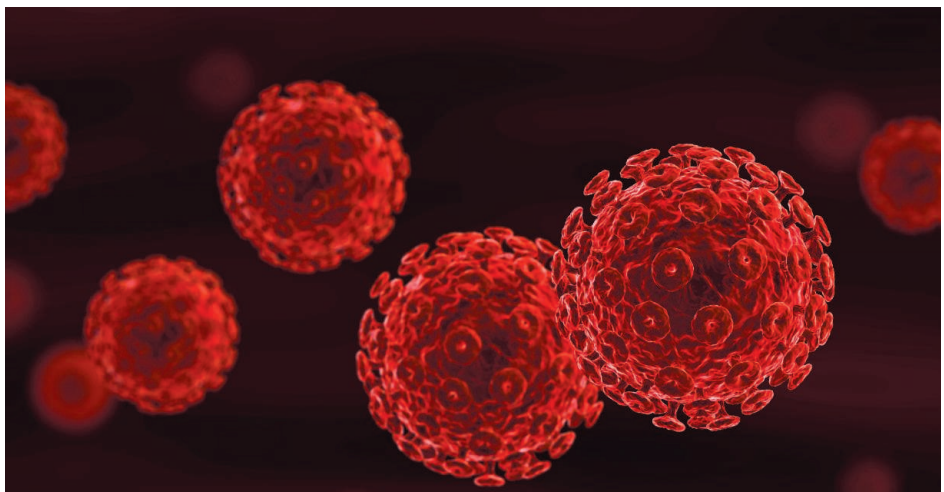
EDITED BY KRISTEN MUELLER AND JAKE YESTON

POLICY

Assessing the Assessments

Though a modern-day Galileo may still woo an occasional Medici patron to support his research, a vast number of scientists depend on much more bureaucratic means to keep their labs running. Since 1986, when the UK Research Assessment Exercise was launched, at least 14 countries have implemented systems to distribute research funding to universities based on evaluation of research output. These have been motivated by the increasing importance of research to economic growth, as well as broader interests in improving public management. Hicks analyzes the systems' rationale, design, and impact. There is a range of assessments (e.g., citation analysis, peer review), across a range of scales (e.g., university, department, individual), that affect a range of funding outcomes (e.g., 25% of UK research support, 2% of Italian block grants). Although distribution of research funding is the putative purpose, direct financial impacts appear small compared to incentives to compete for public prestige. Some values widely associated with universities, such as diversity and equity, may suffer under systems focused solely on excellence and international competition. Though touted as critical to economic success, the systems do not appear to be well designed to meet that goal. — BW

Res. Policy **41**, 251 (2012).



VIROLOGY

Replication Restricted

Despite its deadly nature, HIV-1 is quite limited in the types of cells that it can infect. HIV-1 primarily infects $CD4^+$ T cells but not many myloid-derived immune cells. This is because most myeloid-derived cells express the viral restriction factor SAMHD1. Although this may seem like an advantage to the host, the virus actually gains the upper hand because it can escape detection by the innate immune system. In support of this, HIV-2 and some SIV strains that do not cause such severe pathology express Vpx, which counteracts the effects of SAMHD1. Little is known, however, about how SAMHD1 prevents HIV-1 infections from taking hold. Lahouassa *et al.* noted that SAMHD1 shares homology with a protein from *Enterococcus faecalis* that has nucleotide metabolism activity. Using a variety of in vitro analyses, they found that SAMHD1 exhibited phosphohydrolase activity for dNTPs and regulated the pool of dNTPs in myeloid-derived cells. SAMHD1 expression lowered the concentration of dNTPs below what is required for productive reverse transcription by HIV-1, thereby blocking infection. Thus, regulation of nucleotide pools may be a means by which cells regulate their susceptibility to viral infection, but hidden benefits for the virus may be lurking, too. — KLM

Nat. Immunol. **13**, 10.1038/ni.2236 (2012).

BIOTECHNOLOGY

Small Sources of Sweetness

The sucrose sourced from sugarcane to sweeten our tea and cake—and more recently, to foster ethanol as a transportation fuel—is the same molecule produced by many microorganisms that potentially face fewer cultivation constraints. The trouble is that the microbes don't release their sugar easily. Ducat *et al.* noted that heterotrophic and autotrophic bacteria induce oppositely directed transmembrane proton gradients, and as such, a gradient-dependent native transporter that pulls sucrose into the former might expel it from the latter. They therefore expressed this sucrose permease in *Synechococcus elongatus* cyanobacteria—known to produce sucrose under osmotic stress—and indeed collected the sugar in the medium. Furthermore, strains incorporating the transporter manifested enhanced photosynthetic productivity, as assessed by measuring oxygen evolution rates and fixation of ^{14}C-labeled tracers. Though scale-up presents a range of challenges, extrapolation of the laboratory results suggests prospective sucrose productivities on par with or even exceeding that of sugarcane. — JSY

Appl. Environ. Microbiol. **78**, 10.1128/AEM.07901-11 (2012).

PHYSIOLOGY

Fructose Sweetens the Deal

Glucose, a metabolic product of dietary sucrose, triggers pancreatic beta cells to release insulin, which in turn allows many different cells types to take up glucose to store or use as energy. The other breakdown product of sucrose is fructose, and Kyriazis *et al.* report that it too plays a role in controlling insulin release. Fructose activates a sweet taste receptor (the TIR2-TIR3 heterodimer) expressed by mouse and human beta cells. This enhances the effect of glucose on insulin release. Mice injected with fructose (or saccharine, another taste receptor ligand) showed a rapid increase in circulating insulin, but only if functional taste receptors were present and only if glucose stimulated beta cells as well.

Fructose-activated sweet taste receptors signal through a phosphoinositide pathway, which elevates cytoplasmic Ca^{2+} concentration

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and depolarizes the membrane via a cation channel, TRPM5. Functional interaction between these two pathways is not yet clear, but because TRPM5 has been implicated in glucose-stimulated insulin secretion, it may be a convergence point. Whatever the mechanism, these findings may have implications for the link between high fructose consumption and the development of metabolic diseases such as obesity and diabetes. — LC

Proc. Natl. Acad. Sci. U.S.A. **109**, 10.1073/pnas.1200797109 (2012).

NEUROSCIENCE

The Reading Brain

Developmental dyslexia, which manifests as difficulty with reading, can have long-lasting and detrimental effects on a child's experience with education, with echoes that persist long into adulthood. Raschle *et al.* have leveraged the indications of familial risk for dyslexia to distinguish differences in brain structure present before reading from those that arise after the battle with reading difficulties is well engaged. The authors studied a total of 36 5-year-old children, all pre-literate, characterized by whether or not their family had a history of dyslexia. Even at these pre-reading ages, functional brain imaging revealed an aberrant signal similar to that found in older children with a confirmed diagnosis of dyslexia. Development of the brain network supporting phonological processing appeared to be delayed. On the other hand, the two groups of children showed no difference in other networks that are hyperactivated when persons with dyslexia are reading. Those networks may instead represent compensation brought into play in the struggle to defeat reading difficulties. — PJH

Proc. Natl. Acad. Sci. U.S.A. **109**, 2156 (2012).

EDUCATION

Getting the Rubric Right

Despite the growing popularity of online learning, how to grade online discussions remains a challenge. Simply modifying existing grading rubrics can be problematic, because the format of online learning discussions differs from that of traditional classroom work. To evaluate the value of a clearly defined online learning grading rubric, or scoring tool, Solan and Linardopoulos developed a rubric that took into account the quantity and quality of posts, timeliness of participation, and communication proficiency, and then surveyed undergraduate and graduate student perceptions. Results indicated that although students are appreciative that a rubric is available, most did not consult it while contributing to online discussions. Teachers therefore may need to emphasize, review, and clearly link the rubric to grades assigned throughout the duration of the course. Students also reported that such a tool needs to be dynamic, because despite attempts to be comprehensive, unexpected scenarios will probably occur. Whether such a tool led to increased student learning is unknown, and, along with faculty perceptions of the rubric's value, should be a topic of future research. — MM

J. Online Learn. Teach. **7**, 452 (2011).

ASTROPHYSICS

Swifter than the Sun

Stars form from the gravitational collapse of clouds of gas and dust. Before they attain a stable radius, they spin faster and faster as they contract, much like a spinning ice skater does as she folds her arms to her body. After gravitational collapse stops, stars start slowing down because of magnetic braking—the loss of stellar angular momentum as material gets removed from the star because of a magnetized stellar wind. Stars thus start as fast rotators and then slow down as they age. Current theories of angular momentum evolution reproduce the rotation data for Sun-like stars, but fail to account for those with masses lower than half that of the Sun. These very low-mass stars don't seem to slow down with age as much as expected, and the lower their masses, the faster they rotate at a given age. Reiners and Mohanty reexamined the theories of angular momentum evolution for low-mass stars, and show that angular momentum evolution must depend on stellar radius if the rotation of a star is related to its magnetic field strength. Stars with lower masses have smaller radii and lower magnetic braking efficiencies, meaning that they will take longer to slow down. — MJC

Astrophys. J. **746**, 43 (2012).

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