LETTERS

Edited by **Jennifer Sills**

Editorial expression of concern

On 10 September 2010, *Science* published the Report "Human SIRT6 promotes DNA end resection through CtIP deacetylation" by A. Kaidi, B. T. Weinert, C. Choudhary, and S. P. Jackson (*1*). On 19 August 2018, the Research Governance and Integrity Officer of the corresponding author's institution, Cambridge University, together with the corresponding author, notified *Science* that the institution was launching a preliminary investigation under the Misconduct in Research policy. They have since notified us that the university has concluded that there is a prima facie case that requires formal investigation. *Science* is publishing this Editorial Expression of Concern to alert our readers while we await the outcome of the investigation.

Jeremy Berg
Editor-in-Chief

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Lack of science support fails Brazil

On 2 September, the world watched in horror as Brazil's National Museum, housing a vast collection of more than 20 million biodiversity and cultural artifacts, was engulfed in flames ("In a 'foretold tragedy,' fire consumes Brazil museum," H. Escobar, In Depth, 7 September, p. 960). The museum's extensive natural history collections, painstakingly accumulated over more than two centuries, documented the change in species identity and distributions over time, recorded the earliest South American inhabitants' culture and native languages, and archived the origin and historical progress of a nation. The magnitude of this loss is staggering—not just for Brazil but for the world. Scientific advancement is based on building blocks from the past, and without those components, scientists are left without points of reference. Museum collections are the foundation on which we recognize cultural and scientific novelty as we strive to understand and better the human condition, to advance our grasp of how nature's pieces came into being and fit together, and even to predict the ecological and evolutionary future of the planet's biodiversity (*1*).

Funding for the museum decreased substantially during the past 5 years (*2*), and calls for renewed investment in renovations, security, and protection have been ignored for decades (*3*). In this sense, the National Museum is an apt metaphor for the current state of science in Brazil: Leaders at all levels have failed to provide even the most basic and crucial infrastructure for preserving genuinely priceless collections and cultural resources. This fire at the National Museum follows the loss of 80,000 specimens in the fire that destroyed the collections at Instituto Butantan in 2010 (*4*) and the complete loss of the Museum of the Portuguese Language in São Paulo in 2015 (*5*), both also attributed to poor investment in infrastructure. Recent years have seen

Increased science funding might have prevented the devastating fire at Brazil's National Museum.

large declines in the budget for basic scientific research and student training (*6*). The loss of unique and irreplaceable collections, owing to lackluster federal investment in science, adds salt to the growing wound.

There is some hope among the ashes. Many of the biological collections, including vertebrates, most of the marine invertebrates, and plants, as well as rare books, were spared because they were in different buildings. Fortunately, no human lives were lost. Curators and museum staff are working around the clock to reorganize and house displaced colleagues and maintain teaching and mentoring in their ongoing graduate programs. The Brazilian government has pledged funds and infrastructure to support rebuilding (*7*, *8*), providing an opportunity for Brazil to correct mistakes of the past.

This tragic loss resonates beyond Brazil. Museum collections are timeless national treasures that represent our histories, cultures, and scientific achievements. Every institution and government should reflect and take heed at this sad moment. We must invest in and safeguard our museums and collections for the benefit of science and society worldwide.

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Relating addiction and psychiatric disorders

In their Research Article "Analysis of shared heritability in common disorders of the brain" (22 June, p. 1313), The Brainstorm Consortium shows that psychiatric disorders share common risk variants, whereas neurological disorders appear more distinct from one another and from the psychiatric disorders. The analyses include 10 psychiatric disorders. Future studies on common risk variants should include addictive disorders as well.

Addictive disorders are among the most common, debilitating, and stigmatized disorders (*1*). Just like other psychiatric disorders (*1*), they are defined in international classification systems for psychiatric disorders like DSM-5 (*2*) and largely based on behavioral symptoms. The high comorbidity between addictive disorders and other psychiatric disorders (*3*) suggests pathophysiological overlap. Combinations of these disorders negatively affect prognosis of both disorders, stressing the relevance for exploring shared mechanisms (*4—6*).

The magnitude of genetic correlations between addictive disorders (including substance use) and other psychiatric disorders is comparable with the genetic correlations between psychiatric disorders reported by The Brainstorm Consortium. For example, the genetic correlation of .52 ($P = 2.18 \times 10^{-20}$) between major depressive disorder (MDD) and attention deficit hyperactivity disorder (ADHD), described in the Research Article (table S7A), is similar in magnitude to the correlation of .57 ($P = 3.1 \times 10^{-4}$) between MDD and alcohol dependence (*7*). Similarly, significant genetic correlations of .53, .38, and .37 for ADHD with alcohol dependence (*7*), smoking quantity, and smoking initiation (table S7B), respectively, are comparable to correlations of .52, .26 and .22 between ADHD and MDD, bipolar disorder, and schizophrenia, respectively (table S7A). Genetic correlations for MDD with smoking initiation ($r_g = .33$, $P = 3.1 \times 10^{-11}$) (table S7B) and cannabis use ($r_g = .21$, $P = 3.0 \times 10^{-4}$) (*8*) are also significant. The Brainstorm Consortium did include smoking as a risk factor for other conditions. However, smoking is an addictive disorder on its own, with high prevalence, morbidity, and mortality (*9*, *10*). It therefore makes sense to look at correlations between smoking as a risk factor and tobacco use disorder as an addiction, as well as correlations between tobacco use disorder and other psychiatric disorders.

These correlations suggest that addictive disorders share a considerable portion of their common variant genetic risk with other psychiatric disorders. This association may result from the same genes influencing multiple phenotypes (horizontal pleiotropy) or from causal relationships (vertical pleiotropy) (*11*). To better understand these associations, substance use and addictive disorders should be included in future studies of the biological pathways underlying psychiatric disorders.
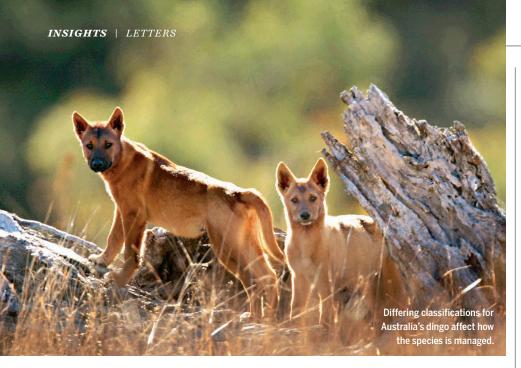
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Differing classifications for Australia's dingo affect how the species is managed.

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Species definitions shape policy

The names we assign to organisms, and why, have important ramifications for our understanding of Earth's diversity and, more practically, how it is managed. For example, wolves, coyotes, domestic dogs, and other canids are often considered distinct (*1*), but their members can, and frequently do, interbreed (*2*). Differing concepts of species—which might take into account morphology, ecology, behavior, genetics, or evolutionary history (*3*)—could describe canids as very few or many species, depending on which concepts are used and how strictly they are applied. Which definition scientists adopt can have political and ecological consequences.

The dingo (*Canis dingo*) has traditionally been considered native in Australia, given evidence of its presence before the year 1400 (*4*) and indications that it has lived in Australia for at least 5000 years (*5*). This designation meant that Western Australia had to have a management strategy in place for the dingo, along with other native fauna. However, a recent paper (*6*) argues that dingoes are in fact *C. familiaris* because they don't satisfy zoological nomenclature protocols nor sufficiently differ genetically or morphologically from other canids, including domestic dogs. The Western Australian government cited this work in justifying its recent decision to declare the dingo a non-native species under the state's Biodiversity Conservation Act (BCA) (*7*). The new order removes the government requirement to manage the species. As a result, dingoes can now be killed anywhere in the state without a BCA license. A potential increase in lethal control of dingoes could have dire consequences for Australia's ecosystems. The dingo is Australia's largest terrestrial top predator [adults typically weigh 15 to 20 kg (*8*)], it fulfils a crucial ecological role, and it has strong cultural significance for Australia's Indigenous people (*8*).

Taxonomy serves a critical purpose for cataloguing and conserving biodiversity, but different interpretations and applications of species concepts can affect management decisions. Policy-makers may use the interpretations that justify their preferred values, such as prioritizing livestock more than biodiversity protection. It is therefore imperative that scientists carefully engage in the policy decision-making process. Scientists must work with policy-makers to convey the multiple dimensions and values that can affect species delineation and make clear the potential consequences of applying such classifications.

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TECHNICAL COMMENT ABSTRACTS

Comment on "DNA damage is a pervasive cause of sequencing errors, directly confounding variant identification"

Chip Stewart, Ignaty Leshchiner, Julian Hess, Gad Getz

Chen *et al.* (Reports, 17 February 2017, p. 752) highlight an important problem of sequencing artifacts caused by DNA damage at the time of sample processing. However, their manuscript contains several errors that led the authors to incorrect conclusions. Moreover, the same sequencing artifacts were previously described and mitigated in The Cancer Genome Atlas and other published sequencing projects.
Full text: dx.doi.org/10.1126/science.aas9824

Response to Comment on "DNA damage is a pervasive cause of sequencing errors, directly confounding variant identification"

Lixin Chen, Pingfang Liu, Thomas C. Evans Jr., Laurence M. Ettwiller

Following the Comment of Stewart *et al.*, we repeated our analysis on sequencing runs from The Cancer Genome Atlas (TCGA) using their suggested parameters. We found signs of oxidative damage in all sequence contexts and irrespective of the sequencing date, reaffirming that DNA damage affects mutation-calling pipelines in their ability to accurately identify somatic variations.
Full text: dx.doi.org/10.1126/science.aat0958

Comment on "The plateau of human mortality: Demography of longevity pioneers"

H. Beltrán-Sánchez, S. N. Austad, C. E. Finch

Barbi *et al.* (Reports, 29 June 2018, p. 1459) reported that human mortality rate reached a "plateau" after the age of 105, suggesting there may be no limit to human longevity. We show, using their data, that potential life spans cannot increase much beyond the current 122 years unless future biomedical advances alter the intrinsic rate of human aging.
Full text: dx.doi.org/10.1126/science.aav1200

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