**Headline:** Robert Sapolsky: Are We Better off Accepting That There’s No Free Will?

**Teaser:** Interviewing the neuroscientist and primate behavior expert on a question that could radically change our understanding of reality.

By Jan Ritch-Frel and Marjorie Hecht

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**[Article Body:]**

The release of *Determined*, a new book by renowned Stanford professor of primate behavior and neuroscience Robert M. Sapolsky, has catapulted him into the middle of an ancient debate: whether humans have free will and agency over their actions. [*Determined*](https://www.amazon.com/Determined-Science-Life-without-Free/dp/0525560971) isn’t just a bio-philosophical treatise: It covers the potential benefits that a society that accepts Sapolsky’s thesis of there being zero free will and agency over our actions will likely become more humane and will be better at understanding and addressing humanity’s challenges.

Sapolsky found fame while teaching the science of stress and anxiety from a neurological perspective and its presence in the wider primate world. His [popular teachings](https://www.youtube.com/playlist?list=PL848F2368C90DDC3D) have enlightened millions and opened new pathways to help people consider the biological causes of their behavior.

Over this year, Sapolsky has rolled a publicity tour in defense of *Determined*’s thesis, including speaking on dozens of nationally known podcasts, and more recently, he has co-launched an informative and witty [YouTube Q&A show](https://www.youtube.com/playlist?list=PLOXn0rUD2D-eBMO44WUaxRFYqwv61pDaa) with his talented daughter Rachel Share-Sapolsky.

We reached out to Sapolsky for an interview about his thinking on how public adoption of science can change perspectives, and his experience as an activist to try and get the world to think differently about the causes of human behavior.

**Jan Ritch-Frel and Marjorie Hecht: You point to the early 1800s in France as a turning point in how society perceived epilepsy, from culpability for behavior during seizures to understanding it as a medical condition. Where are you seeing similar green shoots today?**

**Robert Sapolsky:** A great example is the recognition that obesity is a biological disorder, rather than some sort of failure of Calvinist self-discipline. It is a biological disorder that is profoundly sensitive to psychological state and social context, but it is nonetheless biological.

To give the most dramatic example, if someone has a mutation in the leptin receptor gene, their brain will simply not process food satiation signals, regardless of how much willpower they have. Currently, stigma about weight is one of the most persistent prejudices in society, and findings like this are just beginning to change attitudes toward obesity.

**Q: What in your research led you to volunteer as a witness for capital trials, and what did your experiences lead you to conclude about making a social impact through your research?**

**Sapolsky:** If you conclude that we have no free will, that we are simply the outcome of the interaction of the biological luck over which we had no control with the environmental luck over which we had no control, the entire premise of criminal “justice” makes no sense intellectually or ethically. However, I’m not of much use if I show up in a courtroom to say to the jury that “hey, we’re all just biological machines.”

So my goals are much narrower with the public defenders I work with. You consider a defendant who has done something horribly damaging, and there’s the option to think of their behavior as an index of their questionable moral worth, or as a measure of the damage that their own nervous system sustained over their lives. So my job is to try to get juries to think the latter rather than the former, to teach them the science that leads one to that conclusion.

In terms of what impact I’ve had—it’s been almost entirely futile; we “lost” 11 of the 13 cases I’ve worked on. The jury sits there and nods their heads in the affirmative when you’re telling them how the frontal cortex works… and then when they go into the jury room and look at the pictures of the corpse, they come back with the maximal sentence.

**Q: What human tendencies and capacities are you counting on for people to make positive use of your conclusion that there is no free will?**

**Sapolsky:** Mostly I would be hoping for ways in which humans can counter their tendencies. By this, I mean to try to resist the lure of exceptionalism, which makes us decide that our needs are special and atypically worthy of consideration.

**Q: Can you discuss how an individual can differentiate right from wrong but be “organically incapable” of appropriately regulating his or her behavior? What happens in the prefrontal cortex to cause this?**

**Sapolsky:** The prefrontal cortex (PFC) can inhibit and restrain emotional impulses: If you are in a situation where you are tempted to do something unethical yet manage to resist, it is because of the PFC. Thus, any circumstance that damages, weakens the PFC makes that sort of self-regulation more difficult.

Thus, you can wind up with someone who knows right from wrong, can write erudite philosophical essays about the difference… yet in a moment of emotional arousal, may not be able to prevent themselves from doing something wrong.

**Q: How do you think therapy helps an individual with depression or other problems? What changes in the brain are possible? How does free will enter into the therapy process?**

**Sapolsky:** Just focusing on depression, its cognitive core is a tendency to distort reality in a negative direction. On an emotional level, it’s perceiving yourself to be helpless in circumstances that are not really the case; on a neurochemical level, it’s probably a shortage of serotonin and a number of other neurotransmitters that produces an inability to anticipate pleasure and to block negative rumination.

These are all different levels of explaining the same thing. On the psychotherapy route, the most effective approach is typically cognitive behavioral therapy, which basically consists of recognizing the reality of some trauma, failure, or rejection in the past, but also recognizing that it is a distortion to assume that you are fated for the same in the future, that you are helpless and hopeless in trying to prevent some manner of reoccurrence.

Where the meds fit in are to facilitate that process. Drugs that boost serotonin, for example, lessen the stickiness, the unstoppable qualities of negative rumination… which allows you the affective breathing space to begin to disassemble the distortions that give rise to the incessant negative affect.

Free will plays no role in any of this. Did you turn out to be the sort of person who makes X amount of serotonin instead of Z, whose pathways of negative rumination in the brain are tightly or loosely connected, whose learning makeup is one that is good or not at building upon efficacy? Did you turn out to be the sort of person who respects introspection, is capable of doing it insightfully, is capable of drawing on those insights as a buffer against negative emotion? Did you turn out to be the sort of person who, in the first place, could accept that they are struggling with depression? And we had no control over any of those.

**Q: Why isn’t what’s called “early readiness potential,” as detected in the brain, as much a form of free will as any subsequent action that carries out the potential?**

**Sapolsky:** Because focusing on the early readiness potential, ERP, is missing the point. When exactly it has occurred with respect to when you form an intention to do something is not ultimately relevant to the issue of free will. Instead the absence of free will is shown looking at the more global question of, “How did you become the sort of person who would form that intent at that moment?”

**Q: In your book *Determined*, you present an example of identical twins, only one of whom is schizophrenic with a very different brain imaging scan. Is schizophrenia then *not* inherited? Could it be the result of an infection?**

**Sapolsky:** Genes are about inheriting tendencies, proclivities, and vulnerabilities in behavior, not about inheriting inevitabilities. Consider someone with schizophrenia and pick another person at random, and there is about a 2 percent chance that they will also suffer from the disease. If instead you pick the person’s identical twin, there is roughly a 50 percent chance that they will share the trait. This is powerful demonstration of a genetic influence.

But the fact that the identical twin will have a 50 percent chance of *not*having schizophrenia is a demonstration that genes are just part of the mix of causes, not anything deterministic.

What are some of the non-genetic factors that contribute to schizophrenia risk? Yes, certain types of infections; perinatal birth complications; prenatal malnutrition; chronic stress; and heavy cannabis abuse as an adolescent.

**Q: In your lecture on religiosity, you rely on twin/adoption studies from the late 1960s and 1970s, which looked at schizophrenia and found that some other family members were what they termed schizotypicals. These individuals were not full-blown schizophrenics but they exhibited “off” behavior characteristic of schizophrenics. How do you respond to the criticisms of the twin/adoption studies, including criticism of the idea of a spectrum of schizophrenia conditions (such as the work of** [**Jay Joseph**](https://jayjoseph.net/the-trouble-with-twin-studies/)**)?**

**Sapolsky:** Twin studies, along with adoption studies, are the backbones of classic behavior genetics approaches, and are subject to some withering criticism that is completely valid.

If twin researchers tell you that genes explain X percent of the variability in some trait, applying the criticisms typically shows that the percentage is actually *lower* than X—not that genes have *nothing* to do with the trait. That has no effect though on the main observation, which is that close relatives of people with schizophrenia are very significantly more likely to display schizotypal traits than the general population.

That shows that there is a shared genetics to schizophrenia and schizotypalism—and implicit in that is precisely Joseph’s point, which is that disorders with schizophrenic elements form a continuum, a spectrum (while not at all supporting his broadest conclusions).

**Q: You’ve mentioned that you came from an Orthodox Jewish family. Did you have any reaction from the Orthodox community to your analysis of the roots of religiosity? Or from any other religious thinkers?**

**Sapolsky:** I have had some very negative reactions from folks in both domains. However, those reactions are entirely based on completely missing what I am saying about the subject. I am not remotely saying anything as absurd as, “Ooh, you have to be psychiatrically suspect to be religious,” or even, “Most/many/some people who are religious are psychiatrically suspect.”

I am saying that it is fascinating that traits that can be incapacitating psychiatric maladies in secular settings can be accepted, given sanctuary, and even be viewed as positives in the context of religiosity.