ment of taste neuronal circuitries, especially in combination with the gene-targeted mutant mice for key molecules.

References and Notes

1. B. Lindemann, *Nature* **413**, 219 (2001).
2. D. E. Berman, Y. Dudai, *Science* **291**, 2417 (2001).
3. H. Matsunami, J. P. Montmayeur, L. B. Buck, *Nature* **404**, 601 (2000).
4. E. Adler *et al.*, *Cell* **100**, 693 (2000).
5. J. Chandrashekar *et al.*, *Cell* **100**, 703 (2000).
6. G. Nelson *et al.*, *Cell* **106**, 381 (2001).
7. G. Nelson *et al.*, *Nature* **416**, 199 (2002).
8. Y. Zhang *et al.*, *Cell* **112**, 293 (2003).
9. G. Q. Zhao *et al.*, *Cell* **115**, 255 (2003).
10. K. L. Mueller *et al.*, *Nature* **434**, 225 (2005).
11. R. Norgren, in *The Rat Nervous System*, G. Paxinos, Ed. (Academic Press, San Diego, 1995), pp. 751–771.
12. H. Herbert, M. M. Moga, C. B. Saper, *J. Comp. Neurol.* **293**, 540 (1990).
13. J. F. Bernard, M. Alden, J. M. Besson, *J. Comp. Neurol.* **329**, 201 (1993).
14. H. Bester, L. Bourgeais, L. Villanueva, J. M. Besson, J. F. Bernard, *J. Comp. Neurol.* **405**, 421 (1999).
15. Y. Yoshihara *et al.*, *Neuron* **22**, 33 (1999).
16. Z. Zou, L. F. Horowitz, J.-P. Montmayeur, S. Snapper, L. B. Buck, *Nature* **414**, 173 (2001).
17. M. Sugita, unpublished results.
18. R. B. Hamilton, R. Norgren, *J. Comp. Neurol.* **222**, 560 (1984).
19. R. Norgren, *J. Comp. Neurol.* **166**, 17 (1976).
20. G. V. Allen, C. B. Saper, K. M. Hurley, D. F. Cechetto, *J. Comp. Neurol.* **311**, 1 (1991).
21. C. L. Yee, K. R. Jones, T. E. Finger, *J. Comp. Neurol.* **459**, 15 (2003).

22. We are grateful to the members in Research Facilities for Laboratory Animal Science, Natural Science Center for Basic Research and Development, Hiroshima University, for supporting animal experiment. We thank H. Ohishi for expert help in calcium imaging, Y. Yoshihara for truncated WGA, S. Offermanns and M. I. Simon for Gα15, and P. Mombaerts for ETLpA-/LTNL. This research was supported by a Grant-in-Aid for Scientific Research from the Ministry of Education, Science, Sports, and Culture of Japan, and the Sumitomo Foundation (to M.S.).

Supporting Online Material

www.sciencemag.org/cgi/content/full/309/5735/781/DC1
Materials and Methods
SOM Text
Figs. S1 to S3
References and Notes

7 February 2005; accepted 10 June 2005
10.1126/science.1110787

The Role of Social Groups in the Persistence of Learned Fear

Andreas Olsson,[1] Jeffrey P. Ebert,[3] Mahzarin R. Banaji,[3] Elizabeth A. Phelps[1,2]*

Classical fear conditioning investigates how animals learn to associate environmental stimuli with an aversive event. We examined how the mechanisms of fear conditioning apply when humans learn to associate social ingroup and outgroup members with a fearful event, with the goal of advancing our understanding of basic learning theory and social group interaction. Primates more readily associate stimuli from certain fear-relevant natural categories, such as snakes, with a negative outcome relative to stimuli from fear-irrelevant categories, such as birds. We assessed whether this bias in fear conditioning extends to social groups defined by race. Our results indicate that individuals from a racial group other than one's own are more readily associated with an aversive stimulus than individuals of one's own race, among both white and black Americans. This prepared fear response might be reduced by close, positive interracial contact.

In classical fear conditioning, a neutral stimulus acquires aversive properties by virtue of simply being paired in time with an aversive event. In general, research on classical conditioning has not emphasized differences between classes of stimuli, instead focusing on principles that apply across different kinds of stimuli (*1*). One important exception is research on selective, or prepared, aversive learning. For both humans (*2*, *3*) and nonhuman primates (*4*), stimuli from certain fear-relevant natural categories, such as snakes and spiders, are more readily associated with aversive events than stimuli from fear-irrelevant categories, such as birds and butterflies (*5*). We investigated whether prepared learning can be extended to fear associated with members of another, as compared with one's own, racial group. Recent studies have observed that race bias and fear conditioning may indeed rely on overlapping neural systems (*6*–*8*), suggesting a potential link in mechanism and the opportunity to use classical fear conditioning as a model for aversive learning in a socio-cultural context (*9*, *10*).

We assessed whether individuals of another race are more readily associated with an aversive stimulus than individuals of one's own race, and whether these effects may be moderated by attitudes, beliefs, or contact with members of the racial outgroup. In humans, prepared fear learning has been most consistently demonstrated as a persistence in the learned fear response to fear-relevant conditioned stimuli (*11*). If representations of racial outgroup but not ingroup members act like prepared stimuli, we would expect that fear responses acquired to outgroup faces would persist during extinction relative to fear responses acquired to ingroup faces. To test this prediction, we conducted two experiments whose procedures differed only with respect to the stimuli used (*12*). The first was designed to recreate the standard preparedness effect for traditional fear-relevant stimuli, and the second was designed to test this effect in the context of human social groups defined by race.

Experiment 1 presented subjects with images of two typically used exemplars of fear-relevant (a snake and a spider) and fear-irrelevant (a bird and a butterfly) stimuli in order to verify that the experimental manipulations effectively replicated previous findings. Experiment 2 presented black and white American participants images of faces of two black and two white unfamiliar male individuals with neutral expressions. During fear acquisition, one stimulus (the reinforced conditioned stimulus, CS+) from each stimulus category was paired with a mild electric shock (the unconditioned stimulus, UCS), which was individually adjusted to be perceived as uncomfortable, but not painful. The other stimulus from each category (the unreinforced conditioned stimulus, CS–) was presented without shock. Each presentation of a CS was 6 s, and the UCS co-terminated with each presentation of a CS+ during acquisition. During the extinction phase that followed, no shocks were administered. Skin conductance responses (SCRs) were measured during both acquisition and extinction trials. The conditioned fear response (CR) was assessed as the differential SCR, that is, the SCR to the CS+ minus the SCR to the CS– from the same stimulus category, thereby reducing preexisting differences in the emotional salience of stimulus categories as a confounding variable. In experiment 2, after completion of the extinction phase, subjects completed implicit and explicit measures of race attitudes and stereotypes, as well as self-report measures of contact with racial ingroup and outgroup members. The within-subject design of the conditioning paradigm allowed us to compute a relative measure of conditioning race bias that could be linked to each participant's relative measures of race attitudes, stereotypes, and intergroup contact.

The mean differential SCRs during acquisition and extinction in experiment 1 are presented in Fig. 1A. During acquisition, there

[1]Department of Psychology and [2]Center for Neural Science, New York University, 6 Washington Place, New York, NY 10003, USA. [3]Department of Psychology, Harvard University, 33 Kirkland Street, Cambridge, MA 02138, USA.

*To whom correspondence should be addressed. E-mail: liz.phelps@nyu.edu

was a significantly greater SCR to the CS+ compared with the CS– for both fear-relevant [$t(16) = 5.81$, $P < 0.0001$] and fear-irrelevant [$t(16) = 4.24$, $P < 0.001$] stimuli, indicating acquisition of a CR to both classes of stimuli. As predicted, in the extinction phase, subjects' CRs to snakes and spiders failed to fully extinguish [$t(16) = 2.81$, $P < 0.05$], whereas their CRs to birds and butterflies did [$t(16) = 0.98$, not significant (NS)]. These results replicate earlier results showing a greater persistence of fear learning for fear-relevant than fear-irrelevant conditioned stimuli (*3*, *11*).

The mean differential SCRs during acquisition and extinction to human faces from social groups in experiment 2 are plotted in Fig. 1B. Overall, there was a greater SCR for the CS+ versus the CS– for both racial ingroup [$t(72) = 5.28$, $P < 0.0001$] and outgroup [$t(72) = 8.10$, $P < 0.0001$] faces during acquisition, demonstrating a CR to both. In extinction, there was a persistent, significant CR to racial outgroup faces [$t(72) = 3.87$, $P < 0.0001$], whereas the CR to ingroup races was fully extinguished [$t(72) = -0.29$, NS]. This persistence of fear learning during extinction for outgroup members mirrors the pattern observed for snakes and spiders in experiment 1 (*13*).

This prepared learning effect is displayed separately for white (Fig. 2A) and black American (Fig. 2B) participants. White participants displayed a greater SCR to the CS+ versus the CS– for both black [$t(35) = 6.03$, $P < 0.0001$] and white [$t(35) = 3.96$, $P < 0.001$] faces during acquisition. As predicted, white participants' CRs to black faces did not fully extinguish [$t(35) = 2.85$, $P < 0.01$], whereas their CRs to white faces did [$t(35) = –0.91$, NS]. During acquisition, black participants displayed a greater SCR to the CS+ versus the CS– for both black [$t(36) = 3.52$, $P < 0.01$] and white [$t(36) = 5.44$, $P < 0.0001$] faces, indicating acquisition of a CR. Following the same pattern of outgroup bias exhibited by the white participants, black participants' CRs to white faces did not fully extinguish [$t(36) = 2.59$, $P < 0.05$], whereas their CRs to black faces did [$t(36) = 1.10$, NS].

The extinction data show that unfamiliar members of a racial outgroup can serve as prepared stimuli in a fear-learning situation. These data concur with studies demonstrating that primates selectively associate stimuli from relevant natural categories with an aversive outcome (*11*). Our findings are also consistent with imaging data linking race bias in evaluating others with subcortical brain systems that mediate fear learning across species (*6*–*8*). The propensity to associate aversive events with outgroup members could lead to more negative evaluations of the outgroup, given otherwise equivalent properties of ingroup and outgroup members. In this respect, the outgroup preparedness finding belongs with other psychological mechanisms that have been identified as contributing to the genesis and maintenance of racial prejudice, especially implicit or less conscious forms of it (*14*–*17*).

We examined whether the conditioning bias to outgroup faces was moderated by attitudes and beliefs about the outgroup or the amount of contact with outgroup members. The only measure found to significantly moderate the conditioning bias was interracial dating [Supporting Online Material (SOM) Text]. Specifically, the conditioning bias to outgroup faces was negatively correlated with the reported number of outgroup, relative to ingroup, romantic partners [$r(68) = –0.29$, $P < 0.05$]. In other words, the conditioning bias to fear racial outgroup members was attenuated among those with more interracial dating experience, consistent with a substantial body of research demonstrating that positive intergroup contact reduces negativity toward outgroups (*18*). Because this is a correlational analysis, this finding could instead indicate that a third variable highly correlated with interracial dating is causally important in the reduction of outgroup preparedness or that those individuals strongest in outgroup preparedness are less likely to date interracially. In this sample, more black participants reported interracial dating (51%) than white participants (28%). Figure S1 and table S4 illustrate the similarity of conditioning effects for black and white participants who had only same-race dating experiences.

What remains to be explained is why individuals associate racial outgroup members more easily with an aversive stimulus, and to this end previous research on prepared fear learning allows a challenge to existing ways of thinking about social learning. Demonstrations of prepared learning have typically been taken as evidence for biologically evolved learning mechanisms that treat certain natural categories of stimuli as prepared to be associated with an aversive outcome (*19*, *20*). This interpretation has received support from a range of findings. Conditioned responses to fear-relevant stimuli are especially insensitive to cognitive manipulations: Instructed extinction fails (*21*), and conditioned responses are elicited even when conditioned stimuli are presented without conscious awareness (*22*). In addition, the prepared learning effect does not extend to most culturally defined fear-relevant stimuli, such as broken electrical outlets and some representations of weapons (*2*, *23*), suggesting that fear relevance alone does not mediate this effect. However, at least one study reports that a fear-relevant cultural artifact (e.g., a pointed gun), when paired with a pertinent UCS (e.g., a loud noise), can produce a resistance to extinction that is comparable to that elicited by natural categories of fear-relevant stimuli (*24*). This result suggests that, under certain circumstances, cultural learning can imbue a stimulus with qualities that engage similar learning mechanisms as do spiders and snakes.

The evolutionary interpretation for the results of experiment 1 is relatively straight-

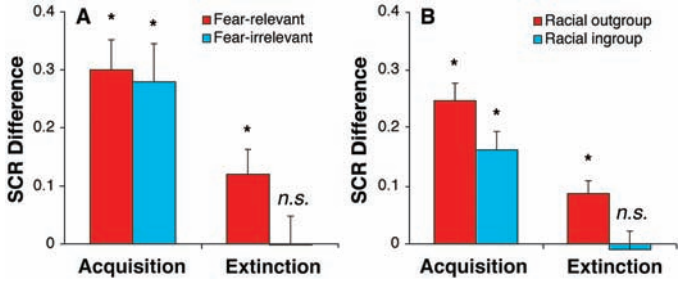

Fig. 1. Mean conditioned response, CR (scaled SCR difference), as a function of stimulus category. Error bars indicate standard errors. Asterisks indicate a statistically significant CR, and "n.s." indicates the CR is not significantly different from zero. (**A**) Experiment 1: there was a CR to both fear-relevant and fear-irrelevant stimuli during acquisition. Only CRs to fear-relevant stimuli resisted extinction. (**B**) Experiment 2: there was a CR to both outgroup and ingroup faces during acquisition. Mimicking the response pattern observed in experiment 1, only CRs to outgroup faces resisted extinction.

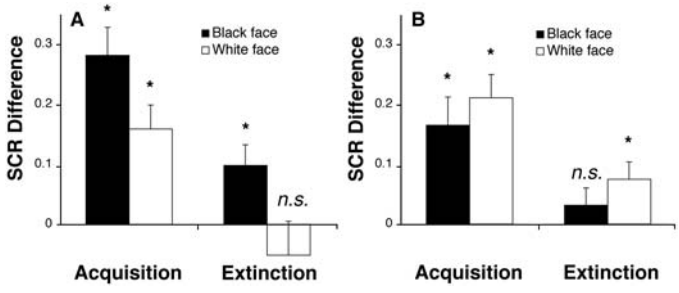

Fig. 2. Mean conditioned response, CR (scaled SCR difference), as a function of race category. Error bars indicate standard errors. Asterisks indicate a statistically significant CR, and "n.s." indicates the CR is not significantly different from zero. (**A**) White participants acquired a CR to both black and white faces, but only their CR to black faces resisted extinction. (**B**) Black participants acquired a CR to both black and white faces, but only their CR to white faces resisted extinction.

forward: Modern primates are predisposed to learn to fear spiders and snakes because such preparedness conferred a selective advantage to our ancestors over conspecifics that were not thus prepared (*11*). A similar argument has previously been made for the superior conditioning effect observed to angry in comparison with happy faces, emphasizing the evolutionary relevance of the face as a means of signaling threat (*25*). The evolutionary interpretation for the racial outgroup bias found in experiment 2 is more nuanced. The differentiation of *Homo sapiens* into what modern humans recognize as distinct races occurred relatively recently in human evolutionary history, by some estimates within the past 100,000 to 200,000 years (*26*). Critically, it is believed that this differentiation occurred precisely because of the mass migration and consequent geographic isolation of different human lineages, meaning that natural selection could not have specifically prepared whites to fear blacks and blacks to fear whites. However, humans might have evolved a more general preparedness to fear others who were dissimilar to them or who otherwise appeared not to belong to their social group because such individuals were more likely to pose a threat (*27*, *28*). If a general preparedness to fear dissimilar others did indeed evolve, then present-day members of another race, with their physical differences and common categorization as belonging to an outgroup, could activate such a mechanism and produce the robust conditioning effect observed in experiment 2.

In other words, because of its relatively recent emergence as an important dimension in human social interaction, race inherently cannot be the basis of the outgroup preparedness result. Instead, it is likely that sociocultural learning about the identity and qualities of outgroups is what provides the basis for the greater persistence of fear conditioning involving members of another group. Most notably, individuals acquire negative beliefs about outgroups according to their local cultures, and few reach adulthood without considerable knowledge of these prejudices and stereotypes (*14*, *29*, *30*). It is plausible that repeated exposure to information about outgroups might prepare individuals to fear newly encountered outgroup members.

Further research will pinpoint the generality and the interpretation of the outgroup bias in aversive conditioning. For now, our finding that close, intergroup contact may reduce this bias suggests that individual experiences can play a moderating role. Millennia of natural selection and a lifetime of social learning may predispose humans to fear those who seem different from them; however, developing relationships with these different others may be one factor that weakens this otherwise strong predisposition.

References and Notes

1. I. P. Pavlov, *Conditioned Reflexes* (Oxford Univ. Press, Oxford, 1927).
2. E. W. Cook, R. L. Hodes, P. J. Lang, *J. Abnorm. Psychol.* **95**, 195 (1986).
3. A. Öhman, M. Fredrikson, K. Hugdahl, P. A. Rimmö, *J. Exp. Psychol. Gen.* **103**, 313 (1976).
4. S. Mineka, M. Davidson, M. Cook, R. Keir, *J. Abnorm. Psychol.* **93**, 355 (1984).
5. In humans, a superior conditioning effect has also been demonstrated with angry compared with happy faces [see (*25*) for a review on faces as conditioned stimuli].
6. E. A. Phelps *et al.*, *J. Cogn. Neurosci.* **12**, 729 (2000).
7. A. J. Hart *et al.*, *Neuroreport* **11**, 2351 (2000).
8. W. A. Cunningham *et al.*, *Psychol. Sci.* **15**, 806 (2004).
9. E. A. Phelps *et al.*, *Nat. Neurosci.* **4**, 437 (2001).
10. A. Olsson, E. A. Phelps, *Psychol. Sci.* **15**, 822 (2004).
11. A. Öhman, S. Mineka, *Psychol. Rev.* **108**, 483 (2001).
12. Materials and methods are available as supporting material on *Science* Online.
13. A mixed analysis of variance (ANOVA) conducted for acquisition trials revealed that participants exhibited greater CRs to outgroup than ingroup faces [$F(1, 71) = 4.03$, $P < 0.05$], an effect not qualified by participant race [$F(1, 71) = 0.85$, NS]. Likewise, a mixed ANOVA conducted for extinction trials revealed greater CRs to outgroup than ingroup faces [$F(1, 71) = 5.59$, $P < 0.05$], an effect not qualified by participant race [$F(1, 71) = 1.70$, NS]. In other words, participants acquired stronger CRs to outgroup relative to ingroup faces, a difference that remained pronounced during extinction.
14. D. L. Hamilton, R. K. Gifford, *J. Exp. Soc. Psychol.* **12**, 392 (1976).
15. C. O. Word, M. P. Zanna, J. Cooper, *J. Exp. Soc. Psychol.* **10**, 109 (1974).
16. Z. Kunda, K. C. Oleson, *J. Pers. Soc. Psychol.* **72**, 965 (1997).
17. H. Tajfel, J. C. Turner, in *The Social Psychology of Intergroup Relations*, W. Austin, S. Worchel, Eds. (Brooks/Cole, Monterey, CA, 1979), pp. 33–48.
18. T. F. Pettigrew, L. Tropp, *J. Pers. Soc. Psychol.*, in press.
19. M. E. P. Seligman, *Psychol. Rev.* **77**, 406 (1970).
20. A. Öhman, *Psychophysiology* **23**, 123 (1986).
21. K. Hugdahl, A. Öhman, *J. Exp. Psychol. Hum. Learn.* **3**, 608 (1977).
22. A. Öhman, J. Soares, *J. Abnorm. Psychol.* **102**, 121 (1993).
23. K. Hugdahl, A. C. Kärkner, *Behav. Res. Ther.* **15**, 345 (1981).
24. K. Hugdahl, B. H. Johnsen, *Behav. Res. Ther.* **27**, 269 (1989).
25. U. Dimberg, A. Öhman, *Motiv. Emot.* **20**, 149 (1996).
26. S. Molnar, *Human Variation: Races, Types, and Ethnic Groups* (Prentice Hall, Upper Saddle River, NJ, ed. 4, 1998).
27. W. D. Hamilton, *J. Theor. Biol.* **7**, 17 (1964).
28. J. H. Manson, R. W. Wrangham, *Curr. Anthropol.* **32**, 369 (1991).
29. A. G. Greenwald, D. E. McGhee, J. K. L. Schwartz, *J. Pers. Soc. Psychol.* **74**, 1464 (1998).
30. D. Katz, K. Braly, *J. Abnorm. Soc. Psychol.* **28**, 282 (1933).

31. We want to thank W. Brennan, D. Fareri, and N. Husain for helpful assistance; J. Eberhardt for providing the face stimuli; N. Shelton for providing the contact items; and A. G. Greenwald, J. R. Hackman, and R. L. Trivers for their helpful comments. This research was supported by the James S. McDonnell Foundation, a 21st Century award (E.A.P.), National Institute of Mental Health grants 1RO1MH57672 and 5R01MH068447 (M.R.B.), and an NSF graduate research fellowship (J.P.E.).

Supporting Online Material

www.sciencemag.org/cgi/content/full/309/5735/785/DC1
Materials and Methods
SOM Text
Fig. S1
Tables S1 to S5
References

13 April 2005; accepted 20 June 2005
10.1126/science.1113551

An Interneuronal Chemoreceptor Required for Olfactory Imprinting in *C. elegans*

Jean-Jacques Remy[1] and Oliver Hobert[2]

Animals alter their behavioral patterns in an experience-dependent manner. Olfactory imprinting is a process in which the exposure of animals to olfactory cues during specific and restricted time windows leaves a permanent memory ("olfactory imprint") that shapes the animal's behavior upon encountering the olfactory cues at later times. We found that *Caenorhabditis elegans* displays olfactory imprinting behavior that is mediated by a single pair of interneurons. To function in olfactory imprinting, this interneuron pair must express a G protein–coupled chemoreceptor family member encoded by the *sra-11* gene. Our study provides insights into the cellular and molecular basis of olfactory imprinting and reveals a function for a chemosensory receptor family member in interneurons.

Olfactory imprinting, which occurs in contexts as diverse as homing behavior in salmon and neonatal attachment in mammals, is a learned olfactory response whose defining features are that the olfactory memory is long-lasting and can only be acquired during a defined developmental time window or during a specific physiological state (*1*). These features distinguish it from other learned olfactory responses, such as olfactory adaptation, which can occur at many distinct developmental or physiological states and usually lasts for a limited amount of time. However, the cellular and molecular basis of olfactory imprinting is poorly understood.

[1]Laboratoire NMDA CNRS UMR 6156, Institut de Biologie du Développement (IBDM), 13288 Marseille Cedex 9, France. E-mail: remy@ibdm.univ-mrs.fr
[2]Howard Hughes Medical Institute, Department of Biochemistry and Molecular Biophysics, Center for Neurobiology and Behavior, Columbia University Medical Center, New York, NY 10032, USA. E-mail: or38@columbia.edu