The ulna-to-fibula ratio as a marker of organizational hormone effects on the motivational brain: A high-powered preregistered replication

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!! Draft version 3.0, 15/10/21. This paper has not been peer reviewed. Please do not copy or cite without author's permission !!

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Data Availability Statement. All data files, the study logbook, analysis scripts for reported results, as well as the output files associated with this research are available at [LINK TO BE INCLUDED IN PUBLISHED PAPER].

Sub-parts of the PSE-data used in this research were also used in a sub-study of (Janson et al., in press), however with a completely different research question.

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Abstract

Objective: We aimed at replicating findings by Köllner and Bleck (2020) regarding their proposed new marker of pubertal organizational hormone effects (OHEs), the ulna-to-fibula ratio (UFR). We tested UFR's sex-dimorphism, independence of body height, interrelationships with other markers, and relationships to the implicit power motive (*nPower*) and activity inhibition (AI).

Method: Our pre-registered, cross-sectional, high-powered study ($N = 250$; 148 women; after exclusions) included the Picture-Story Exercise (*nPower*, AI) and anthropometry of ulna and fibula length, facial width and height, shoulder/waist/hip circumference, and 2D:4D digit ratio.

Results: UFR was sex-dimorphic ($d = 0.37$; outliers excluded), independent of body height, and significantly associated with other possible markers of pubertal OHEs, including facial width-to-height ratio, waist-to-hip ratio, and shoulder-to-hip ratio. As predicted, a “sex-typical” (high for men, low for women) UFR was associated with the inhibited power motive (outliers excluded). Neither *nPower*'s sex-dimorphic relationship with UFR, nor the sex-dimorphic relationship of the inhibited power motive with UFR asymmetry (deemed unreliable and already omitted from the preregistration) reported by Köllner and Bleck (2020) were replicated.

Conclusions: Our findings bolster UFR's status as a marker of pubertal OHEs: It is sexually dimorphic, unrelated to body height, related to other markers, and shows sex-dimorphic associations with the inhibited power motive. In conjunction with findings by Schultheiss et al. (2019) for prenatal OHEs, there is also accumulating evidence for hormonal contributions to implicit motive development.

Keywords: implicit motives, ulna-to-fibula ratio, organizational hormone effects, power motive, puberty.

The ulna-to-fibula ratio as a marker of organizational hormone effects on the motivational brain: A high-powered pre-registered replication

Recently, Köllner and Bleck (2020) proposed the ulna-to-fibula ratio (UFR), ulna length divided by fibula length, as a possible indicator of pubertal hormone exposure. We will check whether their key findings for UFR (sexual dimorphism, independence of body height, convergent validity with other indicators of hormone exposure during development) as well as its relationships to the *implicit need for power* (*nPower*), a capacity for deriving pleasure from emotional, mental, or physical influence on others (Schultheiss & Köllner, 2021), can be replicated in a preregistered, large-sample study.

Organizational hormone effects and markers of pubertal hormone exposure

Starting with Phoenix et al. (1959), theory and research in behavioral endocrinology is based on the realization that hormones can have lasting, permanent *organizational hormone effects* (OHEs) in addition to transient, activational effects on brain and behavior during mammalian development (for review, Arnold, 2009). Recent research reveals that not only prenatal development, but also puberty is an important phase of lasting brain and behavioral organization by steroid hormones in humans and non-human animals (Nelson & Kriegsfeld, 2017; Schulz et al., 2009; Schulz & Sisk, 2016; Sisk & Zehr, 2005). In particular, there seem to be pubertal contributions to dominance motivation and aggression when using facial width-to-height ratio (fWHR; bizygomatic width divided by upper face height; identified by Weston et al., 2007, while examining skulls of humans and primates) as an index of pubertal sex steroid exposure in humans: Meta-analyses show associations of fWHR with outcomes like male aggression (Haselhuhn et al., 2015) and threat behavior, as well as dominance behavior across both sexes (Geniole et al., 2015).

Markers of OHEs

In almost any study with human participants (e.g., due to the ethical impossibility of direct manipulation, Köllner et al., 2019; or due to direct measurement being unworkable,

McIntyre, 2006), OHEs are retrospectively assessed with morphological *markers*. The marker approach assumes a simultaneous effect of OHEs on (a) the brain, and thus on later adult behavior, and (b) the growth of hormone-sensitive body parts during a given developmental window (e.g., prenatally or during puberty). If such body markers remain stable after this developmental window, correlations between adult behavior and markers can be interpreted to reflect OHEs on the brain during development (cf. Hönekopp et al., 2007).

For pubertal OHEs, most researchers use fWHR as an indicator of pubertal testosterone (T; Carré & McCormick, 2008) and possibly also estradiol (E2; Janson et al., 2018). During puberty, sex-dimorphic growth patterns produce a larger fWHR in men compared to women. Its use as an indicator of pubertal T is also supported by causal effects of sex hormone administration on craniofacial growth in humans (Verdonck et al., 1999) and animals (Barrett & Harris, 1993).

However, markers are subject to an ongoing debate among behavioral endocrinologists. As a case in point, digit ratio of the length of the second and fourth digit (2D:4D) is assumed to reflect prenatal OHEs, and its proponents, like Breedlove (Swift-Gallant et al., 2020) and Manning et al. (2014), cite solid experimental data from animal (Zheng & Cohn, 2011) and large genome-wide association studies (Warrington et al., 2018) as evidence for its validity. But other researchers raise statistical and replication issues (Leslie, 2019) and criticize the wide-spread practice of atheoretically correlating digit ratio with any given outcome (McCormick & Carré, 2020). Compared to digit ratio, even less corroborating research exists for markers of pubertal OHEs.

To make things worse, fWHR, the most frequently used pubertal marker, shows at best only weak sexual dimorphism in meta-analyses (Geniole et al., 2015; Kramer, 2017; but see Köllner et al., 2018). But sexual dimorphism is a crucial criterion for a marker: If a body dimension does not reflect the large sex differences in steroid hormone exposure during development (Ober et al., 2008), it is implausible to assume that it reflects more gradual

differences between individuals within each sex (cf. Hönekopp et al., 2007; McIntyre, 2006, using the early sex-hormone-driven dimorphism in 2D:4D as a basis for postulating the marker function; but see Lefevre et al., 2013, for a divergent opinion regarding fWHR involving different facial growth-mechanisms within-sex). This inspired Köllner and Bleck (2020) to propose long bone growth as a sexually dimorphic marker involving the whole body as an alternative to subtle variations in fWHR (further distorted by fat deposits in the cheeks; cf. Lefevre et al., 2013). In addition to countering the replication problems of marker research with pre-registered hypotheses, the present study will further test UFR's marker potential in the domain of dominance motivation as a theoretically well-founded outcome.

***n*Power and its links to OHEs**

From a biopsychological perspective, motivational processes are closely tied to hormones (for review and the subserving brain structures, see Schultheiss, 2013), rendering them an ideal choice for testing marker-outcome-associations. However, self-reported (i.e., explicit) dominance motivation yielded weak results for pubertal OHEs. Systematic associations with fWHR are absent for self-reported aggressive behavior (Özener, 2012) or personality dispositions (Kosinski, 2017). Similar weak associations were observed for UFR and self-reported motivation (Köllner & Bleck, 2020).

More clear-cut findings are obtained for indirect (or implicit) measures of dominance motivation like the *n*Power content-coding measure based on imaginative stories testees narrate about picture stimuli. There are documented links of markers of prenatal (2D:4D; Schultheiss et al., 2019, $N = 618$; Schultheiss & Zimni, 2015, $N = 50$) as well as of pubertal OHEs (Janson et al., 2017; 2018, fWHR, $N = 213$; Köllner & Bleck, 2020, UFR, $N = 126$) to *n*Power.

Implicit motives like *n*Power are affect-based, respond to reward and punishment, operate outside of conscious awareness, and are aimed at attaining specific incentives and avoiding specific disincentives (Schultheiss & Köllner, 2021). Measures of implicit and

explicit motivation do not overlap substantially (Köllner & Schultheiss, 2014; Spangler, 1992). Implicit motive measures based on content coding of imaginative stories, namely the Picture-Story Exercise (PSE; McClelland et al., 1989) used for measuring *nPower*, were derived from experimental arousal of motivational states (Schultheiss & Köllner, 2021) and hence feature causal validity (Borsboom et al., 2004), a property that most questionnaires, including explicit motive measures, do not share.

nPower predicts various individual (e.g. attentional orienting, Janson et al., in press; implicit learning; Schultheiss, Pang, et al., 2005) and biological outcomes, like sex hormone changes (E2 in women, T in men) in response to winning or losing a dominance contest (Schultheiss, Wirth, et al., 2005; Stanton & Schultheiss, 2007). This property, combined with the above-mentioned associations with markers of OHEs, inspired a biopsychosocial model of implicit motive development and hormone-motive-relationships in adulthood (Köllner & Janson, 2017; Köllner et al., 2019), hypothesizing on the brain basis of marker-motive-relationships (e.g., the dominance-relevant, sex-hormone-sensitive anterior hypothalamus).

The previously mentioned association between *nPower* and markers (2D:4D, fWHR, UFR) pertains to the *inhibited power motive* (IPM), a combination of high *nPower* and high activity inhibition (AI). AI is measured by counting negations in stories (“not”) and seems to mark functional rightward brain lateralization (Schultheiss et al., 2009). AI frequently moderates results involving *nPower*, and the IPM represents a more context-sensitive, adaptive style of dominance compared to *nPower* coupled to low AI (Schultheiss & Köllner, 2021). This moderation pattern is also consistently found in studies on OHEs and *nPower*, which is all the more interesting, as OHEs are relevant for prenatal T-driven brain lateralization processes favoring the right hemisphere (Geschwind & Galaburda, 1987). For example, there seems to be an overall association of „male-typical“ low 2D:4D with the IPM (Schultheiss & Zimni, 2015), and a parallel finding was obtained for „male-typical“ high fWHR (Janson et al., 2018). The latter result was moderated by sex in follow-up analyses,

with the effect being predominantly driven by women. Sex-dimorphic findings were also obtained in a high-powered sample aggregated from four individual studies (Schultheiss et al., 2019), where the IPM was related to 2D:4D-asymmetry in women.

Ulna-to-fibula Ratio

Due to the above-mentioned motive-related findings and problems with fWHR, our research focuses on the IPM and on UFR as a promising new marker candidate for pubertal OHEs. As stated above, a marker should be sexually dimorphic. Independence from body height is desirable to rule out the possibility of the marker being an artifact of general body height (cf. Leslie, 2019). Further, intercorrelations with other pubertally determined body parameters (especially fWHR) are conceivable, but not strictly necessary for a new marker due to slightly different time windows and tissue sensitivities involved in bone growth patterns.

For UFR, sexual dimorphism (male > female) and independence from overall body height was observed, as well as indications of convergence of UFR-asymmetry (UFR_{r-l} , formed in an analogy to D_{r-l} for 2D:4D asymmetry as an additional marker of T vs. E2, see Manning et al., 2014) with fWHR and 2D:4D measures (Köllner & Bleck, 2020). However, apart from rare exceptions (Köllner & Bleck, 2020; Köllner et al., 2019), little research on this length ratio exists. In contrast to subtle variations in digit ratio and fWHR, potentially more susceptible to measurement error and variations in the measurement method, UFR represents a more robust measure of long-bone growth (Köllner & Bleck, 2020). The long bones ulna and fibula can reliably be measured on the body surface without much interference of fat deposits. Both are the target of biphasic effects of E2 (or T converted to E2 via aromatization with similar effects on long bone growth; Vanderschueren et al., 2004), which in low doses mediate the pubertal growth spurt and in high doses stimulate closure of the epiphyseal growth plates, thus ending long bone growth (Cutler, 1997; Juul, 2001). Also, the ratio between ulna and fibula seems to carry additional information about “sex-typical” steroid

hormone exposure: Hypogonadic men with Klinefelter syndrome show a “feminized” UFR growth pattern, with legs elongated relative to the arms (in addition to a “feminized” 2D:4D; Chang et al., 2015; Manning et al., 2013). The sex-dimorphic ratio best captured the variance in the data by Köllner and Bleck (2020), with the upper limb/ulna identified as the marker “signal” (i.e. dimorphism; cf. Purkait, 2001) and the lower limb/fibula serving as the reference limb for overall long bone length (as an analogy to 2D:4D, where the second digit is the reference limb for the signal-carrying fourth digit; Zheng & Cohn, 2011). Further reasons for choosing ulna and fibula were equivalent positions on the respective limbs and ontogenetic homology of forearm and shank bones (see Köllner & Bleck, 2020).

Consistent with the notion that UFR captures variations in pubertal gonadal steroids that also impinge on the brain and its motivational systems, Köllner and Bleck (2020) found a sex-dimorphic effect of *n*Power on UFR scores (cf. sex-dimorphic fWHR-results for the IPM by Janson et al., 2018) as well as indications of bivariate within-sex relationships of UFR to *n*Power (men: $r = -.26, p < .10$, women: $r = .19, p = .10$). In addition, as a striking similarity to earlier 2D:4D asymmetry results (Schultheiss et al., 2019), the IPM was related to “sex-typical” (i.e., high scores in men, low scores in women) UFR-asymmetry. However, Köllner and Bleck (2020) only tested 126 participants and thus called for replication of their results.

Overview of the present research

Based on Köllner and Bleck’s (2020) findings regarding UFR’s marker potential for pubertal OHEs on the motivational brain, we conducted a preregistered, high-powered study in healthy adults using essentially the same testing protocol regarding long bone measurement (UFR) and implicit motive assessment (PSE). We additionally assessed other body parameters that may be suitable markers of OHEs. While 2D:4D and fWHR were already investigated in the context of OHEs and implicit motive development, we also investigated circumference-based measures (shoulder, waist, hip; see Hughes & Gallup, 2003), namely waist-to-hip ratio (WHR), shoulder-to-hip ratio (SHR), and shoulder-to-waist ratio (SWR), as they develop

during puberty in a sex-dimorphic way (e.g., WHR as a signal of pubertal testosterone-estrogen ratio, Fink et al., 2003; in general, differential growth of hips and shoulders influenced by sex-hormones during puberty, Rogol et al., 2002). SWR was only tested exploratively, as in contrast to WHR and SHR (e.g., Fink et al., 2003; Gallup et al., 2007), we are not aware of any research investigating links between SWR and OHEs.

Our preregistered hypotheses (<https://aspredicted.org/blind.php?x=er2pd2>) were derived from Köllner and Bleck (2020). We predicted that UFR (but not UFR_{r-l}) would be sex-dimorphic, with larger values for men compared to women (*Hypothesis 1a*). As a (non-preregistered) validation check for UFR, we predicted no significant association of UFR with body height (*Hypothesis 1b*). Regarding implicit motivation, we expected sex-dimorphic associations of *nPower* (*Hypothesis 2*) and the IPM (*Hypothesis 3*) with UFR, respectively. *Hypothesis 3* did, in contrast to Köllner and Bleck's (2020) results, not include body asymmetry, as we were cautious regarding UFR_{r-l} 's absent sexual dimorphism in Köllner and Bleck's (2020) findings. To test convergent validity, we predicted significant relationships of UFR with fWHR, SHR, SWR, and WHR, but not with 2D:4D (*Hypothesis 4*, see 8. in preregistration). Finally, similar predictions were tested for other possible markers (2D:4D, fWHR, SHR, SWR, WHR).

Method

Aside from including additional potential markers and minor adjustments, materials and procedure were identical to Köllner and Bleck (2020).

Sample

Psychology students or psychologists were not admitted to the study. We used the following inclusion criteria by Köllner and Bleck (2020) to maximize comparability in our replication study: (1) Minimum requirements for valid PSEs (average word count of 30 words per story, i.e., 180 words overall; no missing stories), (2) BMI over 17.5 (to ensure normal/finished bone growth; see Köllner & Bleck, 2020). The latter precondition also

required the exclusion of cases for which body height and/or weight were missing. In addition, participants for which one or more marker variable(s) were missing (constituent variables for computing UFR, 2D:4D, fWHR, as well as SHR and WHR) were excluded. Minimum age again was 18 years, but we specified no maximum age to obtain more generalizable results.

Power analyses were based on $1 - \beta = .80$, with a p -level of .05. Regarding sexual dimorphism, Köllner and Bleck (2020) reported a d around 0.50 (suggesting 128 participants, or 64 per cell). But for n Power, most effect sizes regarding UFR, our primary target marker, are unknown, especially given that Köllner and Bleck (2020), in contrast to our preregistration, focused on an asymmetry-based effect involving UFR_{r-l} . Thus, we expected the usual motive-outcome correlation ($r = .22$; see meta-analysis by Spangler, 1992), which corresponds to the average effect size of $r = .21$ in social psychology (mega-analysis by Richard et al., 2003).

Power analyses based on these effect sizes suggested a sample size of 176.

To prevent test power loss due to attrition, we aimed at a sample size of 200 participants (80 plus three times 40 participants collected by a doctoral candidate and three students as requirements for their theses, respectively), but experimenters voluntarily went beyond this number without knowledge of the study outcomes. Overall, 261 participants were recruited via online advertisement and flyers. There were one PSE-based and two BMI-based exclusions, as well as four additional participants with missing marker variables. Two pregnant participants were excluded due to possible distortion of the circumference measures. Two additional non-preregistered exclusions were necessary, as one participant reported to be an XX-man and another one seemed to be intoxicated. Preliminary analyses revealed no UFR or UFR_{r-l} values clearly exceeding normal outliers and thus representing wrong measurements (all values within ± 4 SDs, i.e. 99.9% of the population). The final sample¹ consisted of 250 participants (148 women, 102 men) with a mean age of 26.20 years ($SD = 8.83$; range: 18-60).

¹ Deviations from preregistration: BMI-based and pregnancy-based exclusions were added to parallel the analyses by Köllner and Bleck (2020) as closely as possible and to avoid distortion of circumference measures, respectively. Due to our unexpectedly large sample and only minuscule number of exclusions, we

Design

Predictions were tested in one single testing session in a correlational, one-factorial design with self-reported sex (male vs. female) as between-subjects categorical variable. As preregistered, to control for differences in body composition not specifically attributable to markers, if a marker variable was significantly related to BMI and/or body height, BMI or height or both were controlled for in hypothesis-relevant regressions. PSE variables (*nPower*, *AI*) were continuous predictors. The dependent variables were scores on potential marker variables assessed via anthropometry (UFR, but also SHR, SWR, WHR, 2D:4D).

Materials

Implicit motives

The computer-administered PSE, the six pictures (e.g., Pang & Schultheiss, 2005), and the standard instructions (see Schultheiss & Pang, 2007) used for implicit motive assessment were identical to those used by Köllner and Bleck (2020). Pictures were presented for 10 seconds each in random order and participants had four minutes to type an imaginative story to each picture.

Two trained (> 85% intercoder reliability for the three main motives *nPower*, *nAchievement*, and *nAffiliation* in coding practice material prescored by experts, see Schultheiss, 2009) coders coded the resulting stories using the *Manual for scoring Motive Imagery in Running Text* (Winter, 1994) and *eXtended coding* rules (Köllner, 2015; no second-sentence-rule: allowing for one or multiple motive-subcategories in each sentence, but not for multiple coding of the same subcategory within sentence as long as no other main motive is coded in between) in the PSE-Coder software (Frisch & Schultheiss, 2012). The PSE-Coder also automatically determined word count and *AI* raw scores (frequency of

decided to base all computations on this final sample instead of working with hypothesis-wise exclusions. Using only cases which were valid on all involved measures also allowed us to keep our analyses and tables as straightforward as possible. All statistical conclusions for all hypotheses were unchanged when retested with the originally preregistered hypothesis-wise exclusions (aside from one single correlation between UFR and fWHR dropping to trend-level when including extremely low BMI-scores ≤ 17.50), see the supplemental at [LINK TO BE INCLUDED IN PUBLISHED PAPER].

German form of the negation “not”, its uppercase version, as well as common misspellings: “nicht”, “Nicht”, “NICHT”, “nihct”). For coding *nPower*, we used the following subcategories (for details and the subcategories of *nAchievement* and *nAffiliation*, see Winter, 1994): strong, forceful actions; controlling or manipulating others; attempts at persuasion; providing unsolicited help; being concerned with fame or prestige; eliciting strong emotions in others.

Motive scores of each participant were then summed up by motive domain. Interrater-reliability (Pearson’s *r*) based on raw scores was satisfactory (*nPower*: .76) to good (*nAchievement*: .83, *nAffiliation*: .82) and thus scores of both coders were averaged. As pre-registered, PSE-scores, which all deviated from normality, were square-root transformed after adding a constant of one, and transformed scores were residualized for word count (all *rs* with PSE-variables $>.45$, $ps > .001$) and then *z*-standardized (cf. Schönbrodt et al., 2021).

Anthropometric measurements

Body weight was measured with a body composition monitor (Tanita BC-601), body height with a measuring tape affixed to a wall. BMI was calculated as weight (kg) divided by squared height (m). In addition, participants’ self-reported age and sex was assessed in a demographic questionnaire.

UFR. Ulna and fibula length on both body sides, respectively, were determined on seated, dressed (but jackets, shoes, watches, if possible also pullovers etc. removed) participants via anthropometry with the same devices (precision caliper: Metrica 12040; arm-length-measuring-apparatus, see Figure 1 in Köllner & Bleck, 2020) and according to the same method as described in Köllner and Bleck (2020) that is further detailed in Bleck (2018). The distance between easily measurable reference points was measured for the ulna (caput ulnae to olecranon) and the fibula (caput fibulae to malleolus lateralis). This method of measuring long bone lengths proved to be highly reliable in previous research (interrater reliabilities from .81 up to .99; Köllner & Bleck, 2020). UFR was calculated as ulna length

averaged across both arms divided by fibula length averaged across both legs. The UFR_{r-l} asymmetry score was determined by subtracting left-body-side UFR from right UFR.

fWHR. fWHR (bizygomatic width divided by the distance between approximated prosthion and nasion) was measured with a sliding caliper according to method c) (nasion-approximation) described in Janson et al. (2018; see this publication for details and typical interrater-reliability scores). This method involves approximating prosthion-nasion-distance by the distance between the highest point in the middle of the upper lips and, as a benefit of direct anthropometry not feasible in photographs, a direct nasion approximation on the skin surface. This is achieved by looking at the participant from the front and from the side, aided by haptic inspection if necessary.

Body circumference measures. Shoulder (circumference at greatest width of shoulder blades; arms relaxed), waist (smallest circumference between iliac crest and rib cage), and hip (largest circumference between thigh and waist) circumference were measured with a measuring tape to one decimal place according to Hughes and Gallup (2003). Participants were instructed to inhale naturally before measurements were taken. SHR (shoulder divided by hip circumference), WHR (waist divided by hip circumference), and SWR (shoulder divided by waist circumference) were calculated from resulting values.

2D:4D. Participants placed both palms simultaneously on a Hewlett-Packard HP Scanjet G4050 and we scanned their hands at 300 ppi. Lengths of second and fourth digit (midpoint of the bottom crease to the fingertip; Schultheiss et al., 2019) were measured from resulting scans in the Face/Palm Measuring Tool (version 1.0.7; Köllner et al., 2017). 2D:4D was calculated by dividing 2D length by 4D length for each hand, respectively, and averaging the results across both hands.

Procedure

The procedure and order of tests was identical for all participants and paralleled the one by Köllner and Bleck (2020): After giving informed consent and answering some

questions unrelated to our research, participants completed computer-administered (Inquisit 5) tasks in separated cabinets, starting with the PSE followed by several tasks unrelated to the present research and finishing with a demographic questionnaire. Afterwards, the anthropometric measurements and hand scans were performed on the participants. Then participants received their compensation (20€ or individual feedback on their motivational dispositions) and a debriefing.

Statistical methods

We tested our predictions using Systat 13 and used *t*-tests, Pearson correlations, or general linear models (GLMs), depending on the respective hypothesis.

Results

Data preparation

Besides the above-mentioned PSE-variable-transformations, we logarithmically transformed ($\ln$) age, BMI, and WHR to improve normality, but, deviating from preregistration, refrained from $\ln$ -transforming other variables if this moved them even further away from normality.

Descriptive statistics

Table 1 (overall) and Table 2 (within-sex) provide descriptive statistics. UFR was positively related to BMI ($p = .04$). Thus, subsequent GLMs were controlled for BMI. BMI was also related to all other body parameters measured, except for 2D:4D and fWHR. Neither *n*Power nor AI were significantly related to any body-related variable, but positively interrelated with each other. Within-sex associations of PSE-variables with body-parameters were absent, aside from a positive relationship between AI and UFR_{r-l} in men. Due to the low sample mean age, age-related statistics are not interpretable and thus not discussed further.

Hypothesis testing

Sexual dimorphism

In support of *Hypothesis 1a*, UFR (but not UFR_{r-l} , $p = .20$) was sex-dimorphic ($p = .02$; for t -tests, see Table 1; for descriptive statistics, see Table 2), with larger values for men than for women and a slightly smaller effect ($d = 0.29$) than the one reported by Köllner and Bleck (2020; $d = .55$). This effect size was increased and highly significant ($t(246) = 2.90$, $p = .004$, $d = 0.37$) after excluding two statistical UFR-outliers (≤ -3 SDs; cf. Osborne, 2013, who deems such scores unlikely to be part of the target population)².

As expected (*Hypothesis 1b*) – and in contrast to other body-parameters like 2D:4D, SHR, or WHR – UFR was unrelated to body height in the overall sample ($p = .80$; $BF_{01} = 12.23$; JASP 0.14.1.0). Thus height was not controlled for in subsequent analyses.

Associations with other markers of organizational hormone effects

Regarding convergent validity, in support of *Hypothesis 4* we found positive associations of UFR with fWHR ($p < .05$), as well as with SHR and WHR ($ps < .001$), but not with SWR ($p = .38$), and, as expected, also not with 2D:4D ($p = .85$; $BF_{01} = 12.41$; JASP 0.14.1.0).

Sex-dimorphic associations to nPower and AI

We expected sex-dimorphic associations of *nPower* (*Hypothesis 2*) and the IPM (*Hypothesis 3*) with UFR, respectively. When predicting UFR from sex, *nPower*, and their interaction while controlling for BMI, there was no Sex x *nPower* interaction, $F(1, 245) = 0.002$, $p = .96$, $\eta_p^2 = .000$, conclusions unchanged after removing one of the above-mentioned outliers). Thus *Hypothesis 2* was rejected. Splitting the model by sex in additional analyses revealed conspicuous male outliers (see above), but also no significant effects (ts for both sexes, respectively < 1.44 , $ps > .15$; even after excluding outliers) for UFR and *nPower*.

² Those two male outliers were identified as they kept popping up while testing *Hypotheses 2* and *3*, one overall and both within-sex, respectively, hinting at highly untypical scores potentially resulting from measurement errors. As the preregistration stated that analyses would be repeated with statistical outliers removed, we inspected them more closely. One had the lowest UFR of the overall sample combined with a BMI of 37, probably interfering with reliable surface measurement of bone dimensions. The other outlier had the next-to-lowest UFR among the men, while both were outliers when looking at the overall sample (≤ -3 SDs).

When adding AI to the overall model, the Sex $\times$ *n*Power $\times$ AI interaction predicted by *Hypothesis 3* emerged at trend-level ($F(1, 241) = 3.44, p = .06, \eta_p^2 = .014$), and was significant after removing the two above-mentioned outliers, $F(1, 239) = 4.66, p = .03, \eta_p^2 = .019$. The pattern illustrated in Figure 1 shows that the IPM was associated with “sex-typical” (low for women, high for men) UFR values. Splitting the model by sex in follow-up analyses yielded suggestive, but non-significant *n*Power $\times$ AI interactions on UFR for men ($t(97) = 1.08, p = .28; B = 0.005, SE = 0.003, t(95) = 1.57, p = .12, \Delta R^2 = .025$ after excluding the two outliers) and women, $B = -0.004, SE = 0.003, t(143) = -1.36, p = .17, \Delta R^2 = .012$. Overall, *Hypothesis 3* was at least partly, if not fully, supported.

In a similar vein, visualizations of additional simple slopes analyses backed the notion that sex-typical UFR-values were associated with the IPM (Figure 2). We also found a positive trend-level association of *n*Power and UFR in men in the context of high AI (+1SD; $B = 0.26, SE = 0.14, t(95) = 1.87, p = .06$) and no association in the context of low AI (−1SD; $B = -0.001, SE = 0.11, t(95) = -0.01, p = .995$), underscoring the role of the IPM in particular for our main finding. For women, the non-significant associations between *n*Power and UFR were also in the expected directions both for high (+1SD; $B = -0.06, SE = 0.12, t(143) = -0.49, p = .62$) and low (−1SD; $B = 0.18, SE = 0.12, t(143) = 1.48, p = .14$) AI.

Other potential markers

Looking at other potential markers, the predicted sex dimorphisms (see Table 1; preregistered *Hypothesis 1a* at <https://aspredicted.org/blind.php?x=er2pd2>) were found for 2D:4D (male < female), SHR, and WHR (male > female respectively), but not for fWHR and SWR. In addition, body height and BMI were clearly dimorphic (male > female). Regarding independence of body height (*Hypothesis 1b*), 2D:4D, SHR, or WHR (and BMI) did not fulfill this criterion. Overall, no other potential marker besides UFR simultaneously fulfilled the criteria required by *Hypothesis 1a* and *1b*.

Regarding interrelationships with other markers (see Table 1, *Hypothesis 4*), fWHR was related to SHR at trend-level and significantly to WHR, and circumference measures were pervasively interrelated, with a positive relationship between SHR and WHR as well as SWR, and negative one between WHR and SHR. However, opposed to *Hypothesis 4*, SHR and WHR were, both negatively related to 2D:4D. SHR was positively related to UFR_{r-l}.

For reasons of space, details on the remaining analyses are presented in the supplement ([LINK TO BE INCLUDED IN PUBLISHED PAPER]). As preregistered, *Hypotheses 2* and *3* were retested for other possible markers (2D:4D, fWHR, SHR, SWR, WHR; with *Hypothesis 2* not predicted for 2D:4D and fWHR). There were no significant results, with one isolated exception for *Hypothesis 3* and SWR that barely passed the .05-threshold ($p = .047$). As SWR was not sexually dimorphic, lacking a key marker criterion, we will not further discuss this result. Additionally, we could not replicate the Sex x *n*Power x AI interaction ($p = .67$) on fWHR reported by Janson et al. (2018).

Explorative analyses

In non-preregistered analyses (see supplement), we retested *Hypotheses 2* and *3* also for UFR asymmetry (UFR_{r-l}). There were no significant results. The Sex x *n*Power x AI interaction predicting UFR_{r-l} reported by Köllner and Bleck (2020) was not replicated ($p = .33$), too.

As a side note, *n*Power was also exploratively replaced by other implicit and explicit motivational constructs (cf. Köllner & Bleck, 2020). As this was not preregistered, related findings are presented in a supplemental only ([LINK TO BE INCLUDED IN PUBLISHED PAPER]). There were almost no meaningful results, which will thus not be discussed further.

Discussion

Confirming earlier results (Köllner & Bleck, 2020), UFR was sex-dimorphic and unrelated to overall body height (*Hypothesis 1a/b*). It was related to fWHR, SHR, and WHR, and unrelated to 2D:4D, but also unrelated to SWR (*Hypothesis 4*). As predicted, a “sex-

typical” (high for men, low for women) UFR was associated with the IPM at trend-level, and significantly after excluding two conspicuous outliers (*Hypothesis 3*). Neither *nPower*’s sex-dimorphic relationship with UFR (*Hypothesis 2*), nor IPM’s sex-dimorphic relationship with UFR asymmetry (deemed unreliable and thus already omitted from the preregistration) reported by Köllner and Bleck (2020) were replicated. Analyses regarding other markers were mostly inconclusive.

Interpretation

Overall, our results provide solid support for UFR’s marker potential for pubertal OHEs and another high-powered piece of evidence for hormonal contributions to implicit motive development.

UFR’s potential as a marker of pubertal hormone effects

Regarding possible “quality criteria” for a body marker of *nPower*-related OHEs (E2, T), UFR performed excellently. First, it showed both sexual dimorphism (*Hypothesis 1a*) and independence from body height (*Hypothesis 1b*), a favorable feature combination not simultaneously displayed by any other potential marker tested here. While sexual dimorphism in our view is a sine-qua-non requirement for any marker used to estimate even more gradual differences in OHE exposure within-sexes, the most frequently used marker of pubertal hormones, fWHR, again showed no sex differences. This result fits with other large-sample (Özener, 2012) and recent meta-analytical research (Kramer, 2017, but see Geniole et al., 2015; Köllner et al., 2018). Using UFR seems to be preferable based on sexual dimorphism.

Second, while other potential pubertal markers (WHR and SHR, but not SWR) even showed clearer dimorphism (cf. Hughes & Gallup, 2003) than UFR, their pronounced relationships to overall body height represent a disadvantage. Considering that for 2D:4D, mathematical models leave open the possibility that the sex difference is an artifact of general finger growth parameters associated with larger hands (and thus of overall between-sex hand size differences; Leslie, 2019), new potential markers should not be susceptible to the same

problem. Regarding UFR, we thus were especially interested in *relative* information associated with sex-hormone-driven differential long bone growth (see above), not with overall bone length or overall differences in body height. UFR's independence of body height consequently is an additional advantage compared to prenatal (2D:4D) and potential alternative pubertal markers (SHR, WHR).

Third, relationships with other potential pubertal markers (WHR, SHR) emerged (except for SWR) as predicted (*Hypothesis 4*), supporting UFR's convergent validity. The absent relationship with SWR does not mitigate this conclusion: In contrast to WHR and SHR (e.g., Fink et al., 2003; Gallup et al., 2007), we are not aware of any research investigating links between SWR and OHEs, and SWR's own marker potential is doubtful considering the absent dimorphism in our sample. As expected, no relationship to 2D:4D was found, separating UFR (but not WHR and SHR) from prenatal OHEs.

In sum, UFR, after first promising results (e.g., dimorphism, unrelatedness to body height) by Köllner and Bleck (2020), again demonstrated its possible marker potential for OHEs in a high-powered, preregistered study.

While UFR performed excellently regarding marker criteria, the associated asymmetry measure did not: UFR_{r-l} was proposed by Köllner and Bleck (2020) in an analogy to 2D:4D asymmetry (Manning et al., 2014) to test for similar pubertal effects on motivation as those reported by Schultheiss et al. (2019) for prenatal OHEs. However, data from two studies now show that UFR_{r-l} is not sexually dimorphic. Findings for convergent validity with other markers (fWHR, 2D:4D) from the first study were not replicated, too: Aside from a positive relationship to SHR, no associations with markers emerged for the overall sample. Thus, UFR_{r-l} should be used with caution and only in preregistered analyses in future studies.

Hormonal contributions to implicit motive development

Based on sex-dimorphic associations of the IPM with UFR as a pubertal marker (*Hypothesis 3*), the case for pubertal hormonal contributions to *nPower* development now is

solidified. Our sample was almost twice as large as the one of Köllner and Bleck (2020), and our analyses were not exploratory, but preregistered, and thus less susceptible to Type I Errors (Cramer et al., 2016).

UFR's relationship to motivation seems to be specific to the IPM as predicted by *Hypothesis 3* (and to sex-typical scores). This fits well with previous findings obtained for implicit motives and OHEs, because aside from rare exceptions (*nPower* and UFR in Köllner & Bleck, 2020; *nAchievement* and 2D:4D in Schultheiss et al., 2019; both findings moderated by sex), all main results for markers of prenatal (2D:4D and IPM; Schultheiss & Zimni, 2015, 2D:4D asymmetry and IPM, moderated by sex; Schultheiss et al., 2019) and pubertal OHEs (fWHR and IPM, Janson et al., 2018; UFR_{r-l} and IPM, Köllner & Bleck, 2020; both findings moderated by sex) involved the IPM and thus AI as a moderator. As stated above, AI may mark functional rightward brain lateralization (Schultheiss et al., 2009), while OHEs are involved in prenatal sex-steroid-driven brain lateralization processes favoring the right hemisphere (Geschwind & Galaburda, 1987). Hormone-influenced processes changing cortical connectivity may also occur in puberty (see Köllner et al., 2019, for details), possibly explaining AI's persisting relevance at this developmental stage and thus the IPM findings obtained here and in other studies.

In addition, most studies' main result was moderated by sex. Considering that 2D:4D and UFR are considered relative indicators of the estrogen-to-androgen ratio (or vice versa in the case of UFR; Köllner & Bleck, 2020; Manning et al., 2014) and that dominance contest studies show that the hormones functionally coupled with *nPower* differ between the sexes (T relevant to male and E2 to female dominance; Schultheiss, Wirth, et al., 2005; Stanton & Schultheiss, 2007), divergent or even reversed findings for men and women in marker studies are plausible. For example, our results suggested that sex-typical UFR scores (i.e. high scores in men, low in women) are coupled to the IPM.

In sum, the case for hormonal contributions to *nPower*/IPM development was already solid for prenatal OHEs (Schultheiss et al., 2019; $N = 618$) and is, after first promising results (Janson et al., 2018, $N = 213$; Köllner & Bleck, 2020; $N = 126$), now further solidified for pubertal OHEs by our high-powered study ($N = 250$). This fits with the biopsychosocial model of implicit motive development proposed by Köllner et al. (2019) that includes prenatal and pubertal OHEs as a biological basis of implicit motives which is subsequently modulated by social learning experiences in early childhood (mainly through parental behavior; McClelland & Pilon, 1983) and probably also during adolescence.

Nevertheless, our results diverge from Köllner and Bleck (2020) in two aspects. First, *nPower*'s sex-dimorphic association with UFR (*Hypothesis 2*) was not replicated. Upon closer inspection, however, this association was counter-intuitive, as UFR scores *untypical* for the respective sex (high for women, low for men) were associated with *nPower*, possibly a spurious finding based on hidden multiplicity in Köllner and Bleck's (2020) exploratory GLMs (Cramer et al., 2016). In addition, as stated above, almost every finding regarding implicit motivation and OHEs pertains to the IPM, which also includes AI, which would have rendered such an association untypical of the literature.

Second, as expected, Köllner and Bleck's (2020) sex-dimorphic association of the IPM with UFR_{r-l} was not replicated. While previous results for the IPM and pubertal OHEs emerged without including an asymmetry measure (fWHR; Janson et al., 2018), UFR_{r-l} was not dimorphic in the first study, questioning its marker potential. Considering that the respective IPM-finding was derived from a five-way interaction in an at-best medium sized sample and being aware of possible hidden multiplicity, we had opted for simpler, easier-to-replicate hypotheses and not even preregistered a respective UFR_{r-l} analysis.

Overall, the mentioned divergent results provide no counter-argument against an association of pubertal OHEs and the IPM.

Regarding other potential pubertal markers, the failure of SHR and WHR to produce motive-related results may mainly result from their large dependence on body fat distribution in upper body and buttocks/thighs (Fink et al., 2003) which should be more susceptible to post-pubertal changes (e.g., through nutrition) and thus less appropriate as a pubertal marker than more bone-based measures like UFR. Regarding the non-replication of earlier sex-dimorphic results for fWHR (Janson et al., 2018), we speculate on measurement issues: First, the role of fat deposits in the cheeks, especially in women, may have obscured true variance associated with facial bone growth (cf. Lefevre et al., 2013). Also, in contrast to all other pubertal marker candidates, our experimenters reported problems in producing converging results for facial width and height when comparing their anthropometric measurements on the same participants in a test sample. We conclude that UFR is a preferable choice in studies on OHEs and implicit motivation.

Limitations and future directions

Discussing limitations of our research, for reasons of space we will not reiterate obvious drawbacks of body marker research on prenatal or pubertal OHEs, like possible confounding variables (e.g. age for fWHR; Hehman et al., 2014), imprecisions in body-surface-based measurements (Manning, 2002), or that estimating OHEs on the brain via markers can only be an intermediate step on the way to looking at the brain itself as soon as sufficiently-powered imaging studies become affordable (cf. Köllner et al., 2018). Until then, those issues can be countered via preregistered hypotheses and carefully checking “quality criteria” (e.g. dimorphism, unrelatedness to potentially confounding variables), and thus provide no counter-argument to using markers. Rather, given these limitations, it is encouraging that research on markers and implicit motives repeatedly yielded promising results, despite the fact that observed relationships probably are underestimations due to for example imprecise measurement (cf. Schultheiss et al., 2019).

However, while UFR's pubertal origin is plausible based on our data (e.g., convergent validity), we currently cannot rule out additional influences of earlier developmental stages on the ratio. Thus, other marker "quality criteria" call for further research: hormonal association and attributability to a specific developmental window. A marker should be related to hormones, at least during the developmental window from which it originates from. While there is sound experimental evidence from animal research for 2D:4D and prenatal hormones (Zheng & Cohn, 2011), results for puberty are sparse, with a low-powered exception for fWHR during development (Welker et al., 2016; 75 male participants) and conspicuous null-results for fWHR in adults for baseline and reactive T (Bird et al., 2016). Future studies thus should test associations of E2 and T with potential markers. UFR is a promising choice for such research, given the quality criteria reported here.

As a related issue, attributability to a specific developmental window is a vital, but heavily underresearched marker criterion, requiring effortful, time-consuming longitudinal studies (see Lutchmaya et al., 2004, combining amniocentesis and recording 2D:4D two years after birth). Future research checking this criterion for UFR and other pubertal markers should assess body dimensions, hormonal status, and behavioral outcomes in prepubertal (low steroid levels, pubertal markers not yet developed and unrelated to hormones and behavior), peripubertal (rising steroid levels, markers starting to develop and correlated with hormones and behavior) and adult participants (high steroid levels, markers fully developed and not necessarily still related to hormones, but still to behavior due to the developmental outcomes of pubertal OHEs). This effortful approach is inevitable, as post-mortem studies (e.g., on skulls; Weston et al., 2007) do not allow behavioral assessment and as animal research is not fully generalizable to humans due to morphological differences.

Conclusions

This high-powered, preregistered replication study bolsters UFR's status as a marker of pubertal OHEs: UFR is sexually dimorphic, unrelated to body height, related to other

markers, and shows sex-dimorphic associations with the IPM. Our confirmatory analyses suggest that UFR may provide a dimorphic, outcome-associated alternative to the often-criticized fWHR. Viewed in conjunction with earlier results for UFR (Köllner & Bleck, 2020) and other findings for prenatal (e.g., Schultheiss et al., 2019; Schultheiss & Zimni, 2015) and pubertal (Janson et al., 2018) OHEs, there is also accumulating evidence for hormonal contributions to implicit motive development.

Author contributions

MGK wrote the manuscript, trained motive coders and supervised the coding process, designed the study, and wrote the associated preregistration. KTJ and LP recruited participants and conducted the experiment. LP and LS coded PSEs. KTJ designed and supervised the experiment and refined the manuscript. MGK undertook statistical analysis, aided by KTJ and LS. All authors contributed to and have approved the final manuscript.

Acknowledgements

We thank Liane Mair, Lola Riehlein, and Nadine Schneider for recruiting participants and conducting the experiment. We further thank Nadine Schneider for conducting the 2D:4D measurements.

Ethical Standards

All participants gave their informed consent prior to inclusion in the study. The manuscript does not contain clinical studies or patient data. Procedures were approved by the ethics committee of the Friedrich-Alexander University in Erlangen (No. 181_17 B).

Conflict of Interest

The authors declare that they have no conflict of interest.

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Table 1*Descriptive statistics and correlations for the overall sample*

	<i>M</i>	<i>SD</i>	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. <i>n</i> Pow	4.75	2.80	---											
2. AI	4.72	3.46	.14*	---										
3. UFR	0.69	0.04	.06	-.01	---									
4. UFR _{r-l}	0.00	0.02	.10	.06	.00	---								
5. 2D:4D	0.98	0.03	.05	.05	-.01	.05	---							
6. fWHR	1.53	0.14	.09	-.04	.13*	.09	.07	---						
7. SHR	1.09	0.10	.11#	.08	.23***	.18**	-.21***	.12#	---					
8. WHR	0.82	0.08	.03	.04	.25***	.08	-.14*	.13*	.48***	---				
9. SWR	1.34	0.12	.07	.03	-.06	.09	-.06	-.02	.45***	-.57***	---			
10. Height	173.18	9.87	.03	.00	-.02	.03	-.20**	-.03	.33***	.31***	.02	---		
11. BMI	23.75	4.02	.01	-.10	.13*	-.15*	.00	.04	-.24***	.32***	-.54***	.15*	---	
12. Age	26.20	8.83	-.11#	-.05	-.15*	-.17**	-.03	-.21**	-.27***	.14*	-.39***	-.04	.34***	---
13. Sex	-	-	0.91	0.28	2.26*	1.28	-3.43***	0.56	8.99***	8.89***	-0.45	17.40***	3.38***	0.76

Note. $N = 250$ ($n = 249$ for values related to age due to one case without related information). Descriptive statistics are derived from the raw scores of the respective variables (e.g., ulna/fibula length in cm). Pearson correlations and t -statistics are computed for transformed variables where necessary, which means square root transformation, residualization for word count and subsequent z -standardization for *n*Pow and AI; as well as $\ln$ -transformation for WHR, BMI, and age. *n*Pow = implicit need for power (extended coding); AI = activity inhibition; UFR = ulna-to-fibula ratio of bone lengths averaged across both body sides; UFR_{r-l} = difference between right and left UFR; 2D:4D = 2D:4D digit ratio averaged across both hands; fWHR = facial width-to-height ratio measured from anthropometry (nasion approximation); SHR = shoulder-to-hip ratio; WHR = waist-to-hip ratio; SWR = shoulder-to-waist ratio; height = body size (cm); BMI = body mass index; age = age (years); sex = t -statistics for sex differences (t -tests; ; $M_{\text{♂}} - M_{\text{♀}}$; df : 248; df for age: 247).

*** $p < .001$. ** $p < .01$. * $p < .05$. # $p < .10$.

Table 2

Separate descriptive statistics and correlations for women (n = 148, sub-n = 147 for age-related values; above diagonal) and men (n = 102; below diagonal)

	$M_{\text{♀}}$	$SD_{\text{♀}}$	$M_{\text{♂}}$	$SD_{\text{♂}}$	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. <i>n</i> Pow	4.88	2.76	4.55	2.87	---	.13	.05	.12	.08	.04	.13	-.02	.13	.05	.00	-.13
2. AI	4.97	3.48	4.37	3.43	.14	---	-.07	-.03	.06	-.03	.05	.05	-.02	-.06	-.08	.00
3. UFR	0.68	0.03	0.70	0.04	.06	.05	---	-.06	-.01	.14#	.18*	.30***	-.12	-.20*	.26**	-.16#
4. UFR _{r-l}	0.00	0.02	0.01	0.01	.05	.21*	.07	---	.11	.03	.19*	.05	.12	.03	-.24**	-.18*
5. 2D:4D	0.98	0.03	0.97	0.03	.03	.04	.06	-.01	---	.09	-.10	.00	-.09	-.05	.06	-.03
6. fWHR	1.53	0.14	1.54	0.15	.15	-.05	.09	.18#	.06	---	.06	.09	-.02	-.13	.10	-.13
7. SHR	1.05	0.08	1.15	0.09	.04	.13	.19#	.10	-.15	.19#	---	.36***	.52***	-.09	-.48***	-.43***
8. WHR	0.79	0.07	0.87	0.07	.04	.00	.09	.04	-.10	.18#	.24*	---	-.60***	-.15#	.24**	.04
9. SWR	1.34	0.12	1.33	0.13	-.01	.09	.04	.04	-.03	-.01	.55***	-.68***	---	.07	-.63***	-.41***
10. Height	167.11	6.91	181.98	6.23	-.13	.04	-.17#	-.20*	-.09	-.01	-.01	-.03	.03	---	-.01	-.13
11. BMI	23.09	3.74	24.70	4.23	-.01	-.15	-.08	-.05	.01	-.04	-.30**	.27**	-.45***	-.01	---	.29***
12. Age	25.98	9.06	26.53	8.51	-.08	-.14	-.15	-.16	.00	-.33***	-.20	.27**	-.37***	-.07	.42***	---

Note. Descriptive statistics are derived from the raw scores of the respective variables (e.g., ulna/fibula length in cm). Pearson correlations are computed for transformed variables where necessary, which means square root transformation, residualization for word count and subsequent *z*-standardization for *n*Pow and AI; as well as *ln*-transformation for WHR, BMI, and age. *n*Pow = implicit need for power (extended coding); AI = activity inhibition; UFR = ulna-to-fibula ratio of bone lengths averaged across both body sides; UFR_{r-l} = difference between right and left UFR; 2D:4D = 2D:4D digit ratio averaged across both hands; fWHR = facial width-to-height ratio measured from anthropometry (nasion approximation); SHR = shoulder-to-hip ratio; WHR = waist-to-hip ratio; SWR = shoulder-to-waist ratio; height = body size (cm); BMI = body mass index; age = age (years).

*** $p < .001$. ** $p < .01$. * $p < .05$. # $p < .10$.

Figure 1

Relationship between the implicit need for power (nPower) and ulna-to-fibula ratio (UFR) dependent on activity inhibition (AI) for men (left; two outliers removed) and women (right)

Note. nPower was square root transformed, residualized for word count and subsequently z-standardized. The same was true for AI (median split). Figure available at [LINK TO BE INCLUDED IN PUBLISHED PAPER], under a CC-BY4.0 license.

Figure 2

Simple slopes analyses showing within-sex associations of ulna-to-fibula ratio (UFR) and the implicit need for power (nPower), for low ($-1SD$), medium (M), or high ($+1SD$) AI in women (left) and men (right; ; two outliers removed)

Note. Z-scores were used for computation. AI and nPower were square root transformed, residualized for word count and subsequently z-standardized. Figure available at [LINK TO BE INCLUDED IN PUBLISHED PAPER], under a CC-BY4.0 license.

The ulna-to-fibula ratio as a marker of organizational hormone effects on the motivational brain:

A high-powered pre-registered replication

SUPPLEMENT

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SUPPLEMENT

Retesting our hypotheses (*H*) with hypothesis-wise exclusions

Originally, we had preregistered hypothesis-wise exclusions, but, due to the reasons stated in the main paper, decided to use overall-exclusions instead. Retesting our hypotheses with hypothesis-wise inclusions meant using only exclusions pertaining to all hypotheses (PSE-based, BMI-based, UFR-data missing, the XX-man as well as the participant with bizarre behavior, see main paper) as a starting ground, yielding a sample of 255 participants. fWHR and circumference measure data was present for all remaining participants requiring no further hypothesis-wise exclusions for the respective analyses (*H2*). Three participants were excluded only for UFR's correlation with 2D:4D (*H2*, $n = 252$). Two pregnant participants were now only excluded for UFR's correlation with circumference measures (*H2*, $n = 253$).

On a hypothesis-wise basis, UFR was still unrelated to height ($r = -.00$, $p = .96$) and related to BMI ($r = .14$, $p = .02$), requiring the same control variable as before. (*H1a*) UFR (but again not UFR_{r-l} ; $t(253) = 1.07$, $p = .28$) was still sex dimorphic ($t(253) = 2.51$, $p = .01$). (*H1b*) UFR was still unrelated to body height (see above). (*H2*) There was still no sex-dimorphic association of $nPower$ with UFR ($F(1, 250) = 0.00$, $p = .98$; conclusion unchanged after removing an outlier), but (*H3*) again trend-level support for a sex-dimorphic association of the IPM with UFR ($F(1, 246) = 3.33$, $p = .07$; conclusion unchanged after removing an outlier) that was rendered significant after excluding the two outliers identified in the main paper ($F(1, 244) = 4.51$, $p = .03$). (*H4*) Finally, conclusions regarding relationships of UFR to other markers were completely unchanged: UFR was still related to fWHR ($r = .14$, $p = .03$), SHR and WHR ($n =$

253; $r_s > .23$, $p_s < .001$), but neither with SWR ($n = 253$; $r = -.05$, $p = .40$) nor with 2D:4D ($n = 252$; $r = -.00$, $p = .98$).

When even dropping the BMI-based exclusions, still testing on a hypothesis-wise basis, UFR was still unrelated to height ($r = -.01$, $p = .90$) and related to BMI ($r = .13$, $p = .03$), requiring the same control variable as before. (*H1a*) UFR (but again not UFR_{r.l}; $t(254) = 1.13$, $p = .26$) was still sex dimorphic ($t(254) = 2.46$, $p = .01$). (*H1b*) UFR was still unrelated to body height (see above). (*H2*) There was still no sex-dimorphic association of *nPower* with UFR ($F(1, 251) = 0.00$, $p = .99$; conclusion unchanged after removing an outlier), but (*H3*) again trend-level support for a sex-dimorphic association of the IPM with UFR ($F(1, 247) = 3.37$, $p = .07$; conclusion unchanged after removing an outlier) that was rendered significant after excluding the two outliers identified in the main paper ($F(1, 245) = 4.57$, $p = .03$). (*H4*) Finally, conclusions regarding relationships of UFR to other markers were completely unchanged, aside from UFR's relationship to fWHR dropping to trend-level ($r = .12$, $p = .06$): UFR was still related to SHR and WHR ($r_s > .22$, $p_s < .001$), but neither with SWR ($r = -.06$, $p = .37$; circumference-based measures here even tested with pregnant individuals included) nor with 2D:4D ($n = 253$; $r = .01$, $p = .88$).

In sum, all conclusions were unchanged when retested with hypothesis-wise exclusions.

Testing similar predictions for other possible markers

As preregistered, the same predictions were tested for other possible markers (2D:4D, fWHR, SHR, SWR, WHR; with *H2* not predicted for 2D:4D and fWHR). As can be seen from Table S1, there were no significant results, with one exception for *H3* and SWR ($F(1, 241) = 4.00$, $\eta_p^2 = .016$), that barely passed the .05-threshold. Splitting the model by sex in follow-up analyses non-significantly revealed a positive association of the *nPower* x AI interaction on SWR

for men ($B = 0.014$, $SE = 0.010$, $t(97) = 1.43$, $p = .16$, $\Delta R^2 = .016$) and a negative association for women (one outlier removed; $B = -0.012$, $SE = 0.008$, $t(143) = -1.55$, $p = .12$, $\Delta R^2 = .009$, see Figure S1). As this result was isolated and pertained to a body dimension that even failed to show sexual dimorphism (see *H1a* and Table 1 in main paper), a key requirement for a marker, we will not further discuss it.

In addition, in non-preregistered analyses we retested *H2* and *H3* also for UFR asymmetry (UFR_{r-l}). As can be seen from Table S1, there were no significant results.

Results for other implicit motives and the explicit power motive

In other explorative analyses omitted from the main paper for reasons of space, we retested the relationships postulated for *nPower* in *H2* and *H3* for the implicit need for achievement (*nAchievement*; $M = 5.26$; $SD = 2.81$), the implicit need for affiliation (*nAffiliation*; $M = 6.91$; $SD = 3.19$), and the self-reported, explicit power motive (*UMSpow*; $M = 2.50$; $SD = 0.97$; $n = 248$) assessed with the power scale of a short version of the Unified Motive Scales questionnaire (UMS-6, by Schönbrodt & Gerstenberg, 2012). The latter variable was normally distributed and thus not transformed.

All three variables showed sex differences (see Table S2), with larger values for female participants for *nAchievement* (♂ : $M = 4.53$; $SD = 2.50$; ♀ : $M = 5.76$; $SD = 2.90$) and *nAffiliation* (♂ : $M = 5.99$; $SD = 2.75$; ♀ : $M = 7.55$; $SD = 3.32$), and larger values for male participants for *UMSpow* (♂ : $M = 2.74$; $SD = 1.04$; ♀ : $M = 2.33$; $SD = 0.88$).

In addition, there were some associations between PSE-variables overall and within-sex (see Table S2 and S3). However, significant bivariate associations to body parameters were conspicuously absent, aside from correlations of SHR and body height with *UMSpow* respectively. Within-sex, there were no significant relationships at all, aside from a positive

association between UMSpow and UFR among men. These isolated significances are probably spurious and thus are not interpreted any further.

When replacing *n*Power with one of these exploratory variables, respectively, and retesting sex-dimorphic associations with UFR (*H2*), in the respective GLMs we obtained no significant Sex x *n*Achievement ($p = .22$, after removal of one outlier) or Sex x *n*Affiliation ($p = .16$, after removal of one outlier) interaction. However, we obtained a significant Sex x UMSpow interaction, $F(1, 242) = 4.22, p = .04, \eta_p^2 = .017$ (same conclusion with outlier included). “Sex-typical” UFR-values (high in men, $B = 0.006, SE = 0.003, t(96) = 1.85, p = .06, \Delta R^2 = .034$; low in women, $B = -0.003, SE = 0.003, t(145) = -1.03, p = .30, \Delta R^2 = .007$) were associated with a higher self-ascribed power motive (see Figure S2).

Retesting *H3* with one of these exploratory variables, respectively, by adding AI to the GLMs, we obtained no significant Sex x *n*Achievement x AI ($p = .46$, after removal of one outlier), Sex x *n*Affiliation x AI ($p = .33$, after removal of one outlier), or Sex x UMSpow x AI interaction ($p = .83$, after removal of one outlier).

Overall, aside from an isolated, unpredicted finding for *H2* and UMSpow that barely passed the significance threshold, there are no meaningful findings of any kind when replacing *n*Power with other motivational constructs like other implicit motives or the explicit power motive (cf. Köllner & Bleck, 2020). UFR’s relationship to motivation thus seems to be specific to the IPM as predicted by *H3*.

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Table S1

Results for Hypothesis 2 (Sex x nPower interaction) and Hypothesis 3 (Sex x nPower x AI interaction) when retested with other marker candidates (p-levels).

	<i>H2</i>	<i>H3</i>
1. UFR _{r-l}	.48	.33
2. 2D:4D	.58	.63
3. fWHR	.40	.67
4. SHR	.29 (1 outlier excluded)	.29 (1 outlier excluded)
5. WHR	.74	.22
6. SWR	.17	.047

Note. Respective analyses for UFR_{r-l} were not preregistered. A significant result for *H2* was not expected for 2D:4D and fWHR in the preregistration, respective analyses were run just to be comprehensive. UFR_{r-l} = difference between right and left ulna-to-fibula bone-length-ratio; 2D:4D = 2D:4D digit ratio averaged across both hands; fWHR = facial width-to-height ratio measured from anthropometry (nasion approximation); SHR = shoulder-to-hip ratio; WHR = waist-to-hip ratio; SWR = shoulder-to-waist ratio.

Table S2*Descriptive statistics for the overall sample: Intercorrelations and sex differences*

	1.	2.	3.
1. <i>n</i> Ach	---		
2. <i>n</i> Aff	.24***	---	
3. UMSpow	-.04	-.09	---
4. <i>n</i> Pow	.10	-.16*	.08
5. AI	-.18**	-.25***	.08
6. UFR	-.05	-.01	.08
7. UFR _{r-l}	.04	.01	.11#
8. 2D:4D	-.07	.05	-.04
9. fWHR	-.03	.02	-.05
10. SHR	-.08	-.09	.14*
11. WHR	-.12#	-.05	.07
12. SWR	.05	-.04	.06
13. Height	-.08	-.12#	.15*
14. BMI	-.07	-.03	.04
15. Age	.06	.01	-.11#
16. Sex	-1.98*	-2.60**	3.36***

Note. $N = 250$ (for values related to: age - $n = 249$, UMSpow - $n = 248$, age & UMSpow, $n = 247$). Pearson correlations and t -statistics are computed for transformed variables where necessary, which means square root transformation, residualization for word count and subsequent z -standardization for *n*Ach, *n*Aff, *n*Pow, and AI; as well as $\ln$ -transformation for WHR, BMI, and age. *n*Ach = implicit need for achievement (extended coding); *n*Aff = implicit need for affiliation (extended coding); UMSpow = power scale of the Unified Motive Scales; *n*Pow = implicit need for power (extended coding); AI = activity inhibition; UFR = ulna-to-fibula ratio of bone lengths averaged across both body sides; UFR_{r-l} = difference between right and left UFR; 2D:4D = 2D:4D digit ratio averaged across both hands; fWHR = facial width-to-height ratio measured from anthropometry (nasion approximation); SHR = shoulder-to-hip ratio; WHR = waist-to-hip ratio; SWR = shoulder-to-waist ratio; height = body size (cm); BMI = body mass index; age = age (years); sex = t -statistics for sex differences (t -tests; $M_{\text{♂}} - M_{\text{♀}}$; df : 248; df for UMSpow: 246).

*** $p < .001$. ** $p < .01$. * $p < .05$. # $p < .10$.

Table S3

Separate descriptive statistics for women (n = 148, sub-n = 147 for age-related values; left) and men (n = 102; sub-n = 100 for UMSpow-related values; right): Intercorrelations

	♀	1.	2.	3.	-	♂	1.	2.	3.
1. <i>n</i> Ach		---			-		---		
2. <i>n</i> Aff		.19*	---		-		.28**	---	
3. UMSpow		.03	-.10	---	-		-.07	.00	---
4. <i>n</i> Pow		.14#	-.09	.03	-		.06	-.26**	.11
5. AI		-.24**	-.28***	.11	-		-.09	-.20*	.03
6. UFR		.03	.09	-.07	-		-.11	-.09	.20*
7. UFR _{r-l}		.00	.03	.04	-		.14	.01	.19#
8. 2D:4D		-.09	.01	.04	-		-.10	.05	-.04
9. fWHR		.02	.13	-.10	-		-.09	-.13	.00
10. SHR		-.04	-.03	-.08	-		.02	.02	.19#
11. WHR		-.10	.01	-.07	-		-.02	.09	-.01
12. SWR		.05	-.03	-.01	-		.04	-.07	.14
13. Height		.05	-.07	.05	-		-.03	.14	-.09
14. BMI		.02	.00	.04	-		-.15	.00	-.05
15. Age		.08	-.03	-.11	-		.06	.12	-.14

Note. Pearson correlations are computed for transformed variables where necessary, which means square root transformation, residualization for word count and subsequent *z*-standardization for *n*Ach, *n*Aff, *n*Pow, and AI; as well as ln-transformation for WHR, BMI, and age. *n*Ach = implicit need for achievement (extended coding); *n*Aff = implicit need for affiliation (extended coding); UMSpow = power scale of the Unified Motive Scales; *n*Pow = implicit need for power (extended coding); AI = activity inhibition; UFR = ulna-to-fibula ratio of bone lengths averaged across both body sides; UFR_{r-l} = difference between right and left UFR; 2D:4D = 2D:4D digit ratio averaged across both hands; fWHR = facial width-to-height ratio measured from anthropometry (nasion approximation); SHR = shoulder-to-hip ratio; WHR = waist-to-hip ratio; SWR = shoulder-to-waist ratio; height = body size (cm); BMI = body mass index; age = age (years).

*** $p < .001$. ** $p < .01$. * $p < .05$. # $p < .10$.

Figure S1

Relationship between the implicit need for power (nPower) and Shoulder-to-Waist Ratio (SWR) dependent on activity inhibition (AI) for men (left) and women (right; one outlier removed)

Note. nPower was square root transformed, residualized for word count and subsequently z-standardized. The same was true for AI (median split). Figure available at [LINK TO BE INCLUDED IN PUBLISHED PAPER], under a CC-BY4.0 license.

Figure S2

Scatter-plot showing the sex-dimorphic association (one outlier removed) between self-ascribed power motive (assessed with the Unified Motive Scales, short version UMS-6) and ulna-to-fibula ratio (UFR).

Note. Figure available at [LINK TO BE INCLUDED IN PUBLISHED PAPER], under a CC-BY4.0 license.