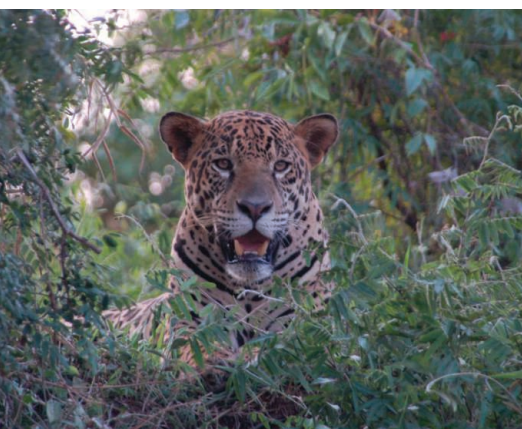


LETTERS

edited by Jennifer Sills

Atlantic Rainforest's Jaguars in Decline



IN HER NEWS FOCUS STORY "PREDATORS IN THE 'hood'" (20 September, p. 1332), V. Morell reported that top predator populations are coming back across much of North America. Meanwhile, predators in Brazil continue to decline. A recent meeting of wildlife experts indicated that the Atlantic rainforest that once stretched along the coast of Brazil and parts of Argentina and Paraguay may soon be the first tropical biome to lose its largest top predator, the jaguar (*Panthera onca*). Researchers estimated fewer than 250 mature jaguars alive in the entire biome, distributed in eight isolated populations (1). Even worse, molecular analyses demonstrate that local effective population size (a critical parameter for the maintenance of

genetic diversity) is below 50 animals (2).

Jaguars are persecuted for their potential impact on livestock, and their prey have been overhunted even in large protected areas (3). Jaguars provide a crucial service in controlling herbivores (capibaras, deer, and peccaries) and smaller predators (pumas, ocelots, foxes, and racoons), and their overall extinction will likely disrupt predator-prey interactions with unpredictable effects on ecosystem function (4). The Atlantic rainforest is a highly fragmented biodiversity hotspot, with less than 12% of the original area left (5). Although 24% of the remaining areas are large enough to support jaguars, jaguar populations can be found in only 7% of the rainforest (4).

Population supplementation and reintroduction programs may provide new hope for jaguars, but uncontrolled hunting of jaguars and their prey is still widespread in most protected areas, threatening the persistence of this important top predator. In the absence of effective protection and management, the fate of the largest predator of the Atlantic forests is bleak.

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Capping Progress
on Invasive Species?

THE EUROPEAN COMMISSION RECENTLY PUBLISHED its long-awaited draft legislation on invasive alien species (1). The proposed regulation implements a key target of the European Union Biodiversity Strategy (2), aiming to bring EU policy in line with the Convention on Biological Diversity targets for 2020, which obliges signatories to identify and prioritize invasive alien species and their pathways of invasion, to control or eradicate priority species, and to manage pathways to prevent the introduction and establishment of new invasive alien species (3).

An EU-wide regulation that coordinates a preventative and responsive system across the member states is a welcome step forward. However, one aspect of the draft risks fundamentally compromising its capacity to tackle the issue: The list of species to which the system would apply is strictly capped at a maximum of 50 species, for at least an initial period of 5 years after adoption (realistically, until 2020). This is only 3% of the 1500 invasive alien species already recognized as present and problematic in the European Union (1, 4), which generate a minimum estimated cost of €12.5 billion annually (5, 6).

The justification for capping the number of priority species is "to provide member states with certainty regarding the extent and

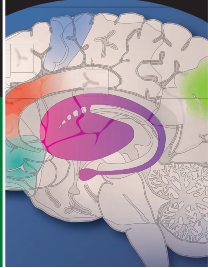
Letters to the Editor

Letters (~300 words) discuss material published in *Science* in the past 3 months or matters of general interest. Letters are not acknowledged upon receipt. Whether published in full or in part, Letters are subject to editing for clarity and space. Letters submitted, published, or posted elsewhere, in print or online, will be disqualified. To submit a Letter, go to www.submit2science.org.



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costs of the actions they will be expected to take.” Given the recognized long-term cost efficiencies of early action on invasive alien species (7), this economic justification is a short-term one, placing the burden of action and a crushing financial bill on future generations. The system should be flexible, responsive, and able to be updated as frequently as needed. Unless the cap is altered, we risk missing a major opportunity, sacrificing longer-term ecological and economic benefits in the name of minimizing short-term input.

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CORRECTIONS AND CLARIFICATIONS

Letters: “Response to ‘In defense of WHO’s blood donation policy’” by N. Lacetera *et al.* (8 November, p. 692). Due to an editorial error, the corresponding letter author, Neelam Dhingra, was referred to once as “he” instead of “she” and three times as “Dinghra.” The HTML and PDF versions online have been corrected.

Reports: “Structure-based design of a fusion glycoprotein vaccine for respiratory syncytial virus” by J. S. McLellan *et al.* (1 November, p. 592). The senior authors did not contribute equally. The HTML and PDF versions online are correct.

Reports: “Parameter space compression underlies emergent theories and predictive models” by B. B. Machta *et al.* (1 November, p. 604). NSF grant DMR 1312160 should have been included in the Acknowledgments. The HTML and PDF versions online are correct.

News Focus: “Varmus’s second act” by J. Kaiser (25 October, p. 416). The article erroneously stated that PubMed Central, NIH’s full-text papers archive, will soon add commenting tools. The tools have been added to PubMed, NIH’s abstracts database. The HTML and PDF versions online have been corrected.

Reports: “Stress-inducible regulation of heat shock factor 1 by the deacetylase SIRT1” by S. D. Westerheide *et al.* (20 February 2009, p. 1063). The authors inadvertently duplicated lanes from Fig. 2A in the bottom panel of Fig. 2B. The corrected Fig. 2B is provided here and does not alter the conclusions of the experiment. In addition, Fig. 3, A and C, and fig. S7 were affected by an insertion in the construct for the HSF1 K80R mutant, which is described in a correction to the supplementary materials. Experiments with the remade HSF1 K80R construct support our finding that acetylation at HSF1 K80 by HSF1 K80R mutant, which is described in a correction to the supplementary materials. HSF1 K80R, a mutation that retains the charge of the lysine without allowing acetylation, exhibits DNA binding activity (see corrected Fig. 3A here), whereas the HSF1 K80Q acetylation mimic does not. Also, the HSF1 K80R construct is able to induce *hsp90* and *hsp25* gene expression as potently as HSF1 wild type (WT) when transfected into *hsf1*^{-/-} cells (see corrected Fig. 3C here). Details are in the revised supplementary materials (www.sciencemag.org/cgi/content/full/323/5917/1063/DC1).

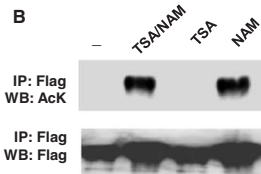


Fig. 2B. Effects of nicotinamide (NAM) and trichostatin A (TSA) on HSF1 acetylation. 293T cells transfected with Flag-HSF1 and p300 were treated with TSA or NAM, or both, and exposed to heat shock, then cell lysates were analyzed by acetylation assay.

Drilling Plans Endanger Yasuní’s Biodiversity

DESPITE BEING ONE OF THE MOST MEGADIVERSE forests and a sanctuary for ancestral indigenous people (1–3), Ecuador’s Yasuní National Park has always faced the looming threat of oil exploitation (“Opponents vow to block Amazon drilling,” News of the Week, 23 August, p. 827). In 2007, in an effort to protect Yasuní’s biodiversity and, more important, mitigate climate change associated with the burning of fossil fuels, Ecuadorian President Rafael Correa promised to keep approximately 850 million barrels of heavy crude oil locked underground to prevent the release of 410 million metric tons of CO₂ into the atmosphere. In exchange, the international community would finance a compensation of US\$3.6 billion (2–5)—about half of the total cost of reducing the CO₂ emissions (US\$7.2 billion) if the oil were to be extracted.

Despite this arrangement, on 15 August 2013, Correa abandoned the moratorium on oil drilling in Yasuní (6). He justified the move by citing inadequate international

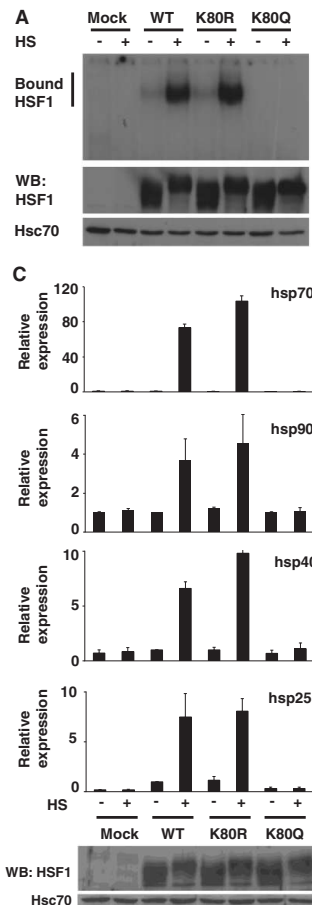


Fig. 3. HSF1 K80Q disrupts the HSR, whereas HSF1 K80R does not. (A) In HSF1, substitution of glutamine at K80 disrupts DNA binding activity, whereas replacement with arginine does not. Electrophoretic mobility shift assay (EMSA) reactions were performed with extracts from *hsf1*^{-/-} cells transfected with the indicated HSF1 constructs treated with or without heat shock (HS) (top). The EMSA probe contains the proximal heat shock element from the human *hsp70* promoter. Western blot analysis was performed on the same samples to show HSF1 and heat shock cognate 70 (Hsc70) levels. (C) Mutation of HSF1 at K80 to arginine rescues the HSR in *hsf1*^{-/-} cells, whereas mutation of HSF1 to glutamine does not. *hsf1*^{-/-} cells were transfected with the indicated versions of human HSF1 and treated with or without heat shock. RNA was quantified using quantitative polymerase chain reaction with primers for the indicated genes. Data are normalized to values obtained for glyceraldehyde 3-phosphate dehydrogenase and are relative to the abundance of each mRNA in WT HSF1 cells treated without heat shock (value set as 1). Experiments in (A) and (C) were performed in triplicate, and error bars indicate $\pm$ SD.

donations from developed nations to protect the park. The presidential decision to exploit Yasuní's oil, estimated to generate \$18 billion, was also portrayed as an opportunity to deal with poverty (6–8). The reality is that poverty was not eradicated during the past four decades of oil exploitation in Ecuador. On the contrary, Big Oil companies (such as Chevron-Texaco) have caused deleterious environmental-social impacts in important areas of Ecuador's Amazon (9), reaching a total liability cost of \$18 billion (10). More than 50% of Ecuador's population disagrees with Correa's decision (6).

The Ecuadorian government should face its responsibility to conserve Yasuní and should implement more sustainable alternatives focused on renewable resources and market diversification (such as ecotourism) to allow for economic growth in the long term. It is not too late for Ecuador to ensure human and environmental rights and keep alive the dream of having a future without oil.

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