The following content is provided under a Creative Commons license. Your support will help MIT OpenCourseWare continue to offer high-quality educational resources for free. To make a donation or view additional materials from hundreds of MIT courses, visit MIT OpenCourseWare at ocw.mit.edu.

**PROFESSOR:** This is the first class since the midterm, but the chemical senses I want you just to read. And I posted questions, I hope. I think I remember posting some questions.

My plan is not to give a quiz today and to come up with some homework. I think I should include something on chemical senses, particularly the olfactory system, but I hadn't settled on exactly what to do yet. So I'll get that done tonight or tomorrow. Because I'd like to spend the next three classes on the visual system, and then two classes on the auditory system.

If you bring your books to the class, I guess it's a little heavy to lug around, but I want to give you-- unfortunately, I've found small errors, one just a typo, in the auditory system chapter. It's a long chapter, so that's why we need two classes. So I'll specify those to you.

It's inevitable, they told me, at the MIT press, that when you publish a book this big, there's always a few things like that that you find out. So they will fix them in the next printing if there is one. I hope there will be.

So where are we now in the outline of this class? We've already done seven parts, ending with the brain states, which is the only part with only one chapter. And so now with sensory systems, readings only for gustatory and olfactory, and now three classes on the visual systems. And notice I used that in the plural, because there's multiple systems for vision. Some of them operate pretty much independent of the others.

So we'll start that today. We'll start with a little bit on the origins of vision, with light detection. And then finally, image formation and the major functions it serves-- predatory escape, which we've been talking about before, orienting towards objects, and then identifying patterns and objects and individuals.

And then I want to start going over the retinal projections in more detail. And next time, we'll look at that in a different way in a little more detail, because I'd like you to be able to remember some of that.

You guys have pretty good memories. Just a little aside here-- I was pleased with the results of the midterm. You figure that 85 to 100 is an A, there were a lot of As. And Bs go all the way down to 70, so that means nobody got below a B. All As and Bs. So I should be clapping up here for you. So keep it up. Keep working hard, and you'll all get As and Bs for sure.

All right. So what in all likelihood was the first functional role of the visual sense? And I want you to be able to describe the nature of the most primitive projection of the eyes to the brain. And I point out here that it was actually the last of the major retinal projections to be discovered. Even after this one was discovered, there's been a few more, and I'll point some of that out in today's class.

So what do I claim is the most primitive function of vision? It was there, even in amphioxus. Sorry?

AUDIENCE: Avoidance of predators?

**PROFESSOR:** Predators? No.

AUDIENCE: [INAUDIBLE].

**PROFESSOR:** Well, the pineal eye is one of the ways this was expressed, that's right-- just detection of light. Think of the most primitive chordate we talked about, amphioxus. He doesn't have a lens. But he can detect light. I'll show you that again today. So we think that's got to be the most primitive sense. So then we look at, well, what are the pathways in the mammal that just do that? OK, so we'll look at that here, two of them.

Why was it so important to detect light? It long preceded image formation and topographic projections. It took a long time to evolve a lens. And we don't know all the details of how that happened.

So that means the only topography in primitive, bilaterally symmetric animals, the only topography was they had a light detector on the right and a light detector on the left. They could see differences in light intensity on the right and left sides. So if some animal was approaching them, attacking them, they might be able to detect, oh, there's something over there. So I need to go the other way.

So we know this is just on the primitive origins that we've gone over before. It was very adaptive for organizing the night and day for efficiency and for safety. If you're a day-active animal, you need a place to get away from other animals at night in order to sleep. If you're a night-active animal, you need a place to sleep during the day. You're inactive. But then if you use vision for predation, you have to have vision in very low lights, and you're more likely to use other senses in addition to vision that might be even more important. Those would be olfactory and auditory. Somatosensory less so, but somatosensory does play an important role in predation too in attack behavior. We'll talk a little more about that later on.

OK. So this is just a reminder. This is amphioxus, the rostralmost end of amphioxus. You find cells with pigment in them. But more importantly, right next to the pigment cells, there are cells that are light receptors. And next to them are cells that have axons that project to other parts of the nervous system of amphioxus.

And you can compare the arrangement of cells in that light spot in amphioxus to the cells in the developing retina. Normally the retina develops as sort of an out-pouching of the sides of the diencephalon. What causes that out-pouching to occur? It's actually induced by things at the surface. Placode, in this case, the lens placode.

And that causes the neural tube to extend out towards the surface. So here you see that. And you see pigmented cells and the cells of the central nervous system here, including the retinal ganglion cells, bipolar cells, and the visual receptor cells.

And this moves back here, so the receptors are actually in the back. The light comes in. It forms a cup-like arrangement. The light comes in this way, goes through the receptors. But they're very transparent. They don't cause much distortion. Seems like a strange arrangement, but that's the way it evolved. So now this is just another representation of the same thing, where they compare a primitive vertebrate with the amphioxus. And I pointed out before that we know from gene expression data now that even amphioxus does have a hindbrain, a midbrain, and a forebrain, but its forebrain is only a diencephalon.

OK. You want to behave differently in day and night. But what if you're spending time underground? Or what if the light is very dim? You don't get the normal signals. Well, it's an amazing thing that evolved that supplements that. You don't need just light, which can vary a lot. You determine your activity by an endogenous process, the cells that have a circadian rhythm of activity. But they are normally every day, they're affected by the light-dark cycle. So we say they're entrained by the light-dark cycle. So they oscillate at 24 hours instead of 24 hours and 20 minutes, or 30 minutes, or whatever--sometimes as long as 25 hours, including in humans, sometimes a little less than 24 is their natural rhythm, but they're all of them, no matter what the natural rhythm of those cells is, it's entrained to the light-dark cycle, normally the light onset.

That's why if you have trouble-- if you're circadian rhythm drifting, you can mess about at MIT by staying up all night and not-- it can be a problem. And one way to help with that is to exposure yourself to bright light in the morning, especially sunlight, especially effective. Some people like to sleep by open windows because of that. And if you're an MIT student, you probably hate to sleep by an open window. You want to be able to sleep in.

So the only thing that's been found that you can counter some of the problems that can cause is to take low doses of melatonin. And I'll say a little more about why I said low doses of melatonin.

So light controls the daily rhythm of secretion of melatonin. It controls daily rhythm of secretion, actually, of a number of things. But one of them is melatonin. It's secreted by the pineal gland. So how does light effect that rhythm of secretion. You can say that, well, it's just by the endogenous rhythm. But actually, no, it's affected by light.

And there's two different pathways in different animals. One is very ancient that you mentioned at the beginning of the class-- the oldest pathway to affect secretion of melatonin. What was it? The pineal eye. A number of animals have that eye.

A number of fish have it, some amphibians have it, and I think even some reptiles. It's an eye on the top of the head, a third eye. It's led to all kinds of fantasies in science fiction, everything else, but all it is a light detector that affects the pineal, it has close connections to the pineal.

What about the other pathway? We don't have a pineal eye. We've got a thick skull. Now, if you're a mouse, the skull is thin, and some light can get right through that skull and affect the pineal. And yes, there are cells that are sensitive to that light coming right through the skull. But a lot of vertebrates have a thicker skull, like us. So even the rat's got a pretty thick skull. So light can't really get through-- or at least, very, very little.

So let's talk about that pathway. But first, let me show you the mammalian suprachiasmatic nucleus, where the cells have this endogenous circadian rhythm. It means circa, dium-- about a day in length, because it varies a little bit. And then we'll talk about the pineal gland and melatonin production, the pathway.

So this is from one of the studies that I did with a postdoc in my lab. We were using a method that's very sensitive compared to earlier methods for tracing pathways in the central nervous system. And here we were tracing them from the retinal ganglion cells. This is the low power picture at the bottom.

So what it's showing here is the suprachiasmatic nucleus and the number of nuclei-- that's what SCN is, suprachiasmatic nucleus. But notice that it spills outside that and goes to these other areas around it as well. In fact, there's even a few axons, to our surprise, went all the way into the olfactory areas of cortex. None of that was known before these methods got so sensitive-- so sensitive that we could trace individual axons. They look like they were stained with the Golgi method, except we get them all, all the retinal ganglion cell axons. And then these are enlargements. The suprachiasmatic nucleus would be here. And here's a sagittal view. This would be suprachiasmatic. But you can see, they're in the anterior hypothalamic nucleus. They're in the nucleus right above, even a little bit in the ventromedial.

If you look at this slide, it's from a rat. And here's the frontal section. This is a Nissl stain, so you see how clear that suprachiasmatic nucleus is. These are the axons down here. They're not stained in this, like they were here. These are the axons at the bottom here. There you'll see them in the chart and here in the photograph. So they're down here.

So we know then, the retina projects heavily-- oh, one other thing I left it. Notice that it's going to both sides. I labeled the axons from only one eye. But they're going pretty much equally to the two sides. And that's true in all the animals that have been studied.

This you don't need to pay attention to. It's looking at a more specific stain for a particular peptide that is found in some of the neurons of the suprachiasmatic nucleus. They've found that they can actually subdivide that nucleus into a number of different parts that do have slightly different projections, and we believe, probably different functions. Some of that is still not fully known.

But look at this complicated pathway that influences of light on the retinal ganglion cells take in order to reach the pineal. Here they go to the suprachiasmatic, and short axons go from there to a more dorsally located hypothalamic nucleus. Paraventricular hypothalamic nucleus-- that's what PDH means.

And there are long axon from that nucleus that connect to the sympathetic nervous system by axons that go right down to the lateral horn in the upper thoracic cord. Remember, the lateral horn cells are the preganglionic motor neurons of the sympathetic nervous system. They're found all the way from T1 down to L2 or L3.

So they get the influence of light. They're activated. Here the axon goes out into the ganglion. SCG means the superior cervical ganglia. It's part of that chain of sympathetic ganglia on either side of the spinal cord. Superior cervical ganglia is right up here. It's way up in your neck. That's where the cervical cord is, of course, below the skull, uppermost spinal cord.

And axons from the superior cervical ganglia provide the sympathetic innervation of the head region. So some of those axons-- now, remember, axons are going to all over from the head. They go to the iris of the eye, for example, and cause pupillary dilation with sympathetic activation.

But this is just one pathway here that goes directly to the pineal gland. And that is how light controls the daily rhythm of secretion of melatonin.

OK. So how about taking melatonin pills? It's sort of popular and all kinds of beliefs about it. Unfortunately, they're not produced by the drug industry. They're produced by various companies that aren't regulated by the Food and Drug Administration. Therefore, the government, NIH, has no say over what they can put in the pills.

And people have this tendency, and I'm sure you've noticed it in yourself, that if a little bit is good, more is better. So people take-- they see these things on the shell, oh, 1 milligram of melatonin, two, three, five, 10. They buy the 10. Here's what happens. You take those big pills, you swamp the receptors, you totally saturate them, and mess up their normal secretion. So you can end up having more problems. So people aren't helping themselves, but they don't know it. They're so convinced that more is better.

So if you want to take melatonin, get the 300 microgram pills, the low-dose melatonin pills. And I've taken them when I go on trips, and it helps me recover from jet lag quite rapidly. So if I go to China, I'm 12 hours off, I take the melatonin pills 20 minutes or 15 minutes before I go to bed. And within a couple days, I'm on Chinese time. Without that, it takes five or six days before I'm fully on-- and even then, I get sleepy at odd times.

So that's just a bonus. I probably didn't put that in the book. I'm sure I didn't. I probably should. But not enough people will read it to make much of a difference.

All right. Second main function of vision, of course, involves image formation, animals with a lens. And I've included the function of predator avoidance and escape here, even though probably before there were lenses, even when animals just could detect something different on the right and left sides of [INAUDIBLE]. But that's the most primitive kind of image you can get.

It's also very useful for detecting novelty. If you can discriminate differences-- if you can use those images to see differences in patterns, differences in shapes, you can detect novelty. Novelty is critically important, because if something is different, it could be dangerous, especially if you're a preyed upon animal. Hence, our visual system is highly attuned to detecting novelty and responding to it.

So next in importance, orienting towards novel objects to explore them. So there's a dual role of novelty there. We have a tendency to explore them. We have a tendency to be afraid of them. So it depends on how novel they are, and the situation, of course. But obviously we orient in order to find food, find potential mates or rivals, and so forth.

And finally, identifying animals and objects, which when we think of vision, that's normally what we're thinking about, not these other functions. It doesn't mean humans don't have them. We do.

All right. So now I want you to recall the hypothesis, using Darwinian logic, concerning the evolution of predominantly crossed representation of visual space. Can any of you remember that when we talked about it before? Let's just go over it.

I claim it was an early evolution of connections that carry information, of things like a sudden appearance of shadows on one side, or triggering escape movements. The most direct, relevant connection from the eye to the escape mechanism for turning away was a cross projection.

And this is the way I represent it. These are the pictures. Here I put the spinoreticular pathway. I discovered this morning that I had this reversed, so I did a mirror image and changed the words. That's why spinoreticular I think in the book is on the left.

But anyway, here's the spinoreticular. It's an ipsilateral pathway, but to a lesser degree, it's bilateral. It goes to both sides, but mainly ipsilateral. Trigeminoreticular is similar-- mostly ipsilateral for input from the face, ipsilateral but some fibers going to the other side. Now, in this picture, on the left, here I show the primitive retinal projections. They've just reached the hypothalamus. They're going to that suprachiasmatic, and I show them just as they are now. I don't think there's been any change. A wide range of animals, they've looked at this kind of pathway. It's always like this, bilateral. And I show it the way it is in the rodents and other mammals they've studied. Pretty much equally to the two sides.

And here's the trigeminoreticular. So to escape from something over here, you want the first movement to be turning away, and you want, at almost the same time, you want it to trigger locomotion, rapid locomtion, this. Turning away and swimming rapidly. And that's exactly-- there's been some detailed studies in the sea lamprey, a very primitive vertebrate, at Northeastern University and some other places as well.

And if you look at their-- they take these rapid video images, you'll see this bending of the animal first, and the tail began to lash to propel the animal forward. So because it's a cross pathway that can reach those neurons first, that gave the evolutionary advantage to the cross projections.

And we know that when they eye got bigger, generally, the bigger a structure is, the more connections it forms in development. So some of the axons extended further. Some of them reach structures that can trigger escape movements. And we'll talk in a minute about what those neurons were early on. But the main thing is that it was faster across.

OK. Now, this is a simple picture on that mammalian brain diagram I've used before in the class, just to show you all of the retinal projections in vertebrates. When I say all, I'm not including these very sparse ones that we know exist here around the suprachiasmatic nucleus, and they do exist around other nuclei too. And there's a few that vary a bit from species to species, and they vary even among animals within a species. So I am not talking about those.

These are the ones you keep seeing in animal after animal after animal-- all the vertebrates. Here's the one to the hypothalamus. This is the lateral geniculate body. And you notice that there's two nuclei there. The one we always hear about is the dorsal nucleus. But that's the one that projects it to the visual cortex up here, and I show that pathway.

Then the axons continue. They reach the area in front of the superior colliculus, or optic tectum in most animals. And then they reach the optic tectum, labeled SC here for superior colliculus, because this is a mammal. Pretectal area has a number of different cell groups that have different functions. But we can lump them together.

It's part-- at least, in the way I treat it in my book, it's part of the epithalamus because there's various structural data that say that both the nuclei and the pretectal cell groups and the posterior commissure region are all part of the epithalamus. But modern molecular studies, they vary a lot. But these people tend to say epithalamus is just [INAUDIBLE], and they call the pretectal area something else. But being an anatomist person first and foremost and not a molecular person that skips over the first 500 years of neuroscience, I say that no, it's all epithalamus. Sorry.

OK. So what is the structure that first controlled escape reactions? Well, here the shortest axons are reaching the suprachiasmatic nucleus. And we've talked about tectum being important for escape movements, but look at all these other structures they go through first. So was the tectum really the first?

So I began to look at this nucleus, the first one beyond the suprachiasmatic nucleus if I don't include this little offshoot here. We know quite a bit about the functions of that. It's the accessory optic tract, and we'll hear more about that next time. Those are axons that leave the main optic tract, so let's just stick with the main optic tract and look at that.

What structure first controlled escape reactions? So we know the midbrain tectum with the superior colliculus, is the dominant structure now. I believe that's true even in humans. If you have the instinctive escape reaction to a novel object.

Most of us don't have any memory of experiencing that, but I was a pretty observant little kid, and I remember suddenly-- I used to also think a lot. I thought too much, so sometimes I wasn't paying attention. And I got right in front of a car once, and he was coming pretty fast right at me. I had a sudden, instinctive reaction. I froze, and I could not move the handlebars at all, even though I wanted to. My cognitive system said turn away, get out in front of this.

But I was frozen. Freezing is a typical anti-predator response of young animals, even big ones, like us. I was pretty young. I froze. I could not move. Thank God the guy at the wheel didn't just freeze. He put his brakes on and he stopped. And he didn't hit me.

All right. So that's a tectal response, and that's what animals do. I've studied it in animals. But then they reach the LGV here, the ventral nucleus of the lateral geniculate. And I discovered that it's part of the subthalamus-well, the more medial part of the subthalamus. And there are connections there, from the visual part, that if you stimulate, you can get very rapid running movements.

Everybody's ignored it. Visual people just focus on the popular things-- superior colliculus, lateral geniculate body, visual cortex. But I love these things that people ignore. In evolution, these things are very important.

It turns out, that area of the subthalamus has direct projections to the midbrain locomotor area. That's how the locomotion, the very short latency pathway, is produced-- from this area. But now we know that it's the pathways from the tectum that also have a rapidly conducting ipsilateral pathway to the midbrain locomotor area that has become dominant, because the tectum is so much bigger.

So often something becomes dominant because of its size. It doesn't mean that the ventral lateral geniculate body is important, but it's become overshadowed by the larger structure. They both have connections to that area.

So next, orienting towards novel objects, for exploring, for finding food, for finding a mate, finding a rival, for finding the doorknob so you can get out the door. All right. This is the question I wrote-- contrast the type of visual orienting for which the midbrain tectum or colliculus has become dominant in most species and the visual orienting for which the pretectal area is important. Now, the problem here is that it's only been discovered in a few species-- mainly two. Discovered first in frogs, verified for a mammal. But let's go through these. First, the main type of orienting. You see something here, you turn towards it. You do that with a hamster. Remember, this is, I think, chapter 13, I talked about the regeneration of the optic tract, and I showed you these videos of hamsters turning. All those turning movements are controlled by the tectum. The hamster can't turn at all without the projection from retina to tectum. There is input coming from visual cortex, but it comes in through this same route, from the front of the tectum into those superficial layers.

So the left eye we want to control something's out here, say a sunflower seed. It's mainly a crossed pathway because of that evolution we talked about earlier. So it's going to the right superior colliculus. So to control, turning to the left, he has to contract the muscles, activate motor neurons on the left side of the spinal cord. He does that by the crossed pathway, tectospinal pathway. So to turn left, it crosses once here, crosses again here.

And was the tectum always dominant? You can ask that same question we asked before. I think the LGV was probably important early on, but it clearly is not so dominant now. So it's this tectum that's the major structure for that. Now, whatever it was in very early evolution.

It reaches subthalamus first. Then it goes through the thalamus. But that, the dorsal thalamus, the lateral geniculate body, doesn't have these same projections. It only projects to the endbrain, mainly to the visual cortex.

So let's talk about the pretectal area. And this is an interesting structure, because remember, I said it had multiple cell groups. And they had different functions. We know, for example, tat there's a superficial part of it that projects into the nucleus of the parasympathetic system that then projects to the ciliary ganglion behind the eye, that when that's activated it causes pupillary constriction.

That's the pupillary light reflex. You shine a light in the eye, the pupil constricts. You're in the dark, that is not active, the pupils expand. Or if you get angry, the pupils expand. You bring in more light at the expense of acuity. It's important to get the maximum light.

And if you're talking to a girl or you're talking to a man, you want to know how interested they are in you, watch their pupils. The pupils will get larger if the sympathetic nervous system--- if they really start to get excited, the pupils will expand. If you're a salesperson, there are salespeople that become sensitive to that, and they can tell how interested someone is. And that's important, because they can make a better deal if they can detect pupils.

But anyway, another function discovered here in frog and treeshrew-- very interesting, because it's also a kind of orienting. If these animals are running along, and there are barriers, there are poles or sticks or trees in front of them, they sidestep them. That's a kind of orienting. They're sidestepping the barrier. They're running this way, they go around. That's not controlled by the tectum. It's controlled by the pretectal area, according to some pretty good studies of the frog and the treeshrew.

So similarly, when the animal has to run rapidly into an opening, he uses that system to control his limbs to get his body aligned during locomotion. It's not been studied very much. Certainly deserving a lot more study. And then the one other function that we know about is a vestibular light function that when you start to fall or when you're locomoting and moving your head, there's stimulation of the entire retina by the flow of visual images over the retina. That flow, things that affect the whole retina, are detected by cells in the accessory optic system and in a nucleus of pretectal area, the nucleus of the optic tract. And that has an important function that we'll be talking about after we finish audition.

So I say here, distinguish between two very different functions in the midbrain tectum, each involving a different output pathway. But which of these functions is precise acuity more important? Well, what are the two functions? We've been talking about them.

Orienting towards something and escaping from something. Those are the two functions. We know they have different pathways. The outputs-- and I talk here about the nature of the pathways. Escape from predators involves an ipsilateral pathway from that tectum. Remember, the fibers have already crossed. The retinal fibers have crossed to get to the tectum. But then there's a short ipsilateral pathway to the local motor regions that control the escape reaction. It's that cross pathway that I showed in this diagram, this one. This controls turning towards things. So those are the two functions.

Now, I ask which of those things requires more acuity? And here, I mean the precision of location, how precisely does the animal know in the sense that his brain is detecting the difference, between a thing in the temporal retina here in front of him, or in the temporal field or nasal retina here or two regions close to each other here? Well, it depends on how precise that topography is, right?

We talk about acuity in the visual cortex likewise. And it's basically visual acuity. How fine a difference you can detect in details of the visual field depends on how much brain there is representing that part of the visual field. So we have a lot more brain representing a lot more area in the cortex devoted to the phobia than areas off the phobia. Periphery gets much smaller representation.

But the same thing is true here for the orienting movements, even though the functions are quite different in the midbrain. You still have a kind of acuity. And here I'm asking, what are the two main methods that have been used by neuroscientists to map the topography of the representations of the visual field and retina in the superior colliculus?

How did I get that map? This comes from my work. And this is actually a summary of a lot of work, with two very different methods. What are they? One's anatomical, the other uses electrophysiology. The most common method is to use electrophysiology, to put the electrode in, record from the neuron there in the superficial tectum, and find out where in the visual field the stimulus has to be in order to activate those cells. That's mapping the receptive field of the cells.

And if we're interested in just location, we don't need to worry about the properties of the stimulus. We just have to find usually a little dark spot on the light background. Or it can be a bright spot on the dark background. The dark spots tend to work the best. You can easily map the visual field.

But you can also make-- if you want to see at once the projection throughout the entire optic tract, you can make a lesion in the eye or an injection of a tracer. You've got to label axons from one part of the retina. So here's the retina, here's the optic disc. Let's say you make a lesion here in the retina. The fibers from out here, though, are all damaged too. So the effective lesion is like that when you make a hole in the retina like that. You can then trace the axons from the retina to a corresponding area. You'll find the axons in the superior colliculus to be in an area like that. So it's only by putting together a number of different cases like that you can come up with the same map. But you will then come up with the map simultaneously for the geniculate body, for the tectum, for all the structures of the optic tract. That's the advantage of the anatomical method.

So finally the function of identifying animals and objects. And I point out here that the colliculus of the midbrain and the visual cortex areas are each important for identification of the [? stimulus ?]. I know we say it's always cortex, and I want to explain why that is. Because identification in the tectum and the pretectum too involves what we call innate releasing [INAUDIBLE]. It's innate behavior, instinctive identification.

For example, the best example is the bug detectors in the frog. A small little area in the visual field that's dark and moving around. There are neurons in the frog tectum that respond. And the frog sits there still and [INAUDIBLE], because his neurons are responding to that fly. But he won't snap with his tongue until it stops. The neurons keep firing for a little while. Then his mouth opens, and the tongue zaps the fly. It's an innate mechanism.

Whereas learned identification abilities with greater acuity follow the invasion of the endbrain by the visual pathways to the striatum and the cortex, especially the cortex. And these are just examples. The hamster learns to use visual landmarks to locomote towards its home or to find food and water. He can't just inherit all that. He might have innate preferences. He has innate preferences to go out at a certain time of day and maybe to go to certain places.

But he also has to learn about where the dangerous places are, where the safe places are, where's the water, or where are the fruits that he can get liquid from, and things like that. Where are the grains? Where's the farmer's grain storage that he can go rob and fill his cheek pouches.

And we know that some animals learn to recognize members of their own species as individuals, just by visual differences. So I'm asking here, why? Relatively recently in evolutionary time have these visual identification abilities evolved so much.

And I point out that with these larger topographic maps, when you have precise topography, you then in the projections from that structure, you just have to combine inputs in specific ways to get some kind of shape and pattern recognition. And that has been studied most extensively for visual cortex in work-- initially it was done in the frog by Jerry Lettvin here at MIT, and then it was done at Harvard in the visual cortex of cats and later in monkeys by [INAUDIBLE]. And in your vision classes, you always learn about those studies.

OK. Visual pathways to the endbrain follow multiple roots. I want you to be able to describe more than just the two traditional roots, because everybody knows about two of them. There's actually, like, six, and I describe them all in the book. And I just summarize them here. Tectum and pretectum each project to the older parts of the thalamus, which project to both cortex and striatum. And they also project to the newer parts of the thalamus of mammals. And then the retina projects directly to the thalamus also, mainly geniculate body.

So that gives multiple projections for visual stimuli to get to the endbrain. So that's just summarized here. And this just shows a picture of the projection from a cell in one of those intralaminar nuclei. And I picked this one because it gets input from the colliculus that I've seen in my studies of hamsters. And here it shows the axon going to corpus striatum and to the neocortex. Most of them don't go to both places. They go to one of those two. But some of them actually go to both. And then I just go through the adaptive advantages of this for reaching the striatum, reaching the neocortex, and the cognitive functions in the projections of those areas. And then [INAUDIBLE] to your vision also, which developed cortical projections better than any place.

So you can read the rest of this. I think it's very clear in the book. And we'll do the retinal projections. We'll look at pictures other than this next time to go through that.

I'll post all these slides. I've already posted them. I may add a few notes to them after the class. So now for next time, read that next chapter. You were supposed to read chapter 20, was it, for today? The first vision chapter.

And then, yeah, 19 was the olfactory chapter. So read chapter 21 also for next time. And I'll come up with some homework.

- AUDIENCE: [INAUDIBLE].
- **PROFESSOR:** No, I don't plan on giving a quiz, but I plan on giving you some homework. I don't know. My impression is that you're happy with the homework and you probably learn more. I can give you an assortment of things.