Archives of Sexual Behavior
https://doi.org/10.1007/s10508-021-02164-9

ORIGINAL PAPER

What Is the Trigger for Sexual Climax?

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Received: 5 January 2021 / Revised: 27 August 2021 / Accepted: 20 September 2021

Abstract
A model is proposed to consider sexual climax in men, women, and animals as a unitary phenomenon. Sexual climax is a stereotyped rhythmic pattern of spinally generated neural activity in the autonomic and somatic nerves innervating pelvic organs. A column of neurons in the spinal cord of the male rat is strongly activated by ejaculation (sexual climax in the male). These neurons project to the thalamus and are therefore called lumbar spinothalamic cells (LSt cells). Comprehensive studies have demonstrated that the LSt cells constitute a central pattern generator of ejaculation. These findings have been extended to female animals. Further studies identified LSt cells in the lumbar spinal cord of men and women. Strong evidence indicates that the LSt cells mediate ejaculation in men. The climax model generalizes and extends these studies. It postulates that LSt cells in the lumbar spinal cord of humans and animals of both sexes generate climax. The LSt cells generate the neural activity driving the pelvic contractions and other responses of climax. The activity is transmitted to supraspinal sites to activate orgasm. The LSt cells receive excitatory and inhibitory projections from supraspinal sites. The descending projections reflect subjective arousal and inhibitions. Spinal sensory neurons from the genitals provide excitatory and inhibitory innervation to the LSt cells. These represent pleasurable and noxious sensations. The supraspinal and spinal excitatory and inhibitory inputs are integrated by the LSt. When the sum of the excitatory inputs, minus the sum of the inhibitory inputs reaches a threshold, the LSt cells generate sexual climax.

Keywords Sexual climax · Orgasm · Ejaculation · Central pattern generator · Spinal cord physiology

There is no topic where the coverage in the scientific literature versus the lay press is as lopsided as the question what triggers orgasm? Both men's and women's magazines devote great attention to the topic. Underlying the discussion in these sex articles is the belief that there is a technique or an anatomical structure that will reliably trigger orgasm in their partner. This has proven to be an elusive goal, or they would not have to return to the topic month after month.

The scientific community has had no greater success in identifying the neural mechanisms leading to the initiation of orgasm. While the lay press is obsessed with the topic, sexual medicine has virtually ignored it. For example, in authoritative reviews written by the foremost experts in sexual medicine, the topic is not addressed in discussions of ejaculation or female orgasm (Rowland et al., 2010; Salonia et al., 2010). This is odd given that the topic is clinically relevant to conditions such as premature ejaculation and anorgasmia. There is little discussion in the medical literature of the neurobiology of triggering orgasm, such as what sensory modalities are involved, what is the location of the initiation of orgasm, and what determines what sexual stimulation is sufficient to evoke orgasm? The real reason why the trigger for sexual climax is ignored is because how the brain generates sexual climax is so little understood (Fig. 1).

One of the largest obstacles to identifying the trigger for climax is the confusing nature of the problem. A very large number of stimuli are known to elicit climax, particularly in women. These stimuli range from several types of pelvic sensations from different pelvic organs to factors involving the highest levels of cognition. There is some disagreement about exactly how climax in men and women are related. What are the similarities and differences, and how do they inform our conceptual understanding? Another question is what is the relationship between climax in humans and in animals?

The central feature of the culmination of sexual excitation in both sexes, in humans and animals, is a synchronized,

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Published online: 18 October 2021

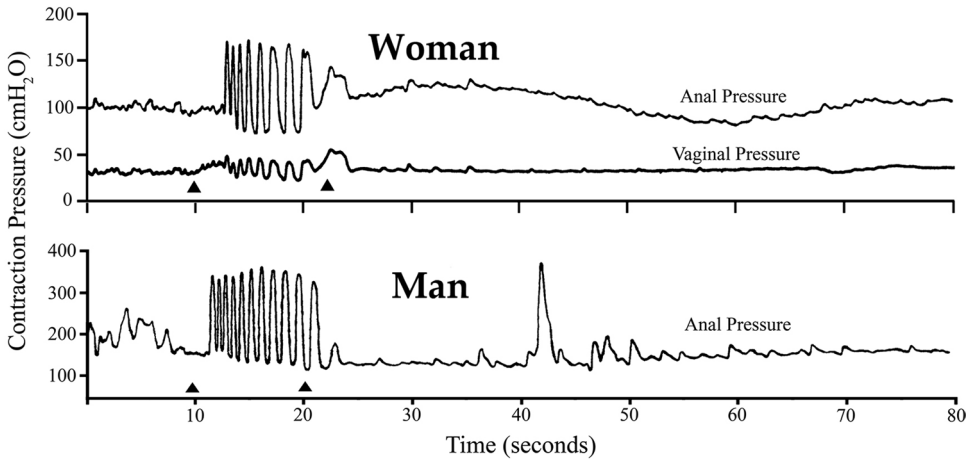

Fig. 1 Physiological recordings of the most common pattern of orgasm in men and women volunteers induced by masturbation. The pelvic contractions of orgasm were measured by the means of balloons inserted into the anus and vagina. Contractions of pelvic muscles are indicated by increases in pressure. Note that the contractions are highly regular with a consistent increase in the interval between each contraction. The arrows indicate the beginning and end of the subjectively experienced orgasm. (McKenna, 2013. Adapted from Bohlen et al., 1980, 1982)

rhythmic activation of the sympathetic, parasympathetic, and somatic innervation of the pelvis. This results in rhythmic smooth and striated muscle contractions in the pelvis. Synchronized contractions occur in all the striated perineal muscles innervated by the pudendal nerve in both sexes, in humans and in animals. Smooth muscle contractions are evoked in the uterus, vagina, urethra, accessory sex glands, and ductus deferens. In the male, the neural and pelvic activity of climax results in ejaculation. In the healthy woman or man, the spinally generated climax is synchronous with intense pleasure, an altered state of consciousness, vocalization, distinctive cardiorespiratory responses, and autonomic responses such as pupil dilation, flushing, sweating, etc. There are strong arguments that animals experience the intense subjective experience of orgasm (Pfaus et al., 2016).

It has been repeatedly demonstrated that the global pelvic activation that is the culmination of sexual arousal is generated in the spinal cord of men and women, and male and female animals. It has been repeatedly demonstrated that both men and women with complete spinal cord lesions can achieve sexual climax by genital stimulation, although the supraspinal component of orgasm is absent (Alexander & Marson, 2018; Chéhensse et al., 2013). Numerous animal studies confirm and extend these findings. The pleasure, altered states of consciousness, and the other psychological components of orgasm are all generated by supraspinal sites. The non-pelvic autonomic and respiratory responses also require supraspinal mechanisms. Thus, the two components of orgasm, the pelvic activation and the subjective experience, dichotomize into spinal and supraspinal mechanisms. The model I am proposing purposefully ignores the details of climax that may be specific to one sex, a particular species, or condition. Therefore, I will adopt a very simple nomenclature to reflect this approach. I will use the term sexual climax to refer solely to the spinal component, the coordinated activation of pelvic organs. The term climax can be applied to humans with spinal cord injury as well as to animals under various experimental situations, including under anesthesia or with neural lesions. The term can be used in future studies of spinal cord physiology *in vitro*. In the healthy male human or animal, sexual climax is a synonym for ejaculation, and in most cases can be used interchangeably. However, this is not always the case. For example, following prostatectomy or TURP surgery (transurethral resection of the prostate) for lower urinary tract symptoms, men achieve climax and experience orgasm, but no ejaculate is produced. In many experimental animal studies, there is also a dissociation between climax and ejaculation. In these cases, sexual climax is a more appropriate term and avoids discussing ejaculation without ejaculate (Fig. 2).

In the most typical case, sexual climax begins abruptly. Activity prior to the initiation of climax is highly variable and stimulus-dependent. In contrast, climax activity is highly stereotyped and regular. Rhythmic neural activity in the autonomic and somatic nerves of the pelvis results in strong, smooth and striated muscle contraction. The inter-burst interval is the shortest (highest frequency) at the beginning of climax. The interval increases throughout climax. The contractions then usually end relatively abruptly, although prolonged after discharges are not uncommon. This pattern of activation has been measured in men and women, male and female rats, and male and female mice. (Bohlen et al., 1980, 1982; McKenna et al., 1991) The only reasonable explanation is that climax seen in male and female animals and humans

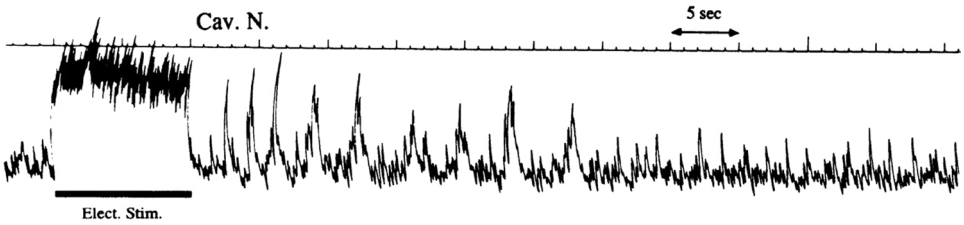

Fig. 2 Example of climax recorded in a female rat. Reflex was induced by stimulation of sensory branch of the pudendal nerve (which is composed primarily of the sensory dorsal nerve of the penis/clitoris) at the time indicated (Elect Stim). Recording is from the cavernous nerve (Cav N). The cavernous nerve supplies vasodilatory control of the erection of the clitoris/penis. The cavernous nerve is an autonomic nerve, and this figure shows that climax includes autonomic activity (McKenna et al., 1991)

are homologous phenomena generated by the same neural mechanisms (Fig. 3).

The dynamics of climax, the rhythmic pattern, and the on and off nature are indicative of the output of a central pattern generator (CPG). CPGs are neural circuits whose output is patterned neural activity. They generate rhythmic neural activity even in the absence of any rhythmic input. CPGs may produce continuous patterned activity, such as respiration, or discrete activity such as swallowing or sneezing. Swallowing is a very precisely timed, sequential activation of pharyngeal muscles. While the output of CPGs is a stereotyped activity, it can be modulated in a variety of ways. For example, the output of the swallowing CPG is modified according to the size of the bolus being swallowed. The output of CPGs can also be changed by neuromodulation, primarily by monoamines. Neuromodulation affects both the intrinsic and synaptic properties of neurons. Monoaminergic inputs result in the activation of G-protein-coupled receptors (GPCRs). This creates a long-lasting (hundreds of msec to minutes) second messenger signaling cascade. This has a profound effect on basic neuronal functions, such as modifying synaptic efficacy, altering firing and bursting activity, modifying membrane currents, etc.

CPGs generate a stereotyped output, which is then shaped by sensory inputs and neuromodulation. It is very likely that the sexual climax CPG can show a range of outputs depending on its inputs and neuromodulation. There is a wide variability of individual patterns of climax in humans. For example, in a minority of women and men, the pelvic contractions are primarily tonic and not rhythmic (Bohlen et al., 1980, 1982). It must be certain that there is a wide variation of climax characteristics in animals given such diverse anatomy and behavior. Nonetheless, the principle is the same. The CPG produces a patterned neural output to the sexual organs, shaped by its afferent inputs and the neuromodulatory tone (Golowasch, 2019; Steuer & Guertin, 2019).

The breakthrough discovery in the study of sexual climax was made in the laboratory of Lique Coolen. She

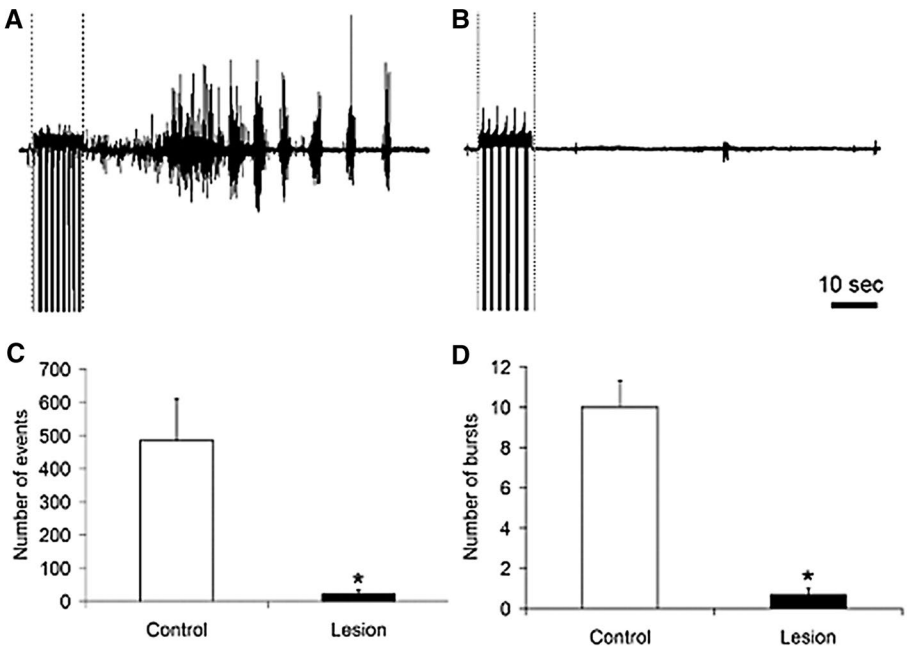

Fig. 3 Example of climax in the male rat and its abolishment by LST cell lesions in the male rat. LSt cell lesions eliminated the dorsal nerve of the penis (DPN) stimulation-induced ejaculatory reflex. The bursting pattern of the bulbospongiosus muscle (BCM) following stimulation of the DPN is illustrated in representative examples of BCM bursting in control (**a**) and LSt-lesioned (**b**) males. Dashed lines in A and B depict the period of DPN stimulation. Quantitative analysis of BCM EMG is shown as numbers of events (**c**) and bursts (**d**). * indicates significant difference from control animals (Staudt et al., 2011)

characterized a group of spinal neurons in male rats which constitutes the central pattern generator that produces ejaculation, i.e., sexual climax in males (Allard et al., 2005; Truitt & Coolen, 2002;). These neurons are located in a column of neurons adjacent to the central canal of the L3–L4 segments of the spinal cord. These spinal segments are located rostrocaudally between the sympathetic outflow to the pelvis in the thoracolumbar segments and the parasympathetic and somatic pelvic efferents in the lumbosacral segments. These lumbar neurons project to an area of the thalamus which is involved in attention and arousal, the medial parvocellular parafascicular thalamic nucleus. The projections from the spinal cord integrate into a forebrain network for ejaculation, which has been elucidated by the Coolen lab (Veening et al., 2014). The thalamic projections are assumed to be responsible for relaying climax information from the spinal cord to activate the forebrain components of orgasm. Because of their location and projections, these spinal neurons are referred to as the lumbar spinothalamic neurons, abbreviated LSt cells. The LSt neurons are immunopositive for the peptide neurotransmitters galanin, cholecystokinin (CCK), and enkephalin. The neurons also express neurokinin-1 (NK_1) receptors, the receptors that are activated by the peptide neurotransmitter Substance P. Immunostaining for galanin provides a reliable method for identifying the LSt cells, since there are no other neurons immunopositive for galanin in this region of the lumbar spinal cord (Fig. 4).

Comprehensive studies carried out by the Coolen lab conclusively demonstrated that the LSt cells are responsible for ejaculation (climax in the male) in the rat. First, the LSt cells are activated during ejaculation in mating studies, as evidenced by the expression of the immediate-early response gene c-fos, a marker for neural activation. No such activation of these cells is seen with other sexual activities such as erection, mounts, or intromissions. In a series of elegant experiments, the LSt cells were lesioned in a very selective manner. This lesion eliminated the ability of the rats to ejaculate in mating studies, without disruption of other sexual behaviors. Following the lesions, there were also severe deficits in an anesthetized, spinalized male rat model of climax. The LSt cells receive direct projection from pelvic sensory fibers. The LSt project to the autonomic and somatic pelvic efferents. The LSt cells are the target of numerous supraspinal projections. Thus, they have all the connectivity that is required to be the generator of climax. Further studies identified the role of several neurotransmitters play in the LSt production of ejaculation, and their disruption by spinal cord injury. Direct injections of excitatory amino acid neurotransmitters onto the LSt cells reliably induced climax in the male rat experimental model. Taken together, these data definitively establish that LSt cells in the lumbar spinal cord mediate sexual climax in the male rat (Kozyrev & Coolen, 2015, 2017; Staudt et al., 2010, 2011, 2012; Truitt et al., 2003; Wiggins et al., 2019).

Further studies in female rats and mice have confirmed key elements of the findings in male rats. LSt cells are activated by climax in females and their connectivity compares to the males (Cai et al., 2008; Carro-Juárez & Rodriguez-Manzo, 2006; Marson et al., 2003; Wiedey et al., 2008). These data indicate that the female rat and mouse have the same climax-generating LSt cells as in the male rat. The climax CPG has been shown to be functional in male neonatal rats (Carro-Juárez & Rodrigo-Manzo, 2005). This indicates that the system is organized at an early stage of development. The number of LSt neurons is sexually dimorphic in neonatal mice (Federighi et al., 2020). A similar sexual dimorphism in the number of LSt neurons in humans has also been identified (Chéhensse et al., 2017) (Fig. 5).

The Giuliano lab has carried out extensive anatomical and physiological investigations of the LSt cells. They made essential contributions to our understanding of the neuroscience of the LSt cells. Transneuronal tracing methods demonstrated that the LSt control pelvic organs of ejaculation. The LSt cells receive projections from the brainstem and hypothalamus. The inputs and projections of the LSt cells, and their physiology were elucidated in many studies (Borgdorff et al., 2009; Chéhensse et al., 2016; Clement et al., 2009; Fachinetti, et al., 2014; Sun et al., 2009, 2013; Xu et al., 2005, 2006). A picture emerges from these many multidisciplinary studies from both labs. The LSt cells receive two major classes of inputs. They receive a dense segmental sensory input, particularly from the pudendal nerve. They receive multiple descending inputs. Among these descending inputs are monoaminergic projections, particularly serotonin and norepinephrine. The LSt cells have two major outputs. The

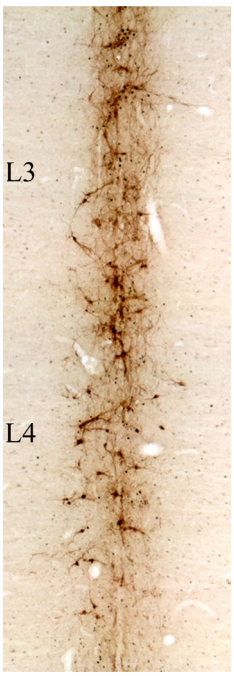

Fig. 4 Horizontal section of spinal segments L3 and L4 in the male rat. Immunohistochemical staining for galanin demonstrates the LSt cells. (McKenna, 2013)

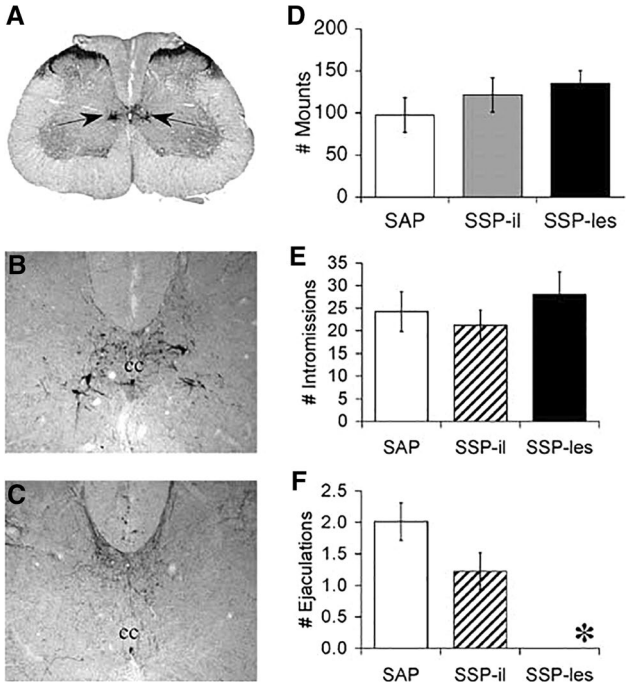

Fig. 5 Effects of LSt cell lesions on rat sexual behavior. LSt cells were specifically lesioned by attaching the neuropeptide Substance P with saporin (SAP), a ribosomal inactivator, to form (SSP-SAP). The complex binds to the NK_1 receptor on the LSt cells, and it is internalized, killing the cell. Native saporin is cell impermeable and does not damage the cells. **a** Confocal image showing the presence of NK_1 receptors (red) in galanin-positive (green) LSt cells. **b** Galanin immunoreactivity showing intact LSt cells in a coronal section of L4 in male rats treated with saporin. **c** Lack of galanin immunoreactivity in L4 spinal cord following SSP-SAP treatment. (**d**–**F**) Numbers of mounts, intromissions and ejaculations, respectively, during 100-min mating sessions in male rats with complete lesions of LSt cells (SSP-les), incomplete lesions of LSt cells (SSP-il) and saporin-treated LSt intact control males (SAP). Asterisk indicates $p < 0.001$ compared to SAP or SSP-il males (Truitt & Coolen, 2002) (Color figure online)

first is a projection to the sympathetic and parasympathetic preganglionic neurons innervating the pelvic organs, and the pudendal motoneurons that innervate perineal muscles. The second projection is to the thalamus (Fig. 6).

A major advance was when the role of the LSt cells in climax extended from animals to men (Chéhensse et al., 2013, 2017). Exhaustive studies in the Giuliano lab compellingly demonstrate a direct correlation between the LSt cells and human ejaculation, providing an even stronger case for the role of LSt in climax. Spinal sections from men and women revealed the presence of a column of galanin-staining neurons in the central gray area of the lumbar spinal cord, similar to the rat. A strong sexual dimorphism was identified in the LSt cells in men and women. The functional significance of the dimorphism is unclear. A comprehensive analysis of a large number of patients with spinal cord injury revealed that damage to the L2-L5 segments was the sole independent predictor of the inability of penile vibratory stimulus to evoke ejaculation. A thorough metanalysis of ejaculation in men with spinal cord injury revealed a direct association between ejaculatory function and damage to the lumbar spinal cord. Ejaculation rates decreased dramatically as the lesion extended into L3 and below. These studies extend the findings in the male rat and provide compelling evidence that the LSt cells mediate climax in men and male and female animals, indicating that this is a highly conserved neural system, as would be expected for vital components of reproduction (Fig. 7).

LSt cells were anatomically identified in the spinal cord of women in the Giuliano studies. As yet, there are no data on the physiological role played by LSt cells in generating sexual climax in women. Given that LSt cells mediate climax in men and in both male and female animals, it would be very surprising if women did not have this same neural system for the generation of climax. A direct test of this model would be high-resolution functional imaging of the lumbar spinal cord while men and women achieve climax.

Taken together, these data suggest an obvious hypothesis. The LSt neurons in the lumbar spinal cord generate sexual climax in men and women and male and female animals. Based on this hypothesis, I propose a model for the generation of sexual climax.

The LSt cells are recipients of several types of inputs. At the spinal level, they receive sensory input from the pelvis, both direct and indirect. Pleasurable sexual stimulation is excitatory to the LSt cells. Other types of sensory stimulation, such as pain, are inhibitory. The LSt cells also receive inputs from supraspinal sites. These descending inputs are both excitatory and inhibitory as well. The excitatory inputs reflect subjective sexual arousal. The inhibitory inputs reflect processes that limit sexual arousal. In addition, descending monoaminergic projections play a neuromodulatory role on the CPG of the LSt cells. The monoamines have an important role in shaping the output of the CPG and affecting its excitability. Pelvic sensory neurons not only innervate the LSt cells directly, but they also project to supraspinal sites. Pleasurable sexual stimulation contributes to subjective arousal which, in turn, excites the LSt cells by descending projections. This pathway, therefore, provides a positive spinal-supraspinal-spinal loop of arousal. Noxious stimuli from the pelvis would have the opposite effect, such as with pelvic pain. The arousing and suppressing loops are both lost in spinal cord injury.

Under this model, an explanation for the trigger for climax suggests itself. When the LSt cell activity reaches a certain threshold, they activate the CPG, producing coordinated activation of the innervation of the sexual organs, that is, sexual climax. Activation of climax occurs when the synaptic integration of the excitatory and inhibitory inputs from supraspinal sites and from peripheral inputs to the spinal cord reaches a threshold.

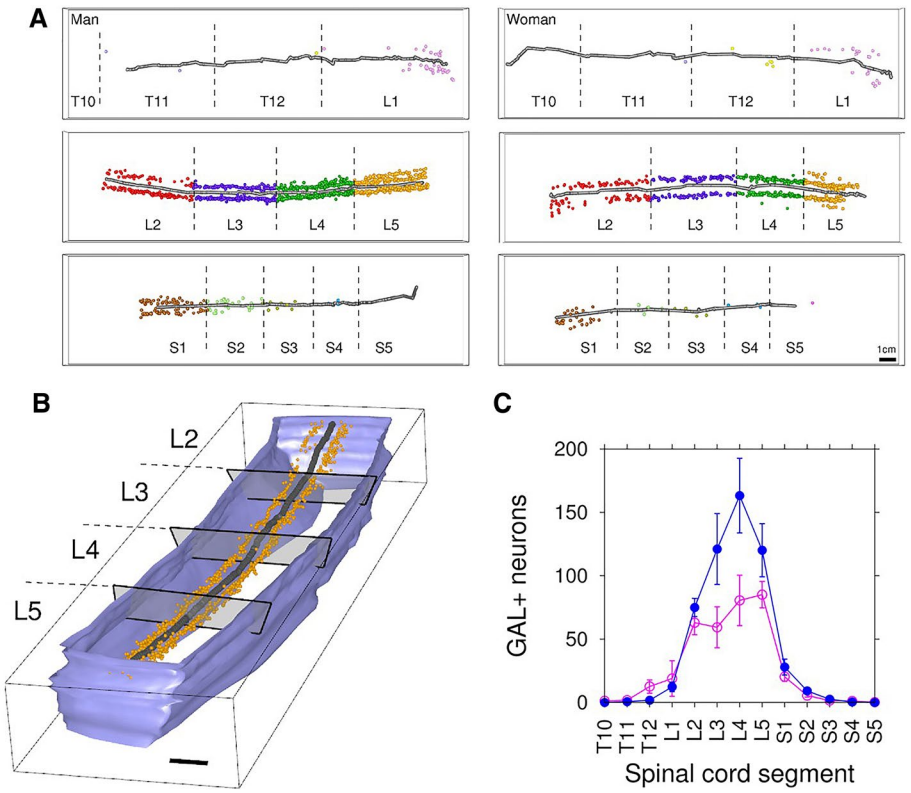

Fig. 6 Segmental distribution of galaninergic neurons in human male and female spinal cord specimens. **a** Left: reconstruction from a male subject. Right: reconstruction from a female subject. Reconstructions were generated from serial longitudinal labeled sections of lower thoracic (top), lumbar (middle), and sacral (bottom) segments. Gray line: reconstructed medial line of the central canal. Colored dots: position of galanin immunoreactive (GAL-ir) neurons. Neuron size was arbitrarily enlarged to enhance visualization. **b** Three-dimensional reconstruction of GAL-ir neurons in L2–L5 spinal segments from a male subject. Scale bar = 1 cm. **c** Average segmental distribution (mean ± standard error of the mean) of the GAL-ir neurons in men ($n = 3$) and women ($n = 3$). (Chéhensse et al., 2017) (Color figure online)

In this model, climax can be stimulated by stimuli, which range from purely spinal to purely cerebral, and combinations in between. Examples of purely cerebral stimuli are nocturnal emissions, imagery-induced orgasm in women (even more rarely in men), and in extreme cases of premature (anteportal) ejaculation. Note, however, that these types of climax are rare. An example of climax induced purely by spinal inputs is climax evoked in men and women with spinal cord injury. Climax can be induced in experimental animal models with complete transection of the spinal cord, as well. However, purely spinal inputs are not very potent in eliciting climax. It appears as if purely cerebral or purely spinal stimuli are not as efficient at generating climax as a combination of stimuli, which is usually the case for almost everyone.

In both humans and animals with spinal cord injury, the pelvic stimuli must be very strong, such as the stimuli used in vibratory ejaculation and the supraphysiological stimuli used in the spinalized rat models of climax. The model predicts this. In severe spinal cord injury, the spinal-supraspinal-spinal arousal loop is interrupted. Therefore, spinal stimuli must be greatly increased to compensate for the loss of descending inputs. Correspondingly, purely cerebral stimuli must be very strong to compensate for the lack of pelvic sensory stimuli. Few people can generate a strong enough central drive to make up for no sexual stimulation.

The activity of the LSt cells is dependent on the descending and segmental inputs. During a sexual encounter, the activity would fluctuate according to changes in central arousal and pleasurable sensations. As central excitement and sexual sensations increase, the activity of the LSt eventually rises high enough to trigger the CPG. That begs the question, what determines the threshold of the LSt cells to elicit climax? That is unknown, and this proposed model is incapable of addressing it. This threshold would be determined by specific details of the LSt CPG. Neurophysiological studies of the LSt cells are necessary to determine both the intrinsic properties of the neurons, their neuromodulation by monoamines, and the dynamics and circuitry of the CPG. The LSt cells are very well suited to physiological studies on spinal cord slices in vitro. Their function can be manipulated by the full array of transgenic manipulations. This approach would be ideal for the identification of therapeutic targets for disorders of climax.

In this discussion, I flew over the data at a high altitude. There are several pieces of the model for which there are gaps in the data, or there is little or no evidence to support it. The arguments rely heavily on analogy and extrapolation. Nonetheless, the result of this speculation is an elegant, unifying hypothesis with vast explanatory power. The model provides a basis for considering climax in men and women, and male and female animals as a unitary neural phenomenon. It puts forward one explanation for climax in everyone. It accounts for the bewildering variety of sensory

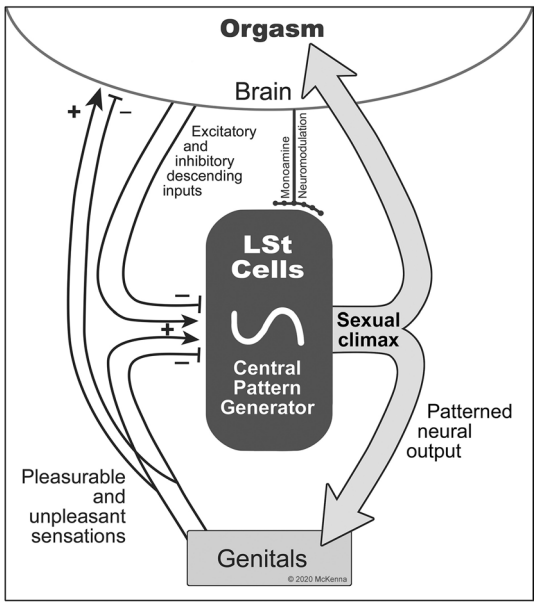

Fig. 7 The Climax Model. Sexual climax is generated and organized by LSt cell central pattern generator (CPG) located in the lumbar spinal cord. The synchronized, rhythmic activity of the CPG evokes the pelvic responses characteristic of sexual climax. The output of the CPG is also transmitted to supraspinal sites to activate the subjective experience of orgasm. LSt cells are the recipients of spinal sensory inputs and projections from supraspinal sites. The spinal sensory inputs may be a variety of pleasurable sexual stimuli or various types of noxious stimuli. Multiple projections from supraspinal sites affect LSt activity. These inputs reflect both excitatory subjective sexual arousal and central inhibitions. The LSt cells CPG is also the recipient of monoaminergic neuromodulatory inputs. This affects the excitability of the LSt cells and shapes the output of the CPG. When the synaptic integration of the aggregate of excitatory inputs minus the sum of inhibitory inputs reaches a certain threshold, the LSt CPG generates sexual climax

and CNS stimuli which have been shown to elicit climax in men and women, and in animals.

The model postulates a highly conserved neural system in both sexes. Male climax, ejaculation, is the indispensable central act of reproduction. To answer the inevitable questions of the purpose of female sexual climax, female climax exists for the exact same reason that male climax does. It is how the central nervous system is wired. Sexual climax is an inherent property of the organization of the spinal cord. The system is indifferent to which set of genitalia it controls.

In hindsight, it should have been obvious that the mechanisms generating sexual climax must be extremely simple. Sexual climax is a vital component of reproduction. The system generating it must have very few moving parts. The irreducible simplicity of the climax model demonstrates this property. In its most skeletal form, you can view the system generating climax as a CPG, which receives inputs from the brain reflecting the behavioral state, and inputs from the genitals reflecting the sensory state. Climax occurs only when both the behavioral and sensory states are strongly sexual. Requiring the alignment of the two states optimizes the chances of successful mating.

This climax model answers the question in the title of the paper. What triggers sexual climax? The answer is sadly anticlimactic. Nothing in particular, and pretty much anything triggers sexual climax. There is no secret place or magical touch that triggers climax. Almost anything can elicit sexual climax, simply by being the final stimulus that causes the threshold to be reached. Supraspinal and segmental inputs converge on the LSt cells. When the summation of these inputs reaches a threshold, the central pattern generator produces the explosive neural discharge of sexual climax. Sexual climax occurs when passion and pleasure embrace. But we already knew that, didn't we?

Acknowledgments This model relies heavily on the work and ideas of others. LSt cells, whose role in sexual function was comprehensively characterized by Lique Coolen, are literally central to the model. This was the key finding to reveal the mechanisms underlying sexual climax. The model depends so much on the work of François Giuliano. His extensive studies of the spinal control of sexual function are foundational to every component of the climax model. It would not have been possible without his contributions. The conceptual perspectives of my dear friends Lique and François have influenced my thinking in unknowable ways over decades. Their ideas are deeply embedded everywhere in the model. I have no idea who contributed what.

Declarations

Conflict of interest The author declares no conflict of interest.

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