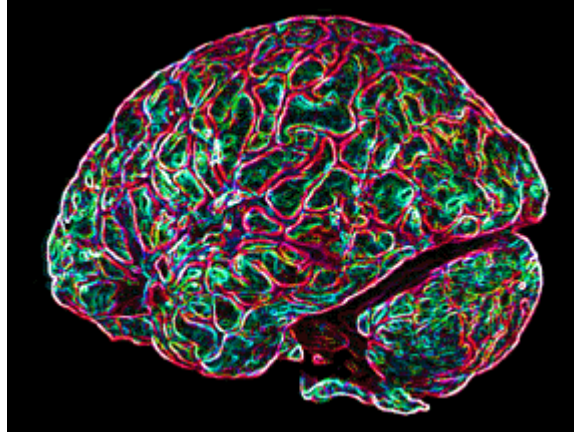


This is Your Brain On Ecstasy

(An MDMA Neurochemistry Slideshow)

Copied from: <http://www.dancesafe.org/slideshow/>



Each slide in this slide-show is rated for its level of technicality with the word BASIC, INTERMEDIATE, or ADVANCED.

Generally, the slideshow progresses from basic to advanced. The advanced slides should not be too difficult to understand if you have gone through the previous ones.

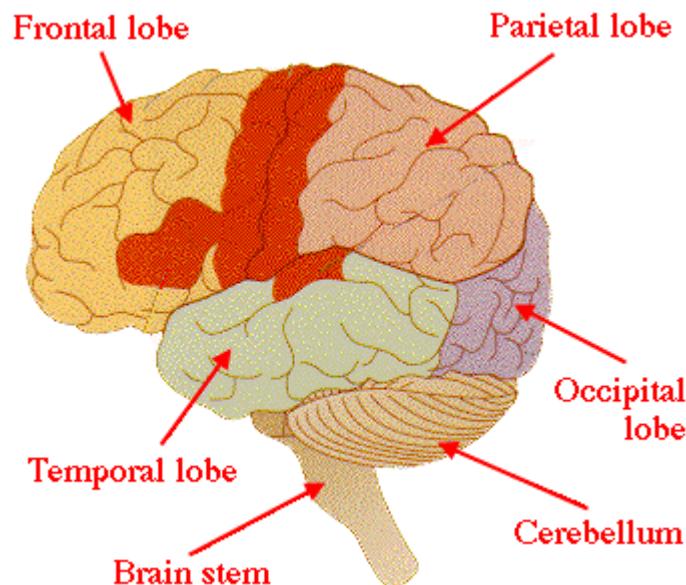
The second part of this slideshow is all about MDMA neurotoxicity, including up-to-date research and current theories.

This Slideshow was created by Emanuel Sferios. Feel free to reproduce any or all of it at will.

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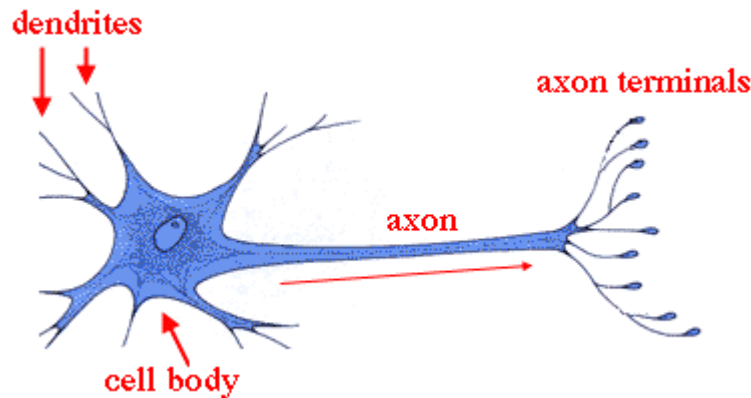
emanuelsferios@dancesafe.org

Slide #1 (BASIC)



This is a model of a typical human brain, showing some of the basic brain areas. You don't need to memorize them all. This is just a warm-up slide to get you started.

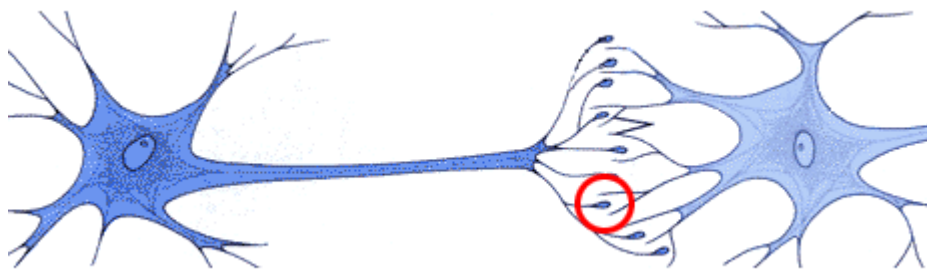
Slide #2 (BASIC)



This is a model of a typical brain cell, or neuron. Your brain contains billions of brain cells. A brain cell consists of a *cell body*, which stores the DNA, *dendrites* which receive chemical signals from other cells, and an *axon*, which carries an electrical signal from the cell body to the *axon terminals*. The axon terminals contain chemicals, called "neurotransmitters," which are released in order for the cell to communicate with nearby cells.

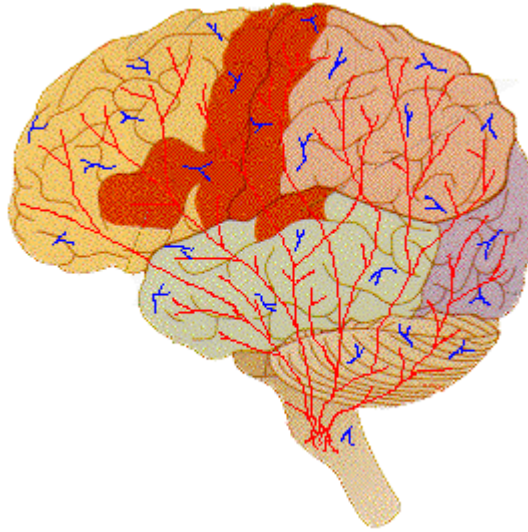
Serotonin is a neurotransmitter, and some brain cells have axons that contain only serotonin. These are called "serotonin neurons." Other brain cells produce and release different neurotransmitters, like *dopamine* or *norepinephrine*, and some produce and release more than one neurotransmitter. However, your serotonin cells only produce and release serotonin.

Slide #3 (BASIC)



Here you can see how the axon terminals, which contain serotonin, lie very close to the dendrites of other, nearby neurons. Notice the gap between the axon terminal of the serotonin neuron and the dendrites of the next neuron. This gap is called the "synapse" and is where the serotonin gets released. Soon we will look at the synapse up close, and see what happens when ecstasy causes large amounts of serotonin to be released there. But first, let's look at how serotonin cells are distributed throughout your brain.

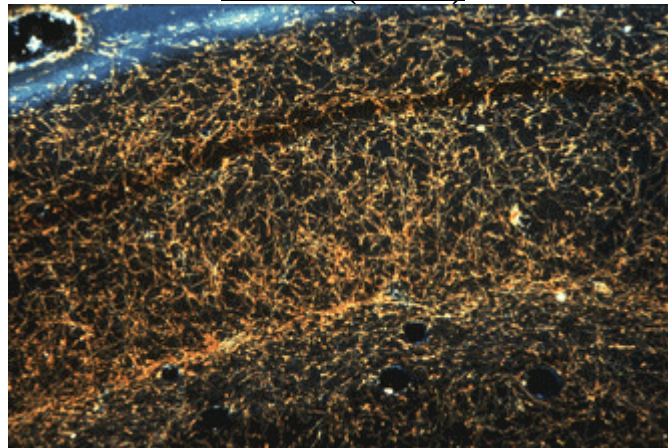
Slide #4 (BASIC)



Most serotonin cells (**in red**) begin in a specific area of the the brain stem called the "raphe nuclei." Their dendrites and cell body's are located here, and they have very long axons that extend into every other part of the brain. Serotonin axons are much denser and have many more tree-like branches than we were able to show in this drawing. They are also much longer than any diagram can easily depict. If you were to stretch out a serotonin neuron on a table in front of you, it might be a foot long, but you still wouldn't be able to see it because it would be so thin. Most people think of brain cells as shorter and confined to particular brain regions (**in blue**). While some brain cells are like this, this is not the case with serotonin cells.

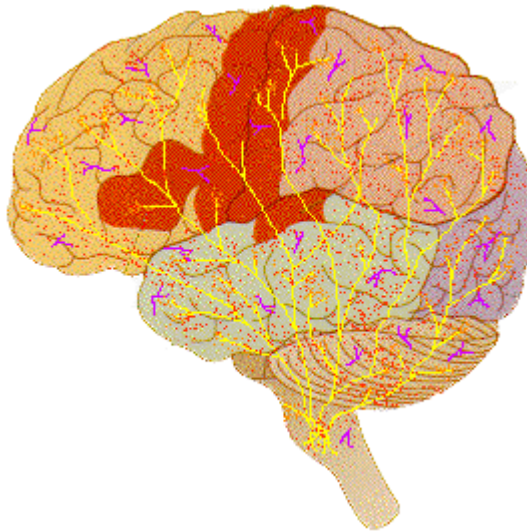
No wonder serotonin plays such an important role in so many brain functions, such as the regulation of mood, heart-rate, sleep, appetite, pain and other things.

Slide #5 (BASIC)



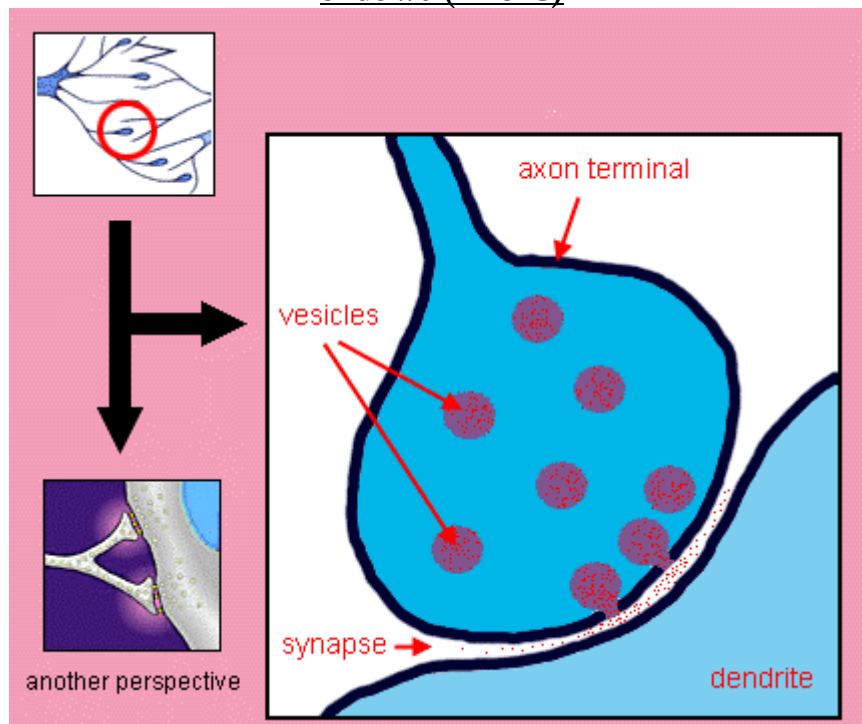
This is an actual photograph of serotonin cells (mostly axons) in a rat's brain. Notice the tree-like branching of the axons. The dark spaces around the serotonin cells are actually densely filled with other brain cells. You can't see them in this picture, however, because only the serotonin cells were stained to make them visible.

Slide #6 (BASIC)



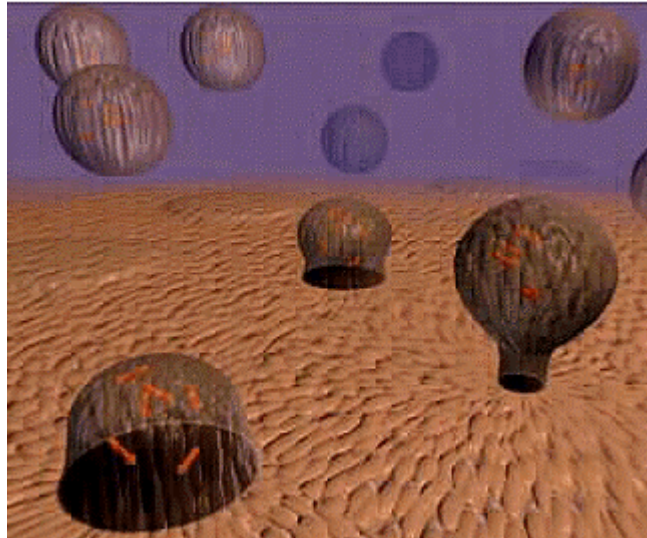
Ecstasy causes your serotonin neurons (yellow) to release large amounts of serotonin (the little red dots), which are stored in the *axon terminals*. This massive serotonin release is responsible for the primary subjective effects of MDMA. MDMA also releases the neurotransmitters *dopamine* and *norepinephrine*. The role of these two neurotransmitters in the MDMA effect will be explained later in the slideshow.

Slide #7 (BASIC)



Inside the axon terminal are small vesicles that contain serotonin molecules. When an electrical charge comes down the axon, these vesicles merge with the outer membrane of the axon terminal and release serotonin into the synapse. We are going to take a closer look at this, because there is a lot that goes on in the synapse. But let's first look at a 3-d drawing of some vesicles inside a serotonin axon.

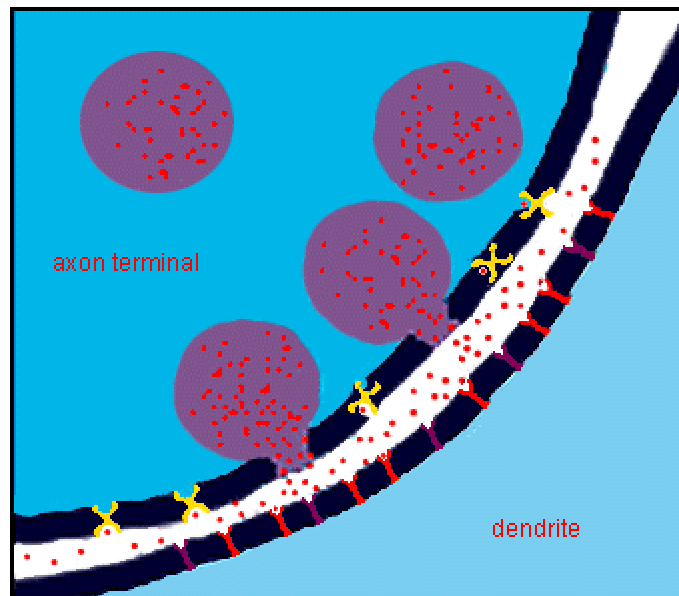
Slide #8 (BASIC)



This is an artists rendition of the view inside a serotonin axon terminal. The vesicles float around scooping up serotonin, and when directed to by an electrical current coming down the axon, they merge with the membrane and release the serotonin into the synapse.

Slide #9 (BASIC)

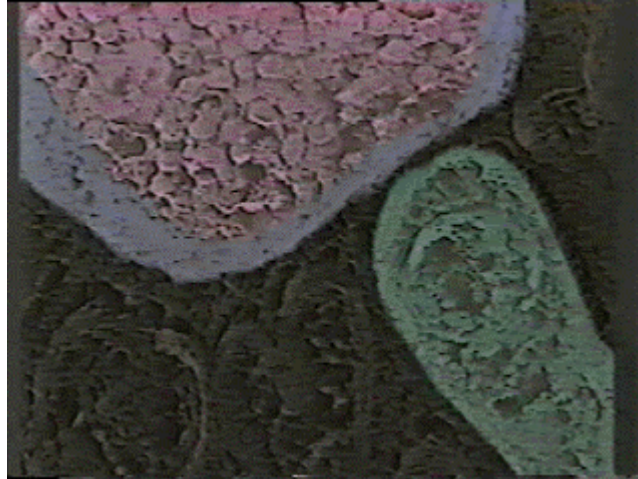
• = serotonin molecule X = serotonin receptor
X = reuptake transporter X = other receptor



Moving in a little closer to the synapse, we can see some serotonin molecules floating around.

We also see some serotonin *reuptake transporters* on the membrane of the axon terminal, as well as *receptors* on the dendrite of the nearby neuron. In order to understand how MDMA works in the brain, and why it produces the effects it does, you need to know what these reuptake transporters and receptors do. But first, just for the fun of it, let's look at an actual photograph of a synapse...

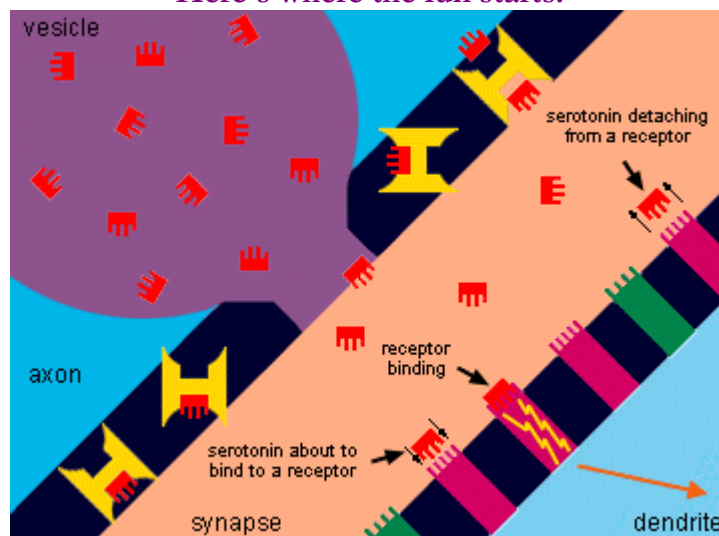
Slide #10 (BASIC)



This is an actual photograph of a serotonin axon terminal (top), a dendrite (bottom), and the synapse in between. Notice the serotonin-filled vesicles inside the axon terminal. You can't actually see serotonin molecules in this picture, nor the reuptake transporters or receptors. This is because they are so small. You can, however, imagine serotonin molecules floating around inside the gray area. Also, notice that some other dendrites are visible even though they haven't been stained like the bright one.

Slide #11 (INTERMEDIATE)

Here's where the fun starts.



This is a closer view of a vesicle releasing serotonin into the synapse.

Serotonin Receptors: The primary reason for Ecstasy's subjective effects.

On the other side of the synapse, attached to the membrane of the dendrite, are these things called receptors. There are receptors for many neurotransmitters. Let's say the magenta-colored ones are serotonin receptors and the green ones are for dopamine. Notice how a serotonin molecule can easily fit into the serotonin receptor, but not into the dopamine receptors (or any other type of receptor for that matter). This is because serotonin receptors are designed specifically for serotonin molecules. When a serotonin molecule attaches to a receptor, which is called *receptor binding*, the receptor sends chemical information down the dendrite to the cell body of the neuron. The cell body then decides, based on the information

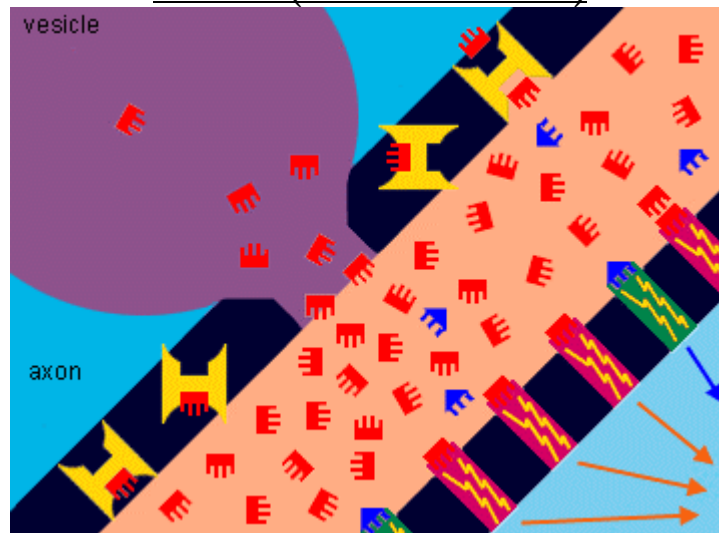
from all its receptors put together, whether or not to fire an electrical impulse down its own axon. If a critical amount of receptor binding occurs then the axon will fire, causing the release of *other* neurotransmitters into *other* synapses. This is how your brain communicates, and something like this is happening in your brain at a normal pace all the time.

Research has shown that your mood is influenced in part by the amount of serotonin receptor binding. When you are happy, it is likely that you have more serotonin receptors activated. Positive events in your life (like falling in love, perhaps) cause greater serotonin release, increasing receptor binding. So does taking ecstasy.

After a little while the serotonin molecule will detach ("unbind") from the receptor and float back into the synapse. When this happens, the receptor stops sending chemical signals to the cell body, and it waits for another serotonin molecule to come along.

(Those yellow things on the membrane of the axon terminal are serotonin reuptake transporters. Don't worry about them just yet.)

Slide #12 (INTERMEDIATE)



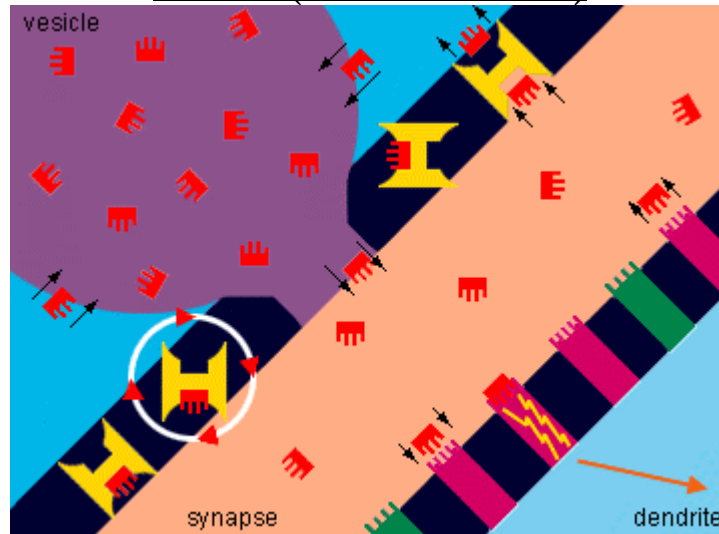
**About an hour or so after you take Ecstasy
(the peak experience)**

When you take Ecstasy, the vesicles release enormous amounts of serotonin into the synapse. This significantly increases serotonin receptor binding (more serotonin in the synapse means a greater chance for some of them to bind to the receptors). This increased receptor activity leads to significant changes in the brain's electrical firing and is primarily responsible for the MDMA experience (i.e. empathy, happiness, increased sociableness, enhanced sensation of touch, etc.). Notice also that there is some dopamine in the synapse as well (the blue things).

MDMA also causes dopamine release (from dopamine cells), but let's not discuss that yet. Keep it in the back of your mind (no pun intended) because it will come up later when we get into neurotoxicity. For now, just notice that the dopamine receptors have also been activated.

The effects of a normal dose of ecstasy last about four to six hours. We will be looking at what happens in the brain during the various stages of an ecstasy experience, as well as some changes that may occur in the brain after long-term, frequent use. But now let's take a look at the "reuptake transporters" (those yellow "H" looking things). To understand how ecstasy works over time in the brain, it is important to know what these things do.

Slide #13 (INTERMEDIATE)

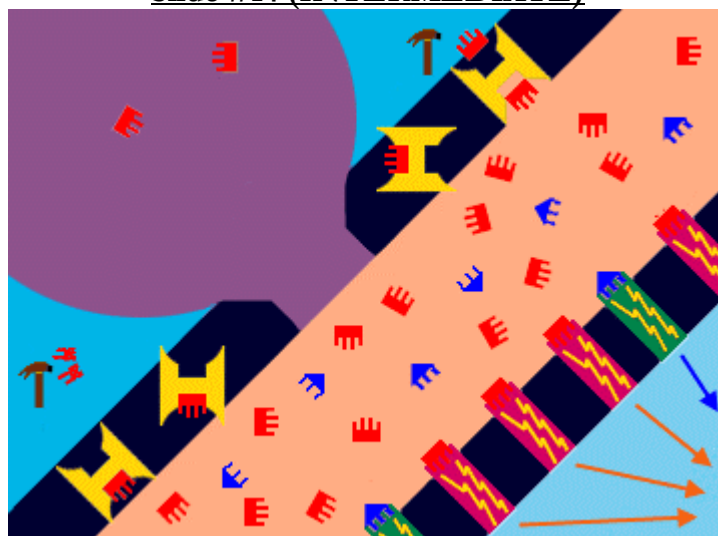


Serotonin Reuptake Transporters

Along with binding to the dendritic receptors, serotonin molecules also bind to "reuptake transporters" on the axon's membrane. These transporters take the molecule and *transport* it back into the axon terminal. They are sometimes called "pumps" and can be thought of as a revolving door. The serotonin enters one side, and the door spins around pushing it out the other side. We have shown here four reuptake pumps in various stages of transporting serotonin. Imagine them spinning and transporting serotonin from the synapse back into the axon.

Reuptake transporters reduce the amount of serotonin in the synapse. Keep in mind that these are *one-way* doors. Serotonin doesn't go through them the other direction. It can only be released into the synapse from the vesicles. As the reuptake pumps are pulling the serotonin back into the axon, some of this serotonin makes its way back into the vesicles, where the MDMA may cause it to be released again. However, some of it gets broken down by Monoamine Oxidase. We show this in the next slide.

Slide #14 (INTERMEDIATE)



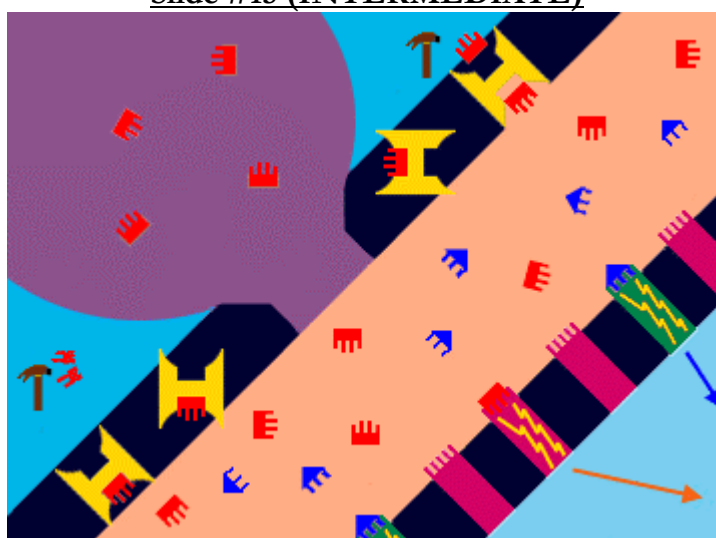
Monoamine Oxidase breaks down your serotonin.

Approximately three hours into your ecstasy experience your serotonin transporters have removed much of the serotonin from the synapse, but there is still plenty around to activate

the receptors, so you still feel the desired effects of the drug. Pretty soon, however, the reuptake transporters will remove most of the serotonin from the synapse, and you will start coming down.

We said in the last slide that some of the serotonin finds its way back into the receptor where the MDMA causes it to be released again. This is true, but notice the hammers inside the axon. This is "monoamine oxidase" (MAO), an enzyme that breaks down serotonin (serotonin is a monoamine, remember). After your reuptake pumps remove the serotonin, MAO breaks most of it down. MAO doesn't really look like a hammer, but thinking of it as a hammer that smashes up serotonin molecules is a good way to remember what it does. (For more information on MAO and the dangers of taking Ecstasy with MAO inhibitors, click [here](#).) Notice too that the dopamine receptors are also still activated as well.

Slide #15 (INTERMEDIATE)



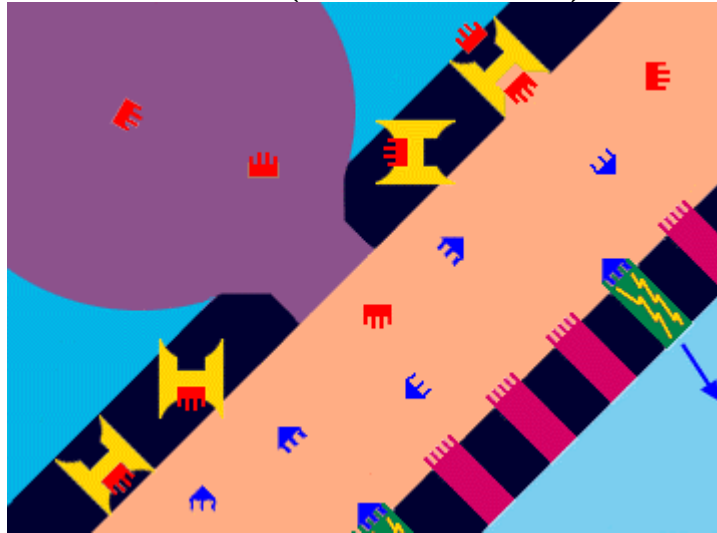
When you start coming down

First, notice that the number of activated serotonin receptors has been reduced because there is less serotonin in the synapse. This means you should be starting to feel somewhat normal again. Also, the uptake pumps are still removing serotonin from the synapse, as usual, and MAO is still doing its job breaking it down. Notice that the dopamine levels in the synapse haven't lowered as much as the serotonin. This is because dopamine replenishes itself much more quickly than serotonin. Notice also that there is a lot less serotonin in your vesicles, and this is mainly why you come down. Simply put, there's no more serotonin left to be released.

The MDMA may still be around trying to make your vesicles release more, but there isn't enough there. In about four hours, Ecstasy has used up most of your serotonin.

You could take more Ecstasy at this point, which a lot of people do. However, this usually doesn't work. You can't just take more ecstasy to regain the ecstasy feeling. Why? Because the ecstasy feeling is really a "serotonin feeling" and you currently don't have enough serotonin left. (It takes time for your brain to build up more, which we will be discussing soon.) Of course, if you took a lower-than-normal dose, you may not have released most of your serotonin, in which case you may feel the effects come on again if you take more. However, you cannot keep doing this repeatedly all night long. There will come a point (sooner rather than later) when you have depleted your serotonin levels so much that taking more Ecstasy will not work.

Slide #16 (INTERMEDIATE)



When you come down some more

Depending on how much MDMA you took, you may end up depleting so much of your serotonin that fewer receptors are activated than before you took ecstasy, when you were in a normal brain state. This is what causes the "ate up" feeling that a lot of users experience when they come down. You can become very depressed at this point, feeling extremely non-social, tired and irritable.

Some people at this point are tempted to take more Ecstasy, because the contrast between how they were feeling an hour earlier and how they feel now is so extreme. But when they take more, it doesn't work. While it may give the user a little more energy (i.e increase the speediness), they won't recapture the empathy and other desirable MDMA effects.

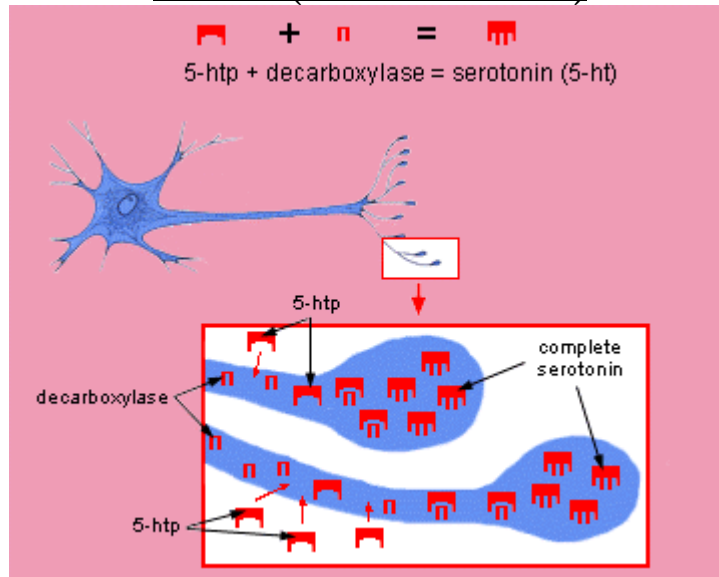
Remember, Ecstasy releases (and then depletes) the serotonin that you already have. It doesn't cause more serotonin to be created.

Your brain needs time in order to rebuild its serotonin levels. This could take up to two weeks. As expected, the larger the dose the greater the serotonin depletion and the longer it takes for your brain to replenish it.

Can these lowered serotonin levels cause depression? Yes. There are a few pharmacological reasons why MDMA use can lead to temporary yet prolonged periods of depression. Perpetually low serotonin levels resulting from weekly MDMA use is one of these reasons. If you take ecstasy on a regular basis, you may be releasing and depleting your serotonin before it has a chance to fully replenish itself. This means you will be operating on lower-than-normal serotonin levels most of the time, and this can lead to depression. Another reason you can get depressed has to do with "receptor downgrading," which we will be discussing soon.

How does your brain make serotonin in the first place, and why does it take so long for it to replenish its stores after they have been depleted by MDMA? Let's take a look . . .

Slide #17 (INTERMEDIATE)



Producing New Serotonin

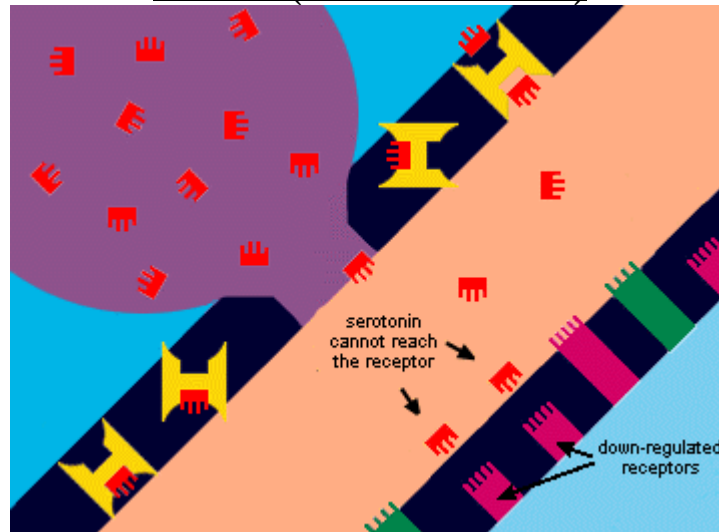
Your serotonin brain cells produce serotonin when an amino acid called *5-Hydroxy-Tryptophan* (5-htp) enters the cell and comes into contact with an enzyme called *decarboxylase*. The 5-htp enters the cell directly through the cell's membrane. It does not have to go through the reuptake transporters, the way previously-released serotonin must. Once in the axon, decarboxylase turns the 5-htp into serotonin, where it enters the vesicles (the vesicles are not shown in this diagram). In other words, after the serotonin is made inside the cell, it moves to the terminal where it is stored in the vesicles ready to be released into the synapse when the time comes.

There's usually plenty of decarboxylase in your cells, but the amount of 5-htp you have can vary depending on your diet. 5-htp is synthesized in your body from another amino acid called *tryptophan*, which is contained in many foods. A diet high in tryptophan-containing proteins can increase the amount of 5-htp in your brain, and thus help your brain build serotonin more quickly.

Normally it takes a long time for your brain to build serotonin. Why? One reason is that tryptophan must go through a number of metabolic changes before it is turned into 5-htp. Another reason is simply that your brain was not made to make serotonin very quickly. Normally, it doesn't need to, because serotonin is not usually released in very large quantities. As a comparison, dopamine is released in larger quantities under normal circumstances, and your brain is thus built to replenish dopamine much more quickly. Researchers say that the dopamine system is "robust" in this sense, while the serotonin system is "delicate."

Some ecstasy users take 5-htp supplements to restore their depleted serotonin levels more quickly. (For information on using 5-htp in this way, see our page on [general health and safety](#).)

Slide #18 (INTERMEDIATE)



Down-regulation of Receptors:

The most probable cause of ecstasy-related depression.

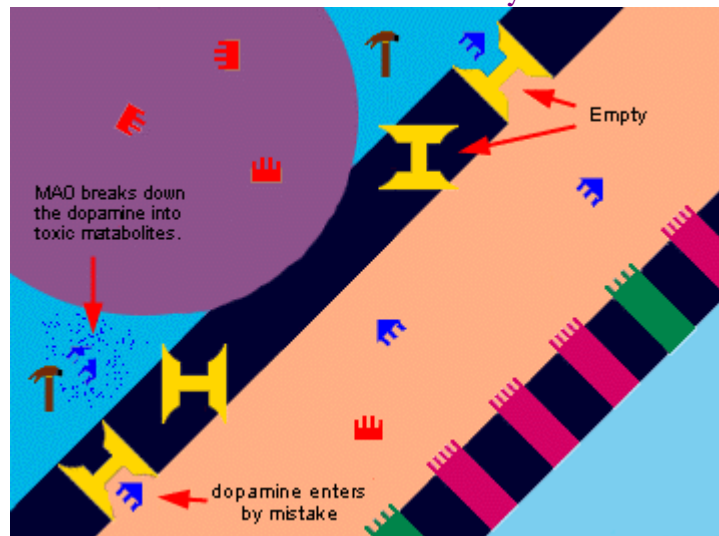
The brain is built to adapt to changing circumstances. One of the ways your brain adapts is through the up-and-down regulation of receptors. What this means is that if your serotonin receptors get hyper-activated by serotonin molecules, they may retreat into the membrane of the dendrite, essentially shutting themselves down for a while. One theory says they do this in order to avoid getting damaged from over-stimulation. Another theory says that it is just a way for your brain to maintain a balanced, normal state. Whichever one of these theories is true, it has been proven conclusively that serotonin receptors will down-regulate over time if bombarded with large amounts of serotonin.

This may lead to depression, even after your brain serotonin levels have been restored, because the serotonin cannot bind to downregulated receptors. Many ecstasy users we have interviewed have reported periods of depression lasting months or even a year or more following a period of heavy use. Keep in mind, however, that most evidence of MDMA-induced depression is anecdotal, based on reports by MDMA users. Since many people experience depression it is difficult to know for sure whether the depression experienced by MDMA users is really related to their MDMA use. While MDMA is known to reduce serotonin levels, serotonin levels varies greatly among healthy, non-depressed, non-MDMA users. Until more research is done, we cannot know for sure whether MDMA users actually have a greater risk of experiencing depression than non-users. (Of course, it is wise to be cautious, and so we have included a section on our [health and safety page](#) about ecstasy-related depression.)

Let's now turn to the issue of neurotoxicity . . .

Slide #19 (Advanced)

Part II: Neurotoxicity



This next section of the slideshow deals with MDMA neurotoxicity. If you have understood everything so far, you should have no trouble with this section. When you are through, you may want to read "the short answer" on the [neurotoxicity](#) page of our site. It contains a more overall analysis.

The current theory

The most current theory of how MDMA causes neurotoxic damage in laboratory animals goes like this:

After MDMA depletes serotonin, the reuptake transporters are left vacant and exposed. When this happens, dopamine enters the transporter and gets taken up into the serotonin axon, where it isn't supposed to be. Studies have shown that dopamine itself is toxic to serotonin cells. But if that weren't enough, MAO comes along and breaks it down into hydrogen peroxide, which is also toxic to the cell. (Yes, the same hydrogen peroxide they put in hair bleach!) The hydrogen peroxide then "oxidizes" certain parts of the cell which don't normally get oxidized ("oxidize," as used here, basically means to break down with oxygen). Researchers sometimes refer to this as *oxidative stress*, and a number of studies have looked at *anti-oxidants* like Vitamin-C as a possible agent to prevent MDMA's neurotoxicity (see our section about pre-loading on our [neurotoxicity page](#) for more info on this).

Once again . . .

To re-cap we have (1) serotonin depletion causing the uptake transporters to become empty. Then (2) dopamine, which exists in higher levels in the synapse now, enters the uptake transporter. (3) This dopamine is broken down by MAO into hydrogen peroxide. (4) The dopamine is toxic to the cell and so is the hydrogen peroxide, by producing oxidative stress.

How did they come up with this theory? And is there evidence for it?

The researchers who first devised this theory (Jon E. Sprague, Shannon L. Everman and David E. Nichols) called it an "integrated hypothesis." They looked at a decade worth of MDMA research and tried to put the pieces together. They came up with this theory in the summer of 1997 and it was published in 1998. To date, it is still the dominant theory of how MDMA causes axon damage in laboratory animals, and would most likely apply to humans as well, should neurotoxic damage in humans be proven conclusively.

Technical details

Below are some rather technical explanations of how they came up with this theory. If you're not interested in such detail, go on to the next slide.

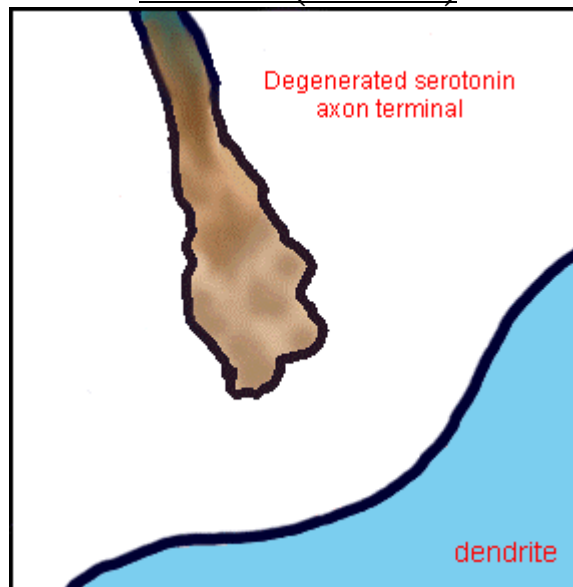
Looking at past studies of MDMA neurotoxicity, it is clear that dopamine plays a crucial role. For example, in 1988, it was discovered that pre-treating rats with α -methyl-p-tyrosine, a substance which inhibits the synthesis of dopamine, prevents MDMA neurotoxicity (Stone et al.). Also, in 1990 a study showed that if you destroy all of the rat's dopamine terminals before giving them MDMA (thus eliminating all their dopamine), they sustain no serotonin axon loss (Schmidt et al.). Furthermore, in the same year they also discovered that if you give the rats L-DOPA, a dopamine precursor, they sustain more neurotoxic damage when given MDMA. And another study in 1991 demonstrated a linear correlation between the amount of dopamine release and the extent of MDMA-induced axon loss in rats (Nash and Nichols).

In 1987 researchers discovered that MDMA itself releases dopamine (Schmidt et al., Steele et al.). Then they discovered in 1996 that serotonin release also increases dopamine release (Gudelsky and Nash). It does this because one of the serotonin receptors (receptor 2A), when activated by serotonin, stimulates the synthesis and release of dopamine (Nash; Schmidt et al., 1990). Also, drugs which block the 2A-receptor have been shown to reduce extracellular dopamine levels.

They also discovered that dopamine actually can get uptaken into the serotonin terminal (Faraj et al, 1994) and that the terminal dose, in fact, contain a type of MAO known to metabolize dopamine (MAO-B).

To further support the theory, in 1995 they discovered that MAO-B inhibitors (L-deprenyl or MDL-72974) reduce neurotoxic damage in rats given 40mg/kg of MDMA.

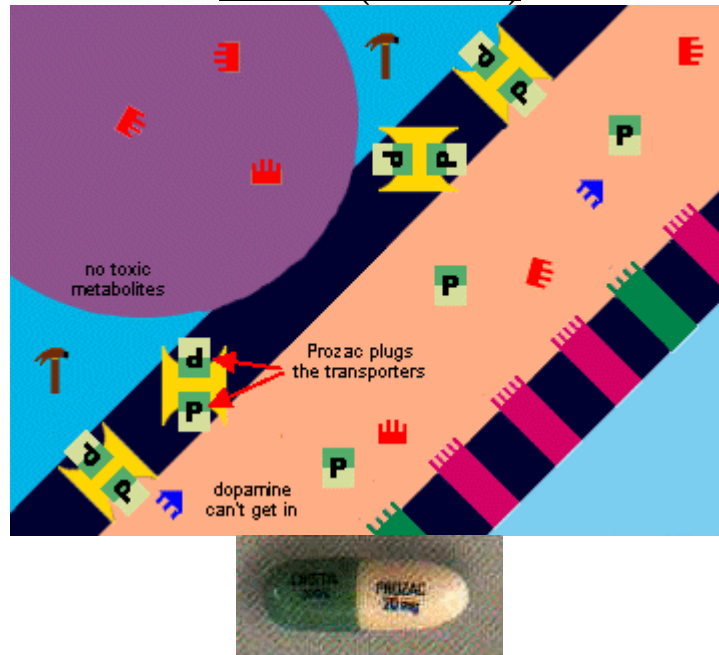
Slide #19b (Advanced)



"All shrivelled up."

This is what a damaged serotonin axon terminal might look like under a microscope using the "Fink-Heimer" silver staining method, or another one called "immunohistochemical" staining.

Slide #20 (Advanced)



Prozac Prevents MDMA Neurotoxicity in Animals

A number of studies have shown that selective serotonin reuptake inhibitors (SSRIs) like Prozac prevent MDMA's neurotoxic effect. The theory as to why this happens is this: SSRIs plug the reuptake transporters, thus preventing dopamine from getting into the serotonin axon terminal (see the previous slide). Notice that the prozac fits perfectly into the transporter. Researchers refer to this as "affinity" and say that prozac has a greater affinity for the uptake transporter than other things in the synapse, including serotonin. In other words, Prozac will bind to the transporter *first*. It will also stay there for a relatively long time. Prozac has a half-life of 30 hours, which means it takes 30 hours for half of it to leave your body, another 30 hours for half of what's left to leave, etc. It is thus called a "long-acting" SSRI. It plugs the transporters for a longer period of time than the other common SSRIs do. Research has shown that brain serotonin levels remain significantly depleted for approximately 24 hours after a large dose of MDMA. It is during this time period (between 6 and 24 hours after dosing) that the brain's serotonin transporters are left empty and vulnerable, and it is during this time that the neurotoxic damage occurs. This means Prozac may be more effective at preventing MDMA neurotoxicity than other SSRIs, which do not last as long in the body (although this is unknown).

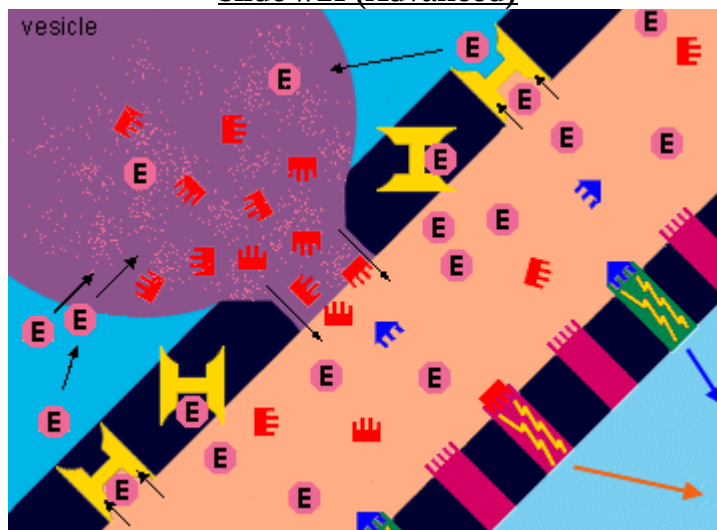
An important observation in these studies was that prozac prevented the neurotoxic damage even when given up to six hours *after* the MDMA. What they did was inject all the animals with MDMA, and then every hour they gave some of them an injection of Prozac. Only the animals who got the Prozac during the first six hours showed no damage. The ones who got the Prozac on the seventh, eighth, ninth and tenth hours (etc.) sustained damage, with the ones who got the prozac later sustaining more.

What about in humans?

While no studies have been done to assess the effectiveness of Prozac as a neuro-protective agent against MDMA neurotoxicity in humans, there is no reason to suspect that human brains react differently than animal brains in this regard.

Next let's take a look at how Ecstasy actually enters the serotonin cell...

Slide #21 (Advanced)



How does Ecstasy cause the release of serotonin?

We've been neglecting this question for a long time, because we didn't want to present too much information all at once, and there wasn't any pressing need early on to show this.

However, we'll show you now.

MDMA enters the serotonin axon terminal by going through the uptake transporters!

Researchers say MDMA has a greater *affinity* for the transporter than serotonin (just like prozac does). This means that the MDMA will be the first thing to get into the axon terminal. Once there, it interacts with the vesicle, causing it to pour its serotonin into the synapse. The

important thing to be aware of is that the MDMA does its thing only after entering the serotonin axon terminal via the uptake transporters. This is important, as we will soon see.

Slide #21-b (Advanced)



MDMA Makes the Serotonin Transporters Work In Reverse!

A new theory is gaining wider acceptance among researchers about exactly how MDMA causes serotonin to be released into the synapse after it enters the axon. It is no longer assumed that the MDMA somehow interacts with the vesicle, causing it to pour its serotonin into the synapse. Rather, the MDMA is thought to make the transporters work backwards, transporting serotonin from inside the axon to the synapse!

Here's the theory:

Once the MDMA enters the transporter, it falls off inside the axon terminal, and leaves the transporter in such a state that a serotonin molecule now binds to the place where the MDMA fell off. The transporter then spins around and deposits the serotonin molecule into the synapse, where another MDMA molecule binds to where this serotonin molecule used to be.

This all happens through a four-step process:

1.

MDMA is released from the transporter into the axon when the transporter undergoes a change in "configuration." (The transporter is basically a group of proteins that can change configuration, or "shape." Depending on its configuration, certain molecules are more likely to bind to it. This is called "affinity." When a molecule with a high affinity binds to a transporter, it changes the transporter's configuration, which eventually causes the molecule to unbind or "fall off," possibly on the other side. This is what makes the transporter capable of "transporting" molecules between the synapse and the axon.)

2.

The transporter now has the correct configuration to attract and bind cytoplasmic serotonin inside the axon.

3.

The bound serotonin is then transported out of the presynaptic cell, and when the transporter changes configuration again, the serotonin falls off into the synapse.

4.

The transporter is now in the correct configuration to attract more MDMA in the synapse, and the whole process is repeated.

Remember, serotonin is produced inside the axon (through the conversion of 5-htp), and under normal circumstances it enters the vesicles, which release it, over time, into the synapse. The reuptake transporters then bring some of the serotonin back into the axon, where it enters the vesicles again and is recycled. On MDMA, however, most of the serotonin enters the synapse directly through the reuptake transporters (in the opposite direction from what is normal).

So now let's go back to a previous slide and look at your brain on ecstasy again.

Slide #22 (Advanced)



This is your brain on ecstasy, really.

If you can describe everything going on here, you're doing pretty well. Let's keep going . . .

Slide #23 (Advanced)



What if you take Ecstasy while you're on Prozac?

Prozac has a greater affinity for the reuptake transporter than MDMA and serotonin both. Most people on Prozac don't feel much when they take ecstasy, because the ecstasy cannot get into the serotonin axon terminal in order to release the serotonin. The ecstasy does cause some dopamine release, as well as norepinephrine, so the user will feel a little something. But the primary effects will be largely inhibited.

Of course, this depends on how much Prozac the person is on, and how big of a dose of MDMA the person takes. Someone on a low dose of Prozac (10mg) will feel more effect than someone on a high dose (40mg). Someone who takes a low dose of MDMA (60mg) will feel less than someone who takes a high dose (150mg).

**This slideshow is an ongoing project. We plan to keep adding pages to it over time.
Please check back soon!**

Upcoming slides:

Details of the Prozac/MDMA experiments on lab rats.

What is the best way to use Prozac for its neuroprotective effect?

What causes MDMA tolerance?

How 5-HTP supplementation prevents a small amount of the neurotoxic damage in rats.

How exactly does MDMA cause the release of serotonin once it is uptaken into the axon?

How is hyperthermia (high temperature) related to neurotoxicity?