Orgasm

Barry R. Komisaruk, Carlos Beyer and Beverly Whipple view the subject of orgasms as an experience that is an integration of body, nervous system and the mind

Why should recent findings about orgasm be of interest to psychologists? If psychologists are interested in the bases of pleasure and pain – in altered states of consciousness, in psychological side-effects of antidepressant and antipsychotic drugs, in some surprising effects of sex hormones, in psychological effects of surgery of the sexual/reproductive system, or in the function of the unique phenomenon of orgasm, then the following brief update may engage and inform you.

question

On the basis that orgasms having different perceptual qualities can be elicited by stimulation of various body regions (e.g. in women: clitoris, vagina, cervix, anus; in men: penis, prostate) what is the perceptual quality of the orgasms when multiple body regions are stimulated simultaneously?

resources

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Orgasm is a compelling, brief event that is an integration of cognitive, emotional, somatic, visceral, and neural processes. Modern definitions of orgasm recognise and incorporate all these levels (see Komisaruk et al., 2006).

Despite bodily differences and some different neural events at orgasm, women's and men's descriptions of the basic feeling of orgasm are indistinguishable from each other (Vance & Wagner, 1976). The scientific study of orgasm in humans was initially focused on measurement of such somatic and visceral events, as exemplified by the pioneering studies of Masters and Johnson (1966) in men and women.

In recent decades orgasm research has entered a new era. The widespread use of antidepressants and antipsychotics, and their significant and mainly untoward effects on sexual responses and orgasm in humans, has provided clues not only to the neurotransmitter bases of orgasm but also to the development of new drugs that can avoid those side-effects (Komisaruk et al., 2006). Furthermore, new technology such as fMRI and PET has been applied to the study of orgasm, enabling us to begin to identify the underlying brain systems toward understanding its compelling nature.

Drugs and orgasm

Psychotropic medications commonly produce anorgasmia as a side-effect. Most of these drugs – antidepressants and antipsychotics – either (a) interfere with the binding or action of dopamine at its D2 or D4 receptors, respectively (Stahl, 1999), or (b) raise the levels of serotonin in some synapses by inhibiting its reuptake.

Dopamine – an orgasm 'accelerator'

Abundant evidence points to dopamine as the key neurotransmitter involved in stimulating orgasm in humans. Thus, administration of the dopamine precursor L-dopa, dopaminergic agonists (e.g. apomorphine), dopamine releasers (e.g.

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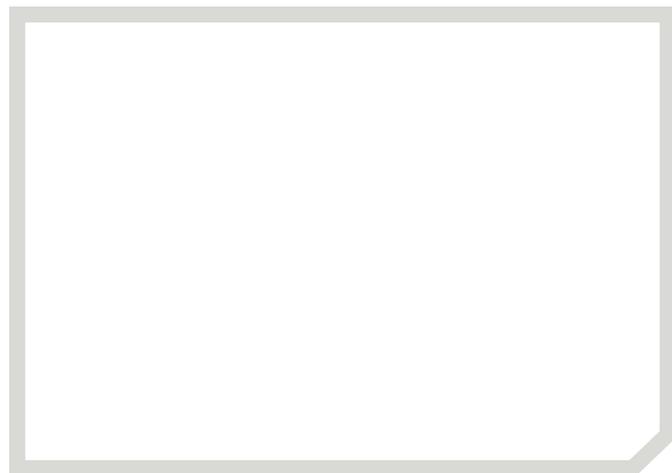
Many different nerve and neuronal pathways are involved in orgasm

amphetamine), or dopamine reuptake inhibitors (e.g. cocaine or bupropion) facilitate the expression of orgasm in men and women. Conversely, administration of antipsychotics impair orgasm, by blocking postsynaptic dopamine receptors (see Komisaruk et al., 2006).

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Dopamine-synthesising neurons that originate in the lower brainstem (specifically the ventral tegmental area) are activated during ejaculation in men, as measured by PET imaging (Holstege et al., 2003). A major projection of the dopamine neuron axon terminals is to the nucleus accumbens of the forebrain. This nucleus is activated during orgasm in women, as measured by fMRI (Komisaruk et al., 2004).

Thus, activation of the dopaminergic system of the brain evidently participates in the production of orgasm in women and men, on the basis of pharmacological functional brain imaging, and neuroanatomical studies. Consistent with this role of dopamine, hypersexuality has been reported in cases of Parkinsonism treated with dopamine precursor or agonist drugs (Bowers et al., 1971). Acute administration of drugs that increase dopaminergic activity only occasionally induces orgasm in the absence of other factors. However, intravenous injection of cocaine, which rapidly increases the release of dopamine at its neuronal terminals in the forebrain, can induce the 'cocaine rush' that individuals report as feeling similar to genital orgasm (Miller & Gold, 1988).

Serotonin – an orgasm 'brake'

Antidepressive drugs (e.g. the SSRIs, which increase the accumulation of serotonin in synapses by blocking its reuptake into the neuron terminals from which it was released) tend to produce anorgasmia. Inhibition of orgasm is mediated by interaction of serotonin with the serotonin-2 receptor subtype (Haensel et al., 1995). This molecular process is critically involved in the inhibition of orgasm – agents such as cyproheptadine that block the action of serotonin almost immediately counteract the inhibitory effect of antidepressants on orgasm.

The 'exception that proves the rule' is found in the case of nefazodone, which, unlike the other SSRIs, does not inhibit orgasm. Nefazodone, in addition to blocking the reuptake of serotonin, also blocks the serotonin-2 receptors, thereby counteracting the effect of the elevated synaptic levels of

serotonin, and thus preventing the serotonin from inhibiting orgasm (Stahl, 1999). Conversely, buspirone, which decreases the release of serotonin into the synapse, facilitates orgasm, thus further supporting the serotonin brake concept.

The braking effect of serotonin on sexual response is reported to be used 'off-label', to therapeutic advantage, by treating premature or early ejaculation with SSRI antidepressants.

The role of sex hormones

By contrast with the action of neurotransmitters, which change neuronal excitability almost immediately upon their release into synapses and thereby generate orgasm, the sex hormones – oestrogens and androgens – characteristically act with latencies of days – providing a facilitatory background for orgasm. In men, a deficiency of sex steroids (e.g. resulting from ageing or following surgical removal of the testes) may lead to anorgasmia and a decrease in sexual interest. The role of sex hormones in women is not as clear. Early studies concluded that bilateral oöphorectomy (i.e. removal of both ovaries) rarely resulted in lack of desire or anorgasmia. However, recent studies report decreases in sexual drive and pleasure after oöphorectomy (e.g. Braunstein et al., 2005).

Oestrogen (oestradiol) treatment does not correct these effects, because they are most likely due to a decrease in plasma levels of testosterone resulting from a decrease in the androgen secretion that normally occurs from the ovaries. Treatment with testosterone, alone or in combination with oestradiol, restores sexual interest and pleasure (orgasm frequency) in most of these women (Bellerose & Binik, 1993).

Androgen (testosterone) therapy is the standard treatment for hypogonadal men complaining of anorgasmia (Steidle et al.,

2003). Transdermal testosterone patches or gels, which slowly and steadily release the androgen into the circulation, have recently been used successfully. But while the efficacy of testosterone to improve sexual behaviour in hypogonadal men is incontrovertible, supplemental dosing with testosterone does not increase the frequency or quality of orgasms in men whose androgen levels are 'normal' (O'Connor et al., 2004).

Hormones are not only stimulatory to sexual desire and orgasm. These components of sexual response are depressed by prolactin, a protein hormone produced by the anterior pituitary gland that is released at orgasm in men and women. Men and women with hyperprolactinemia – elevated blood levels of prolactin – typically show anorgasmia and a low level of sexual desire (Bancroft, 1984).

Indeed, it has been proposed that some antidepressant (serotoninergic) and neuroleptic (anti-dopaminergic) drugs depress orgasm by elevating prolactin secretion. Moreover, some evidence, admittedly inconclusive, suggests a role of the prolactin released during orgasm in the production of the characteristic ('refractory') periods of sexual inactivity following ejaculation in men. In one case, a man who did not show prolactin release during ejaculation had three experiences of vaginal

The little death

Kinsey et al. (1953, p.613) mention, and provide numerous references to the observation that 'at orgasm, some individuals may remain unconscious for a matter of seconds or even for some minutes'. They point out that this phenomenon has been described as 'petite mort' (little death), and 'la mort douce' (the sweet death). While the underlying mechanism is not known, some have suggested contributing factors to be hyperventilation, insufficient blood flow to the brain resulting from irregular heartbeat and/or low blood pressure (Mann et al., 1982), and aortic constriction (Needles, 1973).

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intercourse with ejaculatory orgasm without intervening refractory periods (Krüger et al., 2005).

Brain imaging of orgasm

Consistent with the above-described role of dopamine in facilitating orgasm, several brain-imaging studies provide evidence that the dopaminergic 'reward' system is activated during sexual arousal and orgasm. This is supported by our fMRI studies showing that the nucleus accumbens region, which receives dopamine-containing axon terminals from neurons that originate in the ventral midbrain, is activated during orgasm in women (Komisaruk et al., 2004). Consistent with these findings, Holstege et al. (2003), using PET, found that the ventral midbrain area, in which the dopamine neurons originate, is activated in men during orgasm. In fMRI studies, Aron et al. (2005) found that men and women who were 'intensely in love', when observing pictures of their beloved, showed activation in this ventral midbrain area and the caudate nucleus to which the dopamine-containing neurons also project.

We have reported that in women, pain thresholds are more than doubled during orgasm (Whipple & Komisaruk, 1985), and that the insular cortex and anterior cingulate cortex in the forebrain are activated during orgasm (Komisaruk et al., 2004). Other investigators report that these cortical regions are activated during painful stimulation (Casey et al., 2001). These findings, considered together, suggest that a significant (active inhibitory) interaction occurs between orgasm and pain in the insular and anterior cingulate cortices, indicating that they are involved in both pain and pleasure.

Could these brain regions have some property that is common to both pain and pleasure, perhaps intense emotional expression – controlling the contorted facial expression that occurs both during painful anguish and similarly during impending orgasm – separate from the actual different feelings of pain versus pleasure? Furthermore, it seems possible that (at least

female) genital stimulation and orgasm, which we have shown attenuates the aversive component of pain, nevertheless may not attenuate the arousing quality of pain. This might help account for the practice of receiving what would appear to be pain-inducing stimulation in a sexually stimulating context, a combination that apparently intensifies pleasure.

Another brain component that we have found to be activated during orgasm in women is the paraventricular nucleus region of the hypothalamus (Komisaruk et al., 2006). The neurons of this nucleus secrete oxytocin, which is released into the bloodstream from the posterior lobe of the pituitary gland in peak amounts at orgasm in men and women (Carmichael et al., 1994). These neurons are activated in response to the vaginal-cervical stimulation that occurs during both vaginal intercourse and childbirth, and also to breast and nipple stimulation during suckling. The oxytocin released by the sensory stimulation that originates in these two different body regions is distributed via the bloodstream to the uterus and the mammary glands. At the uterus, the oxytocin stimulates the contraction of the smooth muscles, increasing the force of uterine contractions. This process has been shown in women to accelerate the transport of radioactively labelled sperm-mimic particles toward the ovary (left or right side) that has released a ripe ovum during that particular ovarian cycle. The finding that women who were pregnant were previously more likely to have shown this selectively directional transport, compared with women who were not pregnant, has led some to conclude that orgasm, while not essential to pregnancy, nevertheless probably facilitates pregnancy (Wildt et al., 1998).

At the mammary glands, the oxytocin
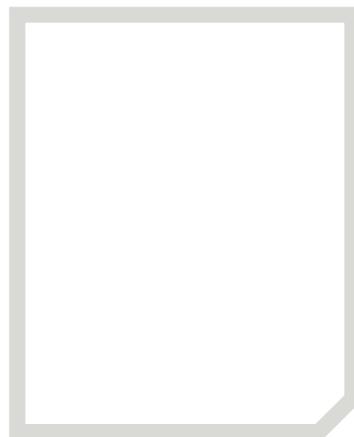
The feeling of orgasm has been described as 'a shower of stars'

stimulates the contraction of the smooth muscle 'myoepithelial' cells that surround the milk-producing alveoli, thereby forcibly ejecting the milk (Komisaruk et al., 2006). The fact that there is convergence of the vaginal-cervical and breast-nipple sensory activity onto the paraventricular nucleus neurons helps to account for the ability of stimulation of each of these organs to produce orgasms, and probably the ability of breast stimulation to modulate the pleasurable perceptual effects of vaginal-cervical stimulation.

Other brain regions reported to be activated during orgasm, and their involvement in other, non-orgasmic, activity, have been reviewed recently and extensively in Komisaruk et al. (2006). How does activation of the neurons in these brain components, such as the nucleus accumbens 'reward area', produce the pleasurable feelings of orgasm? We do not know. But that is just one case of the ultimate question in neuroscience – how do neurons produce any conscious awareness and their uniquely different perceptual qualities – pleasure, pain, light, colour, sound, taste, aroma.

Non-genital orgasms

'Non-genital orgasm' is not an oxymoron. Stimulation of pelvic organs – e.g. clitoris, vagina, cervix, uterus, anus, rectum, prostate and penis, are reported to produce orgasmic sensations. Orgasm elicited from vaginal stimulation has been described as 'deep, heaving', orgasm; from cervical stimulation as a 'shower of stars'; from clitoral stimulation as more restricted to the clitoral region; and from these organs in combination as 'blended', i.e. combining

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their qualities (Ladas et al., 2005).

Sensory activity from these organs is conveyed by a variety of nerves (see Komisaruk et al., 2006). For example, pleasurable orgasmic sensations from the rectum and prostate described by some men are conveyed by the pelvic and hypogastric nerves, respectively (Komisaruk et al., 2006). Orgasmic sensations during defecation reported in the case of a man (Van der Schoot & Ypma, 2002) were probably conveyed by the pelvic nerve. Stimulation of this nerve that occurs during passage of the fetus through the vagina during childbirth has been reported to produce both orgasmic sensations and the urge to defecate, indicating a convergence or commonality of effect of vaginal and rectal sensory activity. It is probably sensory activity via the hypogastric nerve that induces orgasmic sensations from stimulation of the prostate during anal intercourse in men, and conversely, prostatectomy has been reported to diminish orgasmic sensation (Koeman et al., 1996).

Stimulation of the hypogastric nerve probably occurs also during uterine contractions at orgasm and during stimulation of the G Spot, a component of which are the Skene's glands, considered to be a homologue in women of the prostate gland. Ejaculation of a fluid (usually 3–5 ml – approximately one teaspoonful) from the urethra in women, which is chemically different from urine (Belzer et al., 1984), is reported to originate from the female prostate gland (Zaviacic, 1999).

While there is an extensive literature on the effects of hysterectomy on sexual response and orgasm, there is considerable variability in the reported outcomes, some studies reporting that orgasmic response is attenuated (Saini et al., 2002), while others reporting that sexual response may be improved (Goetsch, 2005). The discrepancy in the literature is likely related to the multiple variable factors, including presenting conditions (e.g. genital pain or heavy bleeding that is reduced post-surgery), variability in surgical procedures (e.g. degree of nerve-sparing), whether the cervix and/or ovaries are removed or

retained, the criteria for assessing sexual response (e.g. sexual satisfaction, orgasm intensity), the type of genital sensory stimulation used to elicit sexual response (e.g. clitoral and/or vaginal), and so on.

Orgasmic sensations are reported to be produced also by stimulation of other body components. Men and women with spinal cord injury commonly describe a region of skin hypersensitivity near the level of the injury. When this skin is stimulated inadvertently (e.g. by clothing brushing it) it feels aversive. However, if the skin region is stimulated in the 'right' way by the 'right' person, it can produce orgasmic feelings. This effect was observed in our laboratory in the case of a woman with spinal cord injury whose hypersensitive skin region was at the neck and shoulder. When she stimulated the region with a vibrator, she reported feeling an orgasm and her blood pressure and heart rate approximately doubled, responses characteristic of genitally stimulated orgasms in able-bodied women (Sipski et al., 1993)

As reviewed in Komisaruk et al. (2006), there are published reports of orgasms elicited by stimulation also of lips, hand, knee and anus occurring during dreaming sleep, of phantom limbs, from electrical or chemical stimulation of the septum, amygdala or thalamus of the brain and of the spinal cord.

Orgasms have also been described by men and women when they suffer epileptic seizures that are triggered by specific activity (e.g. brushing the teeth: Chuang et al.,

"sociocultural and funding impediments to studying orgasm scientifically are gradually being breached"

2004), or that occur spontaneously. While these epileptic orgasms are in some cases described as 'unwelcome' (Reading & Will, 1997), others describe them as pleasurable, one woman refusing anti-epileptic medication for that reason (Janszky et al., 2004).

We have measured autonomic and brain activity during orgasms that women have produced by thought alone. During the thought orgasms, the magnitude of the increases in heart rate, blood pressure, pain threshold, pupil diameter, and brain regions are similar to those that we observe during vaginal or cervical self-stimulation-induced orgasms (Whipple et al., 1992). It is not surprising that in those cases of thought-induced orgasms, the specific genital sensory thalamic and cortical, and specific limb-motoric regions, are not activated.

Conclusion

We have but scratched the surface of orgasm's potential as an entity for analysis by physiological, pharmacological, endocrinological, immunological, evolutionary, cognitive, social neuroscience and other lenses. It is evident from a burgeoning literature that the sociocultural and funding impediments to studying orgasm scientifically are gradually but inexorably being breached.

We hope that psychologists will heed the reports of men and women that they experience pleasure, and even orgasmic experiences, from stimulation that has been considered as unconventional. We hope that professionals will acknowledge that there are many ways in which men and women experience sensual and sexual pleasure including orgasm, and thus validate their richly varied sexual experiences.

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