



The Malicious Serpent: Snakes as a Prototypical Stimulus for an Evolved Module of Fear Author(s): Arne Öhman and Susan Mineka Source: *Current Directions in Psychological Science*, Vol. 12, No. 1 (Feb., 2003), pp. 5-9 Published by: Sage Publications, Inc. on behalf of Association for Psychological Science Stable URL: http://www.jstor.org/stable/20182821 Accessed: 18-01-2017 00:39 UTC

JSTOR is a not-for-profit service that helps scholars, researchers, and students discover, use, and build upon a wide range of content in a trusted digital archive. We use information technology and tools to increase productivity and facilitate new forms of scholarship. For more information about JSTOR, please contact support@jstor.org.

Your use of the JSTOR archive indicates your acceptance of the Terms & Conditions of Use, available at http://about.jstor.org/terms



Sage Publications, Inc., Association for Psychological Science are collaborating with JSTOR to digitize, preserve and extend access to Current Directions in Psychological Science

bral cortex: Vol. 7 (pp. 391–440). New York: Plenum Press.

- Kolb, B. (1995). Brain plasticity and behavior. Mahwah, NJ: Erlbaum.
- Kolb, B., Forgie, M., Gibb, R., Gorny, G., & Rowntree, S. (1998). Age, experience, and the changing brain. *Neuroscience and Biobehavioral Reviews*, 22, 143–159.
- Kolb, B., Gibb, R., & Gorny, G. (2000). Cortical plasticity and the development of behavior after early frontal cortical injury. *Developmental Neuropsychology*, 18, 423–444.
- Kolb, B., Gibb, R., & Gorny, G. (2003). Experience-dependent changes in dendritic arbor and spine density in neocortex vary with age

and sex. *Neurobiology of Learning and Memory*, 79, 1–10.

Robinson, T.E., & Kolb, B. (1999). Alterations in the morphology of dendrites and dendritic spines in the nucleus accumbens and prefrontal cortex following repeated treatment with amphetamine or cocaine. European Journal of Neuroscience, 11, 1598–1604.

The Malicious Serpent: Snakes as a Prototypical Stimulus for an Evolved Module of Fear

Arne Öhman¹ and Susan Mineka

Department of Clinical Neuroscience, Karolinska Institute, Stockholm, Sweden (A.Ö.), and Department of Psychology, Northwestern University, Evanston, Illinois (S.M.)

Abstract

As reptiles, snakes may have signified deadly threats in the environment of early mammals. We review findings suggesting that snakes remain special stimuli for humans. Intense snake fear is prevalent in both humans and other primates. Humans and monkeys learn snake fear more easily than fear of most other stimuli through direct or vicarious conditioning. Neither the elicitation nor the conditioning of snake fear in humans requires that snakes be consciously perceived; rather, both processes can occur with masked stimuli. Humans tend to perceive illusory correlations between snakes and aversive stimuli, and their attention is automatically captured by snakes in complex visual displays. Together, these and other findings delineate an evolved fear module in the brain. This module is selectively and automatically activated by once-threatening stimuli, is relatively encapsulated from cognition, and derives from specialized neural circuitry.

Keywords evolution; snake fear; fear module

Snakes are commonly regarded as slimy, slithering creatures worthy of fear and disgust. If one were to believe the Book of Genesis, humans' dislike for snakes resulted from a divine intervention: To avenge the snake's luring of Eve to taste the fruit of knowledge, God instituted eternal enmity between their descendants. Alternatively, the human dislike of snakes and the common appearances of reptiles as the embodiment of evil in myths and art might reflect an evolutionary heritage. Indeed, Sagan (1977) speculated that human fear of snakes and other reptiles may be a distant effect of the conditions under which early mammals evolved. In the world they inhabited, the animal kingdom was dominated by awesome reptiles, the dinosaurs, and so a prerequisite for early mammals to deliver genes to future generations was to avoid getting caught in the fangs of Tyrannosaurus rex and its relatives. Thus, fear and respect for reptiles is a likely core mammalian heritage. From this perspective, snakes and other reptiles may continue to have a special psychological significance even for humans, and considerable evidence suggests this is indeed true. Furthermore, the pattern of findings appears consistent with the evolutionary premise.

THE PREVALENCE OF SNAKE FEARS IN PRIMATES

Snakes are obviously fearsome creatures to many humans. Agras, Sylvester, and Oliveau (1969) interviewed a sample of New Englanders about fears, and found snakes to be clearly the most prevalent object of intense fear, reported by 38% of females and 12% of males.

Fear of snakes is also common among other primates. According to an exhaustive review of field data (King, 1997), 11 genera of primates showed fear-related responses (alarm calls, avoidance, mobbing) in virtually all instances in which they were observed confronting large snakes. For studies of captive primates, King did not find consistent evidence of snake fear. However, in direct comparisons, rhesus (and squirrel) monkeys reared in the wild were far more likely than labreared monkeys to show strong phobiclike fear responses to snakes (e.g., Mineka, Keir, & Price, 1980). That this fear is adaptive in the wild is further supported by independent field reports of large snakes attacking primates (M. Cook & Mineka, 1991).

This high prevalence of snake fear in humans as well as in our

Copyright © 2003 American Psychological Society

primate relatives suggests that it is a result of an ancient evolutionary history. Genetic variability might explain why not all individuals show fear of snakes. Alternatively, the variability could stem from differences in how easily individuals learn to fear reptilian stimuli when they are encountered in aversive contexts. This latter possibility would be consistent with the differences in snake fear between wild- and lab-reared monkeys.

LEARNING TO FEAR SNAKES

Experiments with lab-reared monkeys have shown that they can acquire a fear of snakes vicariously, that is, by observing other monkeys expressing fear of snakes. When nonfearful lab-reared monkeys were given the opportunity to observe a wild-reared "model" monkey displaying fear of live and toy snakes, they were rapidly conditioned to fear snakes, and this conditioning was strong and persistent. The fear response was learned even when the fearful model monkey was shown on videotape (M. Cook & Mineka, 1990).

When videos were spliced so that identical displays of fear were modeled in response to toy snakes and flowers, or to toy crocodiles and rabbits (M. Cook & Mineka, 1991), the lab-reared monkeys showed substantial conditioning to toy snakes and crocodiles, but not to flowers and toy rabbits. Toy snakes and flowers served equally well as signals for food rewards (M. Cook & Mineka, 1990), so the selective effect of snakes appears to be restricted to aversive contexts. Because these monkeys had never seen any of the stimuli used prior to these experiments, the results provide strong support for an evolutionary basis to the selective learning.

A series of studies published in the 1970s (see Öhman & Mineka,

2001) tested the hypothesis that humans are predisposed to easily learn to fear snakes. These studies used a discriminative Pavlovian conditioning procedure in which various pictures served as conditioned stimuli (CSs) that predicted the presence and absence of mildly aversive shock, the unconditioned stimulus (US). Participants for whom snakes (or spiders) consistently signaled shocks showed stronger and more lasting conditioned skin conductance responses (SCRs; palmar sweat responses that index emotional activation) than control participants for whom flowers or mushrooms signaled shocks. When a nonaversive US was used, however, this difference disappeared. E.W. Cook, Hodes, and Lang (1986) demonstrated that qualitatively different responses were conditioned to snakes (heart rate acceleration, indexing fear) than to flowers and mushrooms (heart rate deceleration, indexing attention to the eliciting stimulus). They also reported superior conditioning to snakes than to gun stimuli paired with loud noises. Such results suggest that the selective association between snakes and aversive USs reflects evolutionary history rather than cultural conditioning.

NONCONSCIOUS CONTROL OF RESPONSES TO SNAKES

If the prevalence and ease of learning snake fear represents a core mammalian heritage, its neural machinery must be found in brain structures that evolved in early mammals. Accordingly, the fear circuit of the mammalian brain relies heavily on limbic structures such as the amygdala, a collection of neural nuclei in the anterior temporal lobe. Limbic structures emerged in the evolutionary transition from reptiles to mammals and use preexisting structures in the "reptilian brain" to control emotional output such as flight/fight behavior and

cardiovascular changes (see Öhman & Mineka, 2001).

From this neuroevolutionary perspective, one would expect the limbically controlled fear of snakes to be relatively independent of the most recently evolved control level in the brain, the neocortex, which is the site of advanced cognition. This hypothesis is consistent with the often strikingly irrational quality of snake phobia. For example, phobias may be activated by seeing mere pictures of snakes. Backward masking is a promising methodology for examining whether phobic responses can be activated without involvement of the cortex. In this method, a brief visual stimulus is blanked from conscious perception by an immediately following masking stimulus. Because backward masking disrupts visual processing in the primary visual cortex, responses to backward-masked stimuli reflect activation of pathways in the brain that may access the fear circuit without involving cortical areas mediating visual awareness of the stimulus.

In one study (Öhman & Soares, 1994), pictures of snakes, spiders, flowers, and mushrooms were presented very briefly (30 ms), each time immediately followed by a masking stimulus (a randomly cut and reassembled picture). Although the participants could not recognize the intact pictures, participants who were afraid of snakes showed enhanced SCRs only to masked snakes, whereas participants who were afraid of spiders responded only to spiders. Similar results were obtained (Ohman & Soares, 1993) when nonfearful participants, who had been conditioned to unmasked snake pictures by shock USs, were exposed to masked pictures without the US. Thus, responses to conditioned snake pictures survived backward masking; in contrast, masking eliminated conditioning effects in another group of participants conditioned to neutral stimuli such as flowers or mushrooms.

Published by Blackwell Publishing Inc.

Furthermore, subsequent experiments (Öhman & Soares, 1998) also demonstrated conditioning to masked stimuli when masked snakes or spiders (but not masked flowers or mushrooms) were used as CSs followed by shock USs. Thus, these masking studies show that fear responses (as indexed by SCRs) can be learned and elicited when backward masking prevents visually presented snake stimuli from accessing cortical processing. This is consistent with the notion that responses to snakes are organized by a specifically evolved primitive neural circuit that emerged with the first mammals long before the evolution of neocortex.

ILLUSORY CORRELATIONS BETWEEN SNAKES AND AVERSIVE STIMULI

If expression and learning of snake fear do not require cortical processing, are people's cognitions about snakes and their relationships to other events biased and irrational? One example of such biased processing occurred in experiments on illusory correlations: Participants (especially those who were afraid of snakes) were more likely to perceive that slides of fear-relevant stimuli (such as snakes) were paired with shock than to perceive that slides of control stimuli (flowers and mushrooms) were paired with shock. This occurred even though there were no such relationships in the extensive random sequence of slide stimuli and aversive and nonaversive outcomes (tones or nothing) participants had experienced (Tomarken, Sutton, & Mineka, 1995).

Similar illusory correlations were not observed for pictures of damaged electrical equipment and shock even though they were rated as belonging together better than snakes and shock (Tomarken et al., 1995). In another experiment, participants showed exaggerated expectancies for shock to follow both snakes and damaged electrical equipment before the experiment began (Kennedy, Rapee, & Mazurski, 1997), but reported only the illusory correlation between snakes and shock after experiencing the random stimulus series. Thus, it appears that snakes have a cognitive affinity with aversiveness and danger that is resistant to modification by experience.

AUTOMATIC CAPTURE OF ATTENTION BY SNAKE STIMULI

People who encounter snakes in the wild may report that they first froze in fear, only a split second later realizing that they were about to step on a snake. Thus, snakes may automatically capture attention. A study supporting this hypothesis (Öhman, Flykt, & Esteves, 2001) demonstrated shorter detection latencies for a discrepant snake picture among an array of many neutral distractor stimuli (e.g., flower pictures) than vice versa. Furthermore, "finding the snake in the grass" was not affected by the number of distractor stimuli, whereas it took longer to detect discrepant flowers and mushrooms among many than among few snakes when the latter served as distractor stimuli. This suggests that snakes, but not flowers and mushrooms, were located by an automatic perceptual routine that effortlessly found target stimuli that appeared to "pop out" from the matrix independently of the number of distractor stimuli. Participants who were highly fearful of snakes showed even superior performance in detecting snakes. Thus, when snakes elicited fear in participants, this fear state sensitized the perceptual apparatus to detect snakes even more efficiently.

THE CONCEPT OF A FEAR MODULE

The evidence we have reviewed shows that snake stimuli are strongly and widely associated with fear in humans and other primates and that fear of snakes is relatively independent of conscious cognition. We have proposed the concept of an evolved fear module to explain these and many related findings (Öhman & Mineka, 2001). The fear module is a relatively independent behavioral, mental, and neural system that has evolved to assist mammals in defending against threats such as snakes. The module is selectively sensitive to, and automatically activated by, stimuli related to recurrent survival threats, it is relatively encapsulated from more advanced human cognition, and it relies on specialized neural circuitry.

This specialized behavioral module did not evolve primarily from survival threats provided by snakes during human evolution, but rather from the threat that reptiles have provided through mammalian evolution. Because reptiles have been associated with danger throughout evolution, it is likely that snakes represent a prototypical stimulus for activating the fear module. However, we are not arguing that the human brain has a specialized module for automatically generating fear of snakes. Rather, we propose that the blueprint for the fear module was built around the deadly threat that ancestors of snakes provided to our distant ancestors, the early mammals. During further mammalian evolution, this blueprint was modified, elaborated, and specialized for the ecological niches occupied by different species. Some mammals may even prey on snakes, and new stimuli and stimulus features have been added to reptiles as preferential activators of the module.

For example, facial threat is similar to snakes when it comes to activating the fear module in social primates (Öhman & Mineka, 2001). Through Pavlovian conditioning, the fear module may come under the control of a very wide range of stimuli signaling pain and danger. Nevertheless, evolutionarily derived constraints have afforded stimuli once related to recurrent survival threats easier access for gaining control of the module through fear conditioning (Öhman & Mineka, 2001).

ISSUES FOR FURTHER RESEARCH

The claim that the fear module can be conditioned without awareness is a bold one given that there is a relative consensus in the field of human conditioning that awareness of the CS-US contingency is required for acquiring conditioned responses. However, as we have extensively argued elsewhere (Öhman & Mineka, 2001; Wiens & Öhman, 2002), there is good evidence that conditioning to nonconsciously presented CSs is possible if they are evolutionarily fear relevant. Other factors that might promote such nonconscious learning include intense USs, short CS-US intervals, and perhaps temporal overlap between the CS and the US. However, little research on these factors has been reported, and there is a pressing need to elaborate their relative effectiveness in promoting conditioning of the fear module outside of awareness.

One of the appeals of the fear module concept is that it is consistent with the current understanding of the neurobiology of fear conditioning, which gives a central role to the amygdala (e.g., Öhman & Mineka, 2001). However, this understanding is primarily based on animal data. Even though the emerging brain-imaging literature on human fear conditioning is consistent with this database, systematic efforts are needed in order to tie the fear module more convincingly to human brain mechanisms. For example, a conspicuous gap in knowledge concerns whether the amygdala is indeed specially tuned to conditioning contingencies involving evolutionarily fear-relevant CSs such as snakes.

An interesting question that can be addressed both at a psychological and at a neurobiological level concerns the perceptual mechanisms that give snake stimuli privileged access to the fear module. For example, are snakes detected at a lower perceptual threshold relative to non-fear-relevant objects? Are they identified faster than other objects once detected? Are they quicker to activate the fear module and attract attention once identified? Regardless of the locus of perceptual privilege, what visual features of snakes make them such powerful fear elicitors and attention captors? Because the visual processing in pathways preceding the cortical level is crude, the hypothesis that masked presentations of snakes directly access the amygdala implies that the effect is mediated by simple features of snakes rather than by the complex configuration of features defining a snake. Delineating these features would allow the construction of a "super fear stimulus." It could be argued that such a stimulus would depict "the archetypical evil" as represented in the human brain.

Recommended Reading

- Mineka, S. (1992). Evolutionary memories, emotional processing, and the emotional disorders. *The Psychology of Learning and Motivation*, 28, 161–206.
- Öhman, A., Dimberg, U., & Öst, L.-G. (1985). Animal and social phobias: Biological constraints on learned

fear responses. In S. Reiss & R.R. Bootzin (Eds.), *Theoretical issues in behavior therapy* (pp. 123–178). New York: Academic Press. Öhman, A., & Mineka, S. (2001). (See References)

Note

1. Address correspondence to Arne Öhman, Psychology Section, Department of Clinical Neuroscience, Karolinska Institute and Hospital, Z6:6, S-171 76 Stockholm, Sweden; e-mail: arne. ohman@cns.ki.se.

References

- Agras, S., Sylvester, D., & Oliveau, D. (1969). The epidemiology of common fears and phobias. *Comprehensive Psychiatry*, 10, 151–156.
- Cook, E.W., Hodes, R.L., & Lang, P.J. (1986). Preparedness and phobia: Effects of stimulus content on human visceral conditioning. *Journal of Abnormal Psychology*, 95, 195–207.
- Cook, M., & Mineka, S. (1990). Selective associations in the observational conditioning of fear in rhesus monkeys. *Journal of Experimental Psychology: Animal Behavior Processes*, 16, 372–389.
- Cook, M., & Mineka, S. (1991). Selective associations in the origins of phobic fears and their implications for behavior therapy. In P. Martin (Ed.), Handbook of behavior therapy and psychological science: An integrative approach (pp. 413– 434). Oxford, England: Pergamon Press.
- Kennedy, S.J., Rapee, R.M., & Mazurski, E.J. (1997). Covariation bias for phylogenetic versus ontogenetic fear-relevant stimuli. *Behaviour Research and Therapy*, 35, 415–422.
- King, G.E. (1997, June). The attentional basis for primate responses to snakes. Paper presented at the annual meeting of the American Society of Primatologists, San Diego, CA.
- Mineka, S., Keir, R., & Price, V. (1980). Fear of snakes in wild- and laboratory-reared rhesus monkeys (Macaca mulatta). Animal Learning and Behavior, 8, 653–663.
- Öhman, A., Flykt, A., & Esteves, F. (2001). Emotion drives attention: Detecting the snake in the grass. Journal of Experimental Psychology: General, 131, 466–478.
- Öhman, A., & Mineka, S. (2001). Fear, phobias and preparedness: Toward an evolved module of fear and fear learning. *Psychological Review*, 108, 483–522.
- Öhman, A., & Soares, J.J.F. (1993). On the automatic nature of phobic fear: Conditioned electrodermal responses to masked fear-relevant stimuli. *Journal of Abnormal Psychology*, 102, 121-132.
- Öhman, A., & Soares, J.J.F. (1994). "Unconscious anxiety": Phobic responses to masked stimuli. *Journal of Abnormal Psychology*, 103, 231–240.
- Öhman, A., & Soares, J.J.F. (1998). Emotional conditioning to masked stimuli: Expectancies for aversive outcomes following nonrecognized fear-irrelevant stimuli. Journal of Experimental Psychology: General, 127, 69–82.

Published by Blackwell Publishing Inc.

Sagan, C. (1977). The dragons of Eden: Speculations on the evolution of human intelligence. London: Hodder and Stoughton.

Tomarken, A.J., Sutton, S.K., & Mineka, S. (1995).

Fear-relevant illusory correlations: What types of associations promote judgmental bias? *Journal of Abnormal Psychology*, 104, 312–326. Wiens, S., & Öhman, A. (2002). Unawareness is more than a chance event: Comment on Lovibond and Shanks (2002). Journal of Experimental Psychology: Animal Behavior Processes, 28, 27–31.

The Relation of Hypertension to Cognitive Function

Shari R. Waldstein¹

Department of Psychology, University of Maryland, Baltimore County, Baltimore, Maryland; Division of Gerontology, Department of Medicine, University of Maryland School of Medicine, Baltimore, Maryland; and Geriatrics Research, Education, and Clinical Center, Baltimore Veterans Affairs Medical Center, Baltimore, Maryland

Abstract

Hypertension is an established risk factor for stroke. However, prior to such a major clinical event, hypertension exerts a more subtle impact on the brain that is revealed by diminished cognitive function. Studies comparing the performance of people with high and normal blood pressure levels have shown that high blood pressure or hypertension is related to poorer performance on tests of attention, learning and memory, executive functions, visuospatial skills, psychomotor abilities, and perceptual skills. Hypertension is also predictive of cognitive decline. Variables that may alter (i.e., moderate) the relation of hypertension to cognitive function include age, education, several biological characteristics of hypertension, and the presence of concurrent diseases. Although hypertensives are not clinically impaired, their diminished levels of cognitive performance could affect their perceived quality of life. Various brain mechanisms may explain the relation of hypertension to lower levels of cognitive function. Further understanding of the relation between hypertension and cognition is critical to the preservation of cognitive function across the life span.

Keywords

hypertension; blood pressure; cognitive function; neuropsychology; brain

Hypertension, or a persistently high blood pressure, is a common form of cardiovascular disease that affects one in every five people in the United States. The current definition of hypertension is a sustained systolic blood pressure of 140 millimeters of mercury (mm Hg) or more, sustained diastolic blood pressure of 90 mm Hg or more, or both. Nearly all adults with hypertension (90-95%) have what is called essential (or primary) hypertension, which means that the specific cause of the elevation in blood pressure is unknown. However, essential hypertension is actually thought to be determined by many genetic and environmental factors. Hypertension due to known medical disease (e.g., chronic renal disease) is called secondary hypertension.

It is well known that hypertension causes damage to many of the body's organs, including the heart, kidneys, eyes, and brain, and it is a major risk factor for coronary heart disease and stroke. However, the impact of hypertension on the brain prior to stroke is presently underrecognized. Even in otherwise healthy people, hypertension can lead to mild to moderate alterations in the brain's structure and function, including its ability to efficiently process information (known as cognitive function). These early hypertension-related changes in the brain can be detected by sophisticated brain scans and by neuropsychological assessment of cognitive abilities. In this article, I examine our knowledge of the relation of essential hypertension (which I refer to hereafter as simply hypertension) to cognitive function.

WHAT IS THE RELATION OF HYPERTENSION TO COGNITIVE FUNCTION?

The relation of hypertension to cognitive function is frequently studied by comparing the cognitive performance of people with normal blood pressure (or normotensives) with that of hypertensives at one point in time. Commonly assessed cognitive functions include attention, learning and memory, executive functions (i.e., self-regulatory behaviors such as planning and organization, mental flexibility, and response inhibition), visuospatial skills, psychomotor abilities, perceptual skills, and language abilities. Typically, the hypertensives in these studies have been diagnosed by physicians and either are unmedicated or stop taking their antihypertensive medication prior to cognitive testing