4D) in Klinefelter’s syndrome. Andrology 1(1), 94–99.

Manning JT, Scutt D, Wilson J and Lewis-Jones DI (1998) The ratio of 2nd to 4th digit length: a predictor of sperm numbers and concentrations of testosterone, luteinizing hormone and oestrogen. Human Reproduction 13(11), 3000–3004.

Manning JT, Trivers RL, Singh D and Thornhill R (1999) The mystery of female beauty. Nature 399(6733), 214–215.

Mosquera PA, San Sebastian M, Waenerlund AK, Ivarsson A, Weinehall L and Gustafsson PE (2016) Income-related inequalities in cardiovascular disease from mid-life to old age in a Northern Swedish cohort: a decomposition analysis. Social Science & Medicine 149, 135–144.

Muller DC, Giles GG, Bassett J, Morris HA, Manning JT, Hopper JL et al. (2011) Second to fourth digit ratio (2D:4D) and concentrations of circulating sex hormones in adulthood. Reproductive Biology and Endocrinology 9(1), 57.

Ozdogmus O, Cakmak YO, Coskun M, Verimli U, Cavdar S and Uzun I (2010) The high 2D:4D finger length ratio effects on atherosclerotic plaque development. Atherosclerosis 209, 195–196.

Reimers S (2007) The BBC Internet Study: general methodology. Archives of Sexual Behavior 36(2), 147–161.

Sadr M, Khorashad BS, Talaei A, Fazeli N and Honekopp J (2020) 2D:4D suggests a role of prenatal testosterone in gender dysphoria. Archives of Sexual Behavior 49, 421–432.

Singh D and Zambarano RJ (1997) Offspring sex ratio in women with android body fat distribution. Human Biology 69(4), 545–556.

Swift-Gallant A, Johnson BA, Di Rita V and Breedlove SM (2020) Through a glass, darkly: human digit ratios reflect prenatal androgens. Hormones and Behavior 120, 104686.

Szwed A, Kosinska M and Manning JT (2017) Digit ratio (2D:4D) and month of birth: a link to the solstitial-melatonin-testosterone effect. Early Human Development 104, 23–26.

Talarovicova A, Krskova L and Blazeckova J (2009) Testosterone enhancement during pregnancy influences the 2D:4D ratio and open field motor activity of rat siblings in adulthood. Hormones and Behavior 55(1), 235–239.

Trivers RL and Willard DE (1973) Natural selection of parental ability to vary the sex ratio of offspring. Science 179(4068), 90–92.

Ventura T, Gomes MC, Pita A, Neto MT and Taylor A (2013) Digit ratio (2D:4D) in newborns: influences of prenatal testosterone and maternal environment. Early Human Development 89(2), 107–112.

Wu XL, Yang DY, Chai WH, Jin ML, Zhou XC, Peng L and Zhao YS (2013) The ratio of second to fourth digit length (2D:4D) and coronary artery disease in a Han Chinese population. International Journal of Medical Science 10, 1584–1588.

Zheng Z and Cohn MJ (2011) Developmental basis of sexually dimorphic digit ratios. Proceedings of the National Academy of Sciences of the USA 108(39), 16289–16294.