

Sleeping too Little (or too Much) Linked to Faster Aging, and Other Science News

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New research suggests that sleeping less than 6.4 hours per night on average — or more than 7.8 hours — speeds aging in the brain, heart, lung, and immune system and contributes to a wide range of diseases. To conduct the [study](#), Columbia radiologist [Junhao Wen](#) and several colleagues developed a novel method of assessing the rate at which individual organs age, based on metabolic and imaging data. They then examined how sleep duration is related to specific conditions, discovering that short sleep is associated with depression, anxiety, obesity, type 2

diabetes, hypertension, ischemic heart disease, and heart arrhythmias. Both short and long sleep, they found, are linked to chronic obstructive pulmonary disease, asthma, and a number of digestive disorders, including gastritis and gastroesophageal reflux disease.

Why are some bosses bullies?

If you've ever had a boss who ruled through threats and intimidation, you might have assumed they knew exactly what they were doing. But new [research](#) by Columbia Business School professor [Daniel Ames](#) and [Dean Baltiansky](#) '26BUS suggests that domineering leaders are often unaware of how they're affecting morale. In a series of laboratory studies, they found that people with a "dog-eat-dog" view of relationships tend to assume that their aggressiveness is seen as normal and harmless, while those with a more harmonious view of the world are better at anticipating the risks of pushing subordinates too hard. The takeaway for managers? "Give yourself an opportunity to test if your assumptions are correct," Ames says, by seeking feedback from colleagues who can reveal blind spots.

Alzheimer's may start with cellular waste

Columbia neurologists led by [Kapil Ramachandran](#) have found new clues about the beginnings of Alzheimer's disease, revealing how tau proteins become misfolded and begin forming the toxic tangles long linked to memory loss. In a [study](#) in mice, the scientists observed that when a cellular-waste-disposal system that Ramachandran discovered a few years ago breaks down, defective tau proteins escape destruction and start clumping together. The researchers also found evidence that the infamous APOE4 gene variation, which doubles a person's risk for Alzheimer's, works in part by damaging the waste-disposal system. They say their findings raise the prospect of developing Alzheimer's drugs that prevent the production of tau tangles at the source.

AI is a team sport

Corporate leaders tend to assume that adopting the latest, most powerful AI tools will make their companies more efficient. But a pair of studies from Columbia Business School reveals that involving workers in decisions about how AI is deployed, and training them to use the technology wisely, is even more important. One [study](#), led by [Stephan Meier](#), found that the single greatest predictor in whether a company successfully integrates AI is how well its managers listen to employees' perspectives about how the technology ought to be used. Another [study](#), led by [David Holtz](#), revealed that investments in AI are only beneficial if employees are continuously supported in learning to manage and operate the tools.



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Etna's surprise fuel

One of the world's most active volcanoes, Sicily's Mount Etna, is known as a gentle giant, with relatively mild eruptions every few years that do little to deter hikers and skiers. But scientists know that Etna erupted violently four thousand years ago, and again in 122 BC. Will they know when the next big one is coming? A new [paper](#) by Columbia geologist [Terry Plank](#) '93GSAS, Anna Barth '21GSAS, and several Cornell

and University of Hawaii colleagues may help. The study reconstructs Etna's ancient eruptions in unprecedented detail and reveals that a buildup of carbon dioxide within its plumbing greatly intensified the 122 BC blast. That finding may surprise some volcanologists, who have traditionally discounted CO2 as a major factor in eruptions.

New weapon against pancreatic cancer

A new targeted therapy for pancreatic cancer, built on years of Columbia research, has proven effective in a large-scale human trial, nearly doubling patients' length of survival from seven months to thirteen months. The drug, daraxonrasib, was developed by the California-based biopharmaceutical company Revolution Medicines with help from Columbia cancer researchers [Kenneth Olive](#) and [Gulam Manji](#). The success of the [trial](#) involving 500 patients, as reported in the *New England Journal of Medicine*, is one of the biggest breakthroughs in pancreatic cancer in decades. Experts say that daraxonrasib may soon become a standard treatment for people whose disease progresses after chemotherapy.



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