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How Does Our Brain Generate Sexual Pleasure?

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ABSTRACT

We present herein an exploratory essay on sexual pleasure, in support of the objective of developing an evidence base of knowledge for the WAS Declaration of Sexual Rights. We have attempted to account for the feeling of erotic sexual pleasure, in terms of what is known about neuronal function. The brain regions that are activated during women's orgasm, and their perceptual and physiological roles, are compared with brain regions related to chemically induced euphoria and craving. The brain regions that are activated at orgasm match those that are activated by *both* euphoria and craving. Based on these findings, we propose that erotic, sensual feeling is a simultaneous activation of euphoria *plus* craving. The importance of sensory stimulation, proprioception, sensations, and feelings is emphasized by evidence that their disruption leads to pathologies. The process of buildup of excitation to a peak and then resolution is proposed as a basic "orgasmic" property of the nervous system shared by multiple systems, as in a sneeze, which we consider to be a non-genital orgasm. We postulate a process by which an excitation pattern feels pleasurable and – at higher intensity – euphoric, if it is congruent with an unconscious dynamic "template," but aversive and at higher intensity painful, to the extent that it is incongruent with the template. Under this formulation, peak neuronal excitation that is congruent with the unconscious, simultaneously "getting what is craved," generates orgasmic, erotic, sexual pleasure.

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Introduction

In this essay, we develop the thesis that pleasure *per se* is a fundamental life force, which drives biologically adaptive behavior, promoting health and well-being. In this view, *sexual* pleasure has evolved as a special case that also promotes procreation and thereby perpetuation of the species. We provide evidence that pleasurable stimulation, and in particular sexual pleasure, is necessary and beneficial to human health. Thus, our contribution fulfills and supports a fundamental objective of the WAS Declaration on Sexual Pleasure, which is to develop evidence-based informed knowledge of the benefits of sexual pleasure as part of well-being in individual and public health, and to inform health promotion policies.

Sexual pleasure is a cognitive experience based on the reciprocal relationship between bodily physiology and nervous system function. That is, specific brain activity stimulates physiological responses in the genital system that in turn generates sensory nerve feedback to the brain, whose neurons generate pleasure. In this essay, we speculate on the process by which known neuronal functions of the brain could account for the cognitive experience of sexual pleasure. We hope that this approach will increase the understanding of the reader unfamiliar with this level of analysis, and illuminate future research.

Regional brain activity during orgasm in women

Our studies of the brain responses to genital stimulation have identified a widespread activation of the brain during the intense pleasure of orgasm (Komisaruk et al., 2004; Wise et al., 2017). We have sought to conceptually "generate" the pleasurable quality of orgasm by consideration of the roles of the same brain regions

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thatare also activated under non-sexual conditions (Komisaruk et al., 2006). While there exist a plethora of roles of each of the following brain regions and of their associated neurotransmitters, specific elements of their roles, considered as converging in concert, can account for known components of orgasm, as follows. One of the most salient is the activation of the Nucleus Accumbens and ventral tegmental area at orgasm, the joint occurrence of which is consistent with dopamine release at orgasm, for which there is pharmacological evidence described below. Another is the activation of the medial anterior hypothalamic region, which is consistent with oxytocin release at orgasm. The activation of the cerebellum is consistent with the intense muscular tension during orgasm. The activation of the amygdala is consistent with the increase in sympathetic autonomic tone which increases heart rate and blood pressure at orgasm. The activation the PAG (periaqueductal gray) and dorsal raphe are consistent with the activation of the descending pain-attenuating system, which could play a significant role in the analgesia of orgasm. The activation of hippocampus may be related to the erotic fantasy commonly experienced at orgasm. The activation of the anterior cingulate and insular cortices is of particular interest, as these two regions are activated not only during orgasm, but also during painful stimulation. This raises questions e.g. of whether their different sub-regions are differentially activated by orgasm and pain, whether the orgasm-related activation inhibits the pain-activated region (fMRI cannot distinguish active excitatory from active inhibitory regions), and/or whether the orgasm-activated regions are involved in generating the facial grimaces characteristic of both orgasm and pain, independent of the experience of pleasure or pain. Thus, many of the physiological and perceptual characteristics of orgasm can be related to the roles of the brain regions that have been identified in non-sexual contexts. We fully appreciate the complexity of the neurochemical and functional properties and effective connectivity of the above brain regions, but it is our intention to try to make sense of the variety of empirical findings by pointing out what seems to be a coherent pattern among these various properties, as they relate to the phenomenon of the pleasure of orgasm.

Role of the nucleus accumbens (NAc) and related components

Consideration of the Nucleus Accumbens-ventral tegmental area activity at orgasm is particularly illuminating. At orgasm, the ventral tegmental area, in which the mesolimbic dopamine (DA) neurons originate, becomes activated, suggesting that DA is being released in the projection zones, including the Nucleus Accumbens (NAc) and the prefrontal cortex. Dopaminergic drugs (e.g. buproprion) potentiate, while dopaminergic antagonists (e.g. haloperidol) inhibit sexual response and orgasm (Komisaruk et al., 2006). Drugs that increase DA levels by facilitating their release and/or blocking their reuptake, e.g. cocaine, amphetamine, and others that potentiate DA function (e.g. opiates, caffeine, nicotine) produce a "high" or "rush" or pleasurable sensations, but also craving by an action on the NAc (Risinger et al., 2005). Based on functional MRI findings in that study, Stein and colleagues reported that self-administered cocaine induced a *decrease* in NAc activity when the users said that the drug induced euphoria, i.e. a "high." Consistent with these findings, Breiter et al. (1997), reported an *increase* in NAc activity when cocaine users reported feelings of "craving." It is important to note that "… the high and craving constructs appear not to be independent. The inverse correlation between high and craving, even across all subjects, suggests that during [self-administered cocaine] these states may have become patterned and impossible for subjects to disentangle … "

This "entanglement" between euphoria and craving is likely to occur during orgasm as well. That is, many of the same brain regions that Breiter et al. (1997) and Stein and colleagues (Risinger et al., 2005) report are activated during *euphoria* or *craving* were *all* activated in our studies during *orgasm*. Specifically, they reported that the insula, caudate nucleus, operculum (Secondary Somatosensory Area: S2), and substantia nigra were all activated during euphoria, whereas the amygdala, anterior cingulate, and

orbitofrontal cortex were all activated during craving. In our studies, we found that during orgasm, all these brain regions were activated, including the Nucleus Accumbens!

Do euphoria ± craving = erotic sensuality?

Thus, when these reports are juxtaposed with our findings of an increase in activation of the Nucleus Accumbens during orgasm (Komisaruk et al., 2004; Wise et al., 2017), it raises the intriguing implication that upon genital stimulation induced orgasm, there is activation of brain regions that generate *both euphoria and craving simultaneously*. We propose that the joint generation of these two distinct responses at orgasm would generate the unique quality of *erotic sensuality*.

Perhaps the quality of erotic sensuality is the simultaneous yearning and having of genital sensation. Or, as characterized by Berridge et al. (Smith et al., 2010) and Kringelbach (2010), simultaneous "wanting and liking," of genital stimulation. There is more to the quality of erotic sensuality than just "pleasure." Thus, the sensory qualities generated by the change in activity of these brain regions *differentially* may generate or "reproduce" the unique erotic sensual quality of orgasm.

Is scratching an itch a "micro-orgasm"?

Technically, the functional MRI method has a very slow temporal resolution – on the order of 4–6 seconds. Therefore, it may be that the euphoric and craving feelings are not simultaneous, but rather alternate, and they may even occur contralaterally in the brain, so it may be more appropriate to consider that they occur just jointly. But the quality of joint occurrence of seemingly disparate sensations generates unique feelings. Perhaps the joint sensations of itching and scratching the itch generates a degree of sensuality; the itch is the craving for stimulation, scratching the itch produces the low level "euphoria" of momentarily stopping the itch, and then cycling the process repetitively. One of us on several occasions experienced the feeling of intensely hot shower water impacting on poison ivy-induced severe itch of the ankles. The burning stopped the itching, while both sensations grew in intensity; the combination felt sensuous and even built to an orgasmic feeling!

Thus, combining two seemingly disparate sensations can synthesize a uniquely different sensory quality – an emergent property different from either one. A common example of our brain performing this process is in our visual system, in which the distinctly different 2-dimensional visual images generated by each eye are unified by our brain into a single 3-dimensional image, which then has the uniquely synthesized property of true depth perception.

A fundamental role for neuronal excitation

We started this essay with the assumption that different brain components can generate the feelings of euphoria or craving, but what about sensation at a much more basic neuronal level? Why does sensory stimulation, which induces neuronal activity feel good? Perhaps more basically, neuronal activation *per se* "feels" (i.e. is "good" or, if more intense, is "euphoric") and a relative low level of neuronal activity creates a state to which, in a negative feedback sense, the nervous system is organized to compensate for by obtaining … by acquiring or generating … neuronal activation, a process we perceive as "craving."

Perhaps the above is the level at which to start to understand feelings and drives, for after all, neurons are basically "just" bags of chemicals. What is fear, expectancy, reward, anxiety, rage, pleasure, or pain to a neuron? It is "just" neurons that occupy the brain regions to which these complex processes are attributed, in the literature on brain imaging, lesioning, stimulation, etc. Consideration of these forms of complex evidence will not get us any closer to an understanding of the neural basis for the *quality* of sexual pleasure than climbing a mountain will get us closer to Mars.

What are "feelings"?

Consequently, we must take a different approach in an attempt to account for the pleasure of genital sensation, by addressing the question of what

is the neural basis for "feelings." What is the significance of the neuronal "currency" of action potentials, excitatory versus inhibitory, in generating feelings. Where do feelings originate? What is a basis for the difference between good and bad feelings? Why can genital sensation feel good, pleasurable, erotic, or painful? We attempt to address these questions, with continuous reference to what is known about the activity of neurons in the brain.

The "hard question" in neuroscience is the process through which neuronal activity is transduced into conscious awareness (Chalmers, 1995). A neurophysiological mechanism based on known physical principles has recently been proposed to answer the hard question (Komisaruk & Rahman, 2020). The easier question is *whether* neuronal activity is transduced into conscious awareness, to which all neuroscientists would answer in the affirmative. None would argue with the consensus that without neuronal activity in the brain there would be no conscious awareness.

The essentiality of neuronal activity

Neuronal activity (i.e. action potentials) is not only necessary for conscious awareness, it is neurotrophic, i.e. the survival of neurons depends on synaptic input from other neurons. If a sensory nerve pathway is severed, "Wallerian degeneration" (Conforti et al., 2014) occurs across several sequential synapses. Ramachandran and Blakeslee (1999) describe a young man who, after amputation of his hand, felt as if his amputated fingers were being touched when his face was touched. There is normally a chain of three sequential sets of neurons in the sensory pathway from the hand to the sensory cortex: the radial nerve, which synapses in the nucleus cuneatus in the medulla oblongata, then the medial lemniscus which synapses in the lateral thalamus, and then the thalamocortical neurons that synapse on neurons of the sensory cortex. The fact that the man felt his phantom fingers being stimulated when his face was touched implies that the sensory cortical neurons were still functional that used to respond to his fingers being stimulated. But the three-sequential-neuron chain from the hand to those cortical neurons had degenerated, allowing the thalamocortical neurons that project to the face to sprout axonal terminals that would synapse at the original finger cortical sensory neurons. Thus, the severing of the sensory nerve at the hand produced Wallerian degeneration up to, but not including, the hand sensory cortical neurons. The reason that the hand sensory cortical neurons survived despite the loss of the input from the hand is that cortical neurons receive prolific input from multiple sources, evidently much more input than the input provided to the three-neuron afferent chain leading to the cortex. Thus, there was insufficient neurotrophic activity in the afferent chain but sufficient neurotrophic activity to the cortical sensory hand neurons. Neurotrophic effect in the visual system was previously demonstrated by Wiesel and Hubel (1963): in the kitten, 2–3 months of monocular light and form deprivation produced a marked atrophy of cells in the lateral geniculate body (i.e. the thalamic visual relay site). These studies demonstrate that without sufficient neuronal activity input, the receptive (post-synaptic) neurons do not survive. Thus, synaptic input resulting from neuronal action potentials is a requirement for neuronal survival and function.

The fundamental importance of sensory stimulation

Extrapolating this principle, sensory stimulation is of fundamental importance for the function of the nervous system, and consequently for our existence. Kaufman (1960, p. 321) made the following insightful observation: "Most gratifications are in fact derived from stimulation, not the lack of it. … Freud said that the child sought this experience (nursing) again for the pleasurable state it produced, which it should be noted is a state of stimulation." Under conditions of severe sensory deprivation, our brain generates neuronal activation in the form of hallucinations (Mason & Brady, 2009). We crave sensory stimulation. In the absence or perceived inadequate level of stimulation from our environment, physical or social, we seek it. An actual hug or its myriad physical stimulation equivalents (idiosyncratic) or social symbolic or metaphorical equivalents (e.g. phone call from a loved one) can provide the

sensory stimulation or the related cognitive neuronal activity (excitation). If we can't get that, we give it to ourselves.

We recall a seminar speaker looking pale and terrified as he spoke with his hand pressed against his chest with what looked like all his strength. He never released his pressing hand for his entire hour-long presentation. It looked as if he was giving himself a continuous hug, physical stimulation, to overcome fear of rejection – or worse –abandonment by us, his audience. Fromm (1956) considered anxiety to be the expectation of isolation in some form, such as separation, abandonment, or ostracism.

Perhaps in its most fundamental form, that is, earliest in our development, physical contact with mother or other caretaker provides us with sensory stimulation – loving, caressing touch, sound, odor – stimuli that are comforting, and that establish a "brain pleasure pathway" – a memory – of loving, comforting stimulation (Del Cerro, 2017). Then as we mature, symbolic social stimulation can substitute for that physical stimulation – e.g. a text message or phone call from a loved one. If that strategy is unsatisfying, we may try to substitute for that stimulation by generating internal stimulation by smoking, eating, drinking, drug-inducing "rush," etc.

Pathological consequences of deficient neuronal activity

Visceral afferent activity converges on the same spinal cord neurons as somatic afferent activity at the same dermatomal level (e.g. convergence between heart and arm inputs to the same spinothalamic neurons), as in the case of referred pain (Foreman et al., 2015). Similarly, the visceral sensation generated by smoking, eating, etc., could substitute for the somatic sensation of a hug or its symbolic equivalent. A colleague described that when he smoked cigarettes, whenever some little undesirable life event occurred … a rejection, a disappointment, a mistake … he immediately craved a cigarette. He said the smoke filled his lungs with stimulation. It was reassuring. The rush provided by cocaine has been described as a blast of stimulation. If the strategies of getting physical or social stimulation or viscerally-induced equivalent stimulation fail, then perhaps our body does its best to provide stimulation to us (Komisaruk, 1982). Asthmatic attacks can be triggered by abandonment or loss of a loved one. The lung congestion provides respiratory resistance, a potent stimulus. Experimentally increasing blood pressure directly by various means induces sleeplike activity in the brain (Bonvallet et al., 1954; Komisaruk et al., 1967); it can actually induce sleep (Koch, 1932), and it induces analgesia (Dworkin et al., 1979). Thus, hypertension may actually be our body's attempt to provide us with stimulation that calms us down. Similarly, ulcer, arthritis, Reynaud's disease, Crohn's disease, perhaps even "inflammation," may be our body's best attempt at providing us with sensory stimulation that we feel we lack and crave. Thus, perhaps if we don't pay attention to those attempts … those visceral feelings … then our body increases the intensity of those stimuli. People who are alexithymic, literally, without words for feelings, typically have associated psychosomatic diseases (López-Muñoz & Pérez-Fernández, 2019). It is as if when they don't pay attention to their body's signals, the signals grow stronger – "shout" – chronically, via increasing activity of the organ generating the sensory stimulation, to the point of pathology. Thus, neural excitatory stimulation is essential – we seek it, it is beneficial; if we lack it, or can't or don't get it, we generate it somehow, externally (i.e. somatically), or internally (i.e. viscerally), and if all those strategies fail, our body gives it to us as best it can, but its efforts can become pathological through hyperactivity if not responded to.

What is the difference between neuronal excitation that feels good versus neuronal excitation that feels bad?"

Hence stimulation, i.e. neural excitation, is necessary and it is beneficial, at all levels. Proprioception provides a primary source of stimulation. Glickman and Schiff (1967) proposed that the feeling of muscular contraction *per se*, as in motor behavior, is reinforcing in and of itself. And by extension to humans, it feels good; our muscular activity generates proprioceptive

stimulation and that feels pleasurable. If the proprioceptive stimulation is intense, it feels particularly pleasurable, as in the case of sneezing, stretching, yawning, or orgasm. But painful stimulation is also intense. Why does pain not feel good?

Perhaps the difference between pleasurable stimulation and painful stimulation is the relative intensity and the contribution of neuronal inhibition. Neuronal inhibition is as crucial to normal neural processes as neuronal excitation. It is estimated that 40% of the synapses in the human brain are inhibitory, utilizing GABA as the neurotransmitter (Bowery & Smart, 2006). Without neuronal inhibition our movements would be spastic. Neuronal inhibition enables us to move gracefully and with precision. At a biologically fundamental level, we have hard-wired inhibitory systems that enable coordinated behavior. Our spinal cord neuronal circuitry enables a noxious stimulus (heat) applied to the finger to elicit a withdrawal reflex (pulling the hand away from the heat). While the spinal cord neuronal hard-wiring activates the flexor motor neurons, e.g. the biceps, which withdraw the hand, the noxious stimulus simultaneously activates hard-wired inhibitory neurons that relax the antagonistic triceps muscles. This is a protective reflex in which the hard-wiring controls these "antagonistic" muscles, so that we don't tear the triceps muscle when we contract the biceps. This system functions even if the spinal cord is severed from the brain, further evidence of the fundamental, role of neuronal active inhibition. Another type of biologically fundamental spinal cord-level protective reflex is the Golgi tendon organ reflex, in which sudden, intense stretch of a muscle immediately inhibits it, as if you try to catch a 100-pound bag of concrete and suddenly drop it. That protective inhibitory reflex prevents a muscle from being ripped from its tendon.

More complex, "higher" levels of neural organization in the brain utilize the same principle of hard-wired active excitation coupled with active inhibition. Thus, painful stimulation activates a pain-inhibitory system that attenuates the pain and at extremely intense levels produces actual blackout … unconsciousness. Similarly, pleasurable stimulation also excites an active inhibitory system. The intense excitation at orgasm excites a pain-inhibitory system and also an inhibitory system that produces the "refractory period" in men, during which somatosensory responses are attenuated (Allen & Komisaruk, 2016). This type of excitation/inhibition has been termed, the "opponent-process" theory (Solomon, 1980).

There is an intriguing parallelism between two processes that are antithetical – pain and orgasm. Both systems share the same neural pathways through the spinal cord and into the brain, where at some (still undiscovered) point they diverge. Both pain and genital afference utilize the spinothalamic system. In cases of intractable abdominal pain from cancer, as a desperate procedure, the spinothalamic tract was surgically severed. The pain disappeared, but so did the ability to experience orgasms. When months later the pain started to recur, so did the orgasmic capacity (Elliott, 1969). The spinothalamic system activates the reticular system and the pathways project to the insular cortex and the anterior cingulate cortex. Both these cortical regions are activated not only by pain, but also by orgasm (Komisaruk & Cerro, 2015). Perhaps they both control facial expressions, which could account for the similarity of facial grimaces during pain and orgasm. Alternatively, perhaps the genital input actively inhibits the pain input, but the fMRI methodology is not able to distinguish between active excitation and active inhibition, or that the neurons in anterior cingulate and insular cortices that respond to orgasm and pain are not identical. The sympathetic division of the autonomic system is activated by both pain and orgasm … the heart rate and blood pressure increase dramatically during both. But obviously, as pain feels different from orgasmic pleasure, the neural pathways for the two phenomena must diverge at some point in the brain (Komisaruk et al., 2006).

Pleasure and pain: balance between excitation and inhibition

Perhaps one difference between pain and orgasmic pleasure is the intensity of the stimulation at which the inhibitory system is activated. The intense activation at orgasm triggers the

sympathetic system at some threshold, which in men activates the ejaculatory reflex and an inhibitory system that turns off the excitation and generates the refractory period (Levin, 2005). In women, the inhibitory system activated is sufficient to inhibit pain (Whipple and Komisaruk, 1985), but it is more gradual and fluctuating, so women can experience multiple orgasms and no obvious refractory period.

By contrast, pain elicits higher intensity activation and the inhibitory system "lags" behind, until situations in which the pain becomes so intense that an inhibitory system is activated that triggers unconsciousness. In the case of sado-masochism, perhaps the induced pain is under control of the recipient in that if it becomes aversive, the recipient signals the inducer to stop and the inducer complies. Another form of controlled pain is voluntary ingestion of hot chili peppers – expected, limited, and hence prepared-for pain, which thus becomes arousing but not aversive. These forms of controlled pain are different from "out-of-control" pain and their controllability enables "play" with the intensity. Perhaps another form of such "play" with pain intensity is itch. Itch utilizes the pain system (Potenzieri & Undem, 2012). Perhaps by activating the inhibitory system by scratching (i.e. activating the Melzack & Wall,1965, pain gate mechanism), it is possible to vacillate at the aversive threshold, both below by active inhibition of the pain, and just surpassing, the aversive threshold repeatedly, thereby "playing" just above and below the pain threshold. Perhaps this tantalizing play at the threshold gives itch its compelling quality, e.g. pain on/pain off-on-off-on-off etc., or, as we suggested above, concurrently activating the "euphoria" and the "craving" neural systems, which can elevate the threshold at which the stimulation is perceived as aversive.

Does pleasure = "do-over"?

How does the pleasure system differ from the pain system? The pleasure system can be characterized as the "do again what you just did" system, whereas the pain system can be characterized as the "stop now" system (Olds & Olds,1963; Valenstein, 1964). When their rats pressed the lever and received electrical stimulation in the septum or medial forebrain bundle of the hypothalamus, they pressed the lever again. Was it pleasure or was it compelled to do again what it just did? Evolutionarily, if a behavior pattern leads to a beneficial or non-aversive consequence, it could be adaptive to repeat it. So the repeat-what-you-just-did system is evidently the "reward" or "reinforcement" system. Perhaps the neural system that generates this repetition of a motor performance is the Nucleus Accumbens system. Its function in the fundamental state of repetition is commandeered as the "pleasure" system in humans. Perhaps pleasure is an extrapolation of this repetition system. It is the antithesis of a "stop what you just did" system, which evidently extrapolates to the pain system.

The "do-over" mechanism may have another characteristic that generates pleasure: predictability. Perhaps the lowered persisting specific behavior threshold that facilitates the repetition creates an expectation of repetition. Fulfillment of the expectation is confirmatory and thereby predictable and pleasurable. If we chance to receive stimulation that matches our sensory state – i.e. stimulation that is "like" our sensory state, we "like" it. "Like is like like" (Komisaruk et al., 2009. Like is the sense of matching – similarity, no difference, no disjunction. So perhaps the zero difference between expectancy of a stimulation pattern and receiving that matching stimulation pattern is what we like; it is like our expectancy. In this context, "love" is receiving the stimulation that matches we like or "want." Perhaps this is the optimal form of stimulation hearkening back to our motherly contact. Also in this context is "happiness," in the sense of "chance," i.e. happenstance; it is not necessary to exert control. It involves the expectation that whatever stimulation that may occur will match my state; no expectation of disjunction. Hence, "happy"; the expectation that continuing to do what I am doing will continue to match my state. I don't have to do anything to exert control over what happens, don't have to try to change it. Perhaps this is an essential feature of "pleasure" – the stimulation matches and hence "pleases" the stimulation that is anticipated.

Non-genital orgasms

A special case of this state is orgasm in its multiple forms … genital and non-genital. We have proposed that there is a fundamental process in our nervous system that is an orgasmic process (Komisaruk & Whipple, 2011). That is, a process in which excitation builds to a crescendo and triggers a high threshold, opponent process, leading to a resolution or calming of the original excitation. Genital orgasm is a special case. The more general orgasmic process underlies a sneeze, a yawn, a stretch, urinating, vomiting, and lower intensity, more subtle processes, such as swallowing. Why do they feel good, why are they pleasurable?

It is pleasurable to experience the feeling (i.e. sensation) of muscles contracting but without the pain of making them contract so strongly, e.g. a sneeze; our chest muscles contract without the effort of making them contract. This is a form of "involuntary" proprioception, our body giving us stimulation that we didn't exert effort to produce. We don't have to exert the conscious effort to contract those muscles so strongly. It is a respite from the effort. It feels good because we don't feel the proprioceptive pain that we might feel if we had to perform the same action voluntarily. For us, our sneeze is the expression of our unconscious, for it takes over the biologically fundamental hard-wired evolutionarily adaptive reflexive process. A sneeze provides us a window into our unconscious.

Our child-like adaptive unconscious

Let us consider that there is an unconscious process in our brain that is responsive to our needs and desires which it manifests by adjusting our bodily "physical plant" accordingly via our autonomic nervous system. It thereby regulates our circulatory system to support the behavior and visceral activity that our unconscious dictates, as if we were expressing our needs and desires in relation to our environment – physical and social – at each moment. A metaphor for the process is that it is the expression of the unfettered child in us. That child's brain would be regulating its bodily functions via its autonomic system. What if that pattern of autonomic activity occurs in real time in our adult body now, independent of our actual overt behavior. To the extent that our actual overt behavior is congruent with the pattern, it is a match – pleasure? However, to the extent that our actual behavior is *incongruent* with the pattern is it discomfort, pain, leading to organic pathology, i.e. psychosomatic disease, if chronic? Metaphorically, it could be viewed as a multi-system homunculus, all systems superimposed on each other, creating a multi-component realtime little representation of ourself that "advises" – tries to drive – our behavior.

Extrapolating from the notion above, a sneeze feels good because it is a direct expression/manifestation of our biological imperative. In a sneeze, our chest muscles contract abruptly, involuntarily and strongly. Prior excitation (nasal irritation) builds to a crescendo, then at the "orgasmic" moment, our expiratory muscles contract suddenly and forcefully. When a sneeze "overtakes" our body – takes complete control of it – all our bodily "homuncular" systems are congruent with the action at that moment. It is the primordial expression of our "instinctive," hard-wired, evolutionarily formed, adaptive nervous system. It is our free-expression, innocent child-like, socially unrestricted, feeling-generated homunculus (i.e. literally, "little person") that is the engine of our behavioral expression. Of course, we normally bring it under control based on our experience and environmental/social pressures. But in a sneeze, it takes over our behavioral expression through (almost) all layers of our bodily homunculi, so there is coherence among proprioceptive, visceral and somatic, sensory and motor excitation homunculi … all serving the same biologic imperative … the sneeze and its evolutionarily adaptive function. It is, in essence, a non-genital orgasm. The sneeze "orgasm" is pleasurable because it is intense sensory activation that expresses all systems of our bodily physiological imperative. *Genital* orgasm can be considered a special case of this fundamental principle of nervous system function.

Thus, orgasm is the highest intensity of excitation that we experience; yet it is below aversive intensity. Hence it is the maximization of stimulation/excitation that we crave –and enjoy – as a

fundamental nervous system driving principle. Perhaps craving is our brain's mobilization to obtain stimulation that we feel is absent. Starting with infancy, our primordial behavior is to obtain and maintain sensory stimulation.

Conclusion

Thus, based on the above principles of neuronal function, can we account for how some neural activity feels good and other neural activity hurts? Perhaps pleasurable stimulation is stimulation that generates excitation that is not so intense that it would activate compensatory inhibitory activity (negative feedback), so it proceeds. We seek stimulation, excitation, we work to generate it, it asserts our existence; our neurons require it for their existence and survival; it is the primordial essence. It perpetuates itself, re-activating the system that just produced it, hence "reinforcing" the activity of that system. It feels good because it feels and does not feel bad. That is, it does not produce stimuli that turns it off or prevents it from recurring. Neuronal excitation is feeling-…is being. It is how we know we exist. Existence is feeling, and feeling and perceiving affirms that we exist. The alternative is: no excitation; our nervous system abhors inactivity, abhors no excitation. In the absence of neuronal activity, we crave it, are biologically compelled to obtain it or generate it. But if and when it becomes excessively intense, it activates its own inhibition. The intense excitation can feel euphoric, orgasmic. But at higher intensities, inhibitory processes are activated to attenuate the intensity. To the extent that we perceive the inhibition as ineffective in the attenuation, it feels painful and aversive.

In conclusion, we have endeavored to "generate" the qualities of genital pleasure in terms of the unitary principle of brain function, i.e. that all neuronal activity is in the form of action potentials, which are either absent or that exert excitatory or inhibitory effects on other neurons. The unique qualities of genital pleasure and orgasm may be accounted for by the integration, with memory and realtime contexts (e.g. association with prior and/or current desired or unwanted stimulation) of activation and inhibition of excitatory, inhibitory, craving and pain systems, which can each be interpreted in terms of the unitary principle of neuronal activity. In this view, genital pleasure is but a special case of the infinite variations of human experience.

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