Review Article

The Oxytocin Released by the Human Female Orgasm Boosts Sperm Transport to Enhance Fertility- a New Review of an Outdated Zombie Concept

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Abstract

The present review examines the continued claims in a number of published articles that the oxytocin released by the human female orgasm is a component to enhance reproductive fitness by facilitating the rate or the amount, or both, of sperm transported to the ovum. The errors in these accounts, both of omission and commission, which undermine the claimed support for this proposed function of oxytocin are highlighted and discussed. Other functions of oxytocin present better candidates for its possible orgasmic actions.

INTRODUCTION

Alice laughed. 'There's no use trying', she said: 'one can't believe impossible things'.'I dare say you haven't had much practice,' said the Queen. 'When I was your age, I always did it for half-an-hour a day. Why, sometimes I've believed as many as six impossible things before breakfast'.

Through the Looking Glass (1872), Lewis Carroll.

The White Queen's famous quote could not be more apt in relation to the many claims that have been made for the reasons for and function(s) of the human female orgasm [1-27]. Extensive reviews (King and Belsky with 156 references [28], Puts, Dawood and Welling, with 205 references [29]), a book chapter (Wheatley and Puts with 181 references [30]), and an experimental paper (King, Dempsey & Valentine with 117 references [31]), have focussed on the contentious issue of the role of oxytocin released by the human female orgasm facilitating sperm transport. As all have deficiencies of omission and commission, some common, it is convenient to examine them together. The criticism of these reviews is not directly related to the 'Is the female orgasm an adaptation or a by-product' debate (see Symons [4], Lloyd [15,16], Puts & Dawood [31], for such critiques) or 'to argue the intricacies of proximal (the how?) and ultimate (the why?) functions' so beloved of orgasm evolutionists (Scott-Phillips, Dickens & West [32]) but to the underlying claim that it has a specific role in human sperm transport through its release of oxytocin.

THE REVIEW OF KING AND BELSKY

This review [28], purports to examine critically a most highly speculative concept role for oxytocin released at so-called 'deep orgasms' as creating 'sperm selected orgasms' functionally' harvesting sperm from preferred mates' as opposed to those orgasms that apparently do not have this function. The authors did not refer to previous earlier reviews [33-35], that examined in some detail the various claims made in studies by a number of authors they quote that the oxytocin released at orgasm is involved in facilitating sperm transport and that cervical stimulation by penis 'buffeting' occurs in normal coitus. A recent, detailed critique of the latter claim has also been published [36]. All the referenced evidence against these postulations are featured in the above quoted reviews and do not need to be repeated in detail. However, a brief summary analysis of these studies reveals that:-

i) The mechanism of vaginal tenting and ballooning where the utero-cervix complex is elevated up into the false pelvis well-removed from the semen's ejaculatory path and subsequent pool in sexually aroused women in their late excitatory phase was completely ignored. This tenting not only makes penile contact with the cervix highly unlikely in ventral-ventral coitus (by far the most usual position for such activity) but also precludes sperm entry created by any uterine 'upsuck' into the cervical os until the orgasm and its uterine contractions have finished. The utero-cervix then can descend into the liquefied ejaculate or liquefying semen pooled in the ballooned upper vaginal reservoir [2,33-35,37]. In the section of the King and Belsky [28], review headed 'Argument for and against female orgasm as an adaptation' there is strangely neither discussion nor even mention of the fundamental involvement of vaginal tenting on sperm transport.

ii)   None of the published studies that used intra-venous (i.v.), oxytocin injections are ecologically valid as the doses used were grossly larger than those released at orgasm and are thus pharmacological and not physiological [34]. Furthermore, they were injected into women who were in their basal state and not when they were sexually aroused which is the physiological condition the uterus is in when orgasm-released oxytocin is purported to act. The response of the uterus in the two conditions has not been explored empirically and may well be very different (see section vii below).The conclusions from these experiments are thus not comparable with what occurs during normal coitus with orgasm. To illustrate just one example of their extreme lack of ecological validity, after the injection of oxytocin in the experiments of Zervomanolakis, Ott, Hadziomerovic, Mattle, Seeber, Virgolini et al. [38], the contractions of the uterus lasted for 20-40 minutes but those expressed during natural orgasms have durations measured at the most in a few tens of seconds (see [39], for duration of female orgasm in a laboratory study).

iii)   Uterine peristalsis occurs without the need of systemic oxytocin released at orgasm, it is a normal feature of the archimyometrium (the smooth muscle layer immediately beneath the endometrium that continues through to the fallopian tubes) with or without sexual arousal or orgasm. The peristalsis (oestrogen supported) moves and directs sperm presented to the cervical os rapidly through the genital tract into the patent, ipsilateral fallopian tube serving the ovulating and oestrogen-secreting ovary within one minute, even without sexual arousal or orgasm, oxytocin released systemically is not involved. The contralateral fallopian tube is closed preventing sperm entry. Leyendecker, Kunz, Herbert, Beil et al. [40], reported that the archimyometrium was not sensitive to systemic oxytocin while Kunz, Beil, Huppert and Leyendecker [41], found that that prior to ovulation the cervico-to-fundal activity of the archimyometrium reached a maximum in intensity and frequency of contractions that could not be enhanced by injected oxytocin (despite it being an unphysiological pharmacological dose of 3 International Units, see section ii above). It was suggested that the refractiveness of the archimyometrium to the injected peptide was due to locally secreted (paracrine) oxytocin produced by the endometrial cells, a view also favoured by Zingg, Rosen, Chik, Larcher et al. [42]. This conclusion had also been expressed by Kunz, Noe, Herbetz & Leyendecker [43], who observed that during normal cycles as well as during injection of oxytocin the uterine peristaltic contractions were always confined to the archimyometrium (subendometrial muscle layer). Interestingly, the intravenous injection and infusion of a dose of atosiban, an oxytocin receptor blocker that would occupy all the receptors for uterine oxytocin and vasopressin, had no effect on uterine contractions [41]. Despite these findings, Kunz et al. [41], still hedged their conclusions about the role of oxytocin in sperm transport with their statement that 'It seems reasonable to assume that oxytocin plays an important, although not critical, role in the mechanisms governing rapid sperm ascension that at least, in humans were developed to rapidly preserve an aliquot of spermatozoa following intercourse' (but see section v below).

iv)   The claim by King and Belsky [28], that (systemic) oxytocin's '….. role in sperm transport is not questioned 'quotes just two references both for swine [44,45], there are no references quoted for a definitive role in the human female, a not surprising feature because there are none. In fact, its role in the human female has been questioned many times previously [16,20,35,36,46,47]. Care has always to be taken with cross-species comparisons when mechanisms in animals are assumed to also be involved in human reproduction. Reproduction is one of the areas of biology where species differences are often extreme; a mechanism important in one species may be non-existent in others. Levin [13], has reviewed a number of these in regard especially to sperm transport, exploring the concept of 'exceptionalism'.

v)   'Flowback', the term first used by Baker and Bellis [9], to describe the leaked loss of semen and its sperm content from the vagina after its ejaculation and liquefaction, may have to be regarded in a different light. Rather than being thought of as detrimental to reproductive fitness it may actually be an under- investigated important feature of the human female's reproductive response to the male insemination as it is in a number of animals [see 8,9,48-50] as a mechanism to reduce:-

a)   the sperm load entering the fallopian tube thus creating conditions that would decrease the chance of polyspermy (more than one sperm entering the ovum) which is detrimental to foetal development,

b)   ovum degradation by the enzymes shed from surrounding capacitated sperm when their acrosome reactions occur,

c)   Possible sperm competition from serial impregnations by non-preferred mates.

It might seem that the concept of increased sperm numbers creating polyspermy is dependent on animal studies but this is not a valid criticism as there is good experimental evidence for it in humans. The experiments of Wollf, Byrd, Dandekar and Quigley [51], showed that polyspermic fertilisation of human ova was directly related to sperm concentration while Englert, Puisant, Camus et al. [52], explored the factors leading to tripronuclear eggs in *in vitro* fertilisation.

vi)   In regard to women being able to identify specifically uterine contractions during some orgasms but not in others, the anatomical, physiological and psychological issues involved are much more complex than presented by King and Belsky [28], from their limited studies with female focus groups and the answers hoped to be elicited from the questionnaire they then developed. The groups of muscles invested in the pelvis and around internal genitals of the female are namely, i) the three strata of the smooth muscles of the uterus (stratum subvasculare, also known as the archimyometrium, stratum vasculare and stratum supervasculare), ii) the two smooth muscle layers of the vagina (circular and longitudinal) and iii) the striated musculature of the pelvis which is complex. Unfortunately, there is controversy about the description and terminology of the latter [53]. A possible characterisation divides the muscles into groups. One surrounds the vaginal introitus (ischiocavernosus and bulbocavernosus), the second supports the perineum (deep and superficial perinei), while the third consists mainly of the levatorani, a group of muscles forming the pelvic diaphragm [54].

The archimyometrium of the uterine smooth musculature residing underneath the endometrium has 'spontaneous' peristaltic contractions even in the non-sexually stimulated state but these do not register in the consciousness of women. It is thus extremely unlikely that they do during sexual arousal [34]. Unlike the archimyometrium, the outer two uterine smooth muscle myometrial layers (neomyometrium) do not exhibit peristalsis but can be contracted by systemic oxytocin which can cause discomfort and even acute pain if they contract into spasm [2]. It has been suggested that the pain is ischaemic due to nerve stimulation from the induced hypoxia, this also occurs during painful menstrual contractions and during labour. The layers are also neurally innervated by vasoactive intestinal peptide (VIP), which inhibits contraction [55], and an adrenergic supply which is pro-contractile [56]. The balance between these neural inhibitory and excitatory innervations and the action of oxytocin during sexual arousal and orgasm is as yet unstudied and certainly incompletely understood; their balance may be very different in individuals, especially the inhibitory role of VIP (see ii above).The smooth muscle of the vagina is known to contract, especially during menstruation, but these contractions are not normally perceived by women [10,39]. While intravaginal pressure measurements at orgasm appear to show contractions [10], this may be because of the indirect impinging pressure from the surrounding contracting pelvic striated musculature. In [10], it was reported found that i.v. injection of oxytocin (150 milliunits) into 3 young women in their basal sexually unstimulated state did not cause any contractions of the vagina nor in a young woman immediately after sexual arousal. The pelvic striated musculature contract repetitively at orgasm in most but not all women and these can often be consciously perceived, the contractions, however, now appear to be driven neutrally [11,57,58], rather than activated by systemic oxytocin. It is known that individuals have widely different sensitivities to oxytocin and to awareness of internal bodily visceralactivity, often described as interoreceptive awareness [59]). It is essential to clarify whether the women who claim to be aware of what they designate as 'detectable internal spasming' are not actually identifying contractions of their pelvic and perhaps even vaginal musculature to those of the uterus. Moreover, if all these muscle contractions are occurring at the same time, which is likely what happens at orgasm, it is difficult to believe that women can isolate and identify anyone from the others especially when both obnubilation (clouding of consciousness) and genital site pareidolia (ascribing arousal mistakenly to a particular site(s)) occur. This problem is the mirror image of the claim that women can designate different types of orgasm generation by anatomical site which has been described as the 'ambiguity problem'; for details and discussion see Levin [34-36,60]. It is not unknown for subjects to make mistakes about their body reactions at orgasm even under laboratory conditions [61,62], let alone when having to recall their private sexual scenarios later for a questionnaire. Anyone, male or female, who has experienced orgasms, knows that they vary in duration, intensity, pleasure and possible perceived visceral activity even with loved sexual partners. While the greater the emotional bond with the sexual partner the more likelihood of the greater physical intensity of the orgasm [63], however, in long term relationships sexual arousal and desire decreases [64]. Ecstatic sexual excitement and orgasm can occur even from extra-pair sexual liaisons [8].

vii) Are there any physiological mechanisms that could be involved that may allow some women to identify uterine contractions (unspecified as to expulsive or in sucking) at orgasm and not others? One proposed explanation involves the actual amount of oxytocin release dat orgasm (so-called oxytocin-rich orgasm) and the uterine muscle sensitivity to the hormone both of which appear to vary considerably between women. However, even in the few investigations that have measured the amount of oxytocin released during the female orgasm none have correlated it with the subjective intensity of the orgasm and its representation by perceived uterine contractions (the 'internal spasming' of King and Belsky [28]. These are clearly, obvious crucial measurement to be undertaken, without them we are dealing simply with speculation. However, even if some women can identify specifically uterine contractions at orgasm, let alone 'sucking' ones, there still are the troika of outstanding problems that King & Belsky [28], have ignored, namely, that there is simply no unequivocal evidence, experimental or descriptive, that the oxytocin released at orgasm in the human female has any role to play in increasing the rate or the amount in the transport of spermatozoa, or that in the human female either the speed or the amount of sperm carried to the ovum is needful of such an oxytocin boost (see item iv) and the obvious involvement of vaginal tenting in preventing sperm upsuck during orgasm .The 'upsuck by orgasm' has been described as a 'zombie' concept because no matter how much evidence there is against it, it never seems to die [13,35,60]. For such phenomenon, where 'people keep saying the same thing no matter how much evidence accumulates that it is completely wrong'[65], the words 'Derpy science' have been devised [66]. 'Derpy' has been borrowed from the YTC cartoon 'South Park'. On balance 'zombie' is more universally understood than 'Derpy' for such undying phenomenon. Thus it appears that King and Belsky's [28], initial basic hypothesis, that some orgasms supposedly proposed to be oxytocin- rich are 'sperm selective' and occur in females with prized specific partners as opposed to other orgasms (oxytocin-poor?) for less prized sexual partners, floats on a raft of unsupported and invalid mechanistic assumptions.

THE REVIEW OF PUTS, DAWOOD AND WELLING

After concluding from their initial review of the latest literature in the field that the 'female orgasm appears to promote conception', Puts, Dawood and Welling [29], posed two ancillary questions. The first asked 'Why, for example, would *exogenous* oxytocin cause sperm transport towards the oviducts, if this response had not evolved to be triggered by *endogenous* increases in oxytocin such as those accompanying orgasm?'(their italics) followed by the second 'And why would these contractions move sperm specifically toward the oviduct with the dominant follicle during the fertile phase of the ovulatory cycle if not to promote conception (see section iii)? The latter aspect is simply a mischaracterisation of the physiological nature of the effect. Injected oxytocin transports sperm-like particles to the fallopian tube of the ipsilateral active ovary because the fallopian tube serving the contralateral ovary is closed, that of the ipsilateral ovary is open because of the oestrogens secreted by the active ovary. Oxytocin, *per se,* has no magical directive properties for spermatozoa, with only one pathway open (the ipsilateral fallopian tube) any contractions occurring will propel them into it.

Subsequently, the motility of the sperm and the archimyometrium, with the facilitation of chemotaxis, thermotaxis and rheotaxis [67], guide the sperm to the ovum. In fact, these questions had their physiological answers in a previous review [34], and as it has been pointed out 'It is clearly illogical to believe females would evolve a number of methods to reduce the injected sperm load and then simply counter these at the same time with oxytocin released by orgasm to enhance the speed and number transported' [11]. Moreover, it has never been shown that actual sperm are transported by such oxytocin injections only inferred by the fact that Technecium labelled spheres are when placed directly onto the cervical os and using pharmacological doses of oxytocin in sexually unaroused women (see ii above)all features making the experiments ecologically invalid. In answer to the first question, despite the fact that it was clearly demonstrated that none of the studies with exogenous i.v. administration of oxytocin in non- sexually aroused subjects were ecologically valid or in any way equal to the endogenous release of oxytocin(see ii above and the review [34]), and that Puts et al. [29], accept 'that this criticism is valid' they still do not acknowledge the fact that during coital arousal in the female, vaginal tenting will remove the cervical os from any contact with the ejaculated semen (and thus any sperm uptake by uterine in suck) until after the female orgasmic contractions have taken place (see viii, the timing of coital orgasm[9]). Sperm transport then likely occurs through peristalsis of the uterine archimyometrium (see iv above) within a minute or so, but there is even some evidence that post-orgasm the motility of the uterus/fallopian tubes are actually inhibited (see viii and Levin [34], for details). In this context Fox, Wolff and Baker [68], in their measurement of uterine motility during and after orgasm, state 'It is possible that the regular uterine contractions, which may occur irrespective of orgasm, and in our tracings are abolished by orgasm'. A previously posed question (Levin [34]) 'what is the likelihood, given such a rapid transfer mechanism, that there would be an evolutionary drive for the development of an oxytocin mechanism which could hardly be much faster?' has not been answered. In fact their first question can be reversed to state "Why would there be a need for oxytocin when within one minute after the resolution of genital arousal the cervix enters the now liquefied or liquefying semen pool and especially with the female's need to reduce sperm loading in the fallopian tube in relation to polyspermy and sperm-released lytic enzymic ovum degradation both reducing fertility (see v above)'. As has been emphasised [34], there is' no data that can support this claim that the sperm transported by the archimyometrial peristalsis….is needful of a boost to ensure reproductive success'. Puts, Dawood and Welling [29], have never been able to quote any published experimental evidence that confirms this need, surely a crucial and essential preliminary requirement for any enhancing mechanisms to be proposed. Indeed, as previously stated, there is good evidence that females try to reduce the ejaculated sperm load.

It is interesting to note in the context of the questions that while oxytocin is implicated in a number of animals in relation to sperm transport as Puts et al., have referred at length but they do not quote that oxytocin-deficient mice and oxytocin receptor knock-out mice are quite capable of reproducing normally [69]. These studies indicate the non-essential role of oxytocin in the mating and progeny of mice.

Puts et al., state that 'sexually antagonist by-products often appear reduced or rudimentary if selection cannot eliminate them entirely. Such reduction is apparent in male nipples'. The claim that the male nipple is functionless in human sexual activity has been voiced previously by a number of authors despite lack of supporting studies [4,70]. However, it is just an opinion as is obvious in that the statement by Puts et al., [29] has no reference quoted in support. In fact the authors have ignored the study by Levin and Meston [71], that asked young male (n=148) and female (n=153) university students the effects of nipple and breast stimulation in their sexual activity. In regard to the women, 81.5% reported that stimulation of their nipples/breasts caused or enhanced their sexual arousal, 78.2% agreed that when sexually aroused such manipulations increased their arousal and 59.1% had asked for their nipples to be stimulated during lovemaking. In regard to men, 51.7% reported that nipple stimulation caused or enhanced their sexual arousal, 39% agreed that when sexually aroused such manipulations increased their arousal and 17.1% asked for their nipples to be stimulated. This was the first study that actually compared nipple/breast stimulation in both males and females and indicated that the nipples of men appear far from vestigial and functionless in over half the males surveyed. The study, moreover, highlights the dangers of speculating on biological sexual function rather than investigating it, a situation similar to speculating on orgasm function and sperm transport.

'FEMALE ORGASM MAY PROMOTE CONCEPTION' −THE CONTINUING BIAS OF WHEATLEY AND PUTS

In Wheatley and Puts's chapter the 'Evolutionary Science of Female Orgasm' in the recent book 'The Evolution of Sexuality' [30], one section heading, 'Female orgasm may promote conception' underscores the authors' obvious bias about the functions of the oxytocin released at orgasm. It repeats much of the claimed evidence for this conclusion that has been previously published in the review by Puts, Daword and Welling [29], criticised above, with a few new suggestions for oxytocin's involvement in sperm transport added. They do this by asking a pointed question and then supplying their answer. They state, 'If orgasm arose in females as a by-product of male orgasm and was subsequently modified for a special function in females, what might that function be? Some of the *strongest evidence* concerns the potential of women's orgasm in increasing the likelihood of conception' (my italics added).

In their section headed 'Female Orgasm may promote conception' errors of omission and commission are present. Briefly these are:-

i)   Quoting only the controversial claims of Brody [72], and Brody and Costa [73], that there were psychological and other differences between women who report having 'vaginal' versus 'clitoral' orgasms without balancing them against the many specific criticisms of these studies by Levin [34-36,60], Prause [74,75], Laan and Rellini [76], which are now further supported by Prause, Kuang , Lee & Miller [77],Therrien and Brotto [78], and Brotto [79].

ii)   The sole use of the report of Komisaruk et al. [80], to describe areas of the female brain activated during orgasm. It has

been previously pointed out that this study suffers from a number of weaknesses that make it ecologically invalid. It used inanimate rods to stimulate the cervices of the subjects which is not only a supra-normal level of stimulation but also one that does not normally occur during natural penile vaginal intercourse (PVI) because of vaginal tenting [59], the few subjects employed were not able-bodied women but those with complete spinal lesions which is known to change the sensitivities of various structures [36] and has various technical shortcomings according to Georgiadis [81]. Moreover, it is not mentioned that there is no consensus among brain imaging studies as to what areas of the brain are activated, inhibited or unchanged during sexual arousal to orgasm.

iii)   The electrical brain stimulation papers quoted that created uterine motility were undertaken in anaesthetised cats by Beyer et al. [82], while the quoted Setekliev [83], paper is an incorrect reference that describes the motility of the uterus to distention not to brain stimulation. The correct reference is that of Setekliev [84], who used anaesthetised, artificially oestrogenised rabbits. Results obtained using such experimental conditions in animals can be very different from unanaesthetised, natural human PVI.

iv)   The statement that 'oxytocin induces peristaltic contractions of the uterus ' quotes for support studies that have been shown to use pharmacological doses of oxytocin far greater than the concentrations secreted into the systemic circulation of females during orgasm as described previously in the above critique of the Puts, Dawood and Welling review [29]. In fact these authors actually agreed in their review that such 'criticisms were valid' a conclusion that Whealey and Puts now appear to have overlooked. Again, to stress the obvious lack of any ecological validity of these experiments with oxytocin injections the quoted Zervomanolakis et al ., paper [38], reported that their oxytocin injections caused uterine contractions for 20-40 minutes but those during natural orgasms last at the most for a number of seconds (see section ii in the above critique of King & Belsky's review [28]).

v)   They ignore the problems involved in sperm uptake from the vagina dealt with in some detail in [34]. If it is due to uterine orgasmic contractions from the oxytocin released at orgasm there will be a bulk or 'en bloc' transport of the ejaculate into the cervical canal which would show up because all types of cells would be present in the cervical fluid (dead and deformed sperms, non-motile sperms, vaginal squamous cells and male urethral cells).This, however, is not the case indicating the unlikelihood of such transport [34].

Despite Wheatley and Puts [30], confessing that 'we may never be able to definitely state that the female orgasm is an adaptation as opposed to an exaption or by product. However, taken together the last several decades researches have produced a great deal of evidence that has advanced our understanding of the phenomenon. Taken together the data strongly suggest that the female orgasm serves a reproductive purpose'. Their final conclusion is that ' Oxytocin appears to play a dual role in orgasm, first facilitating its occurrence, then surging at the orgasm peak to activate the mechanisms that transports sperm toward the ovum and perhaps to facilitate the pair bond'(which actually describes a triple role!). A simple question arises from this latter suggestion namely 'How can oxytocin (presumably systemic?) facilitate the occurrence of orgasm if it is not released until the orgasm occurs?'

THE REVIEW OF KING, DEMPSEY & VALENTINE

The review of the role of oxytocin in human sperm transport that prefaces the experimental study of the possible role of uterine orgasmic upsuck contains a number of errors of omission and commission, these and other features have been discussed by Levin [13], and they will not be repeated here. The study was described as measuring 'sperm backflow' following female orgasm but this is an inaccurate description because what they actually tried to measure was not 'sperm backflow' but the uptake of a fluid inserted into the vagina. Their attempt to mimic the claimed orgasmic-induced uptake of semen in their pilot experimental study used 6 females who induced the so-called sperm-retaining 'deep orgasms' [28], at their homes using a powerful vibrator and compared these with sexual arousal induced without orgasm. However, the fluid inserted into their vaginas was not the human ejaculate but a commercially available lubricant (Liquid Silk) used as a proxy to act as a stimulant fluid for the human semen. This stimulant fluid contains substances not found in the ejaculate and lacked any of the active agents known to be present in the ejaculate and any particulate content to represent the spermatozoa [13].

As in so many undertakings, especially those experiments conducted at home without laboratory supervision, the devil is in the detail. Relying on the subject's self-reports is not evidence, as has been criticised in other situations [85]. King, Dempsey and Valentine [31], have assumed that it is only the orgasm-induced contractions that can cause the fluid's uptake. Other explanations could account for the relatively small different uptakes. For example , while the duration that the fluid was left in the vagina after the arousals before its final collection and removal was said to be about 2 hours but this time maybe crucial as the uptake of the fluid by spontaneous uterine peristalsis via the activity of the archimyometrium will be in operation. The induction of the orgasms will end the arousal and the vaginal tenting [2], so the cervix will then quickly descend into the fluid but in the case of the sexual arousal without the orgasms a longer period will occur before the descent of the cervix into the fluid. Thus more fluid will appear to be taken up after the orgasm scenario than with the arousal alone scenario. This may explain their finding that on average some 15.7% more liquid stimulant, a small to medium effect, was retained in the orgasm condition compared to that in the no orgasm one. It could be an experimental artefact. Another protocol weakness is that no control recovery experiments were conducted on instillations of the 5 ml stimulant in the sexually unaroused vagina, full recovery of fluid from the vagina is notoriously difficult because of its structural rugosity. Because of the very restricted selection of participants, their small number, the use of a stimulant fluid as a semen substitute without particulate matter, the induction of orgasm by vibrator and unsupervised timings the ecological acceptability of this study and its result is open to serious question.

It should be noted that this was not the first time that investigators have tried to study experimentally the uptake

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of a 'simulated' ejaculate from the vagina during orgasm but unfortunately all have been shown to have flawed features and not to be ecologically valid. Grafenberg [86], and later Masters and Johnson [2], both used a radio-opaque fluid, the latter however, was matched to many of the characteristics of semen but with no particulate content to match the sperm. It was held in a cervical cap against the cervix but neither found any uptake into the cervical canal or uterus with coital (the former) or self-induced orgasm (the latter). Critics claimed that the caps were too tight and occlusive and prevented such uptake. Wildt, Kissler, Licht, Becker [87], used non-sexually aroused infertile patients to explore the uptake of radioactive-labelled microspheres similar in size to spermatozoa placed in the posterior vaginal fornix with the intravenous injection of pharmacological doses of 3 IU of oxytocin in non- aroused subjects. The problems with this study are discussed in section ii in the review of King & Belsky [28].

FINAL COMMENTS

An obvious fundamental flaw in all the evolutionary–based above reviews despite their extensive references is that none of them offer any hard evidence that spermatozoa in the human ejaculated semen after coitus need any facilitation by orgasm-released oxytocin of their transport to the ovum as opposed to their own inherent swimming ability and the independent -of-orgasm contractions of the uterine archimyometrial smooth muscle layer [34,47]. However, there is good evidence that hastening their transport can create premature arrival of uncapacitated spermatozoa unable to fertilise ova and that too many spermatozoa around the ovum can inhibit fertility by polyspermy (multiple sperm entry into the ovum) and ovum degeneration by the local release of enzymes during their acrosomal reaction [35].

While the complexities of the possible functions of oxytocin in women are well-illustrated by the studies of Taylor, Saphire-Bernstein and Seeman [88], who found that the hormone was elevated when there was distress in their pair –bond relationship while the contradictory finding that the exogenous administration of oxytocin reduced psychological and biological stress responses [89]. Such differences might well depend on the level and the duration of oxytocin created in the blood stream. The anxiolytic functions of oxytocin [11], and other affiliative functions of orgasm [74], present better candidates for its possible orgasmic actions.

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