3.2.2.4.2 Dopaminergics

3.2.2.4.2.1 Sexual enhancement approaches

Pfizer's Blue is a medication specifically for erectile dysfunction. It will do nothing for other aspects of good sexual function: libido (desire, arousal, excitement), orgasm, and ejaculation.

Erections and libido are interconnected, but they are also amazingly independent from each other. Erections are mostly a matter of the vascular system. When the parasympathetic nervous system is in control, blood flow is directed to internal functions such as digestion, as well as to reproductive availability (the facilitation of erections). Partially, this works through the regulation of the adrenergic system (epinephrine and norepinephrine, which, at the same time, work as hormones and neurotransmitters). Adrenergic impact usually causes vascular constriction (making erections impossible). Yohimbine is a peripheral alpha-2-adrenergic receptor blocker, which means that it prevents the adrenergic hormones' effects on alpha-2-adrenergic receptors, which are mainly located in the abdominal and genital areas. If the vasoconstrictive impulse of adrenergic hormones in the abdominal and genital areas is inhibited, erections happen with ease.

But in the human body, there are usually alternative avenues to achieve a desired effect. The efficacy pathway of Pfizer's Blue is very different from that of yohimbine.

Pfizer's Blue works on an enzymatic level. It suppresses the enzyme phosphodiesterase type 5 (PDE5), which naturally occurs in erectile tissue. Phosphodiesterase type 5 (PDE5) breaks down the body chemical known as cyclic GMP. Cyclic GMP is produced during arousal and causes muscular and vascular changes, which lead to an erection. Men who don't produce a sufficient amount of cyclic GMP will have problems achieving an erection, and men with high levels of the enzyme phosphodiesterase type 5 (PDE5) will have problems maintaining one. In both cases, Pfizer's Blue provides a solution by keeping phosphodiesterase type 5 in check.

This has little to do with libido (desire, arousal, and excitement), orgasm, and ejaculation.

Libido is to a certain extend, dependent on testosterone, kept in a fine balance with a number of other hormones. When testosterone is elevated, sexual fantasies are more daring, and they occur at a higher frequency.

The most dramatic effect of elevating testosterone is seen in men who have clinically low testosterone levels, caused by non-functioning testes or pituitary tumors.

However, in men in whom impotence is caused by problems of the vascular system in the genital area, and not by endocrine insufficiencies, a phosphodiesterase type 5 inhibitor will work much better than testosterone therapy.

But the diagnosis of impotence is usually obscured by the fact that there is a parallel decline of a number of bodily functions. For example, men with vascular problems may also have low testosterone levels. While declining testosterone levels may play a role in declining health, it is not that low testosterone levels are the direct cause of vascular problems in the male genitals. The two syndromes just tend to emerge at around the same time.

A lack of capability to achieve an erection, or generally weak erections, are the symptoms by which the affected male usually defines impotence. Which is why “erectile dysfunction” is a more precise term for the condition he primarily wants ameliorated.

Pfizer’s Blue can take care of the erectile dysfunction. While men on phosphodiesterase inhibitors can have erections all right, sex still is not the same as it was in their 20’s. They become aware of the fact that their libido, too, isn’t what it used to be.

Working on one’s testosterone levels can have a distinct positive effect on libido, but not all possible methods work equally well.

My own experience with direct testosterone replacement therapy (Andriol, Proviron) as well as an indirect approach via the hypothalamus and pituitary glands (clomiphene, anastrozole) is that it is hard indeed to differentiate whether all of this does any good.

The connection between testosterone and libido is also not as obvious as the one between phosphodiesterase inhibitors and erections. It's not that one could just apply some testosterone (as gel, patch, or injection) or take a medication that activates testosterone synthesis, and an hour later, one would be ready with heightened libido. If you do take medications to elevate testosterone levels, you can never be quite sure whether and when an effect will kick in. On a regular regimen of testosterone elevation, there may be situations, randomly occurring, when sexual fantasies will, rather suddenly, occupy one's mind.

I do not doubt that elevating testosterone has an anabolic effect. Proviron (toxic to the liver) and tongkat ali can make for quicker muscle gains during a weight exercise program, and help with body fat control. And for men grossly deficient in testosterone (hypogonadism), rectifying testosterone supply can be a great help for sexual function. But as a treatment for standard, age-related impotence, or for plain sexual enhancement, just supplying the body directly with testosterone (e.g. with testosterone patches) has practically no value.

Try another approach.

Libido is a mental and neural affair. And while hormones such as testosterone do work on the frame of mind for sex, we have to be aware that evolution has designed humans as an animal species that is primarily guided by sensual input (mainly sights and sounds) and the cognitive processing of this sensual input.

Which is why the right kind of cognitive processing has the greatest potential to positively affect libido. That sounds like a job for a psychologist. But going to the shrink may not be the right move.

The function of psychologists in modern society is to make us good members of this society, so that we won't cause any problem, neither to public security, nor to the public health system. It's not their aim to make us ready to pursue the ultimate sexual experience, as this may, when multiplied by hundreds or thousands of men, result in all kinds of social and public health problems. The shrink will condition you to be a monogamous family man, not a wild playboy.

I guess that most of us have clear evidence from own experience on how the right sensual input and the right cognitive processing work wonders on libido. Usually, new sensual signals are more powerful than repeat signals. Which is why our libido usually is stronger when with a new partner. Also, forbidden sensual input, and cognitive awareness that it is forbidden, have a stronger impact on libido than sanctioned or routine sensual input. Which is why, so often, boredom rules conjugal bedrooms.

Need an example for the libido power of cognitive processing? Take jealousy. It's all just perceptions. No medication, no therapy needed.

If your wife loves, or makes love with, another man, your blood will rage, and you will want to establish your rights by sexual penetration. Just imagine how she goes along in bed with her lover, and your ears will feel hot, and your loins ready.

Because jealousy has such a profound effect on libido, and because heightened libido is such a gratifying state of mind, I have been working for years on my own personal strategy of supplying just the right amount of jealousy to my daily love life. This, of course, requires a special kind of relationship. It's not sufficient that my partners give me reason to be jealous by having, or having had, relationships with other men. I must also first perceive a particular partner as my property. Which means that I will have to invest in her, mentally and probably also economically.

But then, I would not want to do so with just any girl with whom I may have casual sexual intercourse. I also have to be free from other close personal relationships.

If I do want to harvest the positive effects of well-dosed jealousy, the person who makes me jealous will have to be the main focus of my love life. Which, in turn, is why it is difficult to be made jealous by more than one woman at a particular period of time.

Jealousy is strong medicine. It can raise libido to previously unknown levels. It's great for sexual enhancement. If only we were able to control its supply to our minds in the same way as we can control the supply of erection medications to our bodies.

Close your eyes and imagine your wife loving, and making love with, another man. Don't feel anything? The point is, you can't deceit your cognitive apparatus. Your mind cannot be aware of a perceived unfaithfulness of your wife just because you willfully want to perceive it. It won't work. It will have to be real. And when it's real, then too, it can be out of control.

Isn't there a simpler method? A pill to swallow for better libido.

You could try Dostinex (generic name: cabergoline). Or, in a broader sense: dopamine agonists. But don't expect too much.

Dostinex is a new drug. But dopamine agonists have been around for many decades, and their pro-libido effect is well established. Apart from cabergoline, the assortment of dopamine agonists includes bromocriptine, pergolide, pramipexole, lisuride, apomorphine, and a few more. Actually, apomorphine (brand name: Uprima) is sold in Europe as a medication for erectile dysfunction.

But it's wrong marketing. Dopamine agonists don't work for erections as reliably as phosphodiesterase inhibitors. They work on libido. Therefore, Uprima typically is a disappointment for men whose problems are primarily vascular. I assume that Uprima is sold as a medication for erectile dysfunction mainly because erectile dysfunction meanwhile is an accepted medical condition, while low libido is not.

I have been using dopamine agonists for sexual enhancement for several years. And not only dopamine agonists.

To summarize my observation: while Pfizer's Blue and yohimbine work on erections, and while elevating testosterone levels has an effect only when I do it with tongkat ali extract, some dopamine agonists can be used, on a limited scale, for enhancing libido and orgasm.

Because dopamine agonists (including tongkat ali) suppress the hormone prolactin, which in turn suppresses testosterone, dopamine agonists can, in people with elevated prolactin levels, function in the same way as a testosterone replacement therapy would. This most clearly happens in patients with pituitary cancer, which typically expresses itself in strongly elevated prolactin levels. Those afflicted by the disease have very low testosterone levels. Thus, for them, Dostinex and other dopaminergic agents work as hormonal therapy. The hormonal effects of Dostinex (cabergoline) are less extreme in healthy subjects.

Dopamine agonists not only strongly support libido; they also tend to enhance orgasm and make for a stronger ejaculation.

But dopamine agonists have their downsides. All the older ones can cause bad nausea.

3.2.2.4.2.2 Dopamine agonists

Dopamine agonists have been around for many decades, and their pro-libido effect has been known for a long time. The assortment of dopamine agonists includes bromocriptine, cabergoline, pergolide, pramipexole, lisuride, apomorphine, and a few more.

Apomorphine (brand name: Uprima) was sold in Europe as a medication for erectile dysfunction. But it was wrong marketing. Dopamine agonists don't work for erections like phosphodiesterase inhibitors. They work on libido, at least for some people who use them.

Uprima typically is a disappointment for men whose problems are primarily vascular. Uprima was sold as a medication for erectile dysfunction mainly because erectile dysfunction is an accepted medical condition, while low libido is not.

While sildenafil citrate and yohimbine work on erections, and while elevating testosterone levels is of an unpredictable pro-sexual nature, dopamine agonists can enhance sexual excitement, though also not reliably.

Because dopamine agonists suppress the hormone prolactin, which in turn suppresses testosterone, dopamine agonists can, in people with elevated prolactin levels, function in the same way as a testosterone replacement therapy would. This mostly happens in patients with pituitary cancer, which typically expresses itself in strongly elevated prolactin levels. Those afflicted by the disease have very low testosterone levels. Thus, for them, dopaminergic agents work as hormonal therapy. The hormonal effects are less pronounced, or totally absent, in healthy subjects.

3.2.2.4.2.3 Ergot – a plant poison used for medications that enhance sexuality

Ergot is a fungus that lives on rye and other grasses and is pathogenic to its host as well as to humans and other animals that ingest it. Ergot is also a great source for the art of healing and the pharmaceutical industry.

Like most great pharmacological resources, ergot is a powerful poison. More specifically, many ergot alkaloids have a poisonous effect on the central nervous system, interfering heavily with neurotransmitter function. And here also lies the great promise of ergot as medicine.

Ergot is an old member of the materia medica. It has been used in traditional medicine and it has been scientifically studied for more than 50 years. Among those studying ergot and its derivatives was the Swiss chemist Albert Hofman whose experiments led to the discovery of LSD, an ergot derivative that strongly interferes with the neurotransmitter serotonin.

In the field of conventional medicine, ergot derivatives are nowadays mostly used for their potential to enhance another neurotransmitter, dopamine. A dopamine deficiency is a common grave medical condition, Parkinson's Disease.

While the ergot derivative LSD is used almost exclusively as recreational drug with practically no use in conventional medicine, dopamine enhancing ergot derivatives are sold in pharmacies around the world. The most common ergot prescription drug is probably Sandoz' Parlodel (bromocriptine by generic name). Even though it's very much a conventional medication, bromocriptine and other dopamine enhancing ergot derivatives have a clear potential as life-style drugs. Not all, but many ergot-based medications for Parkinson's Disease have a profound sexuality enhancing (side) effect.

All dopamine-enhancing medications can be used in the treatment of Parkinson's Disease, but not each and every dopamine enhancement produces pro-sexual (side) effects. On a similar level, while many medications used for serotonin enhancement (in the treatment of clinical depression) have anti-sexual effect, this anti-sexual effect is not an unavoidable side effect of serotonin enhancement.

The answer to the puzzle lies in dopamine and serotonin receptor sites. Not all dopamines and all serotonins are alike. The effect of some dopamine binding to specific sites is pro-sexual, and the binding of some other dopamines to other sites may be neutral at best, or even anti-sexual. The same holds true for serotonin enhancing drugs and serotonin binding sites.

Until now, many ergot derivatives are considered "dirty" drug. They are named like this because their action is not all too specific. They have the therapeutic effect for which they are prescribed, but they have many other effects, too. From the perspective of conventional medicine, the pro-sexual effects are a side effect.

3.2.2.4.2.4 Prolactin, the multiple culprit

This article builds on the scientific hypothesis (or, for the less scientifically minded, the assumption) that the hormone prolactin is a multiple culprit.

Obviously, prolactin also has its useful sides. As its name indicates, it is essential to initiate and maintain lactation in women who are about to give birth, or have been given birth. It also stimulates the secretion of progesterone, which has, as this hormone's name indicates, an important function in gestation.

Other effects of prolactin are less useful to modern man (which, grammatically and linguistically correct, includes the females of the species). I am referring to prolactin's power to down-regulate the effect of several other important hormones (especially testosterone), as well as some neurotransmitters (such as dopamine).

Prolactin is the ultimate sex drive killer. Women who have given birth, and men who have been the sexual partners of such women, know that a woman's sex drive can almost completely disappear for some time after having given birth and as long as breast-feeding is maintained.

And it is no secret to the medical community that the sex drive of men and women who suffer from a prolactinoma (cancer of the pituitary gland that causes gravely increased output of the hormone prolactin) is practically nil.

While only a certain percentage of all people suffer from prolactinomas, there is a clear tendency for almost everybody to have gradually increased levels of prolactin levels as one progresses in age. As is determined by our evolutionary development, it is best for the human species in its fight for survival (against other species) when species members of the age between puberty and, let's say, 30 are those who normally produce offspring.

Therefore, it should come to no surprise if physiological traits have developed over time which assure that younger members of the species are biologically better positioned to produce offspring than the older generation. Increased prolactin levels as we age certainly are one mechanism that works to this effect.

But what is best for mankind is not best for each and every individual. By means of genetic make-up, we, as individuals, are designed to vacate the surface of this planet when we have reached an age of, normally, less than 100 years. Crocodiles and parrots, and especially turtles, live much longer, up to hundreds of years, and some trees make it well beyond 1000 if they aren't murdered by man.

There is no logical reason why we should not imagine to live for thousands of years, once we have defeated mischievous mother nature, who, among other tricks, uses prolactin levels, which increase with age, to make sure that we don't compete with younger generations for the right and pleasure to have sexual intercourse. Prolactin's role, apart from initiating and maintaining lactation, clearly is one of down-regulating sexual instinct.

It doesn't do so only by interfering with testosterone. High prolactin levels are also reversely related to our sense of well-being, or directly related to depression. There is a correlation between sufficiently high testosterone levels and sufficiently high levels of the neurotransmitters dopamine.

When in the US, Andrea Yates murdered her children, the term "post-partum depression" became a household word. Her irrational behavior was linked to this condition. Post-partum depression is not uncommon, though it doesn't usually lead to infanticide. And it develops parallel to increased prolactin levels, necessary to initiate and maintain lactation.

I have mentioned initially that this article is based on the scientific hypothesis, or the assumption, that prolactin is a multiple culprit in age-related sexual dysfunction. I am not preaching this theory as the final truth.

However, in order to progress with cognition, we will have to proceed through hypotheses. Hypotheses are based on circumstantial evidence. And there is plenty of circumstantial evidence that indeed, prolactin regulation is the key to many conditions of sexual dysfunction.

Strong circumstantial evidence also is provided from the use, and experimentation, with medications that effect prolactin levels. Lowering prolactin levels is commonly achieved with dopaminergic medications, used in the treatment of Parkinson's. Some of these medications, such as lisuride, cabergoline, bromocriptine, and L-dopa have the reputation to be useful for sexual enhancement.

I have tried some of these, and I definitely cannot recommend them for casual use. Some of them make healthy people feel terribly nauseated.

Probably the most gentle way to reduce high prolactin levels in men and women is via tongkat ali.

Tongkat ali (Eurycoma longifolia by scientific name) has been shown in several scientific studies to lower prolactin. Everybody can search for such studies via: scholar.google.com

Tongkat ali prolactin-lowering effect, of course, is a direct result of tongkat ali's capability to increase testosterone. Testosterone and prolactin are reversely corelated. When one is up, the other is down.

After years of experimenting, I now think that very short cycles of tongkat ali usage (3 days on, 2 days off) work best for most people.

The aim should be to throw a permanently high prolactin level or permanently low testosterone level off balance.

What a human body considers an age-related testosterone and prolactin homeostasis is genetically set. For practically everybody, the bias is towards testosterone in his or her youth, and towards prolactin at a later age.

It is unlikely that any medication or herbal will tilt this balance permanently. Even the most powerful Parkinson's medications work well just a few months.

What we can achieve, though, is to disturb the balance of sluggishness. Then we can have high testosterone levels, and low prolactin levels at least sometimes.

And during these episodes of low prolactin, we can have the most satisfying sexual experiences of our lifetimes.

Prolactin levels may return to their high baseline the next day, or even after a few hours. This does not matter. Because the memory of an extraordinary, really satisfying sexual experience can give happiness many days or even weeks.

When one selects tongkat ali as a route to control high prolactin levels, one should be aware of the following: when tongkat ali consumption increases testosterone, the body will answer with synthesizing more prolactin. In some individuals, this effect will set in rather quickly, so that they may not even feel the elevated testosterone.

But when the tongkat ali is all of a sudden withdrawn, that same body (actually the pituitary gland) will notice too high prolactin, and reduce production.

In such a therapy, the body will likely overshoot targets. Whether during on cycles or off cycles, there will be prolactin peaks and prolactin valleys.

During these tongkat ali-induced prolactin valleys, this is when we can experience orgasms as they would otherwise not be possible (and many female users have their first orgasms ever). For some two third of users, these orgastic events happen during short, 3-day on-cycles. For about one third of tongkat ali users, the prolactin lows occur during off cycles, as a response to the tongkat ali withdrawal, when the pituitary also radically cuts prolactin synthesis.

Even if this radical prolactin low happens only once or twice a month, a few occasions of the best sex ever have a more positive effect on our well-being than boring sex every night.

3.2.2.4.2.5 Bromocriptine

Bromocriptine is a well-established drug for two conditions, increased levels of the hormone prolactine and parkinsonism. The best-known brand name is Parlodel. Bromocriptine also has a sexuality enhancing effect, though it is not commonly sold for that purpose. Nevertheless, there is little doubt that in many people, bromocriptine will increase sexual response.

The sexually enhancing effect of bromocriptine is very different from the effect of sildenafil citrate. The sildenafil works primarily on the sexual organ, providing chemically for better rigidity, or some rigidity in the first place. Bromocriptine, on the other hand, primarily works on the brain, making a person more receptive for sexual stimulation and creating a frame of mind for more powerful orgasms. Both effects are a logical consequence of the way, bromocriptine is traditionally used to lower levels of the hormone prolactin, and to increase levels of the neurotransmitter dopamine.

High levels of prolactin are associated with a decreased sex drive. So, by lowering levels of prolactine, especially when they are high, bromocriptine is regularly credited with increasing the interest in sex.

While the increase in sex drive caused by bromocriptine may be hard to measure, the effect on orgasms is more obvious. A considerable number of people who have tried bromocriptine have reported that orgasms become more powerful ironically because they are better controlled. There may be several almost-orgasms before the real orgasm happens, and the real orgasm may be accompanied by a histamine reaction which is more clearly felt (stuffed nose).

3.2.2.4.2.6 Sexual enhancement with bromocriptine

Bromocriptine has been the first dopaminergic medication I have been using for sexual enhancement. And indeed, bromocriptine can be a definite enrichment to the sex life especially of a novice user. The main disadvantage of bromocriptine is that it will only work well for sexual enhancement for 20 or 30 times. Already after the 3rd usage, the effect wears off.

Unfortunately, increasing the dosage to compensate for the wear-off effect does not work well for bromocriptine. Higher dosages of bromocriptine will likely cause nausea… a feature bromocriptine shares with all pharmaceutical dopaminergics.

3.2.2.4.2.7 Bromocriptine not just for Parkinson's disease

In view of the enormous marketing success of Pfizer's Blue, many pharmaceutical companies may be tempted to distribute substances that could be proven to enhance sexual response. However, for old drugs, the patents of which have expired, there is little incentive to invest into the necessary clinical trials.

The sexually enhancing effect of bromocriptine is very different from the effect of phosphodiesterase inhibitors. The phosphodiesterase inhibitors works primarily on the sexual organ, providing chemically for better rigidity, or some rigidity in the first place.

Bromocriptine, on the other hand, primarily works on the brain, making a person more receptive for sexual stimulation and creating a frame of mind for more powerful orgasms. Both effects are a logical consequence of the way, bromocriptine is traditionally used to lower levels of the hormone prolactin, and to increase levels of the neurotransmitter dopamine.

High levels of prolactin are generally associated with a decreased sex drive. So, by lowering levels of prolactin, especially when they are high, bromocriptine is regularly credited with increasing the interest in sex.

A similar effect is achieved by bromocriptine through the neurological route. Bromocriptine is used as a medication in Parkinson's because it will cause higher levels of the neurotransmitter dopamine. Parkinson's is a disease caused by dopamine levels that are too low. Low dopamine levels normally also cause a loss of interest in sex, and an increased sex drive is a common "side effect" of many Parkinson's medications. But one person's side effect is another person's cure.

While the increase in sex drive caused by bromocriptine may be hard to measure, the effect on orgasms is more obvious. A considerable number of people who have tried bromocriptine have reported that orgasms become more powerful, ironically because orgasms are better controlled. There may be several almost-orgasms before the real orgasm happens, and the real orgasm may be accompanied by a histamine reaction, which is more clearly felt (stuffed nose).

Bromocriptine is a prescription drug most everywhere, though in many countries of the world, prescription drugs can be bought over-the-counter. In countries where prescription drugs are indeed only sold on prescriptions, it is within a physician's discretion to prescribe a drug for conditions for which it has not originally been approved.

For a substance to be approved as a medication, an illness has first to be defined for which it is a cure. Nowadays, there are many newly defined illnesses, such as clinical depression, attention deficit disorder, erectile dysfunction conditions, which have previously not been considered illnesses but just part of the individuality of a particular human being.

Some members of the species are smarter than others, and some are happier, and some of the males are more virile than their neighbors. Not to be as smart as a genius, and not to be as virile as one's neighbor aren't diseases in the classical sense. But new illnesses are constantly defined, primarily when the pharmaceutical industry has on hand a medication to overcome the condition.

So, if there will soon be a medical condition named Weak Orgasm Syndrome, or Clinical Sex Drive Loss, bromocriptine is a sure medication candidate among probable new entries to the market that are sold at ten or twenty times the price.

3.2.2.4.2.8 How does bromocriptine feel – personal experience

How does bromocriptine feel? Probably the first effect it has on me is a slight desire no, not for intercourse but to lie down. This may set in as early as one to one-and-one-half hours after ingesting it. I do have to stress that at that point, I do not feel nauseated if, and only if, I have ingested the bromocriptine with a generous amount of food.

I know that the bromocriptine is kicking in when I feel a stuffed nose. If at that point, I have the opportunity to be sexually active, I have a great time.

I have received readers' mail complaining about exactly this: the stuffed-nose effect. It's not disturbing to me, because I feel it is strongly correlated to the pro-sexual effect of bromocriptine.

The stuffed nose is a pronounced histamine reaction. The histamine released into the nasal tissue causes the tissue to swell, just as in allergic reactions. The word "anti-histamines" is probably better known than the word "histamines". Anti-histamines are a category of drugs that are prescribed for people suffering from allergies, and even as remedy for the common cold. It is known that anti-histamines can interfere with sexual function.

My best climaxes have always been accompanied by a good histamine reaction, even when I did not enhance my sex life pharmacologically. So, when I had a real good time, I typically had a stuffed nose, too.

A long time ago, I have read in a scholarly report (was it of Masters and Johnson?) that a good number of women have a tendency to develop red spots between the neck and the breast when they experience an orgasm, especially if they are among those women who seldom reach a climax. Such red spots certainly would also be a histamine reaction.

The sexuality-enhancing effect of bromocriptine is specific to the wiring, not the plumbing of male sexual function. Therefore, in vascular insufficiencies, it will not be of much help.

An increase in libido, of course, is difficult to measure, compared to erection circumference or persistency. Even empirical studies on intercourse frequency with bromocriptine and placebo use will not tell the full story. There is too wide a range of inhibiting mechanisms.

The following I feel as an increase in libido after bromocriptine usage: 1.) I am more likely to really get going when I'm already at it. 2.) A controlled high-plateau phase that would be impossible to reach without bromocriptine, or, for that matter, with any other sexuality-enhancing drug I know (including yohimbine).

Bromocriptine does not make me sexually more interested in general, or before I am engaged in a sexual act. I don't know of any drug that would achieve this. If one were to be found, it surely would be a hit.

Of course, certain pro-sexual agents can provide enforcement for sexual interest. Both sildenafil citrate and yohimbine can cause erections out of the blue. Most people will think that because they have an erection, they must be horny. They will check their minds for sexual thoughts because they believe they must be present, otherwise they wouldn't have an erection. And when they are searching for sexual thoughts, they may indeed discover them.

But it's still wrong logics. It's just that both sildenafil citrate and yohimbine open the faucet for penile blood inflow, and close it for outflow. Voila, you have an erection. Many men (and women) will take the erection in itself as proof of sexual stimulation, but it's plain physiology, just as nighttime erections that do not have to be accompanied by sexual dreams.

By the way, while sildenafil citrate is purely plumbing, yohimbine does have its effects on the wiring. Yohimbine does enhance the moment of orgasm, while with sildenafil citrate, no such effect can be derived.

On the sildenafil alone, orgasms can be disappointing, especially when a larger dose was used (which may have produced a world-class erection).

The controlled high-plateau phase which I mentioned above as a bromocriptine effect is worth to be described in more detail. It sets in at a moment at which one would normally ejaculate, but, oh wonder, one can go on while the pressure in the urethra builds up and up. While usually, the point of no return is definite (a certainty that lasts for only a few seconds), it is much prolonged on bromocriptine. If you are skillful enough, you can experience this plateau for several minutes, thanks to bromocriptine. It's really one of the most rewarding feelings in life, and it is usually followed by an ejaculation with a lot of force, reaching half a meter if unobstructed, even for an older (but healthy) man.

This is very different from the orgasm enhancement that can be felt on yohimbine. The pre-climax pressure on yohimbine is not so much in the urethra but in the penile tissue that reaches wood-hard rigidity. The plateau phase may or may not be enhanced, but when orgasm comes, you'll experience very pleasant shivers up the spine.

Yohimbine also gives me a histamine release effect, but it's different from bromocriptine's. On yohimbine, skin flashing is likely even before or without sexual activity. But a stuffed nose will occur only at and after orgasm. With bromocriptine it is present from the onset of sexual activity.

As long as one isn't too used to bromocriptine, this is.

Unfortunately, the full pro-sexual effect of bromocriptine, wonderful as it may be, cannot be enjoyed day after day. Actually, in me it wanes rather fast. After usage on five or six days in a row, I should take a week's break at least to get sober. The effect will return after the pause, though it is likely that a lasting tolerance will develop over time.

If all of this reminds you of heroin, you're on the wrong path. Bromocriptine definitely is non-addictive.

Bromocriptine is a dopaminergic drug, which means that its principal use is in the treatment of Parkinson's. The case of waning effectiveness of single dopaminergic drugs is well documented in the treatment of this brain disease, which is why new, alternative drugs are constantly being developed.

3.2.2.4.2.9 How to avoid the bromocriptine nausea

When I first started taking bromocriptine for sexual enhancement, I just used a quarter of a 2.5 mg tablet, and even at this minimal dosage, I felt a slight nausea.

I did have great sex, though.

The first time was absolutely great, and I remember it in every detail. If I could repeat this experience every day, I would be the happiest man in the world.

Unfortunately, as with all dopaminergics, the effect wanes as one's body gets used to the medication.

Of course, one can offset the increased tolerance by increasing the dosage. But for me, and many others, increasing the bromocriptine dosage also causes the most common side effect to become more severe: nausea.

I have been able to use bromocriptine more regularly only after I discovered what to do to avoid the nausea. It's a recommendation that is easy to follow: eat a generous meal immediately before taking the bromocriptine.

For me, in order to avoid the nausea, this is essential. I can stomach a full 2.5 mg tablet of bromocriptine, provided the stomach isn't empty when doing so.

3.2.2.4.2.10 Dostinex – another anti-prolactin ergot derivate

Dostinex (cabergoline by generic name) is a comparatively new medication used in the treatment of Parkinson's disease and prolactinomas (tumors of the pituitary gland). Because another medication used for the treatment of the same conditions, bromocriptine, can have a sexuality-enhancing effect if properly used, I imagined that Dostinex would work in the same direction.

Cabergoline is a medication based on ergot alkaloids. Ergot, of course, is the fungal disease of rye and other grasses, and a potent neurotoxin. Ergot alkaloids heavily interfere with neurotransmitter activity. Probably the best-known ergot derivate is LSD, which strongly messes with the neurotransmitter serotonin.

There are a good number of ergot alkaloids that are used in conventional medicine. Usually, these medications are developed for their dopaminergic capabilities. Such medications are needed to treat the severe deficiency of the neurotransmitter dopamine that leads to Parkinson's.

The ergot alkaloid bromocriptine can be used for its sexuality enhancing properties. Actuality, of all substances I have tested on myself for their sexuality-enhancing properties, bromocriptine is among the few for which I can attest some, albeit limited effectiveness. However, bromocriptine has to be taken in a specific manner to avoid the nausea that otherwise overshadows its sexuality-enhancing properties.

It's the pharmaceutical similarity to bromocriptine, which makes Dostinex such an interesting substance. Of all dopaminergic medications used for the treatment of Parkinson's, Dostinex resembles bromocriptine the most, with its double action of enhancing dopamine levels and inhibiting the secretion of the hormone prolactin from the anterior pituitary gland.

This dual action could be crucial to sexual enhancement. Prolactin, the hormone, which has been named for its function of inducing lactation in women, directly interferes with sex drive in both women and men. It controls to a certain extend the secretion of gonadotropin, the hormone which, one step further down the chain, controls the secretion of testosterone in both men and women.

Regardless of which hormonal constellation in this sequence is responsible for a lowered sex drive, the reversal of the sequence through drugs like bromocriptine theoretically supports sexual desire.

As sexuality is the main source of happiness for practically all forms of higher living, including man, and as utmost sexual satisfaction can only be experience as a sequence to sexual desire, it is a logical quest to provide ourselves with a better long-term hormonal profile than intended for by our genetic blueprints. Anti-sexual changes of our hormonal levels as a consequence of the aging process is an unacceptable provision, which nature, our enemy, has designed in order to install the specific generation turnover rate of humans (20 to 30 years).

Increasing prolactin levels and, at the same time, decreasing testosterone levels are two inter-linked causes why aging men don't have as much sex drive as younger men, why they don't have orgasms as powerful as they used to have, and why they don't get as much pleasure out of their sex lives as when they were in their 20's.

To interfere with this chain of events isn't easy. Testosterone supplementation often doesn't do the trick. Each person's body has its own genetically set ideas what the person's age-appropriate testosterone levels ought to be. Supplying additional testosterone will, in healthy subjects, just provoke the body to down-regulate its own testosterone production, as well as to initiate other measures by which the testosterone balance returns to the aforementioned genetically set levels.

Willfully oversupplying testosterone, or supplementing with synthetic steroids is usually useless, at least for men. Women, if they don't mind the androgenic side effects (increased muscle buildup), may be able to increase desire and orgasmic capacity through both testosterone and synthetic steroid supplementation.

But for men, forcing testosterone levels beyond the genetic set points through the supplementation with synthetic steroids, has some very counterproductive side effects. It will, for example, result in shrinkage of the male organ, as well as in problems to achieve an erection.

My own experimentation with the supplementation of testosterone (Andriol capsules) had no measurable effect on sexual desire at all, not immediate and not long-term.

There is are enormous number of compounds, both herbal and synthetic, which can kill sex drive and interfere with sexual performance. There are also a good number of products, both herbal and synthetic, which are sold with the promise that they help to overcome a lack of sexual desire or so-called erectile dysfunction.

I have been writing about pharmacological sexual enhancement for years, and I take the matter seriously. I have tried almost everything that is available under the sun, whether from below (ginseng) or above (ginkgo) the grass line, and both natural and synthetic (such as sildenafil citrate).

While Pfizer's Blue works for erections, I feel that it alone does not provide any kick in the desire or orgasm departments.

The best option for better sex is still tongkat ali. It does raise testosterone levels, as has been shown in numerous animal and human studies, and by raising testosterone also lowers prolactin. This in turn allows more dopamine impact on libido.

And unlike dopaminergics, tongkat ali is practically free of negative side effects.

And it has a long history as an aphrodisiac in Malaysia and Indonesia. If you do buy some, just make sure you do not fall for any of the widespread tongkat ali scams.

3.2.2.4.2.11 Cabergoline and testosterone

Most dopaminergic drugs do have an effect on testosterone. This is caused by the dopamine agonist's power to inhibit the hormone prolactin. Prolactin has a negative-feedback function in the regulation of testosterone. Increasing levels of prolactin typically cause testosterone synthesis to drop, which is why hypogonadism often accompanies pituitary tumors that express themselves in increased prolactin levels.

Cabergoline is one of the strongest prolactin-inhibiting drugs around. One should therefore suspect that it causes testosterone levels to rise. I have received the following inquiry from a woman in Australia; her account of what happened to her suggests that cabergoline may indeed cause testosterone synthesis to rise sharply. The mail is quoted with permission, though name and email address are withheld.

Cabergoline seemingly causing excessive muscle growth

I was doing some research online regarding pituitary tumors and stumbles upon your site when searching for bromocriptine. I am a 33 year old female who was diagnosed with a pituitary tumor 5 years ago and prescribed bromocriptine by my endocrinologist. But after several attempts to tolerate the drugs side effects, I gave up taking it. I have only recently been prescribed cabergoline, which I was told had less side effects than the traditional treatment of bromocriptine. I started on 1/2 a tablet weekly and have increased the dose to currently 1.5 tablets weekly, and my prolactin levels are currently just over the maximum [normal level]. I have been taking cabergoline for 5 months now.

The only side effects I can mention are within two days of taking each weekly dose I get a little emotional, as in teary not angry of aggressive.

And as for my sex drive, well until about 4 months ago, I never had one. I have not had much of a sex drive since the birth of my last child 12 years ago, my breast milk never dried up from his birth, so I would assume from this, the tumor has caused me problems from that time.

But now, at last, thank god, my libido has returned. And I am a little out of control to put it mildly. I was actually thinking of going to the Dr to see what's wrong with me and perhaps have my testosterone levels checked, but I have stumbled upon your site and now have an answer to my new found sex drive.

Serge I have a question also that you maybe able to answer for me. I started training at the gym weight resistance training 10 months ago. And in a very short amount of time, lifting very minimal weights (bench pressing only the bar) I was gaining a ridiculous amount of muscle size, while training only 3 times a week. After 4 months of training, I looked as if I had been training for years, and people were starting to ask if I was using steroids….even the gym staff. From week to week, the other gym members were noticing growth. Is this something to do with the pituitary tumor as well? I did ask my endocrinologist, but he didn't seem to think so.

3.2.2.4.2.12 Low-price cabergoline

As they are the sole patent holder, Pharmacia & Upjohn can decide, rather arbitrarily in most countries of the world, at what prices cabergoline is sold. There are few exceptions. India is one. India does not recognize patents for pharmaceutical substances, so that, in theory, any Indian pharmaceutical company could start manufacturing cabergoline tablets, and sell them at least in India. Of course, supply would trickle through to other countries, as does the supply of sildenafil citrate.

Alas, I am not aware yet of any Indian cabergoline supplier, and Indian cabergoline supply is not the topic of this article.

The much cheaper cabergoline is supplied by Pharmacia & Upjohn themselves.

Pharmacia & Upjohn decides for specific marketing strategies based on a rather complex set of considerations. Obviously, they want to make as much money as possible. Nothing wrong with that, in principle. (Though I, for myself, prefer to buy for as low a price as possible.)

There are competing products for the same conditions (prolactinoma, Parkinson's disease, restless leg syndrome), such as bromocriptine, for which patents have expired, and which, for this reason, are produced by a number of pharmaceutical companies. That keeps prices down.

There are also government regulations to comply with, and they vary from country to country. In some countries, prices have to be officially approved if Pharmacia & Upjohn want cabergoline prescriptions covered by insurance.

Furthermore, a drug has to be approved for certain conditions separately in each country where it is marketed. In some countries, it's easy; in others, it's tedious.

All of this adds up to sometimes stark differences in the price of specific drugs.

Cabergoline is one of them.

First of all, cabergoline's label indications are rather limited in the US. By FDA approval, the drug is indicated in the treatment of prolactinomas (pituitary cancers that express themselves in increased prolactin levels).

However, in Europe where Pharmacia & Upjohn sell cabergoline under the brand names Cabaser (UK, Switzerland, and others) or Cabaseril (Germany), the approved use is not just in prolactinomas but also in the treatment of Parkinson's disease. That makes a huge marketing difference.

The cabergoline dosages needed for the treatment in prolactinomas are rather small: 0.5 to 2 mg per week. The dosages needed to treat Parkinson's disease are typically much higher, which is why cabergoline is always cheaper in countries where it is officially approved for the treatment of Parkinson's disease.

So, while in the US, Pharmacia & Upjohn sell cabergoline under the brand name Dostinex (sold for the treatment of prolactinomas) in tablets of just 0.5 mg, they market Cabaser and Cabaseril (for the treatment of prolactinomas and Parkinson's disease) in tablets of 0.5 mg, 1 mg, 2 mg, and 4 mg.

For cabergoline as Parkinson's disease medication, Pharmacia & Upjohn could not possibly charge 27.25 US dollars per 0.5 mg. Treatment at such tablet prices would cost a Parkinson's patient, or his insurance company, tens of thousands of dollars per year. That's unrealistic. So Pharmacia & Upjohn sell the drugs at much lower prices wherever it is approved for the treatment of Parkinson's disease. 16 times cheaper in most of Europe, and up to 34 times cheaper in some countries, such as Switzerland. If one buys the 4 mg tablets, not the 0.5 mg ones.

It is not uncommon that different-strength tablets of medications are sold at practically the same price. Pfizer's Blue, for example, costs almost the same for the 25 mg, 50 mg, and 100 mg tablets. Which is why those who have to pay themselves for their sildenafil citrate (as opposed to those for whom insurance shoulders the bill) buy the 100 mg version and just split it with any ordinary scissors.

For cabergoline, too, this is the right approach.

As mentioned above, the lowest price for cabergoline is charged in Europe's richest country: Switzerland. In Switzerland, the government-approved price for 16 tablets of Cabaser at a strength of 4 mg is 151.80 Swiss Franks, which converts to about 100 US dollars.

That's 64 mg of cabergoline for about 100 US dollars, less than 80 US cents per 0.5 mg.

In the US it's 218 US per 8 tablets of 0.5 mg, which is 27.25 per 0.5 mg.

The US price is 34 times higher for exactly the same cabergoline, produced by exactly the same pharmaceutical company, Pharmacia & Upjohn.

I wonder whether pharmaceutical companies choose to sell the same medication under different names in different countries so that the price difference doesn't become too obvious to the average patient and consumer?

Obviously, Swiss pharmacies require prescriptions for prescription drugs. While in Germany and Australia, overseas prescriptions are not usually accepted, there is no such impediment in principle with Swiss online pharmacies.

And of course, a US physician is free to prescribe an approved drug for other conditions than those listed on the package brochure. This is called an "off-label prescription". A US physician prescribing cabergoline for Parkinson's disease would be a typical example.

Swiss pharmacies are usually diligent in answering email (I haven't had a single email that remained unanswered), and English is not a problem in principle. However, many of these pharmacies are not accustomed to sending medications abroad, and they may decline orders because they are not familiar with international credit card transactions, or just shay away from the bureaucratic effort. Most Swiss pharmacies will answer that they accept abroad prescriptions, but that the Cabaser will have to be purchased personally at their pharmacy.

On the other hand, at least one Swiss pharmacy, the Victoria Apotheke in Zurich, has been shipping medications worldwide for many years. Their URL is:

http://www.pharmaworld.com/

Their website states:

"Costs: The medicine will be charged at the official retail price. Prices may vary slightly according to currency fluctuations. Additional costs for shipping and handling will be calculated according to the weight of the consignment."

The government-approved Swiss retail price is 151.80 Franks. And this is the price I have paid for my Swiss Cabaser, bought on a German prescription, and I have been quoted exactly the same, 151.80 Franks, by all the Swiss pharmacies I contacted. Please note: the German / Swiss word for pharmacy is: Apotheke

The Victoria Apotheke requires that the original prescription is sent by mail.

EU residents who want to receive their Cabaser through the postal services, and have communication problems with the Vioctoria Apotheke, can order at roughly double the Swiss price (which is still much, much lower than the US price) at the Farmacia Meritxell online pharmacy, based in Andorra.

They are effectively organized, and on working days, they answer mail quickly. Their replies are in Spanish, but those parts you have to comprehend in order to order are easily understood even for people who do not speak Spanish. This primarily concerns data like credit card numbers, and the amount of money to be remitted.

Their address details are:

Farmacia Meritxell Dr. Nequi n' 7 Andorra la Vella Principat d'Andorra

Phone: + 376 826060 Fax: + 376 862086

mail@farmaciameritxell.com

Their website:

http://www.farmaciameritxell.com/

16 tablets Cabaser 4 mg cost 219.37 Euro, which is roughly the same price as one pays for 8 tablets Dostinex 0.5 mg in the US. Therefore, ordering from Andorra is 16 times cheaper, and it's still the same cabergoline, produced by Pharmacia & Upjohn.

Farmacia Meritxell will answer your inquiry with an order form and a proper bill. You will have to fill in your doctor's name and code, as well as your credit card information. I haven't seen a clear note of the requirement for a prescription, but I assume that one will be needed.

Finally, cabergoline is also sold as veterinary medicine. The brand is Galastop, by Boehringer Ingelheim. I haven't seen a price tag.

3.2.2.4.2.13 Apomorphine for sexual enhancement

When Uprima (apomorphine by generic name) was launched in Europe as the first medical treatment for sexual dysfunction after Viagra, it was suspected that it could repeat the success of Viagra. It did not. Which doesn't mean that it would be an entirely useless drug.

Apomorphine is a dopaminergic drug. It stimulates dopamine receptors. And dopaminergics are a blessing. If you know how to take them, and what to suspect. And if you can purchase dopaminergics at a lower price than the 10 dollars per milligram charged by official Uprima selling sites, or at US pharmacies for other dopaminergics such as Dostinex.

Unlike sildenafil (Viagra), apomorphine and other dopaminergics exert their pro-sexual effect not upon the erectile organ but upon the brain. Apomorphine provokes erections not by interfering with the plumbing of male sexual function (speak: blood supply to the penis), but the wiring necessary for arousal.

That Viagra only affects the plumbing, puts clear limits to its potential as a lifestyle drug. Viagra will do little for men whose plumbing doesn't leak. On the other hand, a good shot of additional desire can be a welcome life enhancement for many people with whom there is nothing wrong physically but who just feel bored with their everyday life. For them, apomorphine can be an enrichment, albeit on a limited scale.

Provided, they can afford it.

I meanwhile have gained experience with apomorphine. I need about 6 mg to have a clear pro-libido effect. Buying official Uprima would cost me 60 US dollars a go.

While apomorphine clearly is a pleasure drug (if taken at dosages below the nausea level), this is about all it has in common with its more famous colleague in name, morphine. Sure, apomorphine can be produced from morphine. But its pharmacological effects are completely different. Morphine is a sedative agent, while apomorphine is a stimulant.

Apomorphine primarily works as a dopamine agonist. Like most dopamine agonists, apomorphine is useful in the management of Parkinson's Disease (PD), a condition characterized by the loss of dopamine-producing neurons, or a decreased function of such neurons. Either way, a dopamine deficiency is the result, leading to severe motor function disturbance.

Apomorphine is a D1 receptor-specific dopamine agonist that makes it different from mostly ergot-derived dopamine agonists, which usually target D2 dopamine receptors, e.g. pergolide and bromocriptine. D3 and D4 dopamine receptors are less often targeted in the management of Parkinson's Disease.

It has long been documented that Parkinson's Disease medications have sexuality-enhancing side effects. It has to be noted that the sexuality-enhancing side effects hold true for many but not all dopamine-enhancing Parkinson's medications. Whether or not a dopamine agonist enhances sexual functions seems to depend primarily on the dopamine receptor sites it targets.

3.2.2.4.2.14 Bulk apomorphine

(Please note: this is an updated version of an article written in 2001. In 2001, I purchased bulk apomorphine from a chemical supply house. The apomorphine I bought was manufactured by Sigma Aldrich. I have no doubt that it was manufactured in accordance with strict quality controls, but it was not considered “pharmaceutical grade”. I do not recommend that people purchase apomorphine which is not pharmaceutical grade, and I myself did not reorder. Instead, I later purchased proper apomorphine sublinguals manufactured for human consumption in accordance with US pharmaceutical regulations, and I obtained a proper prescription for it.

Suppliers of official Uprima that sell the medication on the Internet charge outrageous prices, 80 Pound Sterling (about 120 US dollars) for four doses of 3 mg apomorphine (30 US dollars a dose!). And, according to my experience, the 3 milligram of apomorphine which in the most potent Uprima version are not as generously measured as Pfizer’s Blue.

100 milligram of sildenafil citrate cost some 10 to 12 US dollars, but half of such a tablet does the job for most men. So, the actual price per dose of sildenafil citrate comes down to 5 to 6 US dollars, which I consider still affordable.

Because of the high price, I myself have started using apomorphine regularly only after I had come across an address though which I could receive apomorphine at less than 10 percent of the official Uprima price, which then is a bargain for sexual enhancement.

Unlike sildenafil citrate apomorphine is not a new medication. It has been around for decades for the treatment of Parkinson’s and as an emetic (a medication used to induce nausea and vomiting). This is important as far as patent rights are concerned.

Sure, Uprima is a patented medication. But the patent covers just the use of apomorphine in the treatment of erectile dysfunction, and the delivery method as sublingual tablets. Apomorphine itself is no longer patented.

For this reason, trade in apomorphine itself is much less restricted than, for example, trade in sildenafil citrate, which is a new substance.

Bulk apomorphine can be ordered from large chemicals companies. I once purchased supply of 250 mg that was manufactured by Sigma-Aldrich (sigma-aldrich.com), but I bought it through a local chemicals distributor in Asia.

There is an online store at sigma-aldrich.com, but looking up a chemicals supplier in the yellow pages of any large city may reveal a source closer to home. Furthermore, Sigma-Aldrich is not the only chemicals company through which apomorphine can be purchased online.

One has to be aware that the dosages used of apomorphine are very, very small. Uprima is sold as sublingual tablets of 2 or 3 milligram apomorphine. The therapeutic dose is in the range of 1 to 10 milligram apomorphine.

Amounts of 2 or 3 milligram are of course hard to measure if one doesn't have access to laboratory equipment. I use a small precision screwdriver to quantify dosages. The tip holds approximately 2milligram, as I established by counting the purchased dosage of 250 milligram into some 120 portions. You do not need a microscope or magnifying glass to see a dosage of 2 milligram.

The delivery route of apomorphine is very important. If it is just ingested, it is very likely to cause nausea. The delivery has to be sublingually, so that the apomorphine reaches the blood stream fast (in the treatment of Parkinson's, apomorphine is usually injected, which, of course, is even faster).

To the best of my knowledge, there is nothing terribly high-tech in supplying dopaminergic agents via the sublingual route. For the manufacture of sublingual tablets, one just has to use a base that dissolves fast and at the same time is firm enough to be pressed into tablet form. Even simple glucose could do the job.

The dopamine agonists apomorphine, bromocriptine, and lisuride are all fairly complex molecules. But they have the ability to pass through the mucous skin of the oral cavity, which is why they are suitable for sublingual application. A hydrochloride form (salt) of the active ingredient may be used for convenience, but apart from that, the apomorphine or other dopamine agonist is not chemically modified for sublingual usage.

I myself just placed the amount of a few milligram (about 5) of apomorphine below my tongue… unmixed with any sublingual facilitator.

Feedback

While I have had no problem ordering Sigma-Aldrich apomorphine in Asia, those who want to order it in the US may face some problems, as has been reported by a reader who tried it. This has been caused by a published abuse of their supply line.

“I tried to order some apomorphine using Sigma-Aldrich’s online order system. Placed the order online with no problem. They call you back to do a screening on all new accounts however, to see if your “facility” is qualified to handle the chemicals, and if the end-use is acceptable. I thought I was very good and creative on this one. I said that I was a dog breeder, and used apomorphine as an emetic for poison overdose (it has been used this way by vets for years).

They said they are not allowed to sell apomorphine for that application since their apomorphine is reagent grade and not pharmaceutical grade, and this grade is not FDA-approved for end use in humans or animals, unless as part of an experiment where the animal is destroyed. So much for my cover story.

So if anyone is going to make it past the screening, they have to say they are a research company or something, and make it acceptable. Even then it might be hard – they may ask for a business license and articles of incorporation as proof. They screen due to increased scrutiny since the Scientific American expose where SciAm ordered all the stuff needed to make Sarin gas from Sigma-Aldrich, with no questions asked, and then published an article about how easy it was to order stuff to make nerve gas over the web. Yikes!!

The Sigma-Aldrich representative pointed out that what they supply is reagent grade, not pharmaceutical grade. The question is whether this makes a difference, quality-wise. Please see the following expert considerations:

“Well it is a small amount being ingested and the synthesis isn’t terribly complex, so the risk may not be large, but who knows what synthesis method they are using. If it involved highly carcinogenic solvents such as benzene or toluene along the way there would be traces left in the final product. Also heavy metals like lead, arsenic, or cadmium could be present. Biological toxins could be present in small amounts. The morphine used as the starting material would also not have to be pharmaceutical quality for the Sigma-Aldrich apomorphine, so any impurities in it would be carried through. Sigma specs their stuff as 99% pure, so there is 1% of a bunch of things in there.

With pharmaceutical grade material there are all kinds of restrictions on what kinds of processes and reagents can be used, and the resulting purity required, with FDA oversight and audits as well to insure that each batch meets the specs. Same applies to all materials and solvents used – all have to be traceable and all suppliers have to meet cGMP specs all the way up the chain.

Without getting disclosure from Sigma-Aldrich as to how they make this stuff and what materials are used as sources, and what impurities are in the final product, there isn’t really much of any way to know how much risk is involved.

You could get a small sample tested somewhere via gas HPLC spectroscopy and mass spectroscopy to see what else is in the stuff, both organics and inorganics, and perhaps assume that future batches will generally be similar.

You also don't know what shelf life to expect or how to store it. It should be pretty stable stuff but it might degrade to dangerous degradation products in a year or two, less if hot and humid. Again pharmaceuticals are all characterized for stability under various conditions as part of the development and registration process and they are packaged and labeled accordingly.

I think it is primarily a regulatory issue, but as I indicated above the actual synthesis, reagents used, and final impurities could also be significantly different.

Apomorphine is used in pharmaceutical grade for veterinary purposes – to induce emesis in dogs that ingest poisons accidentally, by placing a large dose right in the eye. You might be able to find a source through some veterinary supply place and get better stuff this way. It should be very cheap in this form as well. Might come as an injectible bottle in liquid form however, rather than a powder, I don't know, but with a small syringe you could just place a drop or two or whatever amount is needed sublingual to get the right dose.

Also of course it is available in injectible form for treating Parkinson's disease, and this would be fully human pharmaceutical quality. Probably very hard to get however, but maybe not too hard where you are. More expensive this way I am sure but probably still an order of magnitude less than Uprima."

Further research

I have written to Sigma-Aldrich to inquire whether they just produce reagent apomorphine, or whether they also sell pharmaceutical-grade apomorphine to companies who then market this apomorphine for the treatment of humans or other animals. I didn't word my inquiry so directly, but rather put it the following way:

“As a journalist and potential buyer of Sigma-Aldrich shares I would like to know whether your company only sells reagents to the pharmaceutical industry, or also bulk pharmaceutical-grade products (especially those of which patens have expired) which are then just repackaged as drugs and marketed under the name of the pharmaceutical company you supply? If you do sell bulk pharmaceuticals, is there an overview of volumes?”

I received the following information from Sigma-Aldrich:

“Sigma-Aldrich sells reagents to pharmaceutical companies for their use in development. We do not sell bulk pharmaceuticals. The pharmaceutical and biotechnology industry provides about 40% of our annual revenue with the remaining 60% coming from universities, government, the chemical and other industries and the diagnostic market.”

I have then sent the following inquiry to a chemical engineer who is an expert on production standards:

“Chemical companies (such as Sigma-Aldrich) produce the same chemicals that are widely used in the pharmaceutical industry. They produce in accordance to current Good Manufacturing Practice (cGMP), but sell these chemicals as reagent grade, not pharmaceutical grade.

“In your opinion, what would be the quality difference to expect in chemicals produced by chemicals manufacturers adhering to cGMP guidelines, and pharmaceutical companies producing these chemicals in pharmaceutical grade.”

I received the following answer:

“Aldrich has a GMP manufacturing unit but I doubt that every chemical in the catalog is made to GMP standards. That is not to say that the chemicals they sell are not very pure which I’m sure they are.

“The quality difference of items made to GMP standards are: that they would come with a C of A, be made according to a validated procedure (at least three times), have a known stability profile, be expiration-dated, be labeled properly with only pre-established claims, etc.

“Other than consistency batch to batch, a chemist should not see any difference especially if the company is certified to ISO9000 or has another internal quality program such as TQM, six sigma, etc. which almost everyone does.”

Sigma-Aldrich is of course not the only possible supplier for apomorphine, and other suppliers do deal in pharmaceutical-grade chemicals.

One of the best web sites on matters of interest to purchasing chemicals is:

http://members.tripod.com/~ChristopherMarrs/

Please specifically see the following page:

http://members.tripod.com/~ChristopherMarrs/GMP_Drugmanufacturer.html

The page lists many companies that sell chemicals, including chemicals designated as pharmaceutical grade.

Additional sources (contributed by J.)

I found two sources so far of pharmaceutical grade apomorphine:

MacFarlan Smith Pharmaceuticals in Edinburgh Scotland

http://www.macsmith.com/

They would have a large minimum order requirement – maybe a kilo or more.

Gallipot Inc. in St. Paul Minneapolis, USA

No web page but info here:

http://members.aol.com/mefrancom/gallipot.htm

Small quantities should be fine here.

A third source which is not pharmaceutical grade but interesting for another reason (see below) is ICN Parmaceuticals:

http://www.icnbiomed.com (Do a search for apomorphine there)

MacFarlan Smith would be an excellent and reputable source – a major supplier, but they deal in bulk only.

“I also found info that apomorphine quickly absorbs water and degrades, turing blue-black in color within a few months to a year. You need to keep it dehydraded in a dessicator to have it remain stable.

“I had another thought about your dose level. Apomorphine is chiral (has a handedness, and rotates polarity of light when in solution) as are most biologically active chemicals – there is an S+ and R- form. The material from Aldrich is certainly a mixture of the two. It is likely that to reduce nausea, TAP/Abbott have chosen to use only the biologically active form, which is the R- form, so that side effects from the inactive form are eliminated. This would reduce dosage by 1/2. ICN sells the R- form, which of course is at a much higher price, $28 for 100 mg, but still cheap. Not pharmaceutical grade however.”

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I have received another reader’s mail on the quality of non-pharmaceutical grade apomorphine:

“By the way, I am not very knowledgeable about chemicals, but I am surprised that your expert expressed a concern about benzene and toluene. Toluene is a common solvent in all sorts of glues and paints, and many painters and factory workers inhale large quantities of it every day. Benzene used to be a popular solvent (and large quantities were inhaled by lots of people), but it is now considered carcinogenic. Nonetheless it is still widely used in manufacturing all sorts of things. The carcinogenic effects are rather weak. The amount of benzene or toluene that you might ingest in a few mg of apomorphine would be tiny, so even if you did it every day I can’t see that it would be dangerous. Note that this is a just lay person’s opinion. I don’t know anything about other possible impurities.”

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Please specifically see the following page:

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“I also found info that apomorphine quickly absorbs water and degrades, turing blue-black in color within a few months to a year. You need to keep it dehydraded in a dessicator to have it remain stable.

3.2.2.4.2.15 Apomorphine instead of yohimbine?

I have been taking yohimbe for years, and I have had memorable experiences with it. I have also tried almost everything else that is marketed as sexual enhancement, and have found almost everything else I tried entirely useless or even counterproductive.

However, yohimbe also has its limitations. For one thing, the side effects are difficult to manage: general over-excitation, heart palpitations, sleeplessness for some 20 hours after ingesting even an amount too little to have any pro-sexual effect.

I have developed some tolerance towards yohimbe and yohimbine, but this is not the principle limitation of the herb / drug. I can overcome tolerance by just increasing the dosage. When I started with yohimbine several years ago, 10 milligram was sufficient for a rock-hard erection for intercourse at least three times a day.

Over the years, I have increased the dosage to up to 50 milligrams. The pro-sexual effect is proportional to the dosage. The more yohimbe, the better the erection. If you go on increasing the dose, you will, for sure, reach the lethal mark. You'll certainly die with a text book-case of priapism, and they'll have to use a saw to make you look not too naughty in your shroud.

But great sex comes from the brain, not from the corpus cavernosum. This is why jealousy is such a great aphrodisiac. Constant yohimbe-use may increase one's susceptibility to jealousy, but daily yohimbe alone is no guarantee that, indeed, one will develop a nice pathological jealousy. There has to be the right relationship for it as well. Once jealousy is induced, no yohimbe or other medication is needed for great sex. When I experienced great jealousy for months on end, I had more sex and better sex than I could achieve with any dosage of yohimbe or yohimbine.

There is nothing essentially wrong with my vital organ. I have good morning erections, and those months on jealousy proved to me that if the mental stimulus is okay, I'm good for 30 climaxes a week.

Unfortunately, that jealousy waned over time, giving me a hard time (or rather a not-so-hard time) when I am in a situation in which I should be enjoying myself. Pfizer's Blue is no solution for that. Sure… Sildenafil citrate eases erections. But erections on sildenafil citrate alone are like nighttime or morning erections.

I have them, and they are not necessarily sexual. Yohimbe is better than phosphodiesterase inhibitors in that there is a mental component.

During jealousy, erections occur because of specific mental constellations… and these reactions are better than those engineered with phosphodiesterase inhibitors or ad-hoc yohimbe.

In the seventies, it was fashionable to diagnose impotency as an entirely psychological problem.

Nowadays, it is fashionable to diagnose impotency as an organic condition. Something that has to do with the selective inhibition of cyclic guanosine monophosphate (cGMP)-specific phosphodiesterase type 5 (PDE5) in the pelvic region. The reason for the diagnostic shift is obvious: the billion-dollar interest in selling Pfizer's Blue. The pendulum will swing backwards. Not quite as far back as in the seventies. But people will realize that good sex and good erections have to come from the brain. This doesn't mean that the problem is psychological.

There are several factors involved, both organic and psychological. They are obviously interlinked. Every mental state is expressed by a so-far largely unresearched biochemical constellation in the brain. But we have to be aware that our organisms are tuned to react on sensual input. What we see is what triggers biochemical processes. I am sure that there is an element of wear and tear with respect to our mental response.

As we grow older, and richer in sexual experience, it is increasingly likely that certain sexual stimuli are no longer capable of triggering the desired response. Sometimes I think, I'd like to undergo some targeted amnesia in order to wipe out sexual experience so that reoccurring stimuli will feel entirely new.

However, getting used to sexual stimuli may not be the only thing that happens as our minds and brains grow older. There is ample scientific proof that sexual agitation is correlated to the activity of the neurotransmitter dopamine and the brain's dopamine receptors.
I have, throughout the years, repeatedly experimented with all kinds of dopamine agonists, such as bromocriptine, deprenyl, lisuride, and others. Bromocriptine had an extraordinarily positive effect for some time, but fast lost its effectiveness in small dosages, while larger dosages induced nausea so bad that I couldn't enjoy sex. (Please see my domain Bromocriptine.com for information on how to avoid the nausea when taking bromocriptine for sexual enhancement.)

According to the books, dopaminergic agents cause nausea because of the effect these therapeutic agents have not only on central dopamine receptors, but also on peripheral receptors. According to the books, the nauseating effect can be countered by taking some domperidone together with the bromocriptine or lisuride or whatsoever.

But domperidone in me reduces the pro-sexual effect of dopaminergic agents.

When I recently tried apomorphine, I was surprised indeed about its side effect-free sexual enhancement. (It doesn't have the reputation of being free of side effects in all people.)

Apomorphine, of course, is what is marketed in Europe as Uprima sublingual apomorphine, which after sildenafil citrate is the only additional pharmaceutical that has been approved specifically for the treatment of so-called "erectile dysfunction".

In the US, Uprima is not FDA-licensed for the treatment of erectile dysfunction. I read somewhere that this is related to a car accident, which had occurred in the testing phase; this accident had been linked to possible side effects of Uprima.

So, what does apomorphine do? First of all, it doesn't give me any negative side effects: no sleeplessness, no heart palpitations, and also no nausea, in spite of the fact that in the official apomorphine literature, nausea is listed as the most common adverse reaction.

I find apomorphine very subtle in effect. I have started with very small dosages, which I consumed in between yohimbine days. Usually, I am not good for any sexual encounter the day after a day with a full yohimbine schedule. So, I was surprised that I had sexual interest strong enough to carry me through intercourse.

I increased the dosages even beyond the 3 mg, and it still didn't provoke nausea. And I feel that the effect of apomorphine is more natural than the effect of yohimbine. When on yohimbine, it's impossible to forget that I'm on yohimbine. On yohimbine, I am drugged. (On a phosphodiesterase inhibitor, I am not drugged, but what do I do with an erection that isn't accompanied by appropriate desire… my problem rather is saturation, lack of libido, not the corpus cavernosum.)

I feel an effect of sublingual apomorphine after about 30 minutes. With yohimbine the first symptoms are increased salivation and an urge for a bowel movement, while the first symptoms of apomorphine are, for me, an urge to yawn. Yawning is, in a funny manner, correlated to the sexual response.

After taking apomorphine, I never feel drugged. I don't feel that I have taken a medication. I just have an increased interest in sex, which results in an increased likelihood of developing an erection.

3.2.2.4.2.16 How apomorphine works

Strictly speaking, Uprima is not a new drug. The active ingredient of Uprima is apomorphine, which has been around for decades, and is used in the treatment of Parkinson's disease and as an emetic in dogs and other domestic animals. (An emetic is a drug that induces vomiting.)

While apomorphine has a definite potential as a pleasure drug, this is about all it has in common with its more famous colleague in name, morphine. Sure, apomorphine is produced from morphine. But its pharmacological effects are completely different. Morphine is a sedative agent, while apomorphine is a stimulant.

Apomorphine primarily works as a dopamine agonist, which accounts for its usefulness in the management of Parkinson's Disease, a condition characterized by the loss of dopamine-producing neurons, leading to severe motor function disturbance. Apomorphine is a D1 receptor-specific dopamine agonist that makes it different from mostly ergot-derived dopamine agonists, which usually target D2 dopamine receptors, e.g. pergolide and bromocriptine. D3 and D4 dopamine receptors are less often targeted in the management of Parkinson's Disease.

It has long been documented that most Parkinson's medications have sexuality-enhancing side effects. I was personally using Parkinson's medications for sexual enhancement long before Uprima was launched. I gained the most experience with Parlodel (bromocriptine), but I have also tested Dopergine (lisuride), Cabergoline (brand name: Dostinex), Mirapex (pramipexole), L-dopa, and deprenyl.

It has to be noted that the sexuality-enhancing side effects hold true for many but not all dopamine-enhancing Parkinson's medications. Whether or not a dopamine agonist enhances sexual functions seems to depend primarily on the dopamine receptor and sub-receptor sites it targets.

Unlike sildenafil citrate, dopamine agonists, whether Uprima or cabergoline (brand name: Dostinex), exert their pro-sexual effect not upon the erectile organ but upon the brain. They provoke erections not by messing with the plumbing of male sexual function (i.e. blood supply to the penis), but by interfering with the wiring necessary for arousal, pleasure, and climax.

That sildenafil citrate only affects the plumbing, puts limits to its potential as a lifestyle drug. Sildenafil citrate will add little for men whose plumbing doesn't leak. On the other hand, a good shot of additional desire would be a welcome life enhancement for many people with whom there is nothing wrong physically but who just feel bored with their everyday life. For them, dopamine agonists could be a real enrichment, and even a medication that saves their marriages.

Dosage for a pro-sexual effect is difficult to determine for all dopamine agonists. This is the case because a dosage that is too high will inevitably result in nausea. This nausea can be so bad that the last thing one fancies is sex. This particularly is a problem with apomorphine, which indeed is commonly used to induce nausea. One of the advantages of cabergoline is that it has far fewer side effects.

Uprima has so far not been approved for marketing in the US. If a FDA endorsement is to be obtained for its marketing in the US as treatment for erectile dysfunction, the primary concern has to be to keep the nausea side effect at bay.

With apomorphine, nausea can be reduced if it gets into the bloodstream quick enough. Parkinson's patients use injected apomorphine. Parkinson's is a serious condition, a matter of life and death, and in such a case, patients can be expected to tolerate injections. But as treatment for non-life-threatening conditions like lack of libido or erectile dysfunction, injection medications have always been a flop.

Tap Pharmaceuticals, the makers of Uprima, try to get around the problem in two ways: by packaging the drug as sublingual, and by keeping the dosage per tablet rather low (2 or 3 mg per sublingual.

The point is: with small doses of apomorphine, the likelihood of nausea will be negligible. But so will the pro-sexual effect.

When I myself use Uprima, I go for 6 mg, which for me is a borderline nausea dosage. With 6 mg of sublingual apomorphine, I'm not really nauseated, but I do have a definite preference for a horizontal position. And at that dosage, I do get a sexual kick out of the medication.

3.2.2.4.2.17 Lisuride causing extreme nausea

I decided to try lisuride after a reader reported a positive experience with lisuride. The reader described his experience as follows:

“Lisuride: I tried this after reading about it in the book Sexual Pharmacology.

It is available in Europe. A friend got some from France. It is an ergotic dopaminergic drug, mainly used for Parkinson’s disease. The packet insert lists impuissance (impotence) as another indication. It has a real effect. It increases desire and sensitivity. It also causes nausea.”

So far the reader’s experience.

I tried half a tablet of lisuride, combined with nothing else, and I felt absolutely lousy. I had to lie down right from the moment I felt the lisuride kicking in, and I had to stay in bed for the whole day.

I didn’t develop any fever, or other measurable symptoms such as increased heart rate. I just felt lousy, as if I wanted to vomit, tough I didn’t reach that point.

3.2.2.4.2.18 How to take lisuride for sexual enhancement

The reader quoted in part 1 of this lisuride series mentioned that he used domperidone (doesn't that sound like a brand of champagne) to counter the nausea caused by the lisuride:

"To offset the nausea, one can take some domperidone ahead of time. Domperidone is available in Europe but not the US. It is sold as an anti-emetic; it works by blocking peripheral dopamine receptors (but not blocking the central ones that are responsible for the sexual effects). I got some over the counter in Holland."

I disagree with his judgment on domperidone. In my own experiments, the domperidone has always countered the sexual effect of dopamine agonists, but never fully suppressed the nausea.

The trick with all dopaminergic agents that are a frontline treatment for Parkinson's disease is to take the with a generous amount of food. Package inserts usually mention that these medications should be taken with food, but usually do not sufficiently emphasize this.

Frontline treatments for Parkinson's disease are drugs such as bromocriptine, lisuride, apomorphine, and L-dopa, all of which I have tried for sexual enhancement, and all of which work to that end.

All of them cause a bad nausea when not taken with food, and are tolerable from the nausea perspective when taken with food. They all enhance sexual parameters in a similar manner, in the brain.

However, if one's sexual dysfunction (or age-related weakness) is rather vascular, they won't do much good. But together with sildenafil citrate or another phosphodiesterase inhibitor, they are powerful tools for sexual enhancement, or as a solution for sexual dysfunction in men. The dopamine agonists enhance desire, while the phosphodiesterase inhibitor delivers the erection.

Because nausea is a problem for many who ingest dopamine agonists for sexual function, especially when not taken with enough food, some people may feel inclined to try other dopaminergic drugs which are less efficient as Parkinson's medications.

I myself have tested the MAO-B inhibitors deprenyl. But for me, deprenyl is too similar to amphetamine. It makes me only hyperactive, not hyper-sexed. And like amphetamine, it causes a shrinkage and a loss of the sense of touch in the male organ.

I tried amineptine (Survector), an anti-depressant that works by enhancing dopamine levels. Though it makes me feel OK, I don't get a sexual kick out of it.

3.2.2.4.2.19 Lisuride combined with Pfizer's Blue

My initial tests with lisuride (Dopergine) primarily resulted in the kind of nausea I suffered during migraine attacks. However, I had conducted these tests before I acquired extensive experience with bromocriptine. I therefore decided to repeat lisuride tests, and especially to try lisuride with Pfizer's Blue.

I used only half of a single Dopergine tablet, and I ingested it with plenty of food. I assumed that what is correct in order to avoid nausea with bromocriptine would also work with lisuride.

And because I do so with bromocriptine, I combined the lisuride with 25 mg of Pfizer's Blue (a quarter of a 100 mg tablet).

Well, this was one test I should have left out. Taking the lisuride with food did not prevent the nausea. It hit with full force.

And the Pfizer's Blue, taken about an hour after the lisuride, only aggravated the terrible condition I was in. I never have heartbeat problems from taking Pfizer's Blue with bromocriptine, and even combined with yohimbine, the tachycardia is tolerable.

But while at this time the lisuride made me so miserable, the Pfizer's Blue topped this by giving me the additional feeling of an immanent heart attack.

I barely made it to a hospital where I spend the next few hours in an emergency room. Surprisingly, the physician on duty found no problems with my vital signs: heartbeat and blood pressure were normal, and so was breathing. But I was so sick, I just couldn't get up from the emergency room bed. Fortunately, as always during nausea attacks, I was able to pass part of the time sleeping.

The strong lisuride impact lasted for about 4 hours, after which I was able to make it home. I was groggy for the rest of the day.

I won’t experiment with lisuride again, so I have nine and one half Dopergine tablets left. Anybody interested?

3.2.2.4.2.20 Pergolide for sexual enhancement

Permax (generic name: pergolide mesylate) is ergot-derived medication with similarity to Parlodel (generic name: bromocriptine) and cabergoline (brand name: Dostinex). Like bromocriptine and cabergoline, pergolide can be used for sexual enhancement, or, more specifically, as support for sexual excitement, orgasm, and ejaculation.

(The following is pharmacological data from the Permax package insert. Please be aware that the dosage information applies to the treatment of Parkinson's disease, not for sexual enhancement. Dosages for sexual enhancement are much lower than those for the treatment of Parkinson's disease.)

Permax (pergolide mesylate) is an ergot derivative dopamine receptor agonist at both D1 and D2receptor sites. Permax is provided for oral administration in tablets containing 0.05 m, 0.25 mg, or 1 mg pergolide as the base.

Pergolide mesylate is a potent dopamine receptor agonist. Pergolide is 10 to 1,000 times more potent than bromocriptine on a milligram per milligram basis in various in vitro and in vivotest systems. Pergolide mesylate inhibits the secretion of prolactin in humans; it causes a transient rise in serum concentrations of growth hormone and a decrease in serum concentrations of luteinizing hormone. In Parkinson,s disease, pergolide mesylate is believed to exert its therapeutic effect by directly stimulating postsynaptic dopamine receptors in the nigrostriatal system.

The major route of excretion is the kidney.

Permax is indicated as adjunctive treatment to levodopa/carbidopa in the management of the signs and symptoms of Parkinson's disease.

Administration of Permax should be initiated with a daily dosage of 0.05 mg for the first 2 days. The dosage should then be gradually increased by 0.1 or 0.15 mg/day every third day over the next 12 days of therapy. The dosage may then be increased by 0.25 mg/day every third day until an optimal therapeutic dosage is achieved.

Permax is usually administered in divided doses 3 times per day. During dosage titration, the dosage of concurrent l-dopa/carbidopa may be cautiously decreased.

In clinical-studies, the mean therapeutic daily dosage of Permax was 3 mg/day. The average concurrent daily dosage of l-dopa/carbidopa (expressed as l-dopa) was approximately 650 mg/day. The efficacy of Permax at doses above 5 mg/day has not been systematically evaluated.

Store at controlled room temperature, 59 to 86 F (15degree celsius to 30degree celsius).

US law prohibits dispensing without prescription.

Side effects

In premarketing clinical trials, the most commonly observed adverse events associated with use of pergolide mesylate which were not seen at an equivalent incidence among placebo-treated patients were:

nervous system complaints, including dyskinesia, hallucinations, somnolence, insomnia;
digestive complaints, including nausea constipation, diarrhea, dyspepsia; and
respiratory systemcomplaints, including rhinitis.

Twenty-seven percent (27%) of approximately 1,200 patients receiving pergolide mesylate for treatment of Parkinson’s disease in premarketing clinical trials in the US and Canada discontinued treatment due to adverse events.

The events most commonly causing discontinuation were related to the nervous system (15.5%), primarily hallucinations (7.8%) and confusion (1.8%). Incidence in Controlled Clinical Trials
The table that follows enumerates adverse events that occurred at a frequency of 1% or more among patients taking pergolide mesylate who participated in the premarketing controlled clinical trials comparing pergolide mesylate with placebo. In a double-blind, controlled study of 6 month,s duration, patients with Parkinson,s disease were continued on l-dopa/carbidopa and were randomly assigned to receive either pergolide mesylate or placebo as additional therapy.

Incidence of Treatment-Emergent Adverse Experiences in the

Placebo-Controlled Clinical Trial Percentage of Patients Reporting Events

Body System/Adverse Event*

Pergolide Mesylate
Placebo
N= 189
N= 187

Body as a Whole
Pain
7.0
2.1

Abdominal pain
5.8
2.1

Injury, accident
5.8
7.0

Headache
5.3
6.4

Asthenia
4.2
4.8

Chest pain
3.7
2.1

Flu syndrome
3.2
2.1

Neck pain
2.7
1.6

Back pain
1.6
2.1

Surgical procedure
1.6
< 1

Chills
1.1
0

Face edema
1.1
0

Infection
1.1
0

Cardiovascular

Postural hypotension
9.0
7.0

Vasodilatation
3.2
< 1

Palpitation
2.1
< 1

Hypotension
2.1
< 1

Syncope
2.1
1.1

Hypertension
1.6
1.1

Arrhythmia
1.1
< 1

Myocardial infarction
1.1
< 1

Digestive

Nausea
24.3
12.8

Constipation
10.6
5.9

Diarrhea
6.4
2.7
Dyspepsia
6.4
2.1

Anorexia
4.8
2.7

Dry mouth
3.7
< 1
2.7
1.6

Hemic and Lymphatic

Anemia
1.1
< 1

Metabolic and Nutritional

Peripheral edema
7.4
4.3

Edema
1.6
0

Weight gain
1.6
0

Musculoskeletal

Arthralgia
1.6
2.1

Bursitis
1.6
< 1

Myalgia
1.1
< 1

Twitching
1.1
0

Nervous System

Dyskinesia
62.4
24.6

Dizziness
19.1
13.9

Hallucinations
13.8
3.2

Dystonia
11.6

Confusion
11.1
9.6

Somnolence
10.1
3.7

Insomnia
7.9

Anxiety
6.4
4.3

Tremor
4.2
7.5

Depression
3.2
5.4

Abnormal dreams
2.7
4.3

Personality disorder
2.1
< 1

Psychosis
2.1
0

Abnormal gait
1.6
1.6

Akathisia
1.6
0

Extrapyramidal syndrome
1.6
1.1

Incoordination
1.6
< 1

Paresthesia
1.6
3.2

Akinesia
1.1
1.1

Hypertonia
1.1
0

Neuralgia
1.1
< 1

Speech disorder
1.1
1.6

Respiratory System

Rhinitis
12.2
5.4

Dyspnea
4.8
1.1

Epistaxis
1.6
< 1

Hiccup
1.1
0

Skin and Appendages

Rash
3.2
2.1

Sweating
2.1
2.7

Special Senses

Abnormal vision
5.8
5.4

Diplopia
2.1
0

Taste perversion
1.6
0

Eye disorder
1.1
0

Urogenital System

Urinary frequency
2.7
6.4

Urinary tract infection
2.7
3.7

Hematuria
1.1
< 1

*Events reported by at least 1% of patients receiving pergolide mesylate are included.

Symptomatic Hypotension

In clinical trials, approximately 10 % of patients taking pergolide mesylate with l-dopa versus 7% taking placebo with l-dopa experienced symptomatic orthostatic and/or sustained hypotension, especially during initial treatment. With gradual dosage titration tolerance to the hypotension usually develops. It is therefore important to warn patients of the risk, to begin therapy with low doses, and to increase the dosage in carefully adjusted increments over a period of 3 to 4 weeks.

Hallucinosis

In controlled trials, pergolide mesylate with l-dopa caused hallucinosis in about 14% of patients as opposed to 3% taking placebo with l-dopa. This was of sufficient severity to cause discontinuation of treatment in about 3% of those enrolled; tolerance to this untoward effect was not observed.

Fatalities

In the placebo-controlled trial, 2 of 187 patients treated with placebo died as compared with 1 of 189 patients treated with pergolide mesylate. Of the 2,299 patients treated with pergolide mesylate in premarketing studies evaluated as of October 1988, 143 died while on the drug or shortly after discontinuing it. Because the patient population under evaluation was elderly, ill, and at high risk death, it seems unlikely that pergolide mesylate played any role in these deaths, but the possibility that pergolide shortens survival of patients cannot be excluded with absolute certainty.

In particular, a case-by-case review of the clinical course of the patients who died failed to disclose any unique set of signs, symptoms, or laboratory results that would suggest that treatment with pergolide caused their deaths. Sixty-eight percent (68%) of the patients who died were 65 years of age or older. No death (other than a suicide) occurred within the first month of treatment; most of the patients who died had been on pergolide for years. A relative frequency of the causes of death by organ system are:

Pulmonary failure/Pneumonia, 35%;
Cardiovascular, 30%;
Cancer, 11%;
Unknown, 8.4%;
Infection, 3.5%;
Extrapyramidal syndrome, 3.5%;
Stroke, 2.1%;
Dysphagia, 2.1%;
Injury, 1 .4 %;
Suicide, 1.4%;
Dehydration, 0.7%;
Glomerulonephritis, 0.7%.

Serious Inflammation and Fibrosis

There have been rare reports of pleuritis, pleural effusion pericarditis, pericardial effusion or retroperitoneal fibrosis in patients taking pergolide. Some patients had experienced similar events while taking the ergot derivative bromocriptine. Pergolide should be used with caution in patients with a history of these conditions, particularly those patients who experienced the events while taking ergot derivatives. Patients with a history of such events should be carefully monitored clinically and with appropriate radiographic and laboratory studies while taking pergolide.

Precautions

Caution should be exercised when administering pergolide mesylate to patients proneto cardiac dysrhythmias.

In a study comparing pergolide mesylate and placebo, patients taking pergolide mesylate were found to have signifiscantly more episodes premature contractions (PACs) and sinus tachycardia.

The use of pergolide mesylate in patients on l-dopa may cause and/or exacerbate preexisting states of confusion and hallucinations and preexisting dyskinesia. Also, the abrupt discontinuation of pergolide mesylate in patients receiving it chronically as an adjunct to l-dopa may precipitate the onset of hallucinations and confusion; these may occur within a span of several days. Discontinuation of pergolide should be undertaken gradually whenever possible, even if the patient is to remain on l-dopa.

A symptom complex resembling the neuroleptic malignant syndrome (NMS) (characterized by elevated temperature, muscular rigidity, altered consciousness, and autonomic instability), with no other obvious etiology has been reported in association with rapid dose reduction, withdrawal of, or changes in antiparkinsonian therapy, including pergolide.

Overdosage

There is no clinical experience with massive overdosage. The largest overdose involved a young hospitalized adult patient who was not being treated with pergolide mesylate but who intentionally took 60 mg of the drug. He experienced vomiting, hypotension, and agitation. Another patient receiving a daily dosage of 7 mg of pergolide mesylate unintentionally took 19 mg/day for 3 days, after which his vital signs were normal but he experienced severe hallucinations. Within 36 hours of resumption of the prescribed dosage level, the hallucinations stopped. One patient unintentionally took 14 mg/day for 23 days instead of her prescribed 1.4 mg/day dosage. She experienced severe involuntary movements and tingling in her arms and legs. Another patient who inadvertently received 7 mg instead of the prescribed 0.7 mg experienced palpitations, hypotension, and ventricular extrasystoles. The highest total daily dose (prescribed for several patients with refractory Parkinson's disease) has exceeded 30 mg.

Animal studies indicate that the manifestations of over-dosage in man might include nausea vomiting, convulsions, decreased blood pressure, and CNS stimulation. The oral median lethal doses in mice and rats were 54 and 15 mg/kg respectively.

3.2.2.4.2.21 Pergolide compared with other dopaminergics

Serge,

I have been on Pergolide now for about 10 days, and have had a chance to try it about 6 times before sex. The bottom line is that it works, and it works pretty well, as a pro-sexual drug for those with dopamine deficiency or dopamine receptor loss.

Here is my report:

Dose: I dose-escalated up to the point where I was taking 0.5 mg (two pills) 2 to 4 hours before sex, PRN, last week. Since I have been trying it almost every day, and since the half-life of pergolide is about 27 hours, this actually equates to about 1.0 mg of pergolide in the system at any given time. This is about 1/3rd the average dose level that is used for Parkinson's treatment of about 3 mg/day. At this dose level I get some slight dyspepsia, dizziness, and nausea for about an hour to two hours after taking the dose, but the side effects fade after that. It appears that the active drug is quickly metabolized to some long half-life active metabolites, which are reported in the literature – the early dizzy phase must correspond to the unaltered drug in the bloodstream. My experience is that the therapeutic window of all of these dopamine agonists (including cabergoline, apomorphine, bromocriptine, and pergolide) is very narrow – one has to reach the level where nausea is about to begin in order to have any positive effects. Pergolide was no exception.

Effectiveness: After reaching the effective dose level, I found that pergolide definitely restored libido and desire. It gave me back what I call that "warm fuzzy feeling" you want to have when you are starting to have sex, and made it pleasurable. In comparison to Dostinex, it was much more effective in this regard. However, the dose level that I was on for Dostinex, 0.5 mg two times per week, was almost certainly much too low for me, so this comparison probably has no validity until Dostinex has been tried at a similar dosage level (i.e., the level just below that which produces a slight nausea or dizziness).

Side effects: Pergolide was very effective, but I would describe it as not being as “clean” for pro-sexual use as Dostinex (again, compared to the low level dose of Dostinex I was on), and it isn’t as clean as Apomorphine (Uprima). By this I mean that it was not effective until there was noticeable dizziness and nausea. In studying the reported binding coefficients at various receptors, it appears this may be due to pergolide having a much higher affinity for serotonin receptors (all subtypes) than either cabergoline or apomorphine, by a factor of as much as 100 times for some receptors. So while pergolide has a better receptor binding affinity profile for dopamine D1 and D2 receptors than either apomorphine or cabergoline (making it a more effective pro-sexual drug), this is somewhat offset by the side effects at other receptors.

Conclusion: I would say that I am satisfied with pergolide, and am more satisfied than I was with the low level Dostinex dose I have been on for the last 8 months. However, I now think that I should cycle this experiment by trying an equivalent, more effective dose level of Dostinex, consistent with the levels I have found are necessary for me for pergolide and apomorphine (about 3x the levels for most normal people). After this trial I could draw a better comparison between the two drugs.

For now, I plan to stop using Pergolide this weekend, flush it out of my system for a couple of days, then begin using the Amantadine next week, and compare how that works. I have some hope that Amantadine may actually do quite well, since it only stimulates dopamine production and acts as a MAO-B inhibitor, so it has little to no effect on serotonin, adrenergic, or other receptors. Unlike all the dopamine agonists, which have partial to full activity at many other receptor types, Amantadine may be more focused on where it counts and thus more successful. I’ll let you know how it turns out.

3.2.2.4.2.22 Pramipexole (Mirapex) tested

I have been able to get some pramipexole (Mirapex) tablets. Pramipexole is a second-generation dopaminergic agent that is not ergot-based (bromocriptine and cabergoline are derived from ergot). I have done my usual sexual enhancement tests for pramipexole.

Unfortunately, the results were nowhere as positive as with bromocriptine and cabergoline, or even apomorphine.

A quarter of a 0.125 mg Mirapex tablet only caused slight nausea, even when taken with food, and had no noticeable pro-sexual effect in me. Taking half of a Mirapex tablet (about two hours before sex) only resulted in a more noticeable nausea no better libido, no better erection, and no effect on orgasm.

I combined the Mirapex with sildenfil citrate. The nausea was not aggravated, and there was no tachycardia (increased heart rate). The sildenafil citrate contributed to the erection, and that was it.

With bromocriptine, I regularly get into a state of mind where I would think: “Oh, boy, I need this so much.” With cabergoline, I could go on and on, and have much more powerful ejaculations than when sober.

I can combine 2.5 mg bromocriptine with 0.5 mg cabergoline and have the benefit from both. I tried a quarter of a Mirapex tablet with 2.5 mg of bromocriptine, and just had a slight nausea with the usual positive bromocriptine effect.

I wouldn’t know what benefit I derived from pramipexole, alone or in combination with bromocriptine and/or sildenafil citrate.

I do recognise that not everybody will react the same way on pramipexole. The following is the assessment of a friend who likewise has many years of experience with dopaminergic agents and yohimbine:

“I take 2 pills, spaced 1 hour apart. When I take them at the same time, my heart starts beating quickly and my blood pressure goes up, so I like to space them out to get a slower onset. I have not read of other people having this problem, so perhaps it is an unusual side effect. I start feeling some sexual effects after one hour, and find the effects are fairly noticeable at 2-4 hours. The effects are definitely enhanced by taking some yohimbe at the same time. However, the yohimbe also increases the heart-rate and blood pressure side effect that I have. With the pramipexole, both erection and sexual sensations are enhanced. When I take 10mg sildenafil citrate and pramipexole together, my girlfriend tells me that I feel extremely large and hard to her (even if she doesn’t know I’ve taken them).”

I still have a supply of pramipexole and would like to exchange it for cabergoline.

3.2.2.4.2.23 Selegiline for better sex

The agenda is better sex. It's that clear and simple. Actually, most things in life are subordinate to better sex. This is the case because, in a very essential way, we, as humans, and probably more as males than as females, derive philosophical meaning for our existence primarily from sexual satisfaction. If sexual satisfaction is no longer available, we are vegetating rather than living.

I do not know of any textbook of sexual enhancement. There are textbooks on crutches. On hairdos. Or on chemistry. Why is there none on sexual chemistry? The term isn't even taken literally by most people. And sexual enhancement, so far, is not a discipline that is thought in medical school or psychology programs.

Sexual chemistry (which is less than sexual enhancement) is covered here and there in medical textbooks. But the coverage is by far not consistent, and the topic is treated rather in footnotes.

For philosophical reasons, I consider sexual enhancement as one of the most important themes in male studies. And it's worthy a dedicated textbook.

I am a scientific researcher, not a MD. I have a background in biology, but as far as sexual enhancement goes, I have a background primarily as having been my own test subject. I am sure that I know more on sexual enhancement than your urologist (who, I am sure, knows more on making a quick buck).

Selegiline is a selective monoamine oxidase (MAO) B inhibitor, used for many years in the treatment of Parkinson's Disease. Parkinson's Disease is a well-defined ailment characterized by the depletion of the neurotransmitter dopamine. Deprenyl, or L-deprenyl, is an alternative generic name for selegiline hydrochloride. The most common brand names are Eldepryl and Jumex.

However, we take interest not so much in the use of selegiline as a treatment for Parkinson's Disease but in the potential, selegiline has as a lifestyle drug. Selegiline's potential as lifestyle drug lies primarily in its sexuality-enhancing power.

3.2.2.4.2.24 Amineptine reported to cause powerful orgasms

The anti-depressant amineptine (brand name: Survector) has repeatedly been reported to alleviate erectile dysfunction and to make orgasms more powerful.

In a 1999 scientific study titled “Sexual dysfunction with antidepressive agents. Effect of the change to amineptine in patients with sexual dysfunction secondary to SSRI”, Angel Luis Montejo (angelluis.montejo@globalmed.es) came to the following conclusion: “Amineptine was shown to be an effective antidepressant in the patients studied, and did not cause secondary sexual dysfunction, and even improved the dysfunction that was present in some patients. In those patients previously treated with SSRI’s, amineptine is able to significantly improve the sexual dysfunction and yet maintain the efficacy of the antidepressive treatment used before these 6 months.”

And Robert Mason Ph.D. reported on smart-drugs.net:

“Amineptine was a drug unique in that it was a dopamine reuptake inhibitor and was proving very popular as an antidepressant, (as a quick review of any of the internet chat-groups will reveal). Unfortunately, it appeared that amineptine helped aid orgasm and as such was considered by the authorities to be a “drug of abuse and potentially addictive”. Many drugs that “interfere” with dopamine have been shown to improve libido, particularly for men (for example deprenyl, L-dopa, and GHB). But perhaps amineptine was even stronger. As a result, it is my understanding that the FDA pressured the foreign manufacturer to remove the drug from the market.”

I also encountered the following newsgroup posting

“statusk@aol.com (status quo) wrote in message news:<1b4c530a.0111220739.372b0cc1@posting.google.com>…

“I wish we could still buy amineptine. It would have been a godsend to us, because it increases libido (via dopamine). SSRIs screw up your ability to have sex, and ejaculate, so I wouldn’t go near them with a bargepole.”

I was able to purchase the drug, and I have run a series of test on myself.

I didn’t have an immediate opportunity to test the amineptine. But when I did, I was quite disappointed.

On a 50 mg dose, I felt about as much as I would feel from a cup of coffee. When I tried a 100 mg dose, I felt the kind of agitation caused (in me) by practically all anti-depression medications, be it trazadone, buspirone, or even St. John’s wort.

OK, amineptine is probably a bit more amphetamine-like. I find that, for example, it suppresses appetite.

I have never tried a huge overdose of amineptine. But even at 100 mg, I find it counterproductive for erections. They just wouldn’t happen on amineptine alone.

I tried it in combination with up to 50 mg of sildenafil citrate. That works, no doubt.

Oh yeah, amineptine was supposed to help orgasms. BS by my judgment. Amphetamine is effective in the treatment of premature ejaculation, and amineptine should work in the same manner. No facilitation of orgasm; rather a delay.

I have spent considerable effort procuring the amineptine, and I did have anticipations, but it’s all hype.