was a senior in college when I carried out my first research project in cell biology, and I found it incredibly exciting to be designing experiments that had important implications in animal and human health.

Nowadays, I’m a biology professor who studies circadian rhythms (the equivalent of roughly a day, or 24 hours) in bread mold. OK, I know what you’re thinking—“just another example of my tax dollars at waste on some esoteric research project with absolutely no societal benefit.” But let me assure you that there is more to my research than meets the eye.

Do you ever wonder why medications to treat high cholesterol, such as statins, are only useful if taken before you go to sleep? Or why you get indigestion if you eat late at night? Nearly all organisms have an internal 24-hour timing mechanism, known as the circadian clock, that drives daily rhythms in behavior and physiology. For example, our heart rate and blood pressure increase around 4 a.m. to prepare us for waking up each morning. In day-active humans, food metabolizing enzymes produced by our liver and fat cells peak in activity during daytime hours. Therefore, if you eat at night, the ability to break down that food is significantly reduced.

Our capacity for learning and memory, sleep onset and our athletic performance also is clock-controlled.

So when it comes to timing, you can forget about what the clock on the wall says, because it’s the circadian clock that rules.

Which brings me to Neurospora crassa, a mold first discovered contaminating bread in French bakeries and the basis of my research in the Department of Biology. Because the Neurospora clock functions similarly to the human clock, it is a model organism for determining what genes and proteins our clock regulates. We can use Neurospora to do preliminary experiments that would be expensive and/or illegal to do on humans in order to fight and prevent human diseases.

In my lab, we are using Neurospora to find new ways to treat cancer. In humans, defects in the circadian clock caused by genetic mutation or by living out of synchrony with the clock (for example, doing shift work) can lead to an increased risk for certain types of cancer. We have found that in Neurospora, the clock regulates the activity of mitogen-activated protein kinases, or MAP kinases. These enzymes play an important role in controlling cell division and stress-related responses. Therefore, drugs that inhibit MAP kinases also inhibit cell proliferation, including the uncontrolled replication of cancer cells. Unfortunately, when you inhibit MAP kinases in normal cells, patients become quite ill. Thus, finding a way to lower the drug dose while still inhibiting the proliferation of cancer cells would open a new avenue to treating patients.

Interestingly, in many human cancer cells, the MAP kinases and other proteins either lose their rhythmicity or show altered rhythms. One possibility is to give cancer patients lower doses of the MAP kinase inhibitor drugs at times of the day when kinases are at reduced levels in normal cells but still at peak levels in cancer cells. This type of therapy is expected to lead to decreased toxicity of the drugs and to increased effectiveness against cancerous cells.

Go figure that the icky old orange mold colonizing neglected bakery goods is proving to be a testing ground for new anti-cancer strategies. And it’s even edible! Next time you’re in Aggieland, drop by my lab for a sample.

—BY DEBORAH BELL-PEDERSEN

PROFESSOR AND ASSOCIATE DEPARTMENT HEAD FOR OPERATIONS
DEPARTMENT OF BIOLOGY
TEXAS A&M UNIVERSITY