On the Abilities of Unconscious Freudian Motivational Drives to Evoke Conscious Emotions

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Human beings use conscious emotions to direct their behaviors. There is some agreement in the scientific community that unconscious motivations are able to evoke conscious emotions. This manuscript focuses on Freudian motivational drives as inductors for unconscious motivation, and also on Panksepp's framework of affective neuroscience for describing the generation of emotions. Recently, it has been suggested that imperative motor factors of Freudian drives (i.e., the hormones ghrelin, testosterone, angiotensin II and adenosine) have the ability to activate both a drive-specific brain area and brain areas of the SEEKING command system. In fact, this manuscript contends that all imperative motor factors have typical SEEKING targets (i.e., so-called receptors) in the brain areas of both nucleus accumbens and lateral hypothalamus. In addition, all imperative motor factors are able to target the central amygdala directly, a brain area classified by Panksepp as the instinctual part of the FEAR command system. Another point of interest may be the evaluation that imperative motor factors of the sexual drive, hunger and thirst can directly activate the RAGE command system by targeting the medial amygdala. Surprisingly, all imperative motor factors are able to modulate Panksepp’s granddaddy mechanism, i.e., to stimulate all seven command systems via the lateral hypothalamus. Orexinergic neurons exclusively located in the lateral hypothalamus have targets for imperative motor factors and project axons to characteristic brain areas of all seven command systems. From the fact that the imperative motor factors of the sexual drive and hunger act in an excitatory manner on orexinergic neurons whereas those of thirst and sleep inhibit such neurons, temporary termination of hunger by thirst may be understood as a very simple example of a co-regulation of Freudian drives. The author wishes to note that there are motivational drives other than the ones described by Freud. Bowlby was obviously the first in describing such drives, and Bowlbyian drive activities cannot be explained with the intermediacy of imperative motor factors. Nevertheless, the ignorance of the magnificent importance of imperative motor factors must be discarded.

Keywords: SEEKING, affective neuroscience, orexin, Bowlbyian drive, unconscious
INTRODUCTION

At present, any consensus on a single unitary definition of the construct of motivation, derived from the Latin words movere and motivus (Gilbert, 2015; Strombach et al., 2016), is lacking in the psychological community (Gneezy et al., 2011). Panksepp noted that a motivation can be described as a process, “in which a bodily need is subserved by a behavior” in contrast to emotions “where no bodily need is evident” (Panksepp, 1998, p. 228). Thus, motivation can therefore be understood as an active movement of an individual initiated by a stimulus as a driving force. Such a view intrinsically predicts that the activity of a (motivational) drive will evoke a motivation. In the past, various classic psychological drive concepts – Hull’s drive reduction theory (Hull, 1943), Lorenz’s hydraulic conception of drive (Lorenz and Leyhausen, 1973), Tinbergen’s hierarchical organization of circuit nodes (Tinbergen, 1950) and Freud’s theory of motivational drives (Freud, 1905, 1915a,b,c) – were developed in order to explain drive-dependent motivations. It should be noted that (at present) a drive cannot be experimentally distinguished from a corresponding motivation when a (Freudian) drive acts as the driving force for this motivation. Adolphs and Anderson noted “The difference between ’drive’ and ’motivation’ is more of an operational and conceptual one than a biological one.” (Adolphs and Anderson, 2018, p. 148). In the present manuscript the construct of motivation will be nevertheless advocated but the author will distinguish metabolic-deficit-dependent motivations and metabolic-deficit-independent ones. Remarkably, Freud [a man of vast reading (Solomon, 1974)] obviously picked-up the idea of “chemical messengers”, with the first hormone identified in 1902 (Bayliss and Starling, 1902). Freud respected the intermediacy of hormones in his motivational drive theory with its known four elements somatic source, aim, object and imperative motor factor (i.e., hormones) (Freud, 1905, 1915a). Very unfortunately, persistent mistranslation of the German nouns Drang (correctly, in the Freudian sense: imperative motor factor) as “motor factor,” Trieb (correctly: drive) as “instinct” and Triebelbre (correctly in the Freudian sense: theory of motivational drives) as “theory of instincts” have given rise to a variety of misunderstandings, especially in those cases where Freud used the German word ‘Instinkt’ which was also (correctly) translated as instinct. For Freud instincts ('Instinkte') are “inherited mental formations” (Freud, 1915c, p. 3017) whereas drives “represent an instigation to mental activity” (Freud, 1926b, p. 4343; Holder, 1970, pp. 19). In 1923, Freud clarified that two types of Freudian motivational drives are constituent elements of Eros: “According to this view we have distinguish two classes of instincts, one of which, the sexual instinct or Eros, is by far the more conspicuous and accessible to study. It comprises not merely the uninhibited sexual instinct proper and the instinctual impulses of an aim-inhibited or sublimated nature derived from it, but also the self-preservative instinct…. ” (Freud, 1923, p. 3974). Thus, according to Freud, three motivational drives (sexual drive, thirst and hunger) are constituent elements of Eros. In order to answer Freud’s question of “What instincts should we suppose there are, and how many?” (Freud, 1915a, p. 2961), we advocated three criteria for identifying a Freudian motivational drive: an imperative nature of the drive as a psychological criterion, orchestration via the lateral hypothalamus as a neurobiological cachet and a drive termination by means of the central release of 5-hydroxytryptamine as a biochemical attribute (Kirsch and Mertens, 2018). By using these criteria, we identified the sexual drive, thirst, hunger (in line with Freud’s prediction) and sleep as Freudian motivational drives with the corresponding imperative motor factors testosterone, angiotensin II, ghrelin and adenosine. These hormones address the frequently ignored problem of drive specificity – “The ability to process and decide between the drives might be lost if each drive is not also an independent generator. In other words, we have to sustain drive-specificity…” (Wright and Panksepp, 2012, p. 18) – because they can simultaneously activate a drive-specific brain area and typical brain areas that are responsible for seeking of resources (vide infra).

In contrast to motivations, a variety of very detailed theories for describing emotions have been outlined so far, i.e., the Appraisal Theory (Scherer, 2004; Lazarus, 1991), Interoceptive Theories (Damasio, 1999; Craig, 2002; Damasio and Carvalho, 2013), Constructed Emotion Theory (Feldman Barrett, 2017), Theory of Emotion (Rolls, 1999), Higher-Order Theory of Emotion (LeDoux and Brown, 2017) and Emotion Systems (Panksepp, 1998; Panksepp and Biven, 2012). The latter framework should be attractive from the perspective of Freudian motivational drives because of the fact that Panksepp’s theory of affective neuroscience tends to emphasize motor-related representations (i.e., drives) in the development of feelings. Panksepp (Panksepp, 1998) classifies seven different types of command systems that may (but do not necessarily have to) evoke special behaviors, e.g., seeking for rewards/resources/sexual partners, lust, caring and affection, loss and panic, rage, fear and play. Special subcortical regions of the brain are involved with the processing to the corresponding conscious emotions,1,2 which are classified as so-called command systems (labeled SEEKING, RAGE, FEAR, LUST, CARE, PANIC, and PLAY) (Panksepp, 1998, 2012; Watt and Panksepp, 2009; Zellner et al., 2011; Solms and Panksepp, 2012; Wright and Panksepp, 2012; Panksepp and Yovel, 2014; Alcaro et al., 2017). Since an activated SEEKING system constantly blends well with all the other command systems by co-regulating them (Wright and Panksepp, 2012), the generation of SEEKING activities (according to Panksepp the SEEKING system is “the ‘graddaddy’ of all the emotional system.” (Panksepp and Biven, 2012, p. 86), are of central importance in the development of conscious emotions.

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1The description of the development of consciousness actually represents a multifaceted problem (e.g., Solms, 1997, 2019; Dehaene et al., 2006). In the present manuscript the author follows the view that core brainstem consciousness is the primary type of consciousness, i.e., so-called affective consciousness (Panksepp, 1998; Damasio, 2018). In any case (affective consciousness and cortical ones), consciously experienced emotions emerged from preconscious processing (Panksepp, 1998, p.34). The Global Neuronal Workspace Hypothesis can distinguish between subliminal, preconscious and conscious processing (Dehaene et al., 2006). Accordingly, a preconscious stimulus that is stored in a so-called temporary preconscious buffer, might achieve conscious state once the central workspace is released to the stimulus.

2A group of modern psychoanalysts has made a distinction between self-conscious emotions and basic conscious emotions. Only the latter type is imbedded in biological determined action tendencies and can therefore be described using Panksepp’s Emotion Systems (Tracy et al., 2007; Schalkwijk, 2018).
Recently, we were able to contend the unexpected possibility that the SEEKING system can be activated by the intermediary action of Freudian imperative motor factors (Kirsch and Mertens, 2018). Since Freud explicitly noted that a motivational drive has an unconscious nature: “I am in fact of the opinion that the antithesis of conscious and unconscious is not applicable to instincts. An instinct can never become an object of consciousness – only the idea that represents the instinct can.” (Freud, 1915c, p. 3000) the question arose how an unconscious stimulus can evoke a conscious sensation? From findings of our last work it is possible to expand a fine idea introduced by Panksepp et al. (Table 1).

Thus, according to Panksepp, the intermediacy of neuromodulators and/or classical neurotransmitters represents a mandatory premise for consciousness. Since it has been mentioned that there are qualitative differences of the nervous system between conscious and unconscious processes (Brakel and Shervin, 2005) and because Freudian drives have an unconscious nature (vide supra) and uses hormones as signal transporters, the unconscious action of the Freudian drives can be determined biochemically at the level of signaling codes (Table 1). For example, an imperative motor factor generated outside the brain [e.g., stomach derived release of ghrelin (Kojima et al., 1999; Stevenard et al., 2017)] cannot be transformed to a neurotransmitter/neuromodulator (i.e., cannot be transformed to signals necessary to achieve consciousness) as long as it circulates in the periphery. After passing the blood-brain barrier, the imperative motor factor can now induce the release of neurotransmitters/neuromodulators by occupying its hormone receptors on various pre-synapses in subcortical brain areas. This release of neuromodulators or neurotransmitters represents – from the perspective of the Global Neuronal Workspace Hypothesis (Dehaene et al., 2006) – the provision of preconscious stimuli that can (but do not necessarily have to) gain access to conscious processing.

Unfortunately, the abilities of imperative motor factors to address the subcortical brain in such a manner, was only deconvolved for the brain areas of the SEEKING system (Kirsch and Mertens, 2018). This option raises the question of whether imperative motor factors have the ability to modulate activities of other command systems, and – if so – the underlying mechanisms would be of interest. This manuscript will report on such totally underestimated capabilities, although they are quite well evaluated.

**DIRECT MECHANISMS FOR THE GENERATION OF COMMAND SYSTEM ACTIVITIES**

The idea of making a direct connection between Freudian drives via their imperative motor factors and Panksepp’s emotional command systems intrinsically required one to locate targets (i.e., so-called receptors) of these hormones at the brain areas of interest (Table 2).

As expected from evaluations of our earlier manuscript (Kirsch and Mertens, 2018), all imperative motor factors can directly generate SEEKING activities because, in addition to targets in their drive specific brain areas, they all also have anchorage grounds in both the lateral hypothalamus and the nucleus accumbens (Table 2). The activation of neurons in the latter area and also in the ventral tegmental area can result in the release of the catecholamine dopamine (Naleid et al., 2005; Abizaid et al., 2006; Jerlhag et al., 2007), and that neuromodulator is a key intermediate in the activation of the SEEKING system (Panksepp, 1998; Panksepp and Biven, 2012; Watt, 2017).

Somewhat surprising was the evaluation that all imperative motor factors have receptors in the central amygdala, a brain area that was classified as a part of the FEAR command system (Panksepp, 1998; Panksepp and Biven, 2012; Watt, 2017). Thus, all imperative motor factors have direct access to both the SEEKING and the FEAR command systems. Panksepp noted that a variety of chemical messengers can activate the FEAR system (Panksepp and Biven, 2012), with the result that the view that imperative motor factors can provide a similar activation

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**TABLE 1 | Types of chemical messenger codes.**

| Type of Codes | Unconscious | Conscious |
|---------------|-------------|-----------|
| Processing    | Freudian Drive | AFFECTIVE<sup>a</sup> | COGNITIVE<sup>a</sup> |
| Signaling code | Hormone codes | Neurotransmitter codes<sup>a</sup> | Neuromodulator codes<sup>a</sup> |

<sup>a</sup>According to (Panksepp and Biven, 2012, p. 8).
is not in conflict with the theory of affective neuroscience. In addition, Panksepp distinguished between conditional FEAR and unconditional ones, stating “Therefore, while the central nucleus of the amygdala is part of the unconditional (instinctual) FEAR system, the other nuclei are not.” (Panksepp and Biven, 2012, p. 196). Therefore, the action of the imperative motor factors on the INSTINCTUAL FEAR system may represent a phylogenetic old mechanism. Of course, as most Freud followers would expect, Freud was aware of a link between his motivational drives and fearful emotions: “So far we have had no occasion to regard realistic anxiety in any different light from neurotic anxiety. We know what the distinction is. A real danger is a danger which threatens a person from an external object, and a neurotic danger is one which threatens him from an instinctual demand.” (Freud, 1926a, p. 4319). The fact that imperative motor factors have direct access to the FEAR command system by targeting the central amygdala may also be an interesting finding for psychoanalysts of other schools because Bowlby noted. “No concept is more central to psychoanalytical theory than the concept of anxiety. Yet it is one about which there is little consensus of opinion, which accounts in no small measure for the divisions between different schools of thought. Put briefly, all analysts are agreed that anxiety cannot be explained simply by reference to external threat: in some way processes usually thought of as internal and instinctive seem to play a crucial role. But how these inner forces are to be conceptualized and how they give rise to anxiety has always been a puzzle.” (Bowlby, 1960). In summary, direct access of Freudian drives to the FEAR command system is not in conflict with either the theory of affective neuroscience or historical predictions by leading psychoanalysts.

Of the other command systems, only RAGE can be addressed via the medial amygdala by three imperative motor factors. The failure of adenosine, i.e., the imperative motor factor of sleep, to activate RAGE can be expected because ongoing rage is obviously counterproductive for the onset of sleep, and therefore such neurochemicals that can be elevated during RAGE (e.g., noradrenaline) are decreased during sleep and vice versa (Watson et al., 2010; Pankseep and Biven, 2012).

Although there are (beyond any doubt) direct mechanisms for the generation of command systems activities, their capability is somewhat limited, as imperative motor factors cannot evoke all types of emotion via that mechanism with the same level of efficiency. Therefore, the question arose as to whether Freud’s imperative motor factors can even do more than generate SEEKING, FEAR and RAGE activities by occupying a receptor in a typical brain region of these command systems.

**INDIRECT MECHANISMS FOR THE GENERATION OF COMMAND SYSTEM ACTIVITIES**

Since all Freudian drives are orchestrated via the lateral hypothalamus (vide supra), the precise targets of imperative motor factors in this brain area have been analyzed (Table 3).

This analysis contains two surprises. Firstly, attractive targets for testosterone on orexinergic neurons are lacking in the lateral hypothalamus (Table 3), although androgen receptors have been detected in that area (Simerly et al., 1990). Beside testosterone, its downstream product estradiol – the enzyme aromatase

TABLE 2 | Targets of imperative motor factors on brain areas of command systems.

| Affective prototype and brain areas | Ghrelin | Testosterone | Angiotensin II | Adenosine | Sleep |
|-----------------------------------|--------|--------------|---------------|-----------|-------|
| SEEKING                           |        |              |               |           |       |
| LH                                | Reference 1 | Reference 2 | Reference 3 | Reference 4 |       |
| NAc                               | Reference 5 | Reference 6 | Reference 7 | Reference 8 |       |
| VTA                               | Reference 9 | Reference 10 | –           | Reference 11 |       |
| RAGE                              | Reference 12 | Reference 13 | Reference 14 | –         |       |
| MmA                               | Reference 15 | Reference 16 | Reference 17 | Reference 18 |       |
| LUST                              | Reference 19 | – | – | Reference 20 |       |
| VMH                               | Reference 21 | Reference 22 | – | – |       |
| CoA                               | – | Reference 23 | – | – |       |
| CARE                              | – | Reference 24 | – | – |       |
| PANIC                             | – | – | – | – |       |
| AnT                               | – | – | – | References 11 and 17 | – |
| PLAY                              | CmT, DmT, PT | – | – | – | Reference 25 | – |

*aClassification according to Watt (Watt, 2017), AnT, anterior thalamus; BLA, basolateral amygdala; BNST, bed nucleus of stria terminalis; CoA, central amygdala; CmT, centromedian thalamus; CoA, cortical amygdala; DmT, dorsomedial thalamus; LH, lateral hypothalamus; MeA, medial amygdala; NAc, nucleus accumbens; PT, posterior thalamus; VMH, ventromedial hypothalamus; VTA, ventral tegmental area. Reference 1, (Mitchell et al., 2001; Toshina et al., 2003); Reference 2, down-stream product of testosterone (Shughrue et al., 1997; Muschamp et al., 2007); Reference 3, (Yoshida et al., 2012); Reference 4, (Svenningsson et al., 1997; Thakhar et al., 2002); Reference 5, (Egecioglu et al., 2010); Reference 6, (Cunningham et al., 2012); Reference 7, (Jenkins et al., 1997; Mendelsohn et al., 1984); Reference 8, (Svenningsson et al., 1997; Rosin et al., 2003; Ferre et al., 2007); Reference 9, (Zigman et al., 2006); Reference 10, (Simerly et al., 1990); Reference 11, (Svenningsson et al., 1997); Reference 12, (Alvarez Crespo et al., 2012); Reference 13, (Simerly et al., 1990; Cunningham et al., 2012; He et al., 2013); Reference 14, (Lenkei et al., 1996); Reference 15, (Cruz et al., 2013; Yoshimoto et al., 2017); Reference 16, (Simerly et al., 1990); Reference 17, (Lenkei et al., 1996); Reference 18, (Goodman and Snyder, 1982); Reference 19, (Alvarez-Crespo et al., 2012; Yoshimoto et al., 2017); Reference 20, (Svenningsson et al., 1997; Rau et al., 2014, 2015; Simes et al., 2018); Reference 21, (Zigman et al., 2008); Reference 22, (Simerly et al., 1990; Cunningham et al., 2012; He et al., 2013); Reference 23, (Simerly et al., 1990); Reference 24, (Cunningham et al., 2012; He et al., 2013); Reference 25, (Mendelsohn et al., 1984; Lenkei et al., 1996).
directly oxidizes testosterone into the estrogen derivative estradiol (Fui et al., 2014) – is also important for male sexual behavior (Cunningham et al., 2012), and estrogen receptors on orexinergic neurons are present in the lateral hypothalamus. The second surprise was the realization that receptors of all four imperative motor factors modulate (in an inhibitory or excitatory manner) the release of the neuropeptide orexin (Table 3). In 1998, two research groups independently identified peptides exclusively produced by neurons located in the lateral hypothalamus (de Lecea et al., 1998; Sakurai et al., 1998). Most scientific journals have now accepted the name “orexins” (instead of orexin) and the lateral hypothalamus as a part of this command system (Watt, 2017), is “the ‘granddaddy’ of all the emotional system.” (Panksepp and Biven, 2012, p. 86). Since the author is unaware whether other neurons can act in a similar manner (but, of course, cannot exclude such a possibility with certainty), and because the activity of orexinergic neurons is under the control of Freud’s imperative motor factors (Table 2), it is concluded that the activity of the granddaddy mechanism, i.e., to evoke emotions via generation of SEEKING-dependent command system activities, can be under the control of Freudian motivational drives.8

It is well known that different motivations can co-regulate (conflict or support) each other (Huang and Bargh, 2014; Gilbert, 2015). Such a co-regulation of Freudian drives can be understood at a biomolecular level with the aid of Table 3 because hunger and the sexual drive can stimulate command systems activities by enhancing orexin-dependent networking, whereas thirst and sleep can operate oppositely. The sense of such a possibility with certainty), and because the activity of orexinergic neurons is under the control of Freud’s imperative motor factors (Table 2), it is concluded that the activity of the granddaddy mechanism, i.e., to evoke emotions via generation of SEEKING-dependent command system activities, can be under the control of Freudian motivational drives.8

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8By expanding Panksepp’s analogy of “granddaddy” the writer of this manuscript comes to the end that the granddaddy lies (sometimes) on Freud’s couch.

### Table 3 | Targets for imperative motor factors of hunger, sexual drive, sleep and thirst on orexinergic neurons located in the lateral hypothalamus.

| Receptor/Imperative Motor Factor | Action on OX Neurons | Reference |
|---------------------------------|-----------------------|-----------|
| GHS-R/Ghrelin                   | Excitatory            | Mitchell et al., 2001; Toshina et al., 2003 |
| AR/Testosterone                 | Excluded              | Simerly et al., 1990; Muschkamp et al., 2007 |
| ER/Estrogen                     | Excitatory            | Shughrue et al., 1997; Muschkamp et al., 2007 |
| A1R/Adenosine                   | Inhibitory            | Svenngisson et al., 1997; Thakhar et al., 2002 |
| AT1R/Angiotensin II             | Inhibitory            | Yoshida et al., 2012 |

OX, orexinergic; GHS-R, growth hormone secretagogue receptor; AR, androgen receptor; ER, estrogen receptor; A1R, adenosine receptor type 1; AT1R, angiotensin II receptor type 1.
too simple for describing such a complex psychological entity.\footnote{One reviewer noted: “In Freudian thinking, repression due to conflict happens because the action associated with the drive is threatening to the ego, which must refer to a threat of the ‘self’-organization of the mind, rather than due to a competing demand that is more important.”}
Thus, the importance of the mechanism of drive co-regulation via orexin-mediated networking for psychological entities cannot be classified at present.\footnote{Notably, Freud expanded the construct of ‘repression’ to affective emotions: “In the first place, it may happen that an affective or emotional impulse is perceived but misconstrued. Owing to the repression of its proper representative it has been forced to become connected with another idea, and is now regarded by consciousness as the manifestation of that idea. If we restore the true connection, we call the original affective impulse an ‘unconscious’ one. Yet its affect was never unconscious; all that had happened was that the idea had undergone repression.” (Freud, 1915c, p. 3081). Here, Freud’s standing “regarded by consciousness as the manifestation of that idea” may be explained by a (partly) cortical processing and in such a case the occurrence of ‘repression’ cannot be solely explained by orexin-dependent subcortical networking.}

**LIMITS OF IMPERATIVE MOTOR FACTORS FOR THE GENERATION OF AFFECTIVE NEUROSCIENCE ACTIVITIES**

Although the indirect pathway – i.e., modulation of orexin-dependent networking by targeting the lateral hypothalamus (Table 3) – expands the capabilities of imperative motor factors, they are unable to cover all possible types of motivations and (corresponding) emotions. A Freudian motivational drive is down-regulated by the cerebral release of 5-hydroxytryptamine (vide supra). Therefore, drives and corresponding motivations that would require the intermediacy of 5-hydroxytryptamine for their processing cannot be regarded as being dependent upon Freudian motivational drives. This gives rise to the puzzler: Do such drives really exist, and where are they operating in psychological situations? Most surprisingly, an entirely unexpected answer is offered by Bowlby’s attachment theory (Bowlby, 1960, 1973). The unconscious motivation of an infant to stay in close proximity to its care provider becomes measureable by expanding the distance between mother and infant for significant periods of time, resulting in distress, anxiety and fear in the infant (Bowlby, 1973). Bowlby mentioned that a drive (even suggested by Freud) is responsible for this motivation of the infant: “Our most conservative conclusion is that Freud was not wholly satisfied with his earlier accounts [i.e., theory of motivational drives]. A more radical one is that, toward the end of his life and imbued with a newly-found but vivid appreciation of the central importance of the child’s tie to his mother, Freud was not only moving away from the theory of Secondary Drive [i.e., motivational Freudian drive] but developing the notion that special drives built into the infant in the course of evolution underlie this first and unique love relationship.” (Bowlby, 1958). Of course, Bowlby’s suggestion of classifying a Freudian drive as a Secondary Drive evoked a number of heavy protests from leading Freud followers, but the one proffered by Anna Freud points to a hitherto unrecognized solution: “He [Bowlby] sets up a controversy between the tie to the mother and the action of the pleasure principle in terms of “primary and secondary drive” and criticizes us for reversing their order of importance, i.e., for regarding the tie to the mother as a secondary, the search for pleasure as a primary instinctual urge.” (Freud, 1960). Most remarkably, Anna Freud accepted Bowlby’s view that there are other drives at work as well as the motivational Freudian ones. It should therefore be helpful to classify these non-Freudian drives as Bowlbyian ones\footnote{The dispute concerning the ranking of the drives is presumably futile since both kinds of drives are obviously essential for the survival of the human species. Thus, by accepting the view that the motivation of the child to stay in close proximity to its mother is the result of a Bowlbyian drive activity, a detailed search in literature would be of interest with regard to the possibility of whether 5-hydroxytryptamine is involved in motivations/emotion connected with attachment in general. The observation that a polymorphism of the 5-HT\textsubscript{2A} serotonin receptor gene – this receptor being one important target for 5-hydroxytryptamine in the brain – is connected with the psychological disorder referred to as ‘avoidant attachment’ (Gillath et al., 2008) currently offers the strongest proof that 5-hydroxytryptamine is in fact involved in the processing (and not in the down-regulation\footnote{Provided that 5-hydroxytryptamine acted like in the Freudian drives as a termination signal, a Bowlbyian drive with a defect 5-HT\textsubscript{2A} receptor would be over-stimulated, in contrast to the observation.}) of a Bowlbyian drive. In addition, 5-hydroxytryptamine increases the secretion of oxytocin (Saydoff et al., 1991; Bagdy and Makara, 1994) and this neuromodulator is obviously highly important for the tie between an infant and its mother (Uvnäs Moberg and Prime, 2012; Watt, 2017).}. The dispute concerning the ranking of the drives is presumably futile since both kinds of drives are obviously essential for the survival of the human species. Thus, by accepting the view that the motivation of the child to stay in close proximity to its mother is the result of a Bowlbyian drive activity, a detailed search in literature would be of interest with regard to the possibility of whether 5-hydroxytryptamine is involved in motivations/emotion connected with attachment in general. 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![FIGURE 1 | Proposed action of Freudian drive-dependent generation of emotions.](image-url)
2013). Since 5-hydroxytryptamine down-regulates the activity of a Freudian drive initiated by imperative motor factors and because 5-hydroxytryptamine supports via increase of oxytocin secretion the processing of a Bowlbyian drive, it can be safely concluded that imperative motor factors are not responsible for Bowlbyian drive activities. Of course, the lack of knowledge of how an impaired or down-regulated Bowlbyian drive is able to activate command system activities – according to Panksepp, an impaired attachment activates the PANIC/GRIEF system (Panksepp and Biven, 2012, pp. 312–313) – needs to be evaluated.

**FAILURE OF EMOTIONS TO INITIATE FREUDIAN DRIVE DEPENDENT MOTIVATIONS**

Experimental psychologists demonstrate that both positive emotionally valent stimuli and negative (aversive) ones can successfully enhance (the motivation) ‘attention’ in patients (Dominguez-Borras et al., 2013; Vuilleumier, 2015). From such experimental findings psychologists have concluded that emotions provide guidance for motivations and are linked with them (e.g., Gilbert, 2014). However, the conclusion that an emotion can initiate a motivation has hardly any means of little significance, given the lack of a generally accepted definition of the term ‘motivation’ in psychology. Since an emotion can inform an individual about the existence of a metabolic deficit (but cannot be responsible for such an imbalance), it is concluded that an emotion cannot initiate a Freudian drive dependent motivation. The expressed example, the putative motivation ‘attention’ is obviously independent of the intermediary action of a Freudian drive and would be (as long as any generally accepted definition of the term ‘motivation’ is lacking) tentatively classified as a metabolic deficit independent motivation that might be liberated by an emotion. The next puzzler, namely whether an emotion can provoke a Bowlbyian drive dependent motivation, cannot be answered yet because the precise architecture of this complex drive still needs to be evaluated.

**CONCLUSION**

This manuscript has been written under the assumption that our recently published update (Kirsch and Mertens, 2018) to Freud’s 100-year-old (but essentially accurate) theory of motivational drives needs to be conclusively expanded in order to exploit its full potential. Not just the SEEKING system, but imperative motor factors of all Freudian drives have targets in the *(median amygdala Table 2)*, a brain area that was classified by Panksepp (Panksepp and Biven, 2012, p. 196) as the instinctual part of the FEAR command system. In addition, the imperative motor factors of the sexual drive, hunger and thirst also have targets in the *(medial amygdala Table 2)*, an area of the brain classified as being part of the RAGE command system (Watt, 2017). Thus, besides directly generating SEEKING activities, all imperative motor factors are able to generate FEAR activities, and three of them can also directly stimulate the generation of RAGE activities. In addition, all drives can indirectly modulate all sorts of command system activities by controlling Panksepp’s ‘granddaddy’ of affective neuroscience, i.e., by modulating the activity of orexinergic neurons in the *(lateral hypothalamus Table 3)*. Because of this, the sexual drive and hunger can stimulate affective neurophysiological activities via orexin-mediated networking, whereas sleep and thirst can inhibit such demands. The opposing actions of hunger and thirst were first used to explain the co-regulation of a Freudian drive. In order not to overrate the deconvolved impressive capabilities of Freudian drives, the astute reader needs to note that essential motivational drive activities described by Bowlby, classified here as Bowlbyian drives, cannot be explained by the intermediary of imperative motor factors. The action of imperative motor factors is basically drafted (Figure 1).

The consideration of actual findings on Freud’s theory of motivational drives (evaluated here and in our previous manuscript) leads to the following assertions:

1. Human beings are directed, but not determined, by Freudian drives in an unconscious manner.
2. The satisfaction of a Freudian drive leads to the release of the neurotransmitter 5-hydroxytryptamine in order to down-regulate the drive.
3. The sexual drive, hunger, and thirst are Freudian drives with an imperative character.
4. The imperative motor factor of a Freudian drive is a signal molecule that directly targets *nucleus accumbens*, *lateral hypothalamus, central amygdala* and a drive-specific brain area.
5. The imperative motor factor of a Freudian drive can directly evoke generation of drive-specific SEEKING and INSTINCTUAL FEAR activities.
6. The imperative motor factors of the sexual drive, hunger and thirst also directly target the *medial amygdala*, thereby evoking the generation of RAGE activities.
7. All imperative motor factors are able to modulate, indirectly, the generation of affective neuroscience activities by targeting orexinergic neurons in the *(lateral hypothalamus)*.

In summary, it can be said that the intermediacy of Freudian imperative motor factors can explain convincingly the modulation of command system activities. Accordingly, the concept of Freudian motivational drives is somewhat underestimated even by Freudian psychoanalysts, although Boag recently mentioned the putative importance of the drives for the *id* and the *ego*, respectively (Boag, 2014).

**AUTHOR CONTRIBUTIONS**

The author wrote and designed the manuscript.
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REFERENCES

Ahiziaid, A., Liu, Z. W., Andrews, Z. B., Shanabrough, M., Borok, E., Elsworth, J. D., et al. (2006). Ghrelin modulates the activity and synaptic input organization of midbrain dopamine neurons while promoting appetite. *J. Clin. Invest.* 117, 3229–3239. doi: 10.1172/JCI29867

Adolphs, R., and Anderson, D. (2018). *The Neuroscience of Emotion: A New Synthesis*. Oxford: Princeton University Press.

Alcaro, A., Carta, S., and Panksepp, J. (2017). The affective core of the self: a neuroarchetypal perspective on the foundations of human (and animal) subjectivity. *Front. Psychol.* 8:1424. doi: 10.3389/fpsyg.2017.01424

Alvarez-Crespo, M., Skibicka, K. P., Forkas, I., Molnar, C. S., Egecioglu, E., Hrabosky, E., et al. (2012). The amygdala as a neurobiological target for ghrelin in rats: neuroanatomical, electrophysiological and behavioral evidence. *PLoS One* 7:e64321. doi: 10.1371/journal.pone.0064321

Amaral, D. G., Price, J. L., Pitkanen, A., and Carmichael, S. T. (1992). *Anatomical Organization of the Primate Amygdaloid Complex*. New York, NY: Wiley-Liss.

Andreti, D. H., Hassell, J., Li, H., Achua, J. K., Guarnieri, D. J., Dileone, R. I., et al. (2014). Anxiolytic function of the orexin 2/hypocretin A receptor in the basolateral amygdala. *Psychoendoocrinology* 40, 17–26. doi: 10.1016/j.psyneuen.2013.10.010

Bagdy, G., and Makara, G. B. (1994). Hypothalamic paraventricular nucleus lesions differentially affect serotonin-1A (5-HT1A) and 5-HT2 receptor agonist-induced oxytocin, prolactin and corticosterone responses. *Endocrinology* 134, 1127–1131. doi: 10.1210/endo.134.8.119151

Bayer, L., Eggermann, E., Saint-Meux, B., Machard, D., Jones, B. E., Mühlethaler, M., et al. (2002). Selective action of orexin (hypocretin) on nonspecific thalamocortical projection neurons. *J. Neurosci.* 22, 7835–7839. doi: 10.1523/JNEUROSCI.22-18-07835.2002

Bayliss, W. M., and Starling, E. H. (1902). The mechanism of pancreatic secretion. *J. Physiol.* 28, 325–353. doi: 10.1113/jphysiol.1902.sp000920

Bisetti, A., Cvetkovic, V., Seradjin, M., Bayer, L., Machard, D., Jones, B. E., et al. (2006). Excitatory action of hypocretin/orexin on neurons of the central medial amygdala. *Neuroscience* 142, 999–1004. doi: 10.1016/j.neuroscience.2006.07.018

Boag, S. (2014). Ego, drives, and the dynamics of internal objects. *Front. Psychol.* 5:0666. doi: 10.3389/fpsyg.2014.00666

Borgland, S. L., Storm, E., and Bonci, A. (2010). Ghrelin increases intake of rewarding food in rodents. *Addict. Biol.* 15, 304–311. doi: 10.1111/j.1369-1600.2010.00216.x

Bischof, S. A., et al. (2012). Yohimbine depresses excitatory transmission in BNST patient with hemispatial neglect. *Neuroscience* 201, 429–438. doi: 10.1016/j.neuroscience.2011.01.010

Craig, A. D. (2002). How do you feel? Interoception: the sense of the physiological condition of the body. *Nat. Rev. Neurosci.* 3, 655–666. doi: 10.1038/nn894

Cruzd, M. T., Herman, M. A., Cote, D. M., Ryabinin, A. E., and Roberto, M. (2013). Ghrelin increases GABAergic transmission and interacts with ethanol actions in the rat central nucleus of the amygdala. *Neuropsychopharmacology* 38, 364–375. doi: 10.1038/npp.2012.190

Cunningham, R. L., Lumina, A. R., and McGinnis, Y. (2012). Androgen receptors, sex behavior, and aggression. *Neuromedecine* 96, 131–140. doi: 10.1159/000337663

Damasio, A. (2018). *The Strange Order of Things*. New York, NY: Pantheon.

Damasio, A. R. (1999). *The Feeling of What Happens: Body and Emotion in the Making of Consciousness*. New York, NY: Harcourt Brace.

Damasio, A. R., and Carvalho, G. B. (2013). The nature of feelings: evolutionary and neurobiological origins. *Nat. Rev. Neurosci.* 14, 143–152. doi: 10.1038/nrn3403

Davis, M. (2000). “The role of the amygdala in conditioned and unconditioned fear and anxiety,” in *The Amygdala*, Vol. 2, ed. J. P. Aggleton (Oxford: Oxford University Press), 213–287.

de Leece, L., Kilduff, T. S., Peyron, C., Gao, X., Foye, P. E., Danielson, P. E., et al. (1998). The hypocretins: hypothalamic-specific peptides with neuroexcitatory activity. *Proc. Natl. Acad. Sci. U.S.A.* 95, 322–327. doi: 10.1073/pnas.95.1.322

Dehaene, S., Changeux, J.-P., Naccache, L., Sackur, J., and Sergent, C. (2006). Conscious, preconscious, and subliminal processing: a testable taxonomy. *Trends Cogn. Sci.* 10, 204–211. doi: 10.1016/j.tics.2006.03.007

Dominguez-Borras, J., Armony, J.-L., Maravita, A., Driver, J., and Vuilleumier, P. (2013). Partial recovery of visual extinction by pavlovian conditioning in a patient with hemispatial neglect. *Cortex* 49, 891–898. doi: 10.1016/j.cortex.2012.11.005

Egecioglu, E., Jerthag, E., Salom, N., Skibicka, K. P., Haage, D., Bohloody, Y. M., et al. (2010). Ghrelin increases intake of rewarding food in rodents. *Addict. Biol.* 15, 304–311. doi: 10.1111/j.1369-1600.2010.00216.x

Fanselow, M. S. (1994). Neural organization of the defensive behavior system responsible for fear. *Psychon. Bull. Rev.* 1, 429–438. doi: 10.3758/BF03210947

Feldman Barrett, L. (2017). The theory of constructed emotions: an active inference account of interoception and categorization. *Soc. Cogn. Affect. Neurosci.* 12, 17–23. doi: 10.1093/scan/nsw154

Ferre, S., Diamond, I., Goldberg, S. R., Yao, L., Hourani, S. M. O., Huang, Z. L., et al. (2007). Adenosine A2A receptors in ventral striatum, hypothalamus and nociceptive circuitry. Implications for drug addiction, sleep and pain. *Prog. Neurobiol.* 83, 332–347. doi: 10.1016/j.pneurobio.2007.04.002

Freud, A. (1960). Discussion of Dr. John Bowlby's paper. *Psychoanal. Study Child* 15, 53–62. doi: 10.1080/00797308.1960.11822567

Freud, S. (1905). “Three essays on the theory of sexuality,” in *The Standard Edition of the Complete Psychological Works of Sigmund Freud*, ed. J. Strachey (London: The Hogarth Press), 1457–1552.

Freud, S. (1915a). “Instincts and their vicissitudes,” in *The Standard Edition of the Complete Psychological Works of Sigmund Freud*, ed. J. Strachey (London: The Hogarth Press), 109–140.

Freud, S. (1915b). “Repression,” in *The Standard Edition of the Complete Psychological Works of Sigmund Freud*, ed. J. Strachey (London: The Hogarth Press), 2975–2988.

Freud, S. (1915c). “The unconscious,” in *The Standard Edition of the Complete Psychological Works of Sigmund Freud*, ed. J. Strachey (London: The Hogarth Press), 2989–3024.

Freud, S. (1923). “The Ego and the Id,” in *The Standard Edition of the Complete Psychological Works of Sigmund Freud*, ed. J. Strachey (London: The Hogarth Press), 3946–3992.
Yoshida, T., Semprun-Prieto, L., Wainford, R. D., Sukhanov, S., Kapusta, D. R., and Delafontaine, P. (2012). Angiotensin II reduces food intake by altering orexigenic neuropeptide expression in the mouse hypothalamus. Endocrinology 153, 1411–1420. doi: 10.1210/en.2011-1764

Yoshimoto, K., Nagao, M., Watanabe, Y., Yamaguchi, T., Ueda, S., Kitamura, Y., et al. (2017). Enhanced alcohol-drinking behavior associated with active ghrelinergic and serotonergic neurons in the lateral hypothalamus and amygdala. Pharmacol. Biochem. Behav. 153, 1–11. doi: 10.1016/j.pbb.2016.12.001

Zellner, M. R., Watt, D. F., Solms, M., and Panksepp, J. (2011). Affective neuroscientific and neuropsychoanalytic approaches to two intractable psychiatric problems: why depression feels so bad and what addicts really want. Neurosci. Biobehav. Rev. 35, 2000–2008. doi: 10.1016/j.neubiorev.2011.01.003

Zigman, J. M., Jones, J. E., Lee, C. E., Saper, C. B., and Elmquist, J. K. (2006). Expression of ghrelin receptor mRNA in the rat and the mouse brain. J. Comp. Neurol. 494, 528–548. doi: 10.1002/cne.20823

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