

# Zombies Among Us

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Members of Western culture have for nearly a century been strangely preoccupied with the theme of zombies. This obsession with fantasy monsters may be an expression of concern about something real but unclear and highly threatening. There is growing awareness that parasites evolve means of redirecting the behavior of their hosts in such a way that it favors their own reproduction. In other words, almost all parasites are able to “zombify” their hosts to some extent, changing their behavior in specific ways that support the parasite’s lifecycle. One better-known example is *toxoplasma gondii*. This parasite is known to change the behavior of its hosts – for example mice – to make them more likely to be eaten by cats, their natural predators. The zombie mice are influenced to act in ways that directly threaten their own interests. A significant percentage of humans are also infected with this parasite, and can be regarded as partially “zombified.” The strange human behaviors we call addictions bear a striking resemblance to the zombie behavior of parasitized animals. Although not likely the result of infection by a biological parasite, addictions may be examples of parasitic habits. Addictions fit into a class of dysfunctional, stereotypic, self-reproducing, parasitic behaviors that spontaneously emerge in every higher animal subjected to environmental constriction.

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**A**s a culture, we are strangely preoccupied with zombies. We have a seemingly endless appetite for books and cinema featuring these nightmare creatures. In this paper I hypothesize that our fixation on unreal zombies may reflect an uneasiness about something that is very real indeed. In the natural world, the real world, some very zombie-like things do exist. They are created by parasites.

Parasites are indeed able to transform their animal hosts into beings that closely resemble the popular image of zombies, half-alive creatures that mindlessly do what the parasite needs them to do. This topic can be pretty scary. Why would we entertain thoughts about it, even as fantasy? Well, we ride roller coasters to experience “danger” in an unreal, safe way. Maybe it makes sense, then, that we would similarly entertain in zombie fantasies our fears about something much more real.

In books and movies, human beings can be transformed by disease or curse into mindless horrors driven either to eat your brains, or – worse – to infect you with their essence so that you turn into a zombie yourself.

According to the dictionary, a zombie is “a will-less and speechless human (as in voodoo belief and in fictional stories) held to have died and been supernaturally reanimated” or “a person held to resemble the so-called walking dead.”<sup>1</sup> Unlike normal people, zombies behave seemingly without awareness, as if they were some kind of malignant robots.

## Some history

The public’s interest in this topic has proved amazingly persistent. Zombie-themed films began appearing almost as soon as movies began – nearly 100 years ago. Early films include “White Zombie” (1932, starring Bela Lugosi), and “I Walked with a Zombie” (1943, Francis Dee and Tom Conway). Through the next decades their continuing popularity was reflected in a dense flurry of similar films, such as “The Plague of the Zombies” (1966, Andre Morell and Diane Clare), and “Zombie Holocaust” (1980, Ian McCulloch), to name just a few. And there is of course the unforgettable 2009 “Zombieland” (Woody Harrelson and Emma Stone).

After all this time the zombie theme has been thoroughly worked over – more than enough to turn it into a threadbare stereotype. By all rights the topic should be intensely boring by now. Yet the fascination continues. Zombie movies, books, and TV shows are still being turned out by the hundreds. To be sure, in our era they are often spruced up by adding secondary themes, as in the 2015 film “Navy Seals vs. Zombies” or the 2016 “Pride and Prejudice and Zombies.” There is a zombie comedy musical – the 2018 Disney film entitled simply “Zombies.” According to the description in the Internet Movie Database, this film is about a zombie and a cheerleader who work together to show people that they can achieve a lot “when they embrace their differences and celebrate what makes them a community.”<sup>2</sup> There’s even a catchy song about zombies that’s used to train aspiring singers!<sup>3</sup>

And there's something else. Oddly, zombies have for many years been the focus of formal philosophical discussions – generally discussions about consciousness. The question for philosophers is whether something that looks, behaves, and speaks as if it were a conscious being, can in fact have no consciousness.<sup>4</sup>

An article from *The Stanford Encyclopedia of Philosophy* says,

*Zombies in philosophy are imaginary creatures designed to illuminate problems about consciousness and its relation to the physical world. Unlike those in films or witchcraft, they are exactly like us in all physical respects but without conscious experiences: by definition there is 'nothing it is like' to be a zombie. Yet zombies behave just like us, and some even spend a lot of time discussing consciousness. Few people, if any, think zombies actually exist. But many hold they are at least conceivable, and some that they are possible.*<sup>5</sup>

### Searching for understanding?

Like a movie zombie, our fascination refuses to die. It has been staggering through the public awareness for a supernaturally long time. Why would we continue to ruminate for decades, with no end in sight? What is it that confers unnatural life upon this strange obsession?

Striving for insight through preoccupation and repetition is a process familiar to psychotherapists, whose job is to help people get clear about patterns in their lives. To illustrate, people who have been severely traumatized often have trouble clearly remembering the traumatic events in a normal way. Instead, their lives become choked with indirect “clues” pointing to those events.

What kind of clues? They have flashbacks, repetitive nightmares, strange bodily sensations, and strong but unexplainable emotions. Further, they are sometimes driven to unconsciously re-enact the events. All of these troublesome symptoms are elements of “traumatic memories,” disconnected fragments of the original traumatic experience. This problem can be addressed through therapeutic techniques that allow the individual to gain understanding of the trauma, and so guide the assembly of those puzzle pieces into a clear image.

Are there people who behave like zombies? Sadly, we often encounter addicts who fit the description perfectly. They stop behaving in their own interest, and instead begin to act in the service of something both alien and malignant. Breaking from the logic of self-preservation, they begin to exhaust their personal resources to support this foreign pattern, in the process often bringing themselves to mental, physical, financial, legal, and social ruin. Strangely, they behave as if they no longer care about the people dearest to them, and those who care the most about them. They willingly drain the resources of family, friends, and associates. Worse, their behavior changes in such a way that the alien

pattern can spread like a disease to those around them. It's as if these unfortunates have been zombified, transformed into a creature radically different from their former selves.

Addictions – alcoholism, drug addiction, addiction to gambling, shopping, pornography, compulsive eating. Can we gain any insight into the causes of these undesirable patterns by comparing addicts to the zombies created by parasites? I believe we can. The comparison helps explain both our fascination with the zombie topic and the self-destructive behavior that is so typical of addicts. To get the full benefit of the analogy, we first need to understand how and why parasites routinely turn their hosts into zombies.

### Parasites need zombies

When Darwin and Wallace conceived the original theory of evolution, they framed natural selection in terms of “survival of the fittest.” The animals that functioned best within their ecological niches were the ones that survived to pass their characteristics on to their offspring.

Consider the struggle between the African wildebeest and its predator the lion. In Darwin's eyes, the strongest, wariest, fastest wildebeest were the ones that escaped the fangs and claws of the lion. So they survived to pass their positive characteristics on to the next generation. By the same token, the fiercest, strongest, most relentless lions ate better than their peers. So they likewise passed their characteristics on to the next generation. This explanation of animal adaptation certainly made sense. Unfortunately, Darwin and Wallace left something critical out of this picture: the influence of parasites.

There are some wonderful advantages to being a parasite. It's easier to ride in a boat than it is to row it. Parasites creep aboard their host organisms, treating themselves to an easy trip down the river of life. So parasitism is a popular lifestyle. So popular that the *majority* of all known species are parasites, from viruses to much more complex life forms. They're everywhere. According to science writer Carl Zimmer,

*Every living thing has at least one parasite that lives inside it or on it. Many, like leopard frogs and humans, have many more. There's a parrot in Mexico with 30 different species of mites on its feathers alone. And the parasites themselves have parasites, and some of those parasites have parasites of their own.... According to one estimate, parasites may outnumber free living species four to one. In other words, the study of life is, for the most part, parasitology.*<sup>6</sup>

In emphasizing the struggle for survival, the authors of evolutionary theory failed to consider the struggle between host animals and their parasites. As it turns out, this omission had consequences far more serious than they could have imagined. As originally presented, their theory suggests that apex predators such as lions (on the land) and sharks (in the ocean) are in control of the action. But in

recent years biologists have discovered that, no matter what ecosystem they study, the parasites are the ones actually running the show.<sup>7</sup>

Parasites controlling ecosystems? How could that be? Most parasites aren't as impressive as lions or sharks. They are generally much smaller than their hosts. Sometimes they are microscopic, as with bacteria, yeasts, and viruses.<sup>8</sup> It's easy to see how Darwin could have failed to grasp the influence of something so diminutive and seemingly insignificant. But the evidence says parasites really do have that kind of power. What kind of power? Simply put, the power to zombify their hosts. Parasites create zombies for the most logical of reasons – survival of their species.

Parasites and their hosts are under constant evolutionary pressure to counteract each other's efforts to control them. The result is an everlasting arms race. Host animals are constantly refining their immune systems to keep their parasites from wiping them out. On the other hand, their parasites are constantly refining their ways of getting around host immunity. All this is a clear consequence of natural selection. Just as lions and wildebeest improve their ability to cope with each other, hosts and parasites evolve ever more sophisticated ways of dealing with each other. Their ongoing struggle includes a fight over the host's behavior. Parasites benefit when the host acts in a way that makes things easy for them.

Let's reconsider the struggle between lions and wildebeest. It's not what it seems. Researchers have determined that a parasite within individual wildebeest influences their behavior. In essence, the parasite converts the unfortunate wildebeest into a zombie that offers itself up to be eaten by a lion. For its part, the lion remains alert for signals that the wildebeest has received this special dinner invitation. If that sounds like horror fiction, please read on.

Changes in host behavior are the rule, not the exception. Biologist Janice Moore stresses that "An animal with a parasite is not likely to behave like a similar animal without that parasite." That statement makes perfect sense from an evolutionary perspective. As she explains,

*Parasites and their hosts have some clear evolutionary assignments. In the case of parasites, they are under strong selection to get to a host, and once there, to use that host in a way that promotes the parasites' survival and reproduction. Likewise, we expect hosts to be under strong selection to avoid colonization by parasites; if they fail at this, they are under equally strong selection to minimize the negative fitness effects of parasites.*<sup>9</sup>

The change in a host's typical behavior can be dramatic. Entomologist David Hughes studied a parasitic fungus that spreads through the Brazilian rain forest by literally transforming carpenter ants (species *O. unilateralis*) into the living dead.

*When one of these insects gets infected by a certain fungus, it turns into a so-called "zombie ant" and is no longer in control of its actions. Manipulated by the parasite, an infected ant will leave the cozy confines of its arboreal home and head to the forest floor – an area more suitable for fungal growth. After parking itself on the underside of a leaf, the zombified ant anchors itself into place by chomping down onto the foliage. This marks the victim's final act. From here, the fungus continues to grow and fester inside the ant's body, eventually piercing through the ant's head and releasing its fungal spores. This entire process, from start to finish, can take upwards of ten agonizing days.*<sup>10</sup>

Clearly, the zombified ant has stopped acting in its own interest. It has begun acting in the interest of the fungus. Examining the zombie ants' corpses, Hughes' researchers found that "a high percentage of the cells in a host were fungal cells." The hapless ant has been remade into a creature that literally is not an ant anymore. Rather, it is part insect, part fungus. The infected ant is not just a metaphorical zombie monster, but a real one.

As a part of their normal lifecycle, certain parasites travel through two or more hosts in succession. These are called "heteroxenous" parasites. Ants seem to be popular targets of this kind of parasite too. One species is host to the trematode, a kind of liver fluke. The trematode's life journey does not begin with the ant, nor does it end with the ant. The ant is just one stop along the way – what's called an *intermediate* host. The trematode's first host is a snail. As the snail moves around, it leaves a trail containing sticky balls with the trematode inside. The ants eat these balls, and get infected. The ant is now the second host. But the parasite's itinerary doesn't end there. To complete its life cycle the trematode must next get the infected ant inside a grazing animal, its final or *definitive* host, within which it will sexually reproduce.

But how does the parasite get the ant inside a sheep or cow? By transforming the ant into a zombie. The parasite takes control of the ant's behavior. It makes the zombified ant to climb to the top of a blade of grass and "freeze" there. Against its will, the insect waits to be eaten along with the grass. Getting chewed to bits isn't so good for the ant, but the parasite survives. Once inside the ruminant animal it moves to the liver, where it becomes a liver fluke. There it reproduces, eventually passing its eggs out with the animal's feces. With its eggs now on the ground, the trematode is ready to infect more snails, and so begin its complex life journey all over again.

### **Mammal zombies**

The ant is an insect. But parasites can make zombies of warm-blooded animals too, as the example of the wildebeest shows. One family of heteroxenous parasites is called *Sarcocystidae*. This species zombifies warm-blooded intermediate hosts, and changes their behavior so that they are more likely to be eaten by their definitive

hosts, which are carnivorous predators. Researchers Seilacher et al write

*The Sarcocystidae, in particular, are characterized by forming cysts and by changing between two warm-blooded hosts (mammals, birds) during their life cycles... In every case, one of the hosts serves for the asexual multiplication (intermediate host, by definition), the other for the sexual multiplication (definitive host). Sarcocystidae do less visible harm to their intermediate (herbivorous) hosts than to the definitive (carnivorous) ones. Nevertheless, they are able to alter the behavior, particularly of the intermediate host, in the interest of the parasites' transmission (bait strategy). This bias is reflected in the fact that infected individuals are more common in the kill than the rest of the prey population.*<sup>11</sup>

*Toxoplasma gondii* is a protozoan parasite that has been in the news a lot lately. It passes through two hosts – for example mice and cats. How does the parasite jump from the mouse to the cat? The transfer happens when the cat eats the mouse. Mice are normally motivated to avoid cats, their natural predators. Anytime they sense a cat nearby, they head for the hills. For example, normal mice freak out when they detect the aroma of cat urine in the air. But *T. Gondii* zombifies the mouse, and the zombie mouse stops acting in its own interest. The infection changes its behavior to benefit the parasite. As a zombie, it is no longer frightened by the scent of cat urine. Judging from its behavior, it finds the aroma intriguing. It may even follow that scent to its source. Hello kitty. Goodbye mouse.

As a team headed by researcher Glenn McConkey reports,

*Striking changes in behaviour have been observed in rodents infected with T. gondii. Infected rodents show a reduction in their innate aversion to cat odour, and though both infected and uninfected rats preferred an area that contained their own scent, the infected rats showed a preference for the cat odour area over an area with rabbit scent (a non-predator), while the opposite was true for the uninfected rats... The innate aversion to cat odour appears to become a potentially fatal feline attraction in T. gondii-infected rats and mice... As well as this 'fatal feline attraction', T. gondii infection also leads to increased activity... and decreased neophobic behaviour in rats.*<sup>12</sup>

How does the parasite make this happen? Well, it might seem like magic, but it isn't. It's biochemistry. *T. gondii* forms cysts that nestle inside the mouse's brain and excrete chemicals that redirect brain activity in a way that favors parasite transmission. Responding to those chemicals, the zombie mouse is attracted to the smell of cats, is less fearful, and is more likely to move around actively – all of these changes announce its presence to cats in an unrodent-like, very reckless way. The upshot is that the parasite twists the mouse's behavior to bring it to the attention

of a cat. Clearly, the zombie mouse is no longer acting to preserve itself, but to serve the interests of the parasite.

## Human zombies

Because humans are rarely eaten by cats, humans are not a natural host to *T. gondii*. So we might think this particular parasite need not concern us. Unfortunately, it is now quite clear that humans do get infected. The reason we are vulnerable is that some of our closest animal relatives are the prey of big cats. Monkeys and apes are often eaten by large felines in Africa. In fact, primates are a large part of their diets. One group of researchers looked at our closest relative, the chimpanzee. They found that a chimp's behavior does indeed change with infection. For example, "Toxoplasma-infected animals lost their innate aversion toward the urine of leopards... their only natural predator."<sup>13</sup> Chimp zombies.

Humans are also primates. So humans are subject to infection as well. In fact, studies confirm that a sizable percentage of the world's population – about one third to one half – harbor the parasite. To be blunt, *T. gondii* has inserted behavior-manipulating cysts into the brains of up to half the people on planet earth. Incidence varies with the country, ranging from 20% to 80% of the country's population. Although the rate in the US is toward the lower end of the range,<sup>14</sup> that's still a minimum of one out of every five citizens.

Does the parasite change human behavior, as with its other hosts? The answer is yes. Because we're close in biology to the rest of the primate group, it makes sense that the parasite would affect our bodies and our behavior in a similar way. Research confirms that *T. gondii* does indeed impact many aspects of human life – a stunning reality that we are just beginning to appreciate. In what way does *T. gondii* change us? Let's start with our biology.

Researchers are able to measure the concentrations of *T. gondii* antibodies in mothers' blood, and have reached several unsettling conclusions. One significant finding is that there exists an "extremely high prevalence of toxoplasmosis in mothers of children with Down syndrome."<sup>15</sup> Another mind-bender is that two and a half times more boys than girls are born to women with the highest concentration of anti-toxoplasma antibodies.<sup>16</sup> *T. gondii* can, in other words, dramatically alter the male-to-female birth ratio. Why in the world would the parasite favor male births? The answer concerns another of its hosts – mice. *T. gondii* spreads faster when there are more male than female mice.

*As males of most rodent species are more exploratory, possess larger home ranges, exhibit more aggression, and consequently are more migratory than conspecific females, we can speculate that overproduction of males by toxoplasma by infected females may considerably facilitate long-range transmission of the infection in a natural situation.*<sup>17</sup>



Another eye-opening finding is that *T. gondii* infection may play a role in the genesis of several mental disorders, including schizophrenia. Researchers Fuller Torrey and Robert Yolken reviewed studies relating to this possibility, and determined there may well be a previously unrecognized connection.

*Recent epidemiologic studies indicate that infectious agents may contribute to some cases of schizophrenia. In animals, infection with Toxoplasma gondii can alter behavior and neurotransmitter function. In humans, acute infection with T. gondii can produce psychotic symptoms similar to those displayed by persons with schizophrenia. Since 1953, a total of 19 studies of T. gondii antibodies in persons with schizophrenia and other severe psychiatric disorders and in controls have been reported; 18 reported a higher percentage of antibodies in the affected person.*<sup>18</sup>

These physical consequences are undeniably concerning. But in this essay we are mostly interested in the zombie effect, so let's look at the impact on human behavior. The alterations in behavior provoked by *T. gondii* infection are in fact numerous, and are substantially different for men and women.<sup>19</sup> Personality changes are detectable with standardized tests such as Cattell's 16PF. Researcher Jaroslov Flegr undertook a review of 11 studies, and concluded that

*Consistent and significant differences in Cattell's personality factors were found between Toxoplasma-infected and uninfected subjects in 9 of 11 studies, and these differences were not the same for men and women... The personality of infected men showed lower superego strength (rule consciousness) and higher vigilance (factors G and L on Cattell's 16PF). Thus, the men were more likely to disregard rules and were more expedient, suspicious, jealous, and dogmatic. The personality of infected women, by contrast, showed higher warmth and higher superego strength (factors A and G on Cattell's 16PF), suggesting that they were more warm hearted, outgoing, conscientious, persistent, and moralistic. Both men and women had significantly higher apprehension (factor O) compared with the uninfected controls.*<sup>20</sup>

Do these personality differences have any real-world consequences? They clearly do. Infection makes men more willing to take risks; studies indicate that infected men are nearly three times more likely than non-infected men to be involved in automobile accidents.<sup>21</sup> This effect is almost completely reversed in women, whose risk-taking declines. There is in addition preliminary evidence that infection can influence the degree to which one is aroused by specific kinds of sexual acts.<sup>22</sup>

### **Zombified by a virus?**

Clearly, *T. gondii* infection is neither benign nor insignificant. But *T. gondii* is a complex multicelled organism. Vi-

ruses are parasites of a much simpler kind. Could a viral infection similarly influence our behavior? Some preliminary evidence gives reason to look further.

We could begin with the homely example of the rhinovirus, which causes the common cold. This virus makes us sneeze. In the course of our sneezing, we spray virus-laden droplets all over everything. This is the way the virus spreads from one person to another. Could the rhinovirus be manipulating us, making us cooperate with its reproductive process? Perhaps. But maybe it's just a coincidence that the virus irritates our nasal passages, causing our noses to run and delivering us into sneezing fits. It could be mere happenstance that sneezing spreads the virus. We need more data.

The flu is caused by the influenza virus. That virus too must travel from host to host in order to reproduce itself. Does the flu virus zombify us to get what it needs? Psychologist Glenn Geher, author of the text *Evolutionary Psychology* says,

*The flu virus might actually manipulate people's nervous systems in a way that increases exposure to a relatively large number of people during the communicable stage of the illness. In other words, perhaps the flu virus makes people unwittingly seek out a relatively high number of social interactions as an evolved mechanism for the virus to get itself into a high number of human bodies.*<sup>23</sup>

A team of researchers headed by Chris Rieber ran a preliminary experiment to test this possibility. "We hypothesized," they say, "that on encountering flu virus, humans would increase their social behavior." But hold on. Common sense tells us that when people start to feel those horrible flu symptoms, they mostly withdraw from others and go to bed. Wouldn't that argue against this idea? Not at all, as it turns out. The study authors point to an interesting, little-known fact concerning when we become infectious.

*Infected individuals are contagious a day before symptom onset and for several days thereafter... Shedding of the virus peaks 2 days after exposure, and symptom scores peak on day 3... Once symptoms appear, transmission via social interactions may be reduced by the onset of sickness behavior. However, no sickness behavior is evident pre-symptomatically, making the social behavior of infected individuals at this time particularly important.*<sup>24</sup>

So the real issue is what people are doing between (a) the time when, still without symptoms, they turn infectious, and (b) the time when they start feeling terrible. During that slice of time are they interacting with more people than they usually do? And if so, by how much?

The researchers were faced with an ethical problem. Before people start getting sick, no one can say for sure whether they've got the flu, or when they got it. One way

to know the exact time of exposure is to deliberately infect subjects with the influenza virus – but that would be both unethical and dangerous. The team came up with an ingenious solution. They used immunization as a proxy for natural infection, knowing that immunization “elicits an immediate immune response similar to that induced by wild-type infection.”<sup>25</sup> That way they could compare subjects’ social behavior immediately before and immediately after immunization.

OK. So what did they find? Was there any difference? According to their report,

*In the two days immediately after influenza immunization, study participants socially encountered almost twice as many other humans as they did in the two days before immunization. Participants were not consciously aware of any changes in their levels of sociability, nor could the changes in their social behavior be accounted for by differences in social patterns associated with particular days of the week. Human social behavior changed on the introduction of viral antigens... To our knowledge, this is the strongest indicator yet discovered out of pathogen-mediated behavioral change and otherwise asymptomatic humans. Our results unambiguously point to increased social interaction after exposure.*<sup>26</sup>

Although subjects didn’t seem to be aware they were doing anything different, their pattern of social interaction changed dramatically. They didn’t attend any more events than before, but they were interacting with more people during each event. The total number of people contacted by each study participant increased from an average of 54 to an average of 101.

Incredibly, the average amount of time they spent with each contact dramatically *decreased* – from over 30 minutes each to *less than 3 minutes each*.<sup>27</sup> The subjects were flitting from person to person like manic butterflies. This change in behavior could hardly be more helpful to a highly infectious virus.

Was this “social butterfly” effect really caused by the virus? Or was it simply that the subjects assumed the experimenters expected them to interact more, so they did. Clearly, the answer to this question is important. But to address it definitively, we need further research, studies comparing an active vaccine to a vaccine placebo.

### **Zombified habits**

The zombie tales reviewed so far illustrate that parasites of all kinds change the behavior of their hosts in ways that favor the parasite’s reproduction. The logic of natural selection itself predicts this. Those varieties of the parasite that happen to influence host behavior in their favor are more likely to survive. So those manipulative varieties are the ones that pass their genes to the next generation. The end result is that infected hosts act like zombies.

Let’s again consider the remarkable conduct of the addict. The behavioral changes we associate with addictions look a lot like the kind of parasite-induced zombie behavior we have been describing. Specifically: (1) the addict undergoes a pronounced and uncharacteristic change in behavior; (2) the addicted person no longer acts in his or her own interest, and instead (3) begins acting in ways that perpetuate an alien and malignant pattern; (4) the uncharacteristic behavior persists despite serious harm, both to the addict and to loved ones; and (5) the malignant pattern often spreads disease-like to others with whom the addict has contact.

Do these facts mean that addiction is caused by some kind of biological disease? That it is the result of an infection like *T. gondii* or a zombie virus like the ones in the movies? Well, it would be hard to defend that idea. For one thing, no such biological parasite has ever been detected. But here’s where the story takes a turn that some will regard as strange. There is another explanation that better fits the facts. It involves a different *kind* of parasite – a non-biological parasite. A *behavioral* parasite.

It is apparent that an addiction is a habit – although an abnormal and destructive one. A useful way of thinking about addictions is that they are “habits gone rogue.” A rogue habit is one that no longer functions in the interest of the behaving animal. Rather, the runaway habit acts simply to keep itself going, to reproduce itself within the animal’s behavior. In essence, a rogue habit is one that has turned into a kind of self-replicating behavior, a parasitic pattern that uses the behaving animal as its host. Is that possible? The evidence suggests that it is.

As brilliant psychologist William James pointed out more than a century ago, “when we look at living creatures from an outward point of view, one of the first things that strikes us is that they are bundles of habits.”<sup>28</sup> Any animal capable of forming habits is as much defined by its body of habits as by its biological body. Habits are learned behavior patterns that operate mostly autonomously, and mostly outside our conscious awareness. By their very nature, habits are like semi-autonomous robots that are restrained only by a flimsy leash of remote supervision. In his 1890 *Principles of Psychology* James said,

*Actions originally prompted by conscious intelligence may grow so automatic by dint of habit as to be apparently unconsciously performed. Standing, walking, buttoning and unbuttoning, piano-playing, talking, even saying one’s prayers, may be done when the mind is absorbed in other things. The performances of animal instinct seem semi-automatic, and the reflex acts of self-preservation certainly are so. Yet they resemble intelligent acts in bringing about the same ends at which the animal’s consciousness, on other occasions, deliberately aims.*<sup>29</sup>

As James emphasized, the big advantage of habits is that they reduce the need for attention to what we’re doing.

This frees our attention, one of our most precious resources, so that it can be applied to other matters that more urgently require it.<sup>30</sup> When we learn a habit, we continue to be aware of what we're doing, but that awareness is no longer acute. As the habit is solidified over time, our awareness of our actions becomes remote, distant. So supervision of an established habit requires only a tiny slice of our attention.

Learned habits provide higher animals a degree of behavioral flexibility that lower animals can't match. But this advantage comes at a cost. James was careful to point out that there are dangers to all this flexibility. The danger is that the behavior of higher animals can easily become dysfunctional. He said,

*The dilemma in regard to the nervous system seems, in short, to be of the following kind. We may construct one which will react infallibly and certainly, but it will then be capable of reacting to very few changes in the environment – it will fail to be adapted to all the rest. We may, on the other hand, construct a nervous system potentially adapted to respond to an infinite variety of minute features in the situation, but its fallibility will then be as great as its elaboration... In short, a high brain may do many things, and may do each of them at a very slight hint. But its hair-trigger organization makes of it a happy-go-lucky, hit-or-miss affair. It is as likely to do the crazy as the sane thing at any given moment.*<sup>31</sup>

Because they are already operating semi-autonomously, habits do sometimes slip the bonds of our attention to become fully autonomous. A habit that has escaped its controls is a dangerous habit. It makes sense to think of such an uncontrolled habit as “diseased.”

### “Cancerous” habits

But how could a habit possibly become diseased? It happens in a way quite like the process by which the cells of our bodies become cancerous. When human cells escape their controls they become cancer cells. Normally, cells act in the interest of the body of which they are but a small part. Each individual cell serves a function that supports the survival of the body as a whole. Their assigned roles are strictly enforced by the body through several mechanisms, most of which involve bodily immune functions. Cancerous cells, throwing off the shackles of these controls, begin to act in their own interest – and abandon the larger interests of the body. They hide from the immune system's agents of enforcement, which would otherwise locate and kill these rebellious cells.

Wilfred Jefferies, Professor of Medical Genetics and Microbiology and Immunology at the University of British Columbia, says “The immune system is efficient at identifying and halting the emergence and spread of primary tumors, but when metastatic tumors appear, the immune system is no longer able to recognize the cancer cells and

stop them.”<sup>32</sup> Cancerous cells stop serving their assigned functions, and instead begin simply reproducing themselves as fast as they can. Reproduction becomes an end in itself. Amazingly, such cells even defy the normal limits on cell division by granting themselves *immortality*.

The immortalization of cells tremendously accelerates the spread of cancers. That's because one of the body's controls on individual cells is a limit on the number of times the cell is permitted to divide. Telomeres are like caps on the end of genes, and this cap is shortened with each cell division. Each of its divisions is a countdown to the end. When the telomere countdown reaches zero, the cell is no longer able to divide. So the length of a cell's telomeres limits the number of times it can reproduce itself. The limit is a cancer safeguard.

Scientists believe cancer cells achieve immortality by turning on an enzyme called telomerase, which lengthens chromosomal telomeres. Because the telomere length of these rogue cells never gets any shorter, they are no longer subject to natural death.<sup>33</sup> Having thus sidestepped the grim reaper, they are free to reproduce themselves without limit. They multiply exponentially. These rebellious cells have truly “gone rogue.”

Habits become diseased in a parallel way. As with the cells of the body, habits are specialized. Individual habits are normally subservient to the larger interests of the individual. But certain conditions make it likely that a habit will escape its behavioral controls, and begin to act in its own interest. Then the normal mechanisms by which habits are refined no longer work to direct them toward the satisfaction of bodily needs. Instead, the rogue habit evolves toward a self-centered goal – simply reproducing itself within the behavior of the individual. Such “habits-turned-cancerous” are self-perpetuating behavioral sequences whose character can range from mostly harmless to decidedly malignant.

Is it really true that habits can go rogue in this way? The answer to this question has been clear for decades. Under specific conditions, addiction-like rogue habits spontaneously arise in all “higher” animals – all those animals that are capable of learning. Animal researchers have precisely identified the circumstances that provoke the development of stereotypical, self-reproducing habits. Such “cancerous” habits are most likely to arise within environments where the animal's opportunities for natural fulfillment have been severely restricted – as when the animal is caged in a zoo, pen, or stall. It is under these abnormally constraining, frustrating conditions that animals develop repetitive, dysfunctional habits reminiscent of addictive behavior. Researchers M. Lewis and colleagues observed,

*Abnormal repetitive behaviors are commonly displayed in animals housed in zoos, farms, and laboratory environments, as well as animals subjected to early social deprivation. Indeed, repetitive behaviors are the most common category of abnormal behavior ob-*

served in confined animals. For example, pacing and route-tracing in birds; sham-chewing and bar-mouthing in pigs; crib-biting and head-shaking in horses; vertical-jumping and backward somersaulting in deer mice; body-rocking and tail-biting in rhesus monkeys; pacing and over-grooming in prosimians; and head-twirling in minks are but some examples of aberrant, repetitive behaviors observed in animals maintained in confinement.<sup>34</sup>

In each and every higher animal tested, severely limiting opportunities for the natural expression of its inherited drives has been found to engender habits that are both stereotypical and abnormally persistent. There appear to be no exceptions.

What does this have to do with addiction? Plenty. Researchers have shown that when animals confined to cages are given access to drugs like cocaine, alcohol, or opiates they are likely to begin using these substances in an addictive way. In other words, where addictive drugs are accessible, imprisoned animals readily incorporate them into their dysfunctional, stereotypical habits. But animals living in more satisfying environments tend not to incorporate the drugs into their habits, even when they have ready access.

The abnormal habits engendered by highly constricted environments are useless to the host animal, yet reproduce themselves within the animal's behavior in a seemingly parasitic manner. The word "parasitic" fits because – like cancerous cells – these cancerous habits exploit the animal's resources to reproduce themselves without providing any benefit to the animal.

To illustrate, a horse confined to a stall may begin to endlessly walk in circles. Once firmly established, this dysfunctional habit may persist even if the horse is transferred to an open pasture. In essence, such stereotypical habits

exploit their host to duplicate themselves without limit, much as cancer cells do. The rogue behaviors increase in frequency until the host animal is doing little else, and appears unable to stop. This process is most often detrimental to the animal.

Humans are not exempt from the development of similarly dysfunctional habits under conditions of environmental restriction. The pattern we call addiction fits well within this type of behavior, a tendency that is virtually universal in the animal world.

The idea of self-reproducing patterns in human thought and behavior will likely be unfamiliar to some readers. But the concept itself certainly isn't new. It was introduced way back in 1976 by Richard Dawkins in the pages of his popular book, *The Selfish Gene*.<sup>35</sup>

Dawkins speculated about self-reproducing, virus-like behavioral patterns he called "memes." He proposed that memes get themselves spread from one person to another by inviting others to *imitate* them. These behavioral "replicators," said Dawkins, occupy the human brain as their host. The meme idea became quite popular, and led to the emergence of a science dubbed "memetics."

As conceived by Dawkins, memes are largely benevolent. In fact, Dawkins and his followers suggest that the complexes they form within the mind are the basis of human awareness and intelligence. Because memes are conceived in generally positive terms, it may be inappropriate to alter the meme concept to make it fit the dysfunctional, zombie-like behavior seen in addiction.

Yet it is clear that rogue habits can acquire characteristics considerably darker than Dawkins's memes were hypothesized to be. It is realistic, then, to think of this pathological process in terms of diseased habits.

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