

Disappearance of  
the mammoths

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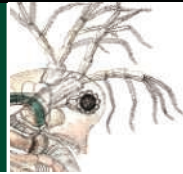
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## LETTERS

edited by Etta Kavanagh

### The Language of Fighting Invasive Species

IN HIS ARTICLE “WINNING THE WAR AGAINST ISLAND INVADERS” (NEWS Focus, 2 Dec. 2005, p. 1410), K. Krajick presents an interesting point of view about eradication of exotic species on islands. However, despite the fact that exotic species are a leading cause of biodiversity loss, there is growing concern about the language (1) and the attitude (2) used by researchers in this area, since they may not be the most appropriate ways to capture the attention and support of laypeople. Gobster argues that the language used is too aggressive and strategies, such as eradication, are too brutal, pushing some organizations to stand against programs controlling exotic species. Public support is a fundamental part of any successful program dealing with exotics, and it has been shown that taking account of invader impacts is an important axis of biological conservation (3, 4).

Biological invasions are complex: Interventions against rats that established on islands several centuries ago may require some discussions on possible nontarget effects and methods, but waiting to see the impact of a recently introduced species before attempting eradication is a crime against ecosystems, given current knowledge on the impact of alien species (5). The challenge today is educating the public on the negative effects of exotic species to obtain their support. Reducing the strength of the language used could increase the support of some groups, together with education to help them understand that, given their effects and the difficulties in predicting the success of exotic species, eradication is in some cases the only logical solution.

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Feral pigs roaming on Santa Cruz Island. A program is currently under way to eradicate this invasive species from the island.

animal welfare issues seriously (as we all should) to understand the ecological considerations that justify such programs. Since humans introduced the pigs to Santa Cruz Island, it makes sense to describe the eradication program simply as an attempt to rectify an earlier ecological mistake, rather than as a war against the pigs.

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### Doing More for Keisha

IN THEIR EDITORIAL “DOING MORE FOR KATE” (16 Dec. 2005, p. 1741), T. Cech and D. Kennedy describe a young woman who might have been a productive scientist, but was transformed into a business major by the anomie of a large research university. Keisha, a valedictorian from a working class high school, was also overwhelmed at a large research university. Rather than changing majors, however, Keisha transferred to a smaller public comprehensive university. There she was soon noticed by an instructor, who drew out her story and introduced her to other faculty. She is now engaged in an undergraduate research project, is academically successful, and seems well on the way to graduate school and a scientific career.

IN HIS NEWS FOCUS ARTICLE, “WINNING THE war against island invaders” (2 Dec. 2005, p. 1410), K. Krajick makes extensive use of the metaphor of warfare in his description of recent attempts to eradicate invasive species such as pigs and goats from island ecosystems. He writes, for instance, of the “war on pigs” on Santa Cruz Island and of a “war room” where professional hunters—no, “terminators”—gather to strategize. Ironically, though, some radical environmental activists who condone ecosabotage also like to use the language of

warfare. Humans, they say, are prosecuting an aggressive war against defenseless nonhuman nature. Krajick ought therefore to be more circumspect in his choice of words. Why play into the hands of those who think of themselves as “eco-warriors,” or of those in the animal rights movement who share their lack of compunction about property destruction? Krajick’s description of an eradication program as an attempt by hired “terminators” to go after “the enemy” in hopes of “wiping out every last invader” will do nothing to help those who take

Although efforts to energize teaching in large classes should be applauded, it is a mistake to believe that this is more than a palliative for the real pathology that afflicts the U.S. scientific pipeline. The anecdotes about Kate and Keisha really say that scientific training and the development of a scientific workforce are intensely personal and human activities. This is not the mission of large research universities, nor are teachers of even the best large classes particularly good at it.

Assuming that intellectual capability in the young is not determined solely by the income of their parents, perhaps the greatest unexploited pool of scientific talent exists among the children of the working class. It is the public comprehensive universities, those invisible and disregarded institutions, that are most likely and best qualified to mentor and develop this impecunious human resource. Yet it is precisely those institutions that are being starved to death by state legislatures nationwide.

If Cech and Kennedy are correct that the U.S. scientific enterprise is threatened, then the science establishment might do even more for Kate and Keisha by attacking the political disease rather than the pedagogical symptoms.

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## Genetic Research into Autism

ACCORDING TO THE EXTREME MALE BRAIN (EMB) theory ("Sex differences in the brain: implications for explaining autism," S. Baron-Cohen *et al.*, *Viewpoint*, 4 Nov. 2005, p. 819), autism is characterized by extreme scores on two dimensions, empathizing (low) and systemizing (high), typical of a very masculine brain. Baron-Cohen *et al.*'s account omits an important part of the causal story: Autism is perhaps the most highly heritable behavioral disorder. Data from our recent twin study speak to the genetic architecture of autistic-like dimensions. The model of two (or more) dimensions underpinning autistic behaviors may be critical for understanding the causes of autism. This is because low correlations exist between social

impairments (related to empathizing) and nonsocial behaviors like restricted repetitive behaviors and interests (related to systemizing) in the general population (1). Moreover, social and nonsocial behaviors are, like autism, both highly heritable, but largely genetically distinct, both in the general population and at the extremes (2). Low genetic correlations suggest that different genes contribute to social and nonsocial autistic traits. However, there is no evidence from twin studies of sex-specific genetic effects, only mean sex differences (well-documented in the article) and an increasing male bias toward the impaired extreme (2, 3). The genetic mechanisms underlying sex differences in relation to fetal testosterone and autism still need to be mapped.

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### Response

WE THANK RONALD *ET AL.* FOR HIGHLIGHTING the need for more genetic research into autism and agree that genes are likely to play a major role in the cause of autism spectrum conditions. It is too early to say whether the relevant genes are sex-linked, sex-limited, sex-influenced, or sex-independent. Although we do refer to genetics on p. 822, because of the strict word limit, we confined our Viewpoint to topics with sufficient evidence for sex-related effects relevant to both the empathizing-systemizing (E-S) and EMB theories. Sex-related effects relevant to the E-S and EMB theories are clear at the psychological level and suggestive at the neuroanatomical level. More work is needed to clarify the role of fetal endocrinology. At present, there is insufficient evidence to identify candidate genes that could give rise both to sex differences in empathizing and systemizing for the general population, and to a hypermasculinization of these in individuals with autism spectrum conditions (1).

Nevertheless, we agree with Ronald *et al.* that psychological, neuroanatomical, and endocrine effects are likely to be downstream of genetic antecedents. We can imagine that a gene such as the androgen receptor (AR) gene on the X chromosome would be a candidate in the EMB model, where variations in the CAG repeat length may be associated with phenotypic variation between the sexes (2) and between autism and controls.

Ronald *et al.* (3) found that social (or what we call E, for empathizing) and nonsocial (or S, for systemizing) traits are largely independent

in a large normative twin sample. In our own studies, we too found that E and S are weakly but significantly negatively correlated in a normative sample ( $r = -0.09$ ), although in an autism spectrum sample, the correlation is stronger and still negative ( $r = -0.29$ ) (4). This may be consistent with Ronald *et al.* (3) in that they observed "All extreme groups had a higher proportion of males than females." So we might hypothesize that the low-E and high-S aspects of autism, although largely independent in the general population, both bear some permissive (although not determinative) relation to maleness when they occur to extreme degrees.

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## Acid Growth and Plant Development

IN HIS PERSPECTIVE "GROWTH BY AUXIN: WHEN a weed needs acid" (7 Oct. 2005, p. 60), M. Grebe asserts that the acid-growth theory describes how the plant hormone auxin (indole-3-acetic acid, IAA) stimulates cell elongation in developing organs such as leaves and roots. On the basis of this statement, he presents the hypothesis that the proton pump AVP1 of the model plant *Arabidopsis thaliana* causes cell elongation via IAA-induced acid secretion. Grebe refers to a single review article (1) by Achim Hager, the spiritual father of the acid-growth concept. Hager's wall acidification model is based on experiments with shoots of grass seedlings (coleoptiles, which are leaf-like axial organs). The hypothesis was proposed in 1971 and thereafter carefully evaluated by scientists (1–4).

The observations that (i) acid buffers (pH 3.5 to 4.0) elicit a rapid short-term growth response in oat coleoptiles, (ii) IAA enhances the rate of proton extrusion so that a pH of about 5.0 is established in the growth-limiting organ walls, and (iii) metabolic inhibitors block both hormone-mediated wall acidification and cell elongation led to the postulate that auxin may initiate coleoptile elongation by rapidly lowering the apoplastic pH value from about 6.0 to about 4.8 to 5.0 (1). However, the fungal phytotoxin fusaric acid (FC), but not the naturally occurring growth hormone IAA, fulfills the predictions of the acid-growth hypothesis of coleoptile elongation (2). FC rapidly activates a plasma membrane-localized proton pump fueled by adenosine triphosphate (P-

### Letters to the Editor

Letters (~300 words) discuss material published in *Science* in the previous 6 months or issues of general interest. They can be submitted through the Web ([www.submit2science.org](http://www.submit2science.org)) or by regular mail (1200 New York Ave., NW, Washington, DC 20005, USA). Letters are not acknowledged upon receipt, nor are authors generally consulted before publication. Whether published in full or in part, letters are subject to editing for clarity and space.

ATPase) and thereby causes an acid efflux into the walls (final apoplastic pH about 3.5 to 4.0). The resulting burst of organ elongation is accompanied by a rapid rise in the rate of cell respiration. These results are in accordance with the acid-growth hypothesis of FC action, which has become a well-supported theory (3). Corresponding experiments with IAA (and FC, used as a tool) led to the conclusion that auxin-induced proton secretion is insufficient to elicit growth: When a wall pH of 4.8 to 5.0 was established by application of a suboptimal concentration of FC, no promotion of coleoptile elongation occurred (2). Alternative concepts of IAA-mediated cell-wall expansion have been proposed that are currently under investigation (3, 5).

In addition, Grebe states that the wall acidification hypothesis of elongation growth also applies to roots, as implicitly assumed by Hager (1). This is not the case (4). In developing roots of grass seedlings, no positive acid (pH 4.0)–growth response has been recorded; at pH 3.5, organ elongation is reduced. In root cells, IAA inhibits rather than promotes the rate of proton secretion (4).

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#### Response

KUTSCHERA COMMENTS ON THE PERSPECTIVE I wrote discussing a study by J. Li *et al.* (“*Arabidopsis* H<sup>+</sup>-PPase AVP1 regulates auxin-mediated organ development,” Reports, 7 Oct. 2005, p. 121). This study provides the first functional genetic evidence that a plant proton pump, the pyrophosphate-driven *Arabidopsis* V-type ATPase AVP1, is required for auxin transport, cell wall acidification, organ growth, and development. Kutschera incorrectly states that I proposed that “the acid-growth theory describes how the plant hormone auxin (indole-3-acetic acid, IAA) stimulates cell elongation in developing organs such as leaves and roots.” I simply posed the question of whether AVP1-mediated regulation of cell wall pH in roots or leaves involves an acid-growth mechanism similar to the one discussed by Hager (1). Additionally, I asked whether AVP1-dependent cell wall acidification may feed back on auxin transport, and whether AVP1 regulates intracellular transport

of auxin carriers and plasma membrane H<sup>+</sup>-ATPase (P-ATPase).

My Perspective has touched off an unsettled controversy among plant physiologists, as it remains a matter of debate as to what extent auxin-induced cell wall acidification contributes to elongation growth (1–5). Studies on several plant species experimentally support Hager’s acid-growth theory (1, 3), originally proposed for stems of grass seedlings (1, 4). Other scientists, including Kutschera, have published evidence questioning the theory (2, 5).

Given the tools and experimental approaches available at the time, most early studies addressing acid-growth employed external application of IAA and/or the fungal phytoxin fusaric acid (FC), which plants do not produce endogenously (1–5). Kutschera correctly points out that FC stimulates cell wall acidification more efficiently than externally applied auxin (2). However, exogenous application of auxin and FC alone cannot determine whether plants intrinsically utilize auxin-dependent cell wall acidification to regulate growth processes.

Li and colleagues focus their discussion of AVP1 function on auxin transport regulation, taking into account that the classic chemiosmotic model of auxin transport has been

strongly supported by genetic and molecular studies (6). Similar to the work by Li and colleagues, genetic loss- and gain-of-function studies of endogenous proteins required for cell wall acidification and auxin responses may now be combined with physiological experiments to address the relevance of auxin-induced acid growth in planta. Studies of acid-

growth responses in *Arabidopsis* seedling stems (hypocotyls) have been initiated (7). Similarly, auxin-stimulated acid growth of tomato hypocotyls as well as its loss in the *diageotropica* mutant has been reported (7, 8). External application of auxin at high concentrations stimulates hypocotyl (7) but inhibits root elongation in *Arabidopsis* and tomato (8,

9), consistent with experiments cited by Kutschera. Intriguingly though, application of external auxin at low concentrations stimulates *Arabidopsis* root growth (9). These findings raise the question of which growth responses are physiological processes regulated by auxin within the range of its endogenous concentrations. Thus, it remains to be revealed exactly where auxin-regulated cell wall acidification constitutes an intrinsic mechanism regulating different aspects of plant growth.

### CORRECTIONS AND CLARIFICATIONS

**Random Samples:** "Asian science on the move" (27 Jan., p. 447). The worldwide gross expenditure on research and development was reported wrongly. It comes to \$830 billion and not \$2.8 trillion as stated.

**Reports:** "Restoration of auditory nerve synapses in cats by cochlear implants" by D. K. Ryugo *et al.* (2 Dec. 2005, p. 1490). On page 1490, the number of nonexperimental subjects conflicts with those listed in the Supporting Online Materials. The SOM is correct: Six congenitally deaf cats and four normal hearing cats were used as controls.

**Research Articles:** "Logic of the yeast metabolic cycle: temporal compartmentalization of cellular processes" by B. P. Tu *et al.* (18 Nov. 2005, p. 1152). Several references were cited in the wrong places in the text. The correct sentences and citations are as follows: "A recent study described a ~40-min respiratory oscillation that produces a genome-wide, low-amplitude oscillation of transcription during continuous culture (11, 12)... About two-thirds of the most periodic transcripts in the YMC encode components of mitochondria (Table 2) (14)... Might it be that an ancestral symbiont (35), endowed with the capacity to generate ATP by both respiratory and reductive pathways, used the two pathways in an oscillatory manner (Fig. 7B)?... Temporal compartmentalization of metabolic function also appears to take place during the circadian cycle of flies and mice (27, 28). The primitive cyanobacterium *Synechococcus elongatus*, which conducts both photosynthesis and nitrogen fixation, uses its circadian regulatory apparatus to ensure that these biochemically incompatible pathways are executed at temporally distinct phases of the circadian cycle (36). The circadian cycle drives the periodic expression of many genes encoding the rate-limiting enzymes of numerous metabolic processes (27, 28). Restricted feeding can entrain the circadian cycle (35, 36), perhaps through metabolic feedback impinging directly on the transcription factors that themselves regulate circadian rhythm (39, 40)."

**Reports:** "Treatment of autoimmune neuroinflammation with a synthetic tryptophan metabolite" by M. Platten *et al.* (4 Nov. 2005, p. 850). The hydroxyl group on the benzene ring at the 3' position on 3,4-DAA (anthranilic acid) in Fig. 1A should not be there.

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