

# We Love to Be Scared on Halloween But Fears and Phobias Are No Laughing Matter

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It seems we humans spend a good deal of time and energy in pursuit of thrills, chills, and spills, especially at this time of year. From haunted houses to ghoulish get-ups, we love to be scared.

Even when it's not Halloween, scary pursuits are commonplace. For fun we jump off bridges tethered to bungee cords or drive fast cars or swim with sharks. Or watch others do it. On the face of it, these fear-seeking behaviors seem contrary to our basic instincts of self-preservation. But it could be that they are vestiges of our genetic and environmental past.

“Many individuals are motivated to seek increased arousal,” explains Fordham University’s Dean McKay, whose research focuses on obsessive compulsive disorder and anxiety disorders. “Intense experiences such as bungee jumping or extreme rock climbing satisfy this need. Most individuals seek this out in some way, whether by direct experience or (vicariously) by observation.”

High jinks and thrills aside, fear is an excellent survival tool handed down by our cave-dwelling ancestors who probably learned fear in order to avoid saber-toothed tigers and woolly mammoths. Today’s world can be quite scary, too; we teach life-preserving fears to our children — warning them about cliff edges and hot stovetops and talking to strangers.

So, fear is a good thing. But like so many good things, too much of it can be a problem. Too much fear

can be debilitating, and the result can be an anxiety disorder or phobia.

At some point in our lives, about three in 10 of us experience an anxiety disorder severe enough to meet diagnostic criteria for impairment, according to APS Fellow and Charter Member David Barlow, Director of Boston University's Center for Anxiety and Related Disorders. And 11 percent of us meet criteria for a specific phobia.

Control is the key, McKay says. Halloween revelers and thrill-seekers have it; phobics and victims of anxiety disorder don't. When systems that serve a critical need malfunction, the result is disease. Think of phobias and anxiety disorders as "diseases" of the brain's "fear system."

Our understanding of phobias has been undergoing a revolution. "For a long time," says Barlow, "we thought that people with phobias must have had some traumatic experience that created the fear. Now we know that's not true. Only a minority of people with specific phobias have actually had a bad incident."

Fear of water can show up in children too young to have any meaningful experience with water, he says. And when you compare people who have a fear of dogs with others who don't, about the same number in each group have had a bad experience with a dog. The dog attack might precipitate phobia in those already vulnerable, but it doesn't cause it.

It's more often quite the opposite, according to research by Ross Menzies, University of Sydney, Australia. Most stuntmen, he found, have histories of serious childhood accidents. Far from making them phobic, they embraced the wellspring of their fear and built a livelihood around mastering it.

McKay says this demonstrates that phobias are not learned: We're born with them and "events previously thought to bring about phobias actually lead to greater approach for the object."

But don't go looking for a "phobic gene." According to Barlow, the inherited DNA codings are a bit more complex than that. "We're now learning that they're not single genes, they're variations in genetic structure, certain genes with certain patterns of alleles that, when they're turned on by events in the environment, would make us more susceptible."

So yes, genetic inheritance does play a role, accounting for perhaps a third to half of the variance between those who develop phobias and those who don't, "but it's misleading to talk like that. The genetic piece means nothing unless it's part of the feedback system."

He likens that system to an "intricate dance" of "triple vulnerabilities," genetics being only one dancer. The other two are psychological vulnerabilities — one generalized, based on early experiences developing a sense of control over events or lack of it, and the other more specific, in which one learns to focus anxiety on specific objects or situations. "When these three vulnerabilities line up, then you're at substantial risk for developing a phobia," Barlow says. "If they don't line up, if you just have one and not the others, you're at much less risk.

"There was always this myth that somehow anxiety disorders were due to chemical imbalance and could be treated with pills, whereas the psychological myth was that it was some distorted cognition or

learning. We now know all these things are in it and interrelated. It's all a system. You have to change the whole system, not just neurotransmitter endings."

One of the most successful ways to counter the genetic vulnerability, he says, is by changing the internal and external environment. This "could be behavior, regulating your emotions, or learning new ways to respond to stress, which in turn will influence brain functioning."

### **Not All Phobias Are Alike**

Of course, not all phobias are alike. They are typically grouped under three headings: specific phobias (like fear of heights, dogs, or water); social phobias, now more often called social anxiety disorders; and panic disorders — inexplicable panic attacks.

More than simple fear is involved, McKay says. It's also disgust, "a pretty powerful emotion for engendering avoidance. In the past two or three years, we've seen a big upswing in this kind of work, where an effort is being made to isolate the way disgust contributes to phobias." He is currently co-editing a book on the subject with Bumni Olatunji of Vanderbilt University, to be published in 2007.

How phobias are treated is another of the major turnarounds prompted by psychological research over the past few years. "Up until recently," Barlow says, "the usual approach was to see patients once a week and have them do a lot of prescribed exercises between sessions." Treatment lasted 10 to 12 weeks. The innovation, pioneered by Lars-Göran Öst, Stockholm University, "was simply to do it all at once in an intensive fashion," says Barlow, who now treats phobic patients using Öst's method — in a single four- to six-hour session, "more equivalent to doing surgery."

In that session, a patient gets intensive exposure to the thing or situation that triggers the terror. A patient might be shown insects, or small animals brought in from a pet store. The acrophobe may be taken to high places and a claustrophobe to enclosed spaces. Guided by the psychologist, the patient learns to "extinguish" the fear by learning to control it.

"The success rates, particularly for adults, seem to be up in the 80 to 90 percent range," Barlow says, "so it's clearly the treatment of choice" for specific phobias. Only about 10 to 15 percent of the patients relapse, and even then "it's fairly easy to go back and have a booster session."

More generalized social anxiety and panic disorders still require long-term therapy or medication, which are about equally effective, says Barlow. The drugs are either antidepressants, such as selective serotonin reuptake inhibitors (SSRIs), or high-potency tranquilizers, although the latter are used much less and typically as adjuncts to therapy because patients can develop dependence on them.

While some patients prefer the "quick fix" of a pill, especially when they see it promoted on television, surveys have shown that around 75 percent prefer therapy that teaches them to master their fears. This is due primarily to the side effects of the medications, but also because relapse is more common with drugs.

"Psychological treatments are more durable," Barlow says. "The patients actually seem to learn something that has lasting benefit." As for combining drugs and therapy for anxiety disorders, he says, "Surprisingly, there does not seem to be any advantage to combining the two. It's more expensive and

there's no evidence that the treatments are additive.”

Exposure therapy teaches patients to regulate and master their emotional response “and to accept that there has to be some confrontation with both the internal and external situation that provoked the phobic response,” Barlow says.

The main disadvantage of the new one-day treatments is that they aren't readily available. “These intensive treatments came into their own in the past five years, but are still not widespread,” he says. “Our biggest obstacle is actually getting them out there to the consumers. They require a lot of training, and that expertise is just not widely available yet.”

Barlow's center at Boston University is now experimenting with intensive exposure therapy for panic disorders and social phobias as well. It recently received a five-year National Institute of Mental Health grant of \$2 million to test one-day exposure therapy on 50 adolescents with panic disorders, comparing their results to patients who received more traditional care. The investigators are beginning with adolescents because the need is greater: It's harder for schoolchildren to get to a clinic every week than for adults.

A similar study using adult patients has also been started, and the center has a pilot project to treat severe social anxiety by putting small groups of patients together for several hours a day for a week. After their group interactions, they are sent out on assignments to interact socially in public, such as at a coffee shop.

### **Relapse is Tied to Context**

The therapy springs from intriguing results in animal research. Mark Bouton, University of Vermont, has been working with laboratory rats since 1979, building on Pavlov's work on conditioned responses in dogs almost a century ago. Bouton demonstrated that a rat learns to fear a certain sound because it is always accompanied by an electric shock, then “extinguishes” that fear response by repeatedly hearing the tone without the shock. But he never “unlearns” the original fear; it lives on alongside the new learning, ready to spring alive again — in the right context.

He discovered this by teaching rats fear in one chamber, teaching them to extinguish it in a different chamber, then putting them back in the original chamber. When the tone sounded in the original chamber, the rats once again froze in fear, even after the fear was extinguished in the second chamber. It also happened if they were placed in a neutral third chamber and received the shock with the bell tone. The fear came rushing back to the fore.

“The fact that the original performance can recover after extinction may be an important insight into understanding relapse after therapy,” Bouton and his colleagues write in their chapter in a newly published book on fear and learning (Bouton 2006). “(M)any manipulations of the context can cause an extinguished fear...to recover or return.”

Their recent research has focused on how to take advantage of this context-dependency to prevent relapse into the original fear reaction, without much success. “(A)s far as we have been able to determine,” they write, “extinction can often still remain surprisingly sensitive to the context (and the original responding thus susceptible to relapse) even after extinction procedures that have been designed

to optimize the new learning.”

The one approach they say “appears especially promising” at preventing relapse is building treatment “bridges,” such as conducting therapy in the very situations that usually trigger the fright.

They offer multiple ways of doing this: “Conducting exposure therapy in the context where relapse is going to be a problem provides the most direct bridge,” they write. “Retrieval cues for extinction provide another kind of bridge in the sense that they bring a piece of therapy to the relapse context. Occasional reinforced trials in extinction are another kind of bridge because they allow extinction in the presence of a cue (a new reinforced trial) that may be a strong stimulus for relapse.”

Bouton is now studying time as context. “Extinction is specific to the temporal context in which it is learned,” he explains, “just as it appears to be specific to the room in which it is learned. We are therefore looking at a lot of implications of this idea that are leading us in some interesting new directions. One practical implication is that extinction trials — therapy sessions — that are widely spaced in time should protect the system from relapse that might otherwise occur at intervals shorter than the interval between extinction trials.”

Given that relapse rates for specific phobias are so low, the research appears to have greater significance for social anxiety and panic disorders, where relapse is more common. “I think of our research as a kind of caution sign,” Bouton says.

### **A Clearer Picture in the Brain**

Thanks both to animals like Bouton’s rats and brain-imaging research, we now also have a better idea of what’s happening in the brain when phobias strike. “It’s very clear that the amygdala is a central player from the animal research,” says Scott Rauch, MD, Associate Chief of Psychiatry for Neuroscience Research at Massachusetts General Hospital, “but it’s just one part of a larger network. At least two other key areas play a role. The ventromedial prefrontal cortex (vmPFC) probably plays a critical role in the capacity to recall extinction memory, whereas the hippocampus plays an important role in the context” in which that memory was acquired.

Raffael Kalisch and colleagues at the University of Hamburg, Germany, are looking in the other direction — removing context from the equation. They have conducted the first study of fear extinction’s context-dependence in humans. “A key feature of this study,” they write in the *Journal of Neuroscience* (in press), “is that our design allows delineation of the neural circuitry involved in that function, using a psychological manipulation that engenders recall of extinction memory in the appropriate context.

“Clinically, contextual restrictions on extinction can considerably complicate anxiety therapy.... For therapeutic purposes, therefore, it often may be desirable to create non-contextualized extinction memories.” They say their data suggest this might be achieved by making the vmPFC-dependent recall of the extinction memory — the therapy — independent of the hippocampus.

A clearer picture of how the three realms of the brain interact is slowly emerging, says Rauch. In patients with post-traumatic stress disorder (PTSD), “quite an array of data from a variety of imaging tools” shows that “the amygdala is hyper-responsive, and that the vmPFC and hippocampus are both structurally small and of reduced function. It is hypothesized (but not yet shown) that the reduced

function results in insufficient inhibition of the amygdala.”

The amygdala also plays a role in learning about safety, he says, but “it seems to take the prefrontal cortex to recall that extinction learning and to suppress the amygdala’s response.”

Rauch and colleague Mohammed Milad also have shown that the thickness of an individual’s vmPFC correlates with the ability to recall the fear-extinction, and that may link it to personality as well. “The thicker the area, the more extroverted a person might be. The idea being, if you’re able to extinguish these adverse associations effectively, it enables you, when you have a bad experience, to limit that to the situation in which it occurred and not to over-generalize it. It allows you to be somebody who is more courageous or outgoing in the way you attack life.”

There are differences, of course. It’s generally believed that anxiety disorders and PTSD have similar brain mechanisms, he says, “but we also know these disorders are distinct one from another, and there ought to be some differences. That’s precisely where the science is right now, trying to understand what the similarities and differences are.”

For example, data from imaging studies “suggest that PTSD and panic subjects show exaggerated amygdala responses to general threat-related stimuli as well as stimuli related to their particular fears, whereas for (specific) phobias, exaggerated amygdala responses may be limited to the stimuli related to their particular fears.”

Panic attacks are also being re-examined in light of human imaging studies, Rauch says. Historically, panic disorder attacks were thought to arise spontaneously, but research shows the amygdala can be activated subconsciously. This opens the door to reinterpreting “spontaneous” panic attacks. “It opens the possibility that, even though the individual is not aware of what stimulus in the environment tweaked their amygdala, it doesn’t mean there wasn’t any stimulus, just that they didn’t know what it was.”

For the most disabling cases that require more aggressive treatments — panic disorders, PTSD, obsessive-compulsive disorder — Rauch says, “as we come to understand underlying mechanisms and brain circuitry, it may be possible to develop newer and better medications, or use treatments to influence the limbic system circuitry, known to play a critical role in anxiety disorders.”

Experimental procedures being investigated include lesions that cut the circuitry at targeted locations; “deep brain stimulation,” in which implanted electrodes modulate the circuitry; electro-convulsive therapy; and trans-cranial magnetic stimulation — putting a magnet against the head to create a magnetic field that influences brain activity.

Whatever technology comes along for extreme cases, however, it is clear that for the foreseeable future behavioral therapy, and especially the innovative intense exposure therapies being developed by psychological science, will be called upon to carry the load in treating most patients with phobias and anxiety disorders.

## **Reference**

Bouton, M.E., Woods, A.M., Moody E.W, Sunsay, C. & García-Gutiérrez, A. (2006). “Counteracting the Context-Dependence of Extinction: Relapse and Some Tests of Possible Methods of Relapse

Prevention, "In *Fear and Learning: Basic Science to Clinical Application* (175-196). In M. G. Craske, D. Hermans, & D. Vansteenwegen (Eds.), Washington, DC: American Psychological Association.