Tolerance for Predatory Wildlife

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ECOLOGY

Tolerance for Predatory Wildlife

Can individual tolerance toward predators affect the success of predator conservation?

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Around the world, populations of many large, predatory animals are declining, with wide-ranging consequences for other species and ecosystem services (*1*). The declines have a variety of causes, but for mammalian carnivores and sharks, direct human causes of mortality predominate (*2*). Scientists and policy-makers have concluded that promoting human tolerance is critical to the success of predator conservation efforts (*1*, *3*–*5*). Yet the factors that affect people's tolerance of wildlife are not well understood.

The terms tolerance and intolerance are widely used to capture both individual-level judgments of predators (such as attitudes and perceptions), as well as individual behaviors (such as poaching) that directly or indirectly influence outcomes for predators (*5*). It is widely assumed that intolerant behavior toward predators—whether in the form of eradication policies (such as bounties) or illegal killing—is motivated by retaliation for real and perceived losses of livelihood (*5*, *6*). Under this assumption, governments and private organizations aiming to protect predators have implemented economic incentives to reduce the perceived costs of predator conservation and raise tolerance for predators.

Sweden's pioneering program that pays Sami reindeer herders to tolerate predators elucidates the limits and potential benefits of economic incentives for predator conservation. The Swedish government appears to be succeeding in protecting wolverines, brown bears, and lynxes by paying Sami villagers for each successful predator reproduction on communal grazing areas (*6*). However, the Sami have thus far refused to accept incentives for protecting gray wolves because these predators are perceived to scare and scatter reindeer widely. As a result, wolves have only recolonized south of the reindeer areas.

Since the early 1990s, livestock producers have been offered subsidies for predator-proof electric fencing and its installation in south-central Sweden. A correlational study of 445 Swedes living in wolf territories found that those who received subsidies tolerated wolves better than those who had not, regardless of the number of verified wolf attacks on sheep or dogs (*7*). However, the study could not rule out that tolerant farmers were more likely to accept government subsidies, or that intolerant farmers rejected subsidies because they took care of predator problems independently and illicitly.

Friend or foe? Human tolerance toward predators such as jaguars depends on social factors and can be influenced by information on the risks and benefits from the predators.

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These studies suggest that economic incentives can be used to increase tolerance for some predators and protect some from poaching. However, Liberg *et al.* concluded that between 1998 and 2009, an estimated 51% of Sweden's wolves died of poaching; 69% of the latter were concealed by the perpetrators (*8*). Thus, incentives may change poaching behavior in some people but are not a panacea. The delivery of benefits may need to be supplemented by social change. Support for this idea comes from a correlational study of Kenyan Maasai livestock herders, which shows that lion killing diminished when compensation was paid for livestock losses and diminished further when trusted community members were paid to protect livestock, warn villagers of the presence of lions, and monitor lion movements (*9*).

The influence of peers and social norms on poaching intentions is revealed by research on Brazilian ranchers living near jaguar territories. On the basis of interviews with 268 Brazilian cattle ranchers about their intentions to kill jaguars illegally, Marchini and Macdonald concluded that social factors were more influential than retaliation for jaguar predation on cattle or perceived threats to humans (*10*). The ranchers' intentions to kill jaguars positively correlated with the size of their land holdings and were best explained by social norms; ranchers who thought that others kill jaguars or expected such poaching were more intent to kill jaguars themselves. The social facilitation that results in areas where poaching is common and accepted can create predator-free zones as neighbors and associates coordinate their actions explicitly or tacitly (*10*).

Because some hunters in North America and Europe historically helped to conserve populations of valued game (such as deer and ducks), policy-makers in these regions often assume that hunters will also help to conserve predators designated as legal game. For example, a program that allowed up to 43 endangered wolves to be killed in Wisconsin had the explicit purpose of fostering greater social tolerance for wolves (*11*). Yet a study of 656 residents of Wisconsin's wolf population range showed a decline in tolerance and an increase in intention to poach wolves between 2001 and 2009, after the implementation of government culling of wolves implicated in livestock attacks (*12*). Tolerance continued to

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decline following the first-ever regulated wolf harvest in Wisconsin (*13*). These results are not consistent with the expectation that legalizing predator killing will reverse intolerance or intention to poach.

Before-and-after comparisons cannot disentangle the effects of coincidental changes in policy and public debate; for that, one needs controlled experiments. A study of individual acceptance of American black bears measured changes in tolerance in four treatment groups that received different information about black bears. Information about the benefits people gain from bears, in combination with information about how to reduce risks posed by bears, increased subjects' acceptance of bears in their region (*14*). Information about how to reduce risks alone (without accompanying information about benefits) decreased subjects' acceptance of bears, possibly by increasing the salience of risks. A correlational study of attitudes among residents around a Nepalese national park came to a similar conclusion, reporting that the strongest predictors of acceptance of tigers were the perceived benefits, none of which involved killing tigers (*15*).

The above studies of jaguars, wolves, lions, and bears challenge the conventional view that intolerance for predators and intention to kill predators result primarily from perceived threats to livelihoods. Although monetary incentives for predator tolerance appear to have been successful in several cases, there is evidence that predator-poaching is influenced more strongly by social factors, with peer group norms and government-sanctioned predator-killing affecting people's intentions to poach predators. We therefore recommend caution in legalizing the killing of predators. Experimentally manipulating monetary and social incentives would help conservationists to determine which factors influence poaching, both among individuals and across cultures that have different histories with various predators. These insights could be highly valuable for future recovery and restoration efforts.

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CELL BIOLOGY

Dysfunctional Mechanosensing in Aneurysms

Cellular sensing of a compliant extracellular matrix may be critical in maintaining structural integrity of the aorta.

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The aorta is the body's main conduit for blood flow, passing through the chest and abdomen. When this artery's wall—thick as a garden hose—weakens, the aorta can dilate abnormally, rupture, and cause life-threatening bleeding. Abdominal aortic aneurysms occur most commonly in individuals between 65 and 75 years old. By contrast, thoracic aortic aneurysms and dissections (TAADs) afflict the young as well and arise primarily from noninflammatory mechanisms that often involve underlying genetic mutations (*1*, *2*). Rupture results from mechanical failure, but what renders the aortic wall vulnerable? It may be that TAADs arise from a failure of cellular mechanosensing.

All large arteries grow and remodel to establish and preserve mechanical homeostasis in response to changing hemodynamic conditions (*3*, *4*). The thoracic aorta (see the figure) is subjected to the largest cyclic circumferential stretch from the distending blood pressure, and axial stretch from gross motions of the heart. Like other large arteries, it responds to sustained changes in blood pressure, but its extreme compliance and elastic recoil allow it to accommodate large changes in pressure-driven blood flow without changing the contraction of the smooth muscle cells within the wall.

Cells of the aortic wall are embedded in an extracellular matrix that bears most of the stress from blood pressure. Whereas wall stresses are typically 100 to 200 kPa, stresses supported or exerted by cells of the wall are about 3 to 5 kPa (*4*). This implies that the matrix shields these cells from high stresses. Yet, cells still must sense altered stresses to initiate appropriate remodeling (*5*, *6*). Matrix proteins must also be prestressed when incorporated within existing stressed matrix to promote mechanical homeostasis (*4*). That is, smooth muscle cells and fibroblasts do not merely secrete collagen fibers; rather, they assemble organized collagen fibrils through force-dependent processes that involve cell adhesion proteins (integrins) and the cytoskeleton (actin and myosin) (*7*). Hence, cell sensing and regulation of a compliant extracellular matrix are fundamental to maintaining proper thoracic aortic function and structural integrity.

The aortic extracellular matrix consists of myriad proteins, glycoproteins, and glycosaminoglycans, but elastin and collagen play particularly important roles in compliance and recoil, and stiffness and strength, respectively. Smooth muscle cells and fibroblasts sense (*5*, *6*) the mechanical state of this matrix through integrins and the cytoskeleton. Transduction of this information to intracellular signaling pathways allows them to control the synthesis of matrix components and alter their cytoskeleton in response to cycles of increased mechanical load (see the figure) (*5*, *6*). This force-regulated matrix remodeling involves factors that are secreted by cells within the aortic wall. Smooth muscle cells and fibroblasts release transforming growth factor–β (TGF-β), a cytokine that binds to the matrix in latent form and is activated by proteases or integrins through force-dependent

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