The Social Brain

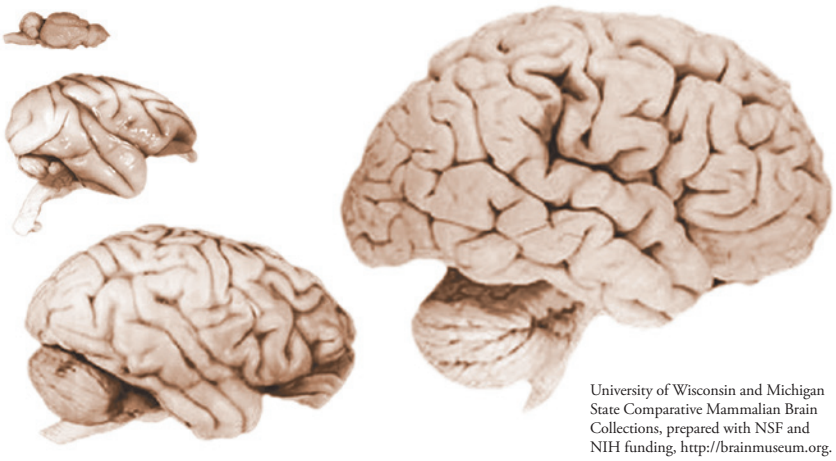
by Ralph Adolphs

The Adolphs lab uses an eye tracker to find out how people look at each other's faces in normal social situations. Caltech may be the only place on the planet where such headgear might actually work as a pickup line. "What's that you're wearing?" "My eye tracker." "Cool!" Tiny cameras at cheek level record the subject's eye movements and send the data to a laptop. The couple in the Rathskeller bar are grad students Jessica Edwards and Dirk Neumann, while postdoc Nao Tsuchiya serves drinks, and undergrad Sam Huang works the laptop.

I'm studying how our brains generate emotions and guide our social behavior. To give you an idea of the kinds of questions my lab is working on, consider the talk that I'm giving to you right now. As I stand at the front of the auditorium, I look at your faces and imagine what you are thinking about me. Are you interested? Are you bored? At the same time, you in the audience are looking at me and wondering how I feel as I'm standing here. Am I nervous? Am I happy? Now, what is very interesting is that none of the answers to these questions are known objectively. It's not like asking what color shirt I'm wearing. You can't see what's going on in my mind, and I can't see what's going on in yours—yet we manage to attribute thoughts and feelings in both cases, and we do so easily and automatically. How do our brains do this?

One feature of essentially any psychiatric disease is an inability to interact appropriately with other people. Some diseases—autism, for example—have this precise dysfunction as their main feature. But I'm also interested in the *normal* processing of social information. Do men and women process social information differently? Do young and old people? What comes into play when we interact in groups? What social factors drive the stock market? And what makes us elect a particular political candidate? By investigating the mechanisms by which the brain processes information about other people, we will gain some insight into questions such as these, and at the same time contribute toward understanding illnesses such as autism.

Let's begin with a brief review of the brains, social behaviors, and success as a species of different mammals in order to highlight what is so unusual about us humans. Primitive mammals, such as European hedgehogs, have a relatively simple repertoire of social behaviors, without a complex social hierarchy. Their brains are very small, weigh only three grams or so, and don't have many folds, which means that there's not much room for many



University of Wisconsin and Michigan State Comparative Mammalian Brain Collections, prepared with NSF and NIH funding, http://brainmuseum.org.

The brains above, in order of size and complexity of folding, are those of a hedgehog, a macaque monkey, a chimpanzee, and a human.

brain cells. Hedgehogs are only found in parts of Europe—outside of pet stores, that is.

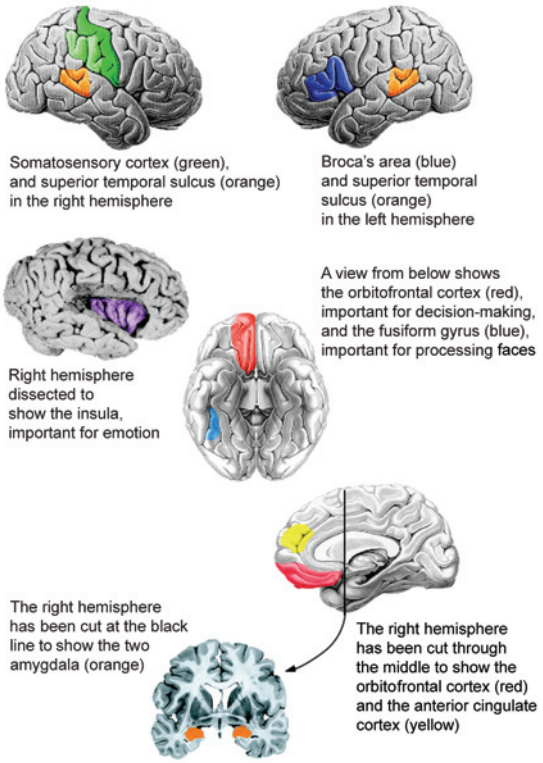
The social behavior of macaque monkeys is substantially more complicated. They live in groups with a hierarchy, and can derive socially relevant information from looking at one another's faces and gestures. Their brains weigh about 100 grams and have a larger surface-area-to-volume ratio than those of the hedgehog, so they have a lot more brain cells. But macaques still haven't done all that well in the global scheme of things, and their range in the wild is relatively restricted.

Our closest living relatives, the chimpanzees, have very complex social behaviors. Like us, they can go to war, and they can make peace. And they also have the precursors to many social emotions, such as shame and guilt. Their convoluted, complex brains weigh around 400 grams, which is about the brain weight that our hominid ancestors likely had four million years ago, and the large surface-area-to-volume ratio means there's a lot of cortex and, consequently, a lot of processing power. Nevertheless, apes haven't done all that well globally, and it's very likely that many—if not all—of them are going to become extinct in my lifetime.

Then we have *Homo sapiens*. Our habitat is the entire planet; in the last 30,000 years or so, especially in the last several hundred, we've taken it over and transformed it. Our brains weigh about 1,300 grams (slightly more, on average, in men than in women), and are even more convoluted than those of the chimpanzee, which means that we have a lot of brain cells packed into the cortex. Our social behavior, as you know from first-hand experience, is very, very complex. We live in huge groups, in institutions, and in countries—in fact, we now have a global society. And because we have culture, we can store and transmit a gigantic amount of knowledge.

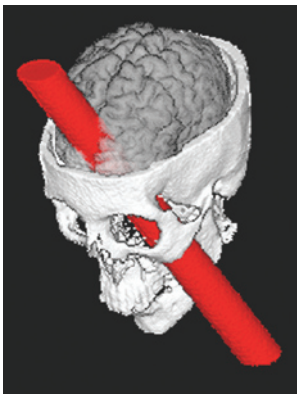
What makes our minds so different from those of any other species that we can generate such complex social structures? One thing that we can do much better than other animals is to think flexibly and abstractly. In particular, we can think of things that are not the case. We can imagine unicorns and dragons, we can recollect things from far in the past, and we can plan years, even decades, into the future. Thus we can adopt points of view that are outside the current context. We can also adopt the point of view of another person and, by imagining what it would be like to be that person, we can empathize with and understand him or her. It is likely that no other animal can do this to the same extent humans can (though apes show some of the precursors of this ability.)

While increase in overall brain size may well be correlated with our complex social adaptations, there is also good evidence that specific regions, shown below, are specialized to process information about other people. Some are involved in language, whose basic social function is, I think, to create a shared consensual point of view between people. But the two main structures my lab is studying are the orbitofrontal cortex, which is located at the base of the frontal lobes, right behind the eyes, and the amygdala, a small structure deep within the brain. These two seem to integrate cognition and emotion, linking what we see in the outside world to an emotional response to it.



The above views of the brain show areas involved in language processing, social perception, decision making, and emotion.

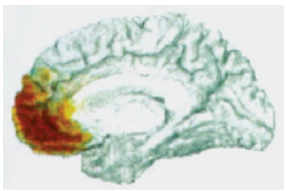
Warren Anatomical Museum, Francis A. Countway Library of Medicine



Damasio *et al.*, *Science* (1994), 264, 1102—1104.

Right: Phineas Gage's life mask and skull are preserved in a Harvard museum, along with the tamping iron that shot through his head (not shown). Far right: In 1994, a team led by Antonio and Hanna Damasio used these exhibits to compute the route the rod must have taken through Gage's brain. They found that it had destroyed his orbitofrontal region.

The role of the orbitofrontal cortex was a mystery until a gruesome accident to a man named Phineas Gage provided the first insight. In 1848, Gage, a railway construction gang foreman, was laying track for the Rutland & Burlington Railroad near Cavendish, Vermont. He had drilled a hole into the rock, put gunpowder in it, and was tamping down the powder with a large metal rod when he accidentally struck a spark, and the gunpowder exploded. The tamping rod shot straight through his head and landed many yards away. Amazingly, Gage survived this severe accident, and lived for many more years. But, although he still seemed to have normal intelligence, could speak, and had a good memory, he had completely changed as a person. Prior to the accident, he was a very polite and diligent young man who cared about other people, had lots of friends, and held down a good job. After the accident, all of this changed. Gage didn't care about people any more, he didn't regulate his emotional responses to them, and it didn't bother him what people thought. He became very rude and profane, and soon lost his job and all his friends.



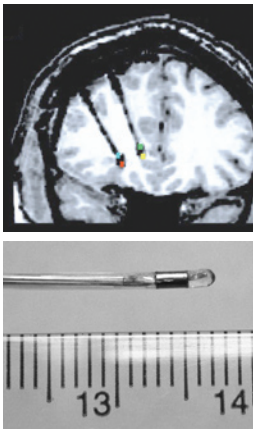
After frontal brain-tumor surgery, some people are left with damage to the regions highlighted on the right. The orbitofrontal cortex is the very bottom part of this area.

One of the areas of the brain damaged in Gage's accident was the orbitofrontal cortex. As a postdoctoral fellow at the University of Iowa working with neurologist Antonio Damasio, I was able to observe patients with similar damage, usually due to the surgical removal of a brain tumor. Like Gage, these patients develop something that's been dubbed "acquired sociopathy." They perform normally on IQ tests, and have normal language, memory, and perception, but are unable to guide their behavior with respect to other people. They can't make decisions that are in their best interests, typically fail to hold a job, and are unable to maintain lasting social relationships.

To study what is going on in the brains of these patients, Dan Tranel of the University of Iowa showed them a series of images that varied in terms of their emotional content, and recorded the patients' emotional response by measuring changes in skin conductance of the palms of their hands. Some of the pictures they looked at were neutral, like landscapes or chairs, some were pleasant, like puppies or babies, and some were highly aversive images of mutilation, disease, or war.

The results were striking. In healthy control subjects, Dan had measured a large response specifically to the emotionally arousing pictures, but in the patients with orbitofrontal cortex damage, there was no emotional response at all. When asked to describe what they saw in the aversive pictures, the patients said things like "It looks like a gunshot wound" or "There's a lot of blood." But when asked how they felt when they were looking at these pictures, they said they didn't feel anything.

To get a more detailed look at what the orbitofrontal cortex does, I teamed up with neurosurgeon Matt Howard and postdoctoral fellow Hiroto Kawasaki. We began experiments in patients with epilepsy who had had electrodes implanted in this region. This allowed us to record the activity of *single* brain cells when the patients looked at the images. Let me stress that we hadn't implanted the electrodes for our research—a surgeon had implanted them in order to figure out where the epileptic seizures were originating, so that he could remove that part of the brain. While these patients were being monitored, often over a week or two, they had to lie in their hospital beds with all the electrodes embedded, so we asked them if they would like to participate in our research. If they agreed, we recorded the electrical responses of their neurons while they looked at our pictures on a screen.



Kawasaki *et al.*, *J. Cognitive Neurosci.*, 2005, 17:20, 1509.

Above: The top image is a brain scan that shows two electrodes embedded in a patient's orbitofrontal cortex to record the activity of single neurons. The image below that is a magnified view of one of these electrodes (the scale is in centimeters). Two tiny metal contacts on either side of the large silver contact on the electrode record the neuronal activity.

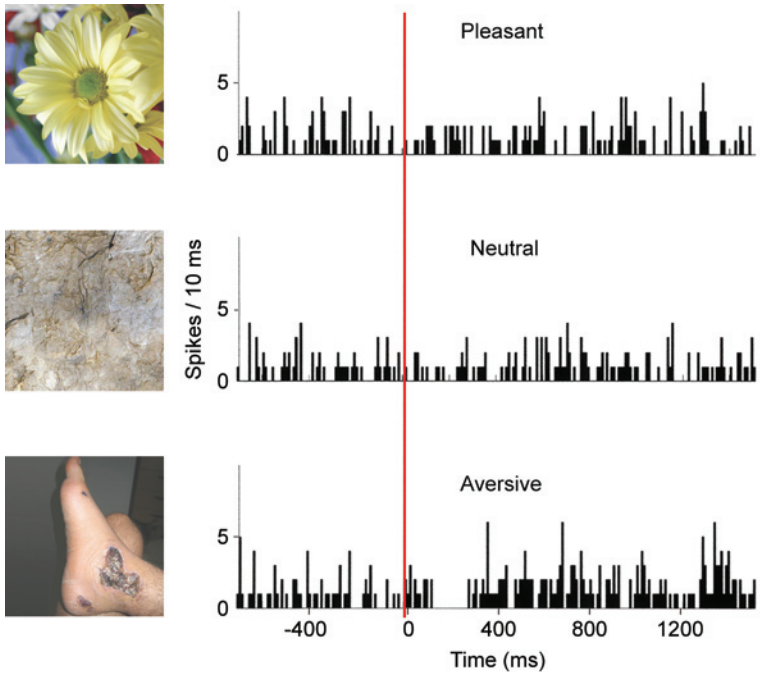
Our results are shown in the graph below, in which the little vertical bars indicate the firing of individual neurons, while the horizontal axis represents time and the red line indicates the point when we showed the subject the picture. When we put a pleasant picture on the screen, the neurons pretty much kept firing in the same way that they had been. The same thing happened with a neutral picture. But when we flashed up an aversive image, we saw, after a short delay, a cessation of firing followed by a prolonged increase in the firing rate. So even *single* neurons in this region of the brain can encode information about the emotions signaled by the stimuli and, moreover, they can do so very rapidly—the time between seeing the picture and the start of rapid firing was about 120 milliseconds. We checked that this effect was not just due to simple visual differences—for example, we made sure the aversive pictures were not simply brighter, or larger, or had more of a certain color in them.

Apart from suffering from severe epilepsy, these patients were normal. But we were also able to do this experiment on a patient whose orbitofrontal cortex had been partially lesioned, and found that the disturbing images had no effect on the firing rate of individual neurons (not shown). Again, this patient failed to be affected emotionally by what she saw.

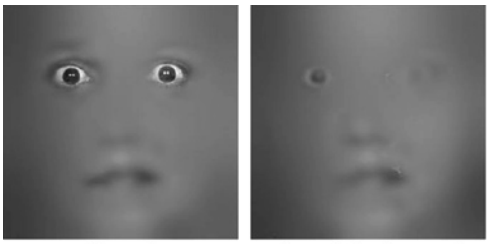
The amygdala is connected to the orbitofrontal cortex, and in many ways serves a similar function. "Amygdala" is Greek for almond, and there are two of these almond-shaped structures inside the brain on either side of the midline. We've been studying a 40-year-old woman who has a very rare genetic disease that results in lesions of the amygdala because of calcification. Patients with this type of lesion can show a selective deficit when they look at the faces of other people. They're unable to recognize one single, specific, emotion—fear.

In order to work out the mechanism behind this,

Kawasaki *et al.*, *Nature Neuroscience*, 2001, 4, 15.



While a patient looked at photos of varying emotional content, recordings were made from a single neuron in the orbitofrontal cortex. The red line marks the point at which the photo was shown. There was no change in neuron activity when pleasant or neutral images were shown, but an aversive image evoked a brief lull in brain cell activity, followed by a burst of firing.



Adolphs *et al.*, *Nature* (2005) 433: 68—72.

Normal people shown only parts of photographs of fearful faces used information from the eyes and mouth to detect the look of fear (left image), whereas a woman with nonfunctioning amygdala used much less information from the eyes (right image).

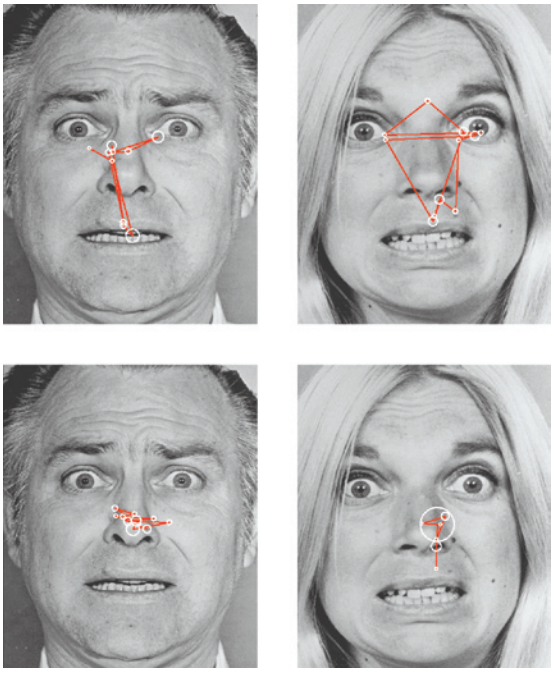
we showed faces that looked either fearful or happy to a wide range of normal people, and asked them to push a button to tell us which of these expressions each face had. But instead of showing the whole face, we made the task much more difficult by manipulating the image so that only little bits were visible at one time. Sometimes the subjects saw an eye, or a nose, or a piece of the mouth, or an ear, and they had to judge happiness or fear from that. After showing thousands of images to a large number of people, we found that some bits are, indeed, more helpful than others. Part of the ear doesn't help, because the ear looks exactly the same whether someone is fearful or happy. A little bit of the mouth is more helpful, and the nose less so. When we put together all the pieces for which people were able to say correctly that it was fear, and subtracted all the bits for which they were not able to recognize fear, it revealed that normal people discriminated fear from happiness mainly by using information about the eyes. Big, staring eyes show fear.

The patient with amygdala damage was strikingly different. Like normal people, she made use of the mouth and nose to some extent, but she failed to make normal use of the eye region. We concluded, therefore, that this patient was impaired in recognizing fear because her brain was unable to use information about the eyes in other people's faces.

Now one explanation that might have occurred to you is that perhaps people with amygdala damage never look at the eyes in the first place. We investigated that possibility by using an eye tracker, which is basically a little video camera that measures with great precision where someone is looking when viewing a picture of a face. Humans make about three or four fast eye movements, called saccades, per second, and normal people, when viewing a face, often sweep it in a triangular arrangement, looking a lot at both the eyes and making frequent excursions down to the mouth. The patient with the lesioned amygdala didn't do that; she just stared at the face and didn't explore it at all. In particular, she didn't look at the eyes. So it seems that people with damage to this part of the brain don't spontaneously look at the eyes—and without the information about the eyes, they're impaired in recognizing fear.

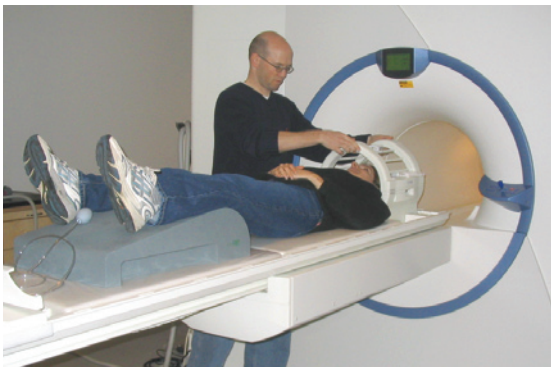
We wondered what would happen once this patient was told to look at the eyes, so we ran the experiment again after instructing her to do this, and found that her performance became completely normal. In this simple way, we were able to "rescue" her impaired fear recognition—in essence, we had instructed her to do something consciously that her amygdala would normally have instructed her to do unconsciously.

We are now pursuing many other lines of investigation with this subject, and with others who have similar damage to the amygdala. This past January, for example, we began brain-imaging studies where we again showed pictures of faces and measured the eye movements, only this time we did so while the subjects were lying inside Caltech's new magnetic resonance scanner in the basement of the Broad Center. In this way, we could see what was going on in their brains while they were looking at the faces. Such research is showing us how the *rest* of the brain changes when a small part of it—in this case, the amygdala—is damaged. Some of the changes reflect impaired functioning, since the



Adolphs *et al.*, *Nature* (2005) 433: 68—72.

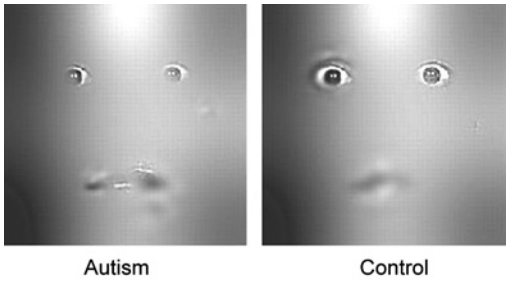
How normal people scan faces is shown in the top row, while the bottom row shows how an amygdala-damaged woman goes about it. The white circles denote the areas at which viewers stare, or fixate; the bigger the circle, the longer they fixate. The red lines represent the eye movements between fixations.



Magnetic resonance technology manager Steve Flaherty prepares a subject for a brain scan in the state-of-the-art Siemens Trio 3 Tesla whole-body scanner housed in Caltech's Brain Imaging Center.

brain is no longer getting normal input from the amygdala, but some changes are compensatory—the rest of the brain can make up, to some degree, for the damage.

In a collaboration with Joe Piven at the University of North Carolina, we're also studying people with high-functioning autism and Asperger's syndrome. These are people who have a clinical diagnosis of autism, but have normal IQs. With funding from the Cure Autism Now Foundation, postdoc Michael Spezio and graduate student Dirk Neumann have been exploring how such people look at faces. When we asked them to detect happiness or fear from bits of faces, and compared the results with those from a control group of healthy subjects with similar IQs, we found that the people with autism made less use of information about the eyes, and somewhat more use of information about the mouth, as shown below. It could be that they're compensating by making more than normal use of the mouth. We hope to extend these studies to see if we can do the kind of intervention that we did with the amygdala patient. Can we change their social cognitive abilities if we instruct them how to look at other people's faces?

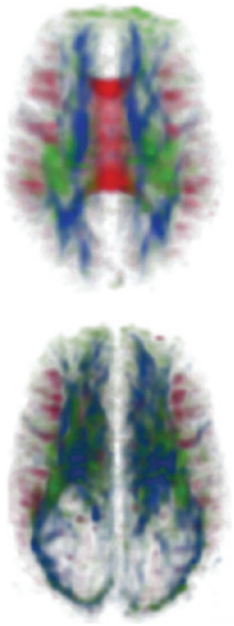


Above: Autistic people, when asked to detect fear in faces, used some information from the eyes and some from the mouth (left), while normal viewers used a lot more information from the eyes and less from the mouth (right).

Joe and I are extending these studies to the parents of people with autism. Given that autism is highly heritable, we believe that it has a substantial genetic component. Do the parents already have some of these abnormal genes, and do they show subtle differences in how they process faces?

We're also studying people born without a connection between the left and right halves of their brain, a line of research made possible by a grant from the Pfeiffer Foundation. The two hemispheres are usually connected by a big bundle of about 200 million or so nerve fibers, or axons, called the corpus callosum, but in these people, it is missing. Using the magnetic resonance scanner, staff member Lynn Paul is taking scans of these unusual brains to get detailed information about their structure with a technique called diffusion tensor imaging that allows us to see in which direction the axons run. Lynn has found that the millions of axons that would normally have crossed the midline to the other hemisphere have grown in a fore and aft direction within each hemisphere. As a result, the cells in each hemisphere may actually be more densely interconnected than in the brains of normal people.

If you ask these patients what their main difficulty in life is, they'll tell you it's social—they can't understand other people's emotions. One thing they have great difficulty with is getting jokes. This makes sense: The left hemisphere processes language-based information, such as reading the joke, while the right hemisphere processes emotional information. The humor in a joke often arises from the mismatch of the verbal and emotional components, so if there's no communication between the two hemispheres, the person can't "see"



Right: In these diffusion tensor images of brains viewed from above, the front of the brain is toward the top of the page. Axons that run from left to right are colored red, and axons that run from front to back are blue. In the top, normal, brain, the corpus callosum is the mass of red axons bridging the hemispheres. The bottom brain, that of a person born without a corpus callosum, lacks this red bridge, but has more blue axons within each hemisphere.

the joke. For the same reason, puns and metaphors are also hard for them to understand. In fact, their impairments in many respects resemble those seen in people with high-functioning autism.

Taken together, these studies will give us a better understanding of how humans behave socially, both in health and in disease. Our findings mesh nicely with single-neuron recordings in animals, where investigators have also found that the orbitofrontal cortex and amygdala play a role in emotion and social processing.

We are now extending our studies to the "real" world. Last summer, two SURF (Summer Undergraduate Research Fellowships) students, Sam Huang and Lisa Lyons, initiated a study in which a subject wore an eye tracker while interacting with another person in a social situation. In this way, we were able to measure how we look at other people in actual face-to-face conversations rather than as faces in a photograph.

We're also trying to study the differences in social judgments made by men and women. Graduate student Jessica Edwards and SURF student Jessica Stockburger have been looking at how we make moral judgments regarding how "right" or "wrong" an action is, focusing on actions that in some way involve cheating on a partner or spouse. The question they asked was: Would men think that it is more "wrong" for women to cheat on their husbands than for men to cheat on their wives? To do this, they took actual moral memories that real people had produced, where they remembered having had an affair with someone else—having cheated on their spouse. Jessica Stockburger took these real-life stories and used both the original versions and also another version that she had made, which switched the genders in the story but kept everything else the same. She then presented these to both men and women and asked them to rate how right or wrong they thought the action was.

As was predicted, for at least some of these stories, they found that men think it is much worse if a woman cheats on her husband or boyfriend than if a man cheats on his wife or girlfriend, even though the actions and details were identical in the two cases. There were also some converse effects for women readers. This study is one example, among several, that also illustrates the rich cross-talk between different disciplines. In this study, we have biologists, psychologists, and philosophers all collaborating to figure out how moral judgments are made.

I hope that the data from all these studies will give us a better picture of how we think about other people. When we interact, what goes on in our minds and in our brains? How do these processes break down in diseases such as autism? And what does it imply about the things that set us apart from other animals? Or, looking at it the other way around, what does it imply about how similar our brains and minds are to those of other animals? If we understand these issues better, we will end up understanding ourselves better. □

A camping trip to one of the Channel islands allowed the Adolphs group to interact socially; Adolphs is sixth from the left. Check out the rest of the group on the lab's website, www.emotion.caltech.edu.

Ralph Adolphs holds a joint appointment as the Bren Professor of Psychology and Neuroscience and professor of biology. He was born in Germany, raised in Canada, and educated in the United States, gaining a bachelor's degree from Stanford in 1986, and a PhD from Caltech in 1992. His graduate work with Mark Konishi, the Bing Professor of Behavioral Biology, was on the auditory brainstem of the owl, but he moved on to the human brain when he joined the University of Iowa as a postdoc of cognitive neuropsychologist Antonio Damasio. Adolphs became an assistant professor at Iowa in 1997, and an associate professor in 2003. He joined Caltech in January 2004 as a half-time professor of psychology and neuroscience, but continued at Iowa for another year to complete his research on patients at the university's medical school. In January 2005, he joined Caltech full time. Adolphs gained a Klingenstein Award in the Neurosciences in 2000, a McDonnell Foundation 21st Century Science Award in 2002, and a National Alliance for Research on Schizophrenia and Depression (NARSAD) Distinguished Investigator Award in 2005.

This article is based on a Seminar Day talk given in May 2005.