

## **The social biopsychology of implicit motive development**

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This chapter summarizes recent evidence for *organizational hormone effects* (OHEs), that is, lasting organizational effects of steroid hormones on nervous system structure occurring during development (Sisk & Zehr, 2005), on the development of non-conscious motivational dispositions. We combine this evidence with earlier research on social aspects into a biopsychosocial developmental model, assuming that OHEs provide a biological basis for non-conscious motivational dispositions, which is then pruned and fine-tuned to varying degrees by later social learning experiences. We focus on human participants (for OHEs in animals please see the chapter by K. Schulz) and on non-conscious motivational dispositions. While there is evidence for influences of interactions between hormones and conscious traits on behavior (e.g., Carré et al., 2016; Slatcher, Mehta, & Josephs, 2011), this is beyond this chapter's scope, because non-conscious and conscious motivational dispositions are clearly distinct, as outlined below.

### **Implicit Motives**

*Implicit motives* are dispositions allowing an individual to perceive the attainment of specific types of incentives as rewarding and the confrontation with specific types of disincentives as aversive (Schultheiss, 2008; Schultheiss & Köllner, 2014). Operating outside of conscious awareness, they select, energize, and direct behavior (McClelland, 1987), partly by interacting with endocrine systems (Schultheiss, 2013).

A large body of research has focused on three implicit motives: (1) the implicit *need for power* (*nPower*), the capacity for deriving pleasure from having impact on others while experiencing others' impact on oneself as aversive; (2) implicit *need for affiliation* (*nAffiliation*), the capacity to derive pleasure from establishing, maintaining, and restoring positive interpersonal relationships, and (3) implicit *need for achievement* (*nAchievement*), the capacity to derive pleasure from autonomous mastery of challenging tasks (see Schultheiss, 2008, for details). We focus on *nPower*, while the latter two needs are only addressed briefly.

Implicit motives differ from *explicit motives* (self-attributed needs and goals; McClelland, Koestner, & Weinberger, 1989), predicting different kinds of behaviors and responding to different cue types (Schultheiss, 2008; cf. meta-analysis by Spangler, 1992). While self-attributed needs predict planned behaviors like judgments and choices and respond to verbal cues, implicit motives predict spontaneous behaviors and respond to non-verbal cues (e.g., Biernat, 1989; see Schultheiss, 2008). Moreover, implicit and explicit motives have no substantial overlap, as demonstrated by 60 years of meta-analytically combined research (Köllner & Schultheiss, 2014; Spangler, 1992), and may depend on neurobiologically distinct motivational systems (McClelland et al., 1989).

An individual's implicit motive strength is not consciously accessible and cannot be measured via questionnaires (see Schultheiss, 2008). Thus, a well-established method for motive assessment is the *Picture-Story Exercise* (PSE; McClelland et al., 1989), comprising four to eight pictures of persons in ambiguous social situations, such as two women working in a laboratory (see Schultheiss & Pang, 2007). These pictures are presented in random order and participants have four minutes per picture to write imaginative stories. The stories are then scored by coders who previously exceeded 85% interscorer-agreement with experts on training materials. Coders analyze the stories for imagery pertaining to specific motive domains like *nPower* (e.g., strong forceful actions, impressing others) following specific

coding rules (e.g., Winter, 1994). Such coding rules are empirically derived by comparing stories from participants with experimentally aroused motives to stories from control participants (see Schultheiss & Köllner, in press, for an overview). These coding systems are causally valid, since resulting scores reflect experimental manipulations of the measured construct (cf. Borsboom, Mellenbergh, & van Heerden, 2004). Scores are summed within every motive domain to yield overall motive scores for each participant and are typically corrected for overall PSE story length (see Schultheiss & Pang, 2007).

### **Predictive validity of the implicit need for power**

Implicit motives possess high predictive validity. For example, *nPower* is linked to various individual (e.g. proximal: behavioral expression of preference for signals of submission, Stoeckart, Strick, Bijleveld, & Aarts, 2017; distal: managerial success, McClelland & Boyatzis, 1982), biological (e.g., sympathetic activation; McClelland, 1982), and societal (e.g., engagement in wars; see Schultheiss, 2008, for an overview) criteria.

A recurrent moderator of *nPower*'s associations with various criteria is *activity inhibition* (AI; McClelland & Boyatzis, 1982; Schultheiss & Brunstein, 2002; see Schultheiss, 2008). AI is measured via PSE (counting the frequency of the negation "not"; Langens, 2010) and considered a marker for greater right-hemispheric and lesser left-hemispheric engagement during stress (Schultheiss, Riebel, & Jones, 2009). High-AI individuals appear to have better access to functions associated with the right-hemisphere, such as emotion-encoding and -decoding competencies (Schultheiss et al., 2009). Thus, high *nPower* paired with high AI, the *inhibited power motive* (IPM), makes individuals particularly successful in the social arena (Schultheiss & Brunstein, 2002). This may be due to their power behavior being more context-sensitive and sophisticated (Schultheiss & Köllner, in press), as can be observed for example in a proficiency in persuasive communication as indicated by factors like gesturing or speaking fluently (Schultheiss & Brunstein, 2002).

An explanation for *nPower*'s ties to a wide range of behaviors is its affect-amplifying function (see Schultheiss & Köllner, 2014). Altering the reinforcer value of specific stimuli by enhancing affective responses to them, *nPower* can have scaling effects on stimulus-driven learning processes (Schultheiss & Köllner, 2014). This facilitates the acquisition of behaviors allowing an individual to exert influence on others, like persuasive communication (Schultheiss & Brunstein, 2002).

Another way for *nPower* to influence behavior is by interacting with endocrine systems in situational contexts that evoke dominance arousal or stress (Schultheiss, 2013; Stanton & Schultheiss, 2007, 2009). *nPower* is tied to activational effects of the steroid hormones testosterone (T) in men and estradiol (E2) in women (Schultheiss, 2013). *Activational hormone effects* are reversible hormone concentration changes promoting the display of certain behaviors (Sisk & Zehr, 2005). For example, T-increases after losing a dominance contest predict an increased inclination among men to engage in another contest, whereas T-decreases predict withdrawal from further competition (Mehta & Josephs, 2006). Although extensively researched, increases in T after winning a dominance contest and decreases in T after losing a contest cannot be found consistently in human samples (see Carré & Olmstead, 2015, for a review), with the overall effect being highly heterogeneous and the effect in lab-settings being restricted to men and very weak (see Geniole, Bird, Ruddick, & Carré, 2017, for a meta-analysis). However, when *nPower* is considered, the expected effects are found in high-power men (Schultheiss, 2013). *nPower* is associated with T-changes after dominance contests in a direction depending on their outcome: high-power, but not low-power, men show T increases after winning and decreases in T after losing (e.g., Schultheiss, Wirth, et al., 2005; Vongas & Al Hajj, 2017). These effects are assumed to be (partly) mediated by interactions of *nPower* with other hormonal parameters, such as epinephrine and norepinephrine release after winning (stimulating effect on testes) and cortisol release after losing a dominance contest (inhibiting effect on testes; Schultheiss, 2013; Stanton &

Schultheiss, 2009; cf. Sapolsky, 1987). In women, *nPower* predicts E2-changes in reaction to a dominance contest in the same pattern as it predicts T-changes in men: High-power, but not low-power, women show E2 increases after winning and decreases in E2 after losing (Stanton & Schultheiss, 2007). A recent study again found that among normally-cycling women high, but not low, in *nPower*, winners had higher post-contest E2 levels than losers (Oxford, Tiedtke, Ossmann, Özbe, & Schultheiss, 2017). Thus, E2 seems to play a similar role in women as T plays in men regarding dominance behaviors and *nPower* (Schultheiss, 2013).

Despite *nPower*'s broad predictive validity, not much research was devoted to its development until recently. Central among past research is a longitudinal study identifying associations of specific early child-rearing practices reported by mothers when their children were five years of age with specific adult implicit motive levels of these children at the age of 31 to 32 (McClelland & Pilon, 1983). For example, severity of toilet training and scheduling of feeding was associated with later *nAchievement* and possibly lacking maternal responsiveness for the child's crying with later *nAffiliation*. Most importantly, parental permissiveness for aggressive and sexual behaviors was linked to childrens' adult *nPower*. At first glance, one might assume that these parenting practices may be the roots of adult implicit motive levels. However, taking *nPower* as an example, this raises the question where those aggressive and sexual behaviors and their underlying inter-individual differences to which parents react to originate from in the first place. Indeed, recent findings hint at even earlier influences on motive development: OHEs (Schultheiss, 2017; Schultheiss & Zimni, 2015).

### **Implicit motives and organizational hormone effects**

#### **Organizational hormone effects and the marker hypothesis**

OHEs differ from activational hormone effects, as the former entail lasting changes to an organism's nervous system structure and shape, especially during important developmental stages (Schultheiss, Schiepe, & Rawolle, 2012; Sisk & Zehr, 2005). Prenatal (Phoenix, Goy, Gerall, & Young, 1959) and pubertal (Sisk & Zehr, 2005) OHEs are particularly relevant for

brain organization and adult behavior. In the following, we refer to prenatal and pubertal “stages”, but only for ease of description: They may not be discrete events, but part of protracted continuing development until adulthood (cf. Schulz, Molenda-Figueira, & Sisk, 2009). While the brain’s sensitivity for OHEs decreases across the protracted postnatal period, increased hormone exposure during these stages renders them critical for development (see model in Schulz et al., 2009).

The brain, especially the hypothalamus, is the origin of hormonal and autonomic nervous system processes involved in motivation (see Schultheiss, 2013, for an overview). As the brain is also the target of the above-mentioned OHEs during development, it is plausible that individual differences in the functioning of the adult motivational brain are related to individual differences in earlier exposure to OHEs. But how can we retrospectively assess OHEs?

Traces of exposure to OHEs are reflected in morphological *markers*, as OHEs also effect bodily changes. Such correlated changes between an organism’s nervous system function and body morphology, triggered by the same organizational hormones, should allow the approximation of OHEs on the developing brain and on brain-dependent motivational processes via body markers (Hönekopp, Bartholdt, Beier, & Liebert, 2007). This renders markers valuable information sources, as experimental manipulation of hormone levels in humans during development is impossible due to ethical reasons.

The ability of markers to reflect differences in OHE exposure during development is underscored by the fact that they are often sex-dimorphic, reflecting sex differences in hormone secretion during development (Ober, Loisel, & Gilad, 2008). For example, sexual dimorphisms were observed for the *second-to-fourth-digit length ratio* (2D:4D), with men featuring smaller ratios than women (e.g., Hönekopp et al., 2007; Zheng & Cohn, 2011). Also, the *facial width-to-height ratio* (fWHR), the bizygomatic width (distance between left and right zygion) divided by upper face height (distance between nasion and prosthion; e.g.

Zilioli et al., 2015; cf. Weston, Friday, & Liò, 2007), is overall larger for men compared to women (see meta-analyses by Geniole, Denson, Dixson, Carré, & McCormick, 2015; Kramer, 2017, but see also Kramer's follow-up-analyses within the same paper).

We argue that it may be fruitful to investigate possible connections between OHEs, reflected in morphological markers, and implicit motive development: First, hormones influence motivational development (e.g., the emergence of new motivational tendencies during puberty; Forbes & Dahl, 2010). Second, organizational hormones and social experiences may interact in shaping for example assertive behaviors (Schulz et al., 2009). Consequently, the above-mentioned parenting practices (McClelland & Pilon, 1983) may not constitute the actual origins of motives, but a social-learning-based modulation of a biological basis previously established by OHEs. Actually, this is very likely, as the evidence from marker studies involving 2D:4D and fWHR shows.

#### **Prenatal hormone effects and implicit motive development: The case of 2D:4D**

Evidence for 2D:4D's marker function for prenatal OHEs comes from several observations: For example, the length of the fourth digit can be experimentally increased by prenatal T and reduced by prenatal estrogen in mice (Zheng & Cohn, 2011). In addition, in humans, the sexual dimorphism in 2D:4D is already present after the first trimester of pregnancy (Malas, Dogan, Evcil, & Desdicioglu, 2006) and remains stable afterwards. Furthermore, 2D:4D is largely unrelated to adult sex hormone levels (Hönekopp et al., 2007).

There are several ways of measuring 2D:4D, including anthropometry and palm scans/photocopies (Kemper & Schwerdtfeger, 2009). Also, 2D:4D has various correlates: For example, a low 2D:4D is substantially related to athletic prowess (Hönekopp & Schuster, 2010). In the case of aggression, while the meta-analytically derived effect sizes are far too small to be considered practically meaningful, a minuscule overall association of 2D:4D with aggression and violent behavior across both genders emerged (Turanovic, Pratt, & Piquero, 2017).

**2D:4D and implicit motives.** A link between motives and 2D:4D was repeatedly found. Currently, two published 2D:4D-samples suggest prenatal OHEs on *nPower* development: Schultheiss and Zimni (2015) observed an association between a more “male-typical” smaller 2D:4D, assessed with a ruler on photocopies of the hands, and PSE-*nPower* in the context of high AI (see Figure XX.1). This moderating effect of AI as an indicator of brain lateralization is not surprising considering the IPMs’ above-mentioned relevance.

<FIGURE XX.1 HERE>

In another study featuring the PSE and hand scans, Schultheiss (2017) again found that a more “male-typical” digit ratio of the right hand was associated with simultaneously high *nPower* and high AI, directly replicating Schultheiss and Zimni (2015). In addition, there were sex-dimorphic relationships between digit ratio (right hand) and *nPower*: Women, but not men, with a higher *nPower* also had more female-typical higher 2D:4D.

Recently, O. C. Schultheiss (personal communication, October 19/20, 2017), corroborated this link in a large aggregation featuring more than 400 participants and including three individual studies, among them the sample of Schultheiss (2017). A sex-dimorphic relationship reflected in a Sex x *nPower* x AI interaction emerged for the asymmetry (left versus right) of 2D:4D, with a stronger *nPower* x AI effect in women. Among women, *nPower* correlated differently with 2D:4D-asymmetry for high-inhibition versus low-inhibition participants. In sum, OHEs seem to contribute to the development of a joint motivational syndrome involving *nPower* and AI, probably in a sex-dimorphic way.

#### **Pubertal hormone effects and implicit motive development: The case of fWHR**

fWHR can be considered a marker of OHEs on the pubertal brain: It is a craniofacial feature of skulls that may develop sex-dimorphically after the onset of puberty (Weston et al., 2007). While steroid hormones influence human craniofacial growth (Verdonck, Gaethofs, Carels, & de Zegher, 1999), fWHR is associated with pubertal T when controlling for age (Welker, Bird, & Arnocky, 2016).

fWHR is measured for example via anthropometry or from photos (see Geniole et al., 2015, Supplementary). It has correlates like aggressive behavior in men (Carré & McCormick, 2008) or fighting ability in professional competitions (Zilioli et al., 2015). Meta-analyses revealed small associations between fWHR and male aggression (Haselhuhn, Ormiston, & Wong, 2015), as well as male threat behavior and dominance behavior across both sexes (Geniole et al., 2015). Geniole et al. (2015) concluded that fWHR may be part of an evolved intra-sexual signal system for dominance among men, but their relationship between fWHR and dominance behavior also included *female* participants. The latter finding is remarkable, especially as Haselhuhn et al. (2015) had a priori excluded women.

**fWHR and implicit motives.** fWHR's correlates suggest a possible link to *nPower*. Thus, we (Janson et al., 2018) tested this relationship using anthropometry and a six-picture PSE in two samples, later combining them for adequate statistical power. Controlling for BMI and age, which both influenced fWHR, we found a pattern much like the one observed by Schultheiss and Zimni (2015), however reversed, as fWHR is positively associated with dominance, not negatively like 2D:4D. The expected *nPower* x AI interaction on fWHR-scores emerged, with the IPM tending to predict high, and a disinhibited *nPower* tending to predict low, fWHR. However, when considering sex, this pattern only held for women (see Figure XX.2).

<FIGURE XX.2 HERE>

This women-specific finding seems to be at odds with fWHR's supposed status as an evolved male within-sex threat and dominance cue informative for the intra-sexual competition among men (Geniole et al., 2015). However, we offer an alternative explanation: First, the expected *nPower* x AI interaction was lower-order than the unexpected moderation and may be easier to replicate. Thus, the moderation by sex needs replication before it is taken seriously. Second, *nPower* does not deal with threat specifically, but more broadly with having influence on others as a precursor of attaining dominance (cf. Schultheiss & Köllner,

in press). Thus, reducing *n*Power to threat or aggression (in men) is inappropriate. Third, dominance behavior, a more appropriate construct when dealing with *n*Power in general, was associated with fWHR *across both sexes* meta-analytically (Geniole et al., 2015). Fourth, E2, a hormone with ties to *n*Power in women, may influence (facial) bone growth, too. Therefore, reducing fWHR to a marker of pubertal T only may be an oversimplification (see Janson et al., 2018, for details). We conclude that our basic *n*Power x AI interaction indicates that pubertal OHEs influence or refine the adult *n*Power in similar ways as prenatal OHEs.

### **The social biopsychology of implicit motive development: A tentative model**

Building on these findings (for an overview of the so-far published studies, please see Table XX.1), we now discuss their implications for the bigger picture of implicit motive development (see Figure XX.3). Combined with evidence for early social influences on motives (e.g., McClelland & Pilon, 1983) they suggest a biological basis (Growth-parts of Figure XX.3) which channels motive development and is later trimmed and fine-tuned by social learning experiences (Prune&Tune-parts; compare Schultheiss & Köllner, in press).

<TABLE XX.1 HERE>

<FIGURE XX.3 HERE>

Prenatal OHEs on motive development (*Growth-I*) were found using 2D:4D as a marker in two published samples and showed up in a large aggregation of existing samples. This represents robust evidence, as consistently (1) *n*Power was associated with 2D:4D when considering (2) AI as a moderator.

How can we explain such marker-motive-relationships? A possible brain basis of *n*Power may be found in a network of subcortical nuclei centered on the anterior hypothalamus, as the latter seems to be important for aggressive dominance in animal studies mediated by E2 and T (Schultheiss, 2013; cf. Nelson & Trainor, 2007). Though human *n*Power as a broad disposition should not necessarily be equated with aggressive dominance in animals, prenatal hormone effects targeting this network may lead to lasting variations in

human *nPower*. These variations may then for example entail inter-individual differences in the aggressive behaviors to which parental practices were found to react to in the study by McClelland and Pilon (1983).

The frequent moderation of the results by AI as a marker for functional hemispheric asymmetry during stress indicates that *inhibited nPower/IPM* specifically is tied to prenatal OHEs. The pervasive moderation pattern may even suggest that high AI at least partly results from lateralized brain development due to early endocrine influences: Prenatal T disadvantages the left compared to the right hemisphere during development (Geschwind & Galaburda, 1987). This may explain why the left cortex is thinner in human fetuses and lateralization stronger for male ones (Kivilevitch, Achiron, & Zalel, 2010). It may also explain why male human fetuses have a larger right hemispheric volume and why early sex steroid exposure influences brain asymmetry in rodents, with a thicker right, compared to left, neocortex found in males (see Toga & Thompson, 2003, for an overview). Thus, prenatal T may contribute to functional asymmetries, for example lateralization of prefrontal cortex (PFC) functions. The PFC modulates human dominance behavior, as reduced PFC-activation seems to be associated with impulsive aggression (cf. Nelson & Trainor, 2007). This may explain why AI is part of the socially effective IPM and a consistent moderator in studies involving motives and markers of OHEs. Also, this may elucidate AI's continued relevance as a moderator when examining pubertal OHEs on motives: Primate studies show dopaminergic and other circuitry changes in the PFC during adolescence, changing cortical connectivity, and almost all such adolescent brain-remodeling mechanisms can be influenced by hormones (Sisk & Zehr, 2005).

The fact that the so-far obtained findings were repeatedly sex-dimorphic (Schultheiss, 2017; O.C. Schultheiss, personal communication, October 19/20, 2017) is another noteworthy aspect of the result patterns. This brings to mind other sex-dimorphic 2D:4D-associations, for example in the BBC Internet Study (Manning & Fink, 2008) where 2D:4D was positively

related to family size and reproductive success for women, but negatively for men. Also, sex-dimorphic findings in the domain of motive development are not surprising, considering what is known about activational hormone effects of the male (T) and female (E2) “dominance hormone”, respectively (Schultheiss, 2013): A “male-typical” 2D:4D should represent high prenatal T-to-E2 ratio, whereas a “female-typical” 2D:4D should represent low T-to-E2 ratio (Lutchmaya, Baron-Cohen, Raggatt, Knickmeyer, & Manning, 2004; but see the partial-at-best replication by Ventura, Gomes, Pita, Neto, & Taylor, 2013).

As a side note, one study also reports some genetic influences on implicit motive strength, predominantly for *n*Affiliation, and on behavioral motive expression for all three motives (Hagemeyer & Kandler, 2014, September). Thus, we include genes in our model, but without drawing far-reaching conclusions based on a single study so far.

Regarding social learning experiences in early childhood (*Prune&Tune-I*), we assume a trimming and shaping of (1) a motive’s strength, previously influenced by prenatal OHEs, and of (2) a motive’s socialization level, especially by the parents, respectively. Schultheiss and Köllner (in press) suggest that the biological basis of power motivation needs further refinement for developing socially acceptable forms, for example via contextual factors or parenting practices. This fits well with the associations of specific early parenting practices with specific adult motive levels (McClelland & Pilon, 1983).

Regarding the psychological mechanism behind this social learning, besides pruning (Schultheiss & Köllner, in press) in the sense of a necessary curtailing of the unrefined power impulses of children (and to some extent also of adolescents, see below), we believe that there is also a “tuning” aspect associated with social interactions: Interactions with the environment set the stage for Pavlovian (learning predictive cues, mediated by the amygdala) and instrumental conditioning processes (via reinforcement of successful behavior leading to incentive contact, mediated by the striatum). The effectiveness of these conditioning processes depends on the incentives reaped from those interactions via a hedonic response,

which in turn depends on the strength of a given implicit motive (see Schultheiss & Köllner, 2014, for the pathways in which motives influence learning, their neuronal underpinnings, and an integrative skill-building model), to which prenatal hormones contribute to in the first place.

Thus, if a child displaying behaviors like screaming or aggressive acts is unsuccessful in attracting attention or attaining influence on parental behavior or even gets punished, such outcomes will be much more aversive if the child is high in *n*Power and the child will try to avoid such unpleasing experiences in the future, inhibiting the behavior (pruning). Searching for alternative impact strategies, it will learn that predictive contextual cues in the appropriate situational context (for which the hippocampus mediates episodic memory that is also modulated by motive-dependent affective responses, see Schultheiss & Köllner, 2014) need to be considered and try more refined behaviors and more contextualized strategies. This way, the child will be rewarded with the desired influence on parental or peer behavior and is conditioned to use such refined behavior in future social interactions (tuning). Such shaping processes are especially likely for the IPM, to which most of the above-mentioned results regarding OHEs pertain: The results of IPM-individuals' highly refined skill-building are later observable in their behavior (persuasive speech, e.g. gesturing, speaking fluently) and life outcomes in management or voluntary office holding (cf. Schultheiss & Brunstein, 2002, for an overview). Motive-dependent skill building in turn may again be influenced by hormones: Effects of *n*Power on implicit learning phenomena may be mediated by steroid hormones under some conditions (Schultheiss & Rohde, 2002; Schultheiss, Wirth, et al., 2005) and hormones can affect all stages of learning (see Nelson & Kriegsfeld, 2017, for an overview).

As stated above, we consider early childhood factors not as the origins of motives, but as pruning and tuning of their prenatal biological basis (cf. Schultheiss & Köllner, in press). For example, regarding *n*Power development, parents can only be permissive towards aggressive impulses if such impulses are displayed – if not, mothers cannot accurately report

their degree of permissiveness (cf. McClelland & Pilon, 1983, for problems related to linking adult traits with early child-rearing practices). In sum, the initial strength of such impulses in interaction with the degree to which parents scale back aggressiveness or channel it into more socially accepted forms of self-assertion should determine the first model-half's outcome.

Pubertal OHEs on implicit motive development (*Growth-II*) are suggested by our fWHR-study (Janson et al., 2018). The findings resemble those of Schultheiss and Zimni (2015) for 2D:4D, suggesting that prenatal and pubertal hormonal effects on motive development may be similar. Based on our results, a connection between pubertal OHEs and adult motive levels is very likely, but further research must corroborate this conclusion, which currently rests on a single, albeit large, two-sample study.

There is currently no research regarding adolescent social learning experiences (*Prune&Tune-II*). However, adolescent social pruning and fine-tuning of the outcomes of pubertal OHEs on motive development is plausible, as social experiences modulate OHEs on behavior during adolescence (Schulz et al., 2009). McClelland and Pilon (1983) state that their early-childhood-correlates only explain 10-30% of the variance and that later experiences during school or adulthood may contribute to specific motive levels. The role of peers during adolescence may be particularly important here, considering the heightened sensitivity to peer acceptance or rejection and an increased affective impact of peer interactions due to their rewarding nature during adolescence (see Kilford, Garrett, & Blakemore, 2016, for a review). This rewarding nature may create the incentives for similar Pavlovian and instrumental conditioning phenomena like those we assume for early childhood: Further refinement of impact strategies that were successful or socially accepted during childhood is necessary to arrive at the highly refined contextualized behavioral patterns underlying successful dominance in adult life. Peer interactions may be the testing ground for the development of such patterns. Also, if dominance is about gaining access to resources and mates (cf. Schultheiss, Pang, Torges, Wirth, & Treynor, 2005), the latter aspect

should only become relevant after becoming able to sexually reproduce after the onset of puberty, demanding corresponding refinement of *nPower* as a precursor of attaining intra-sex dominance and thus becoming attractive to the other sex (intrasexual selection; see Wilson, 1980, p. 159). However, these deliberations are speculative until direct evidence is available.

Our model does not deny other possible influences on adult *nPower* expression. For example, having younger siblings, and later having children, seems to channel *nPower* expression away from impulsive and towards more responsible behaviors (Winter, 1988). In addition, social environmental factors assessed at the age of five may be associated with later IPM specifically: For boys, primary involvement of the mother versus of the father in child-rearing seems to be associated with a disinhibited *nPower*, high *nPower* (higher than *nAffiliation*) combined with low AI, versus the “imperial-power-motive syndrome”, high *nPower* (higher than *nAffiliation*) combined with high AI, respectively (McClelland & Pilon, 1983, p. 571). Moreover, our focus on critical developmental stages does not preclude the possibility of motive changes later in life, as brain development continues long after the end of adolescence (Pujol, Vendrell, Junqué, Martí-Vilalta, & Capdevila, 1993).

Finally, further research considering (markers of) OHEs when investigating motives and activational hormone effects is particularly promising, as the adult strength of motive-related activational hormone effects may depend on the outcomes of the two organizational stages (cf. Janson et al., 2018). Besides those mentioned above, there are many other links between motives and hormones, for example between *nAffiliation* and progesterone, and specific motives fluctuate across the menstrual cycle, where changes in hormone concentrations occur (Schultheiss, Dargel, & Rohde, 2003). Also, motive arousal with motive-relevant movies (e.g., the Godfather II for *nPower*) is associated with changing hormone levels (e.g., elevated T in high-T men; Schultheiss, Wirth, & Stanton, 2004). Checking if the strength of such motive-hormone-links in adult life depends on the outcomes of brain organization during development may lead to a better understanding of the neural

underpinnings of implicit motives and ultimately to an endocrinological life-span model of implicit motives (cf. Köllner & Janson, 2017)

### **Limitations and further research**

Some general limitations regarding marker research are beyond this chapter's scope, like statistical problems with ratio scores like 2D:4D and fWHR (Cohen & Cohen, 1983) and the potential for relevant effects being obscured by other factors influencing marker scores, as has been shown in the case of BMI affecting fWHR (Lefevre et al., 2012). However, these problems are addressed to some extent in our own research findings, as Janson et al. (2018) report their fWHR-results controlling for age and BMI. Nevertheless, the relationships we obtain from less-than-optimal markers like 2D:4D measured on the body surface may attenuate and thus underestimate the relationship between *nPower* and OHEs: X-ray and photocopy-assessment of 2D:4D produces a correlation of only .45 (136 individuals; Manning, 2002, p. 3&4) and finger fat may also affect 2D:4D measurement (Wallen, 2009).

Regarding chapter-specific limitations, there is a general lamentable dearth of research on implicit motive development over the life course and the factors that have the most impact on it. As a consequence, our model has blind spots: There is no research regarding social learning experiences in adolescence (Prune&Tune-II), for example. In addition, the model rests heavily on *nPower*-related findings, especially regarding prenatal and pubertal OHEs on motive development (Growth-I/II). Nevertheless, there are some cues for an association between prenatal OHEs (Growth-I) and the development of *nAffiliation* (Schultheiss & Zimni, 2015;  $r = .17$ , "female-typical" 2D:4D non-significantly related to *nAffiliation*) and findings for social learning experiences in early childhood (Prune&Tune-I) regarding *nAffiliation* and *nAchievement* (McClelland & Pilon, 1983). Consequently, we believe that the model holds for motives in general, but generalizations at this point remain speculative.

### **Future research**

Thus, future research is essential, like testing other motives' associations with markers or our model's utility to test predictions. Such research should include additional markers potentially reflecting the influence of steroids, allowing us to map OHEs on implicit motive development onto a comprehensive marker framework.

**Linear bone growth.** One such possible marker is long bone length: While *nPower* is associated with E2 in women and T in men, estrogens and androgens are also associated with organizational influences on linear bone growth (see Juul, 2001, for an overview; cf. Cutler, 1997). Estrogens have a biphasic effect on linear bone growth in puberty. Low levels mediate the pubertal growth spurt and increase long bone growth velocity, whereas high levels stimulate epiphyseal closure and cessation of linear bone growth (Cutler, 1997; Juul, 2001). T, the main representative of androgens, is highly connected to E2, an estrogen. It can be converted into E2 by aromatization and thereby exert similar effects on bone growth as estrogens (Vanderschueren et al., 2004).

The relevance of estrogens (or T-converted-to-E2) during puberty renders long bone length a potential marker for OHEs of E2 and T (cf. Bleck, Fenkl, Jägel, & Köllner, 2016, October). We thus expected the length of long bones like ulna and fibula to be negatively associated with *nPower*, because high levels of E2 and T lead to shorter bones and higher *nPower*. As the 2D:4D-findings by O. C. Schultheiss (personal communication, October 19/20, 2017) demonstrate sex-dimorphic patterns moderated by AI for the left-right-asymmetry of a bone measure, we tested our prediction with bone (ulna vs. fibula), side (left vs. right), sex, *nPower*, and AI as predictors while controlling for body height. The overall five-way interaction was significant in our sample including more than 100 participants, possibly indicating similar influencing variables on long bone length and 2D:4D. Among other lower-order effects, two notable patterns emerged:

First, we obtained a Bone x Sex x *nPower* interaction, which persisted when omitting Side and AI in a simplified model. This indicates sex-dimorphic effects on bone length.

Correspondingly, Bone marginally interacted with *n*Power in both sexes: Women with a longer ulna and shorter fibula were higher in *n*Power while for men the pattern was reversed. Building on bone ratios like 2D:4D and as ulna and fibula interacted with *n*Power in a different way, we thus compiled an *ulna-to-fibula ratio* (UFR) by dividing ulna length by fibula length. UFR was highly sex-dimorphic, with higher scores for men compared to women, and predicted by a Sex x *n*Power interaction. As this may hint at a marker function, we will report on UFR's relationship with variables like *n*Power or established markers elsewhere soon.

Second, we obtained a Side x Sex x AI interaction, which marginally persisted when removing Bone and *n*Power from the main model. This result may be tempting regarding speculations on body (Side) and brain (AI) lateralization driven by OHEs. However, in follow-up analyses a Side x AI interaction emerged only in men and additional analyses remained inconclusive.

Nevertheless, it is an interesting pattern that *n*Power interacted with bone type (ulna vs. fibula) while AI interacted with body side in the above-mentioned two sex-dimorphic interactions. Sexual dimorphisms in bone length, with interactions involving AI or *n*Power, respectively, tentatively indicate similar influencing variables on bone length and 2D:4D (cf. O.C. Schultheiss, personal communication, October 19/20, 2017). As this was the first study concerning relationships of *n*Power and AI and long bone length, replication is needed before explaining our findings theoretically.

**Other possible future research.** While the evidence for prenatal OHEs on *n*Power development based on 2D:4D is sound, it should be tested if the findings extend to other possible markers of prenatal hormones like anogenital distance (AGD; Dean & Sharpe, 2013). Also, more published 2D:4D-data would clarify possible connections to *n*Affiliation.

Corroborating research on pubertal OHEs on motive development is needed, especially replication of our fWHR and UFR-findings. Also, other sex-dimorphic

characteristics should be tested, for example facial features like cheekbone prominence or lower face/face height (Lefevre et al., 2012) or waist-to-hip ratio (Cohen-Bendahan, van de Beek, & Berenbaum, 2005).

We currently conduct several studies to broaden the range of markers possibly associated with implicit motives. For instance, we reanalyze pictures of participants taken in two earlier studies for various facial characteristics (e.g., fWHR, cheekbone prominence) to check if pubertal changes in facial morphology can again be linked to adult motive levels. Ongoing studies include photographing participants and assessing 2D:4D via palm scans, using a newly developed software for morphometric hand and face measurements (Köllner, Schmiedl, Waßer, & Schmiedl, 2017).

However, in the long run, we should move away from only looking at less-than-optimal markers of influences on brain development and look directly at the brain itself (cf. Köllner, Janson, & Schultheiss, 2018). Do these morphological body markers, for example, co-vary with actual structural, functional, and connectivity features of the hypothalamus which we implied as a possible brain basis of *nPower*? Exploring anatomical variations in hypothalamic structures as well as their functional connectivities via brain scans and doing functional magnetic resonance imaging (fMRI; Huettel, Song, & McCarthy, 2014) studies assessing their role in processing dominance-related information would be a more straightforward way of pinpointing the brain basis of human *nPower*.

### **Conclusion**

In sum, there is growing evidence from marker research for OHEs on the development of adult implicit motive levels. Several 2D:4D-studies consistently point to prenatal influences on *nPower*. Pubertal OHEs on further development and refinement of *nPower* are also likely, as our fWHR-study suggests. AI consistently emerged as a moderator of these results, suggesting that brain lateralization is an important factor for further theorizing. These promising results warrant further research. We consider it remarkable how much evidence for

a biological basis of motives is obtained by simply looking at proxy measures like biological markers, especially given the minuscule size (Turanovic et al., 2017; meta-analysis on 2D:4D and aggression) or even absence (Kosinski, 2017, fWHR and self-reported behavioral tendencies in more than 137000 participants) of substantial relationships obtained in conventional marker research. The consistent, repeatedly-found patterns extend the success story of the PSE-based alternative approach to conceptualizing and assessing human (dominance) motivation from studies investigating activational hormone effects to OHEs. Finally, the biopsychosocial model of implicit motive development may serve as a framework to test blind spots in motive development, such as social influences during puberty.

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Table XX.1

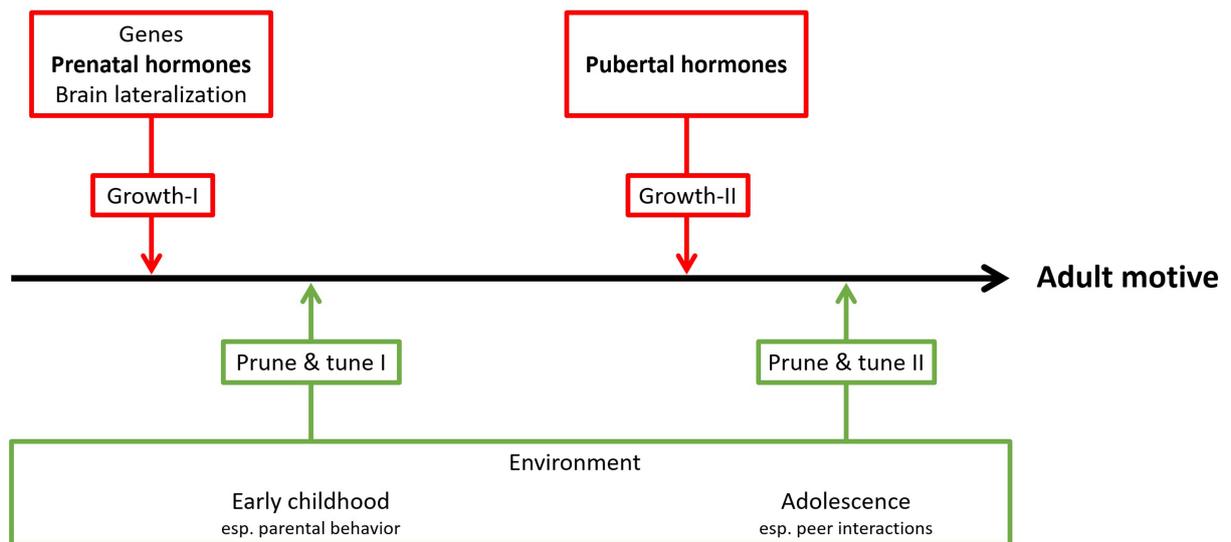
*Overview of the so-far published findings regarding implicit motives and organizational hormone effects*

Study	<i>N</i>	Marker	Main association	Other associations/findings
Schultheiss & Zimni (2015)	50	2D:4D	Low 2D:4D with IPM	-
Schultheiss (2017)	144	2D:4D	Low right 2D:4D with IPM	Higher right 2D:4D with high <i>n</i> Power in women
Janson et al. (2018)	213	fWHR	High fWHR with IPM	Main finding moderated by sex, pattern holds for women only

*Note.* *n*Power = implicit need for power; IPM = inhibited *n*Power; 2D:4D = second-to-fourth-digit length ratio; fWHR = facial width-to-height ratio.

**Note on Figure XX.1.** This figure was a reprint of copyrighted material from another source and thus cannot be made available in this preprint. The original version of the Figure is available in Schultheiss and Zimni (2015, Figure 2; p.398).

**Note on Figure XX.2.** This figure was a reprint of copyrighted material from another source and thus cannot be made available in this preprint. The original versions of the Figures is available in Janson et al. (2018: Figure 1; p.31 – top half of deleted **Figure XX.2** & Figure 3; p.32 – bottom half of deleted **Figure XX.2**).



*Figure XX.3.* Tentative biopsychosocial model of implicit motive development. Prenatal and pubertal organizational hormone effects (Growth-I/II) provide a biological basis for an emerging implicit motive which is later “pruned and tuned” by social learning experiences in interactions with the environment during early childhood or adolescence, respectively (Prune&Tune-I/II). Until now, Prune&Tune-II is based on grounded speculation.