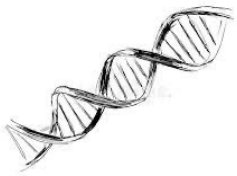
that happened since then has supported the notion of punctuated equilibrium.



Next he moves onto DNA. When we look at a DNA strong, there are periods that code for genes interspliced with large sections (95% or so is non-coding) that function as an "instruction manual."
The genes themselves are not always coded for in just one snippet. Often multiple areas on the DNA will code for parts of the same gene. So you can have a section that codes for the first third of a protein followed by a long stretch that has nothing to do with that protein.
This is then followed by a section coding for the next third. And so on. Each of these sections is called an exon. The in-between stuff is called introns. People deduced and then discovered something called splicing enzymes.

the splicing enzymes would come along & snip out the intron sections so that the first third of the exon connected to the middle 3rd & final third to produce a clear read-out.

this is a massive deviation from the concept that one gene specifies one protine. Diffrent splicing can thus create very diffrent results. the more they goes into the deeper

David Baltimore was the first to introduce the concept that this makes the genes modular and opens the door to massive information within the DNA universe. Because of this flexibility, DNA would then have the potential to abandon the original A-B-C model and create,

for example, an A-C combination. This will give you 7 different ways to combine these exons, which means there are 7 different proteins that can result (pacing mutations of course).

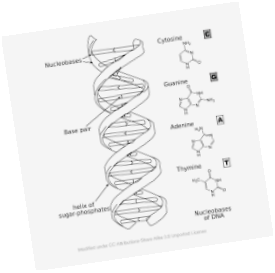
the more they've realised diffrent point of view. diffrent enzyems splicing at diffrent spots. So we have different items being created by the same Basic DNA original set due to diffrent splicing enzymes being activated & activated at diffrent times of life.

The instruction booklet part of DNA is all about when and under what circumstances to activate and start and stop creating proteins.

(For example, human growth hormone is released throughout life but has peak periods.) For better or worse this means that DNA doesn't "know" what it's doing. Instead it's a read-out that's under the control of lots of other factors. Among these are the regulatory sequences upstream from the gene.

These might be called promoter or repressive sequences that promote or repress the expression of DNA snippets downstream. They are like switches. And they are turned on when the right event (internal or external) happens. These events are triggered by transcription factors. These might turn on single genes or whole networks in the DNA.
On the flipside, any given gene can have a whole bunch of different promoters that it's waiting to hear from before it does its thing.

So who is in charge?
=> well, whoever or whatever controls these trascpritionfactor. including the enviornment. which has something to do with genetic effects!



So what qualifies as environment?
=> It could be something inside the cell

For example maybe the cell is getting low on energy. This could release a transcription factor that would result in the cell being activated to take up more energy.

Or it could be something from outside the cell,

such as a hormone floating around in the bloodstream. A hormone is a blood borne chemical messenger.

Testosterone is used as an example. It would float far and wide and have its effects and those effects would increase significantly when the male hits puberty resulting in changes in lots of areas in the body.

You could have massenger outside from body as well. Such as scary sight or an olfactory massenger, like a pheromone.

& as a consequence of this, Sapolsky notes that the most interesting stuff with DNA now is not the specific nature of the protines but rather when it does its thing & what elements trigger it.

DNA is covered, stabilized and protected by chromatin. And so there is a whole world of messengers that inform the chromatin of where and when to open up and allow the transcription factors through. Changes can also happen that will permanently impact the chromatin.

For example, mothering styles in rats have been shown to permanently change elements in the chromatin in areas relating to anxiety. This leads into the field of epigenetics. Research with monkeys has shown a change in one area impacting 4,000 other areas!

So moral of the story — fertilization is all about genetics while development is all about epigenetics.

So if you have a mutation in one of these splicing enzymes or transcription factors, the kind of changes that would result could well fit into the punctuated equilibrium (not gradual) model of evolution.

LECTURE 5

as we know from the previous lacture DNA as the bigboss man is undermine as we learn that 95% of the DNA is simply the instruction manual, this transcprition factor has a huge impact in a if-then manner.

that splicing & epigenetic effects impact growth & on & on. Here he highlights ways in which things are interconneeted- enviroment, gene, etc. &

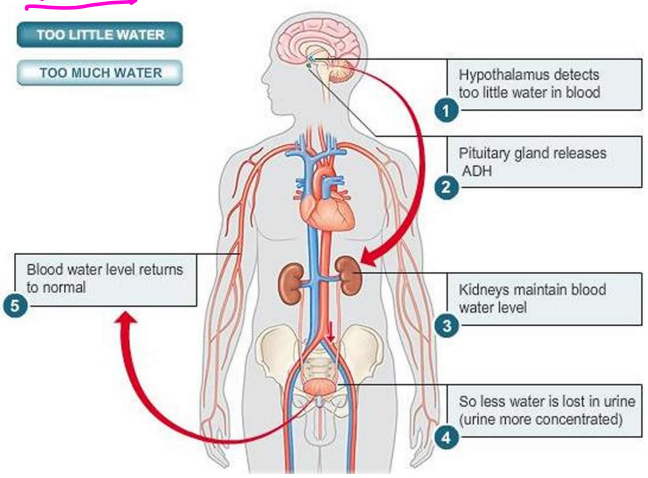
Here come to the most fun part of Sapolsky — Principal element of life in which there is a bit of randomness & chances in even the most structured system.
(chaos theory / Heisenberg Uncertainty)

https://youtu.be/ovJcsL7vyrk

https://youtu.be/TQKELOE9eY4

& there is also a bit of structure in the seemingly chaotic. This might be an important theme in evolution.

ADH



https://youtu.be/u1k5186FJNO

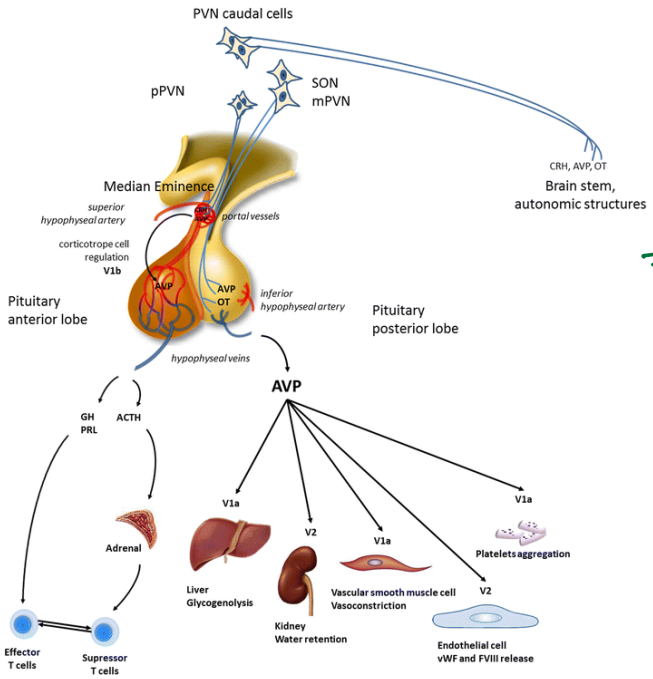
As promoters change, transcription factors change. Splicing enzymes can change their behavior and create entirely new proteins. Changes in transcription factors can activate entirely different gene sequences. Little changes can have big results, especially when those changes cascade.

oneversion of the promotar stimulates release of more vasopressin. coreluted with this is an increase in monogamous

mating.

vasopressin & vols & perhaps humans

↓



— The more vasopressine receptors the more likely the vol is to be monogamous & poligamous vols, when given vassopressin behave monogamously.

⇒ There are some evidence that Impacts human behaviour too. Sapolsky mentions a study that suggested that the type of vasopressin promoter you have provided some predictive power of the likelyhood of you getting divorced down the line.

Naturally there are 3 million confound here, but it gives one pause in terms of the concept of free will.

One the other side of Dinorphin

Promoters seem to relate to ease of addiction to pain killing drugs in rat. The more promoters that the rat is to exhibit addictive behaviour

toward, killing drugs when given the opportunity.

Changing transcription factors changes gene networks. He notes that a disproportionate share of the differences in the genetic code between chimps and humans lie in the genes that code for transcription factors. This leads to the suggestion that the most interesting evolutionary changes are going to be those found in changes in the regulatory structure of the genes, not in changes to the DNA itself.

the more genes you found in a species, the grater the percentage of those genes that code for transcription factors.

for example, you have gene A. you have one transcription factor A

but if you have A, B. you have 3 transcription factor

A, B, AB & so on down the line,

Microevolution is about the protines
Macroevolution is about networks

A PLANT GENETICIST NAMED MARGARET MCCLINTOCK. HE GOES THROUGH A HISTORY OF ONE OF HER EXPERIMENTS IN WHICH SHE ARGUED FOR TRANSPOSABLE GENES IN PLANTS,
I.E. GENES THAT ARE ACTUALLY MOVING AROUND ON THE DNA LINE, CREATING NEW PROTEINS, NETWORKS, RESULTS. THIS AMAZING FEATURE IS ALSO SEEN IN THE HUMAN IMMUNE SYSTEM WHICH ADAPTS ITSELF CONSTANTLY IN ORDER TO COMBAT PATHOGENIC INVADERS (AND SOMETIMES, UNFORTUNATELY, TO COMBAT THINGS LIKE THE INSULIN PRODUCTION CELLS IN THE ISLETS OF LANGERHORN - GIVING THE PERSON TYPE I DIABETES).

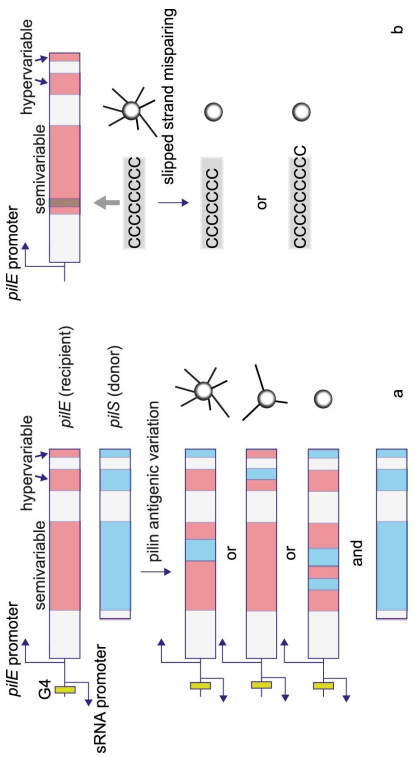
A PLANT CAN'T RUN AWAY FROM TROUBLE, SO IT HAD TO EVOLVE ANOTHER WAY TO HANDLE THE WORLD'S DIFFICULTIES. SO THEY HAVE FANCY STRESS RESPONSE TRICKS, SUCH AS CHANGING GENES AROUND TO HANDLE NEW ENVIRONMENTS AND CHALLENGES.

This is done by activating transposaze, which is splicing enzymes that slices out section of the genes so that they can jump around.

& same kind of gene you found in animal

Predictably, & unfortunately, pathogens also get to utilize this trick.

Trypanosoma brucei is a nasty protazoan that causes sleeping sickness in humans. It invade the body & in order to evade the host's immune response, uses jumping genes to change it's protine coating. So the adaptive immune system stays a step behind cause just as soon as it figures out how to kill the original coating, the trypanosoma has changed it's shild.



The adaptive immune system takes out pathogens in a sort of lock and key function, but if the pathogen changes the locks faster than the immune system can chisel out the keys, you're in for real trouble.

This is called antigenic variation.

In essence the pathogen has numerous shells (for this parasite the estimate is in the thousands) and shuffles through them as it replicates itself. It puts the immune system at a distinct disadvantage.

imagine you are a Detective & you can only

catch your suspect if he's wearing the exact same outfit he had on when he committed the crime.

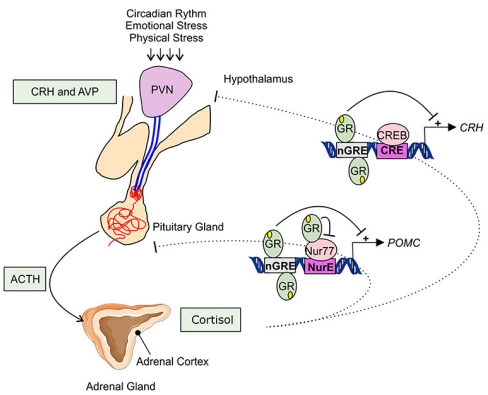
if he has **1 shirt, 1 pair of pants, 1 pair of shoes.** you will catch him immidiately.

if he has —

1 shirt,	1 pair of pants	, 2 pair of shoes	= 2 days
15 "	15 "	5 "	= 1,125 days

this same shit happens in body also. Nuroprogenitor cells can also jump around - this is the cells in your body that have the most to do with determining who you are being the least constrained by genetic determinism.

So a hormone has 2 receptor on it. One - on the hormone side to trigger it & other that connects to the promoter. these can be mixed & matched so that hormone can be triggered & then go out & attach to an entirely new promoter. this is a new if then clause.



the downside is that the immune system recovers it sometimes overshoot the original mark & endsup getting hyper. & in it hyper state it became over reactive. & the next thing you know you have an auto-immune disease, which is more common after pregnancy. this could be dangerous & some autoimmune disorder, such lupus, are severe enough that the affected will be advised to avoid pregnancy.

Glucocorticoids are stress hormones that suppress the immune system (there's a lot more to it, but in brief, they suppress it by reducing the inflammatory response). A slight clip and a little shuffling and you can create the new if-then clause if there's progesterone around suppress immunity. What's this about? Pregnancy. This if-then statement prevents the immune system from attacking the fetus.

Next up are copy number variants. This is the world of multiple copies of the same gene. This can allow for experimentation with one back-up copy. At the same time, there can be problems linked to it, such as is seen with schizophrenia.

The multiple copies of genes may account for "irreducible complexity," i.e. how can an eye pop up out of nowhere? If the organism has multiple copies of sensory genes and is able to experiment with one without sacrificing the other, it could develop a feature incrementally, slowly growing an eye while using sound and tactile information for guidance until such time as the eye starts working. (This can account for evolution's production of vision while leaving a very big door open in regard to what's out there that we haven't evolved to see.

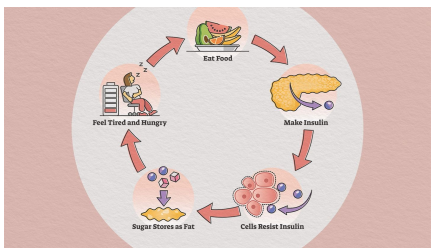
This is the whole world of intuition and spiritual belief. This is the whole world of wackos that claim they can sense things others can't. Or is it?)

this is where Spritual guru's form their belief.

for the most part these changes will not be beneficial overall since they have to coordinate with so many diffrent gene network. Therefore it's generally a stabilizing selection in which you won't see much change. However, when the genes stumble onto somethin good, you may see a rapid change.

now moveon to Insulin resistance.

In brief, the hominid body is designed to store nutrients. these days our foods are loded with everything, all kinds of things. So we are seeing a huge increase in arange body mass, cause body is storing all the goods & bad stuff.



The more wasteful your metabolism the better it is.

Get yourself a body that isn't used to a western diet and you are a candidate for type II diabetes as your body grows beyond what it's supposed to. The fat cells get full and start ignoring insulin. Insulin gets angry and calls on the pancreas to make more insulin to help force the fat cells to do their job. The fat cells relent a little but demand ever greater amount of insulin to listen and pretty soon you've got a blood sugar problem and are well on your way to burning out your pancreas.

— this problem is now more common than ever.

Sometimes there are surprising effects from genetic variation, such as the story of the Silverfox

The Dutch Hunger winter is a great example of this. Due to Nazi shuffling of food, the Dutch experienced a winter of starvation. The women who were carrying babies gave birth to "thrifty" babies whose metabolisms had learned to hold tightly onto whatever nutrients floated by.

Thus they are more at risk for all the metabolic problems in adulthood - hypertension, diabetes, excessive weight gain, etc. And so are their offspring since they gestated within a mother's body that was very thrifty and thus shared less nutrients.

MRSA, VRE, smallpox, our friend trypanosoma, all with a capacity to evolve faster than our drugs. So there's a continual battle between the cells of our body and the pathogens that want to crash the party.

the last fear is that of Antibiotic Resistance

LECTURE 6

gibberish => rubbish / useless

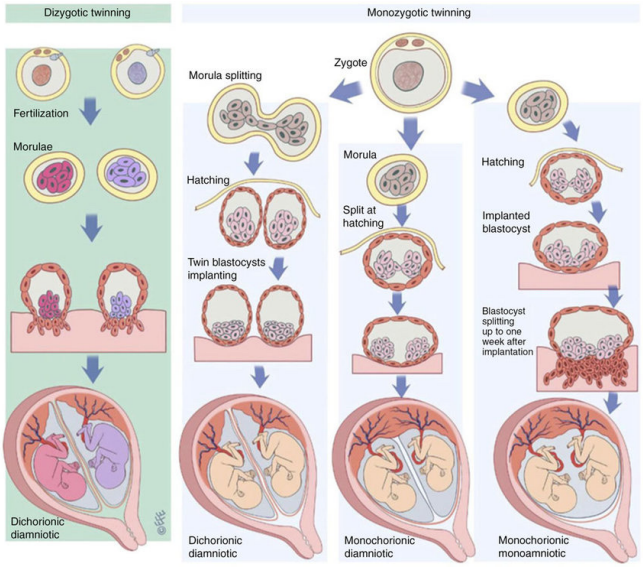
In brief, this is a field in which scientists look for patterns of shared traits among individual that have different levels of shared genes influence from that!

the basic notion being that if you have a behavioral trait that is more common. the closer you are genetically, you can infer that the behavior is driven by the peron's gene.

due to concern over commitment's effects. the study focus on several variants that help control for environmental influence.

for instance, comparing identical twins to fraternal twins or comparing siblings that are raised in different enviorments. Unfortunately this also has flaws.

for example, he notes that twins are not treated the same; the environment is much more similar for monozygotic twins.

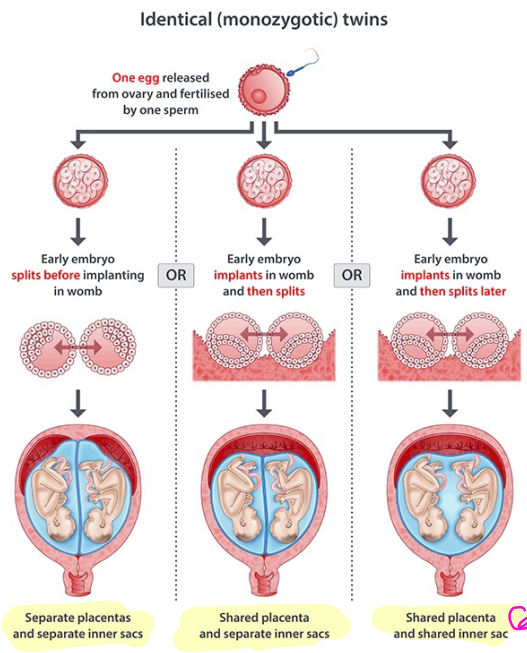


Identical twins (also called monozygotic twins) result from the fertilization of a single egg by a single sperm, with the fertilized egg then splitting into two. Identical twins share the same genomes and are always of the same sex. In contrast, fraternal (dizygotic) twins result from the fertilization of two separate eggs with two different sperm during the same pregnancy. They share half of their genomes, just like any other siblings. Fraternal twins may not be of the same sex or have similar appearances.

the environmental differences can start early - if they split within first 5 days after conception each will have it's own Placenta, if they split in the 5-10 days Range. there is a shared placenta. means there will be difference in the extent to which they share the same bloodstream.

He mentions a Johns Hopkins study that examined differences in math ability between boys and girls. The data set suggested that boys were better than girls at math, with a 13:1 ratio in the upper levels. However, in more equal societies, such as our friendly Scandanavians, the difference is not only diminished, but slightly reversed with girls scoring higher. The lower a society's score when it comes to gender equality, the greater the difference between the sexes on tests of mathematical aptitude

Johns hopkin study



at this point in time coughing girl comes on to the scene. She'll be coughing in the background for the next few lectures.

As he notes the differences in environment for 13 year old boys and girls, it's easy to see that his viewpoint is that this field is, at the very least, difficult to prove scientifically and, more realistically, ludicrous.

Simply people don't ever share the same environment. There are thousands of different expriences that shape us & influence how we handle situation.

A big study on schizophrenia based on Danish citizens shows genetic influence in the development of schizophrenia. Using adoption studies and statistical measures, they found a 1% chance of being schizophrenic among the population on the whole, but with no biological basis while being raised in a schizophrenic household the number goes up to 3%. When raised in a household that did not have a schizophrenic parent but in which the biological parent(s) do, the number jumps to 9%.

And for the truly bizarre situation in which the kid had a genetic legacy of schizophrenia and managed to get adopted into a household with a schizophrenic adoptive parent, the rate goes all the way to 17%. He notes that this synergistic effect will come up again.

Sapolsky also states that this study was the first time a genetic basis was shown for a psychological disorder. As such it's a landmark event because a genetic psychological problem is a medical problem, not just a mere adjustment to society issue.

To correct for this, adoption studies are used. Here siblings with similar genes that are raised in different environments are compared. The thinking is relatively straightforward -

if these siblings are more like each other than they are like the siblings in their adoptive homes, genes are playing a role.

⇒ no biological basis but schizophrenic household the number is 3%.

⇒ biological parents have but living household doesn't the number is 9%

⇒ with both it's 17%

Now here are some problems —

1. Under the cleanest circumstances the baby would have been whisked away second birth, thus preventing any shared environment with mother. However this is often not the case.

2. Prenatal effects – the prenatal environment shared with mom, including levels of various hormones in the blood stream —

To get around this (perhaps speciously) the argument is made that they can measure the frequency with which the trait is shared with the mother or father.
If there's a 17% correlation with the mom but only 10% with the father, then the 7% difference is attributed to the prenatal effects.

3. Adoptive family placements are not random - efforts are made to place the child in a similar type of home. thus the adoptee share a lot of biology with the new family, screwing up the notion that enviornmt & genetics have been separated.

The new gold standard study model is the identical twins separated at birth model. From this group, the research suggests about 50% heritability of IQ, about 50% heritability of where you are on the introversion-extroversion scale, and about 50% heritability for degree of aggression.

anxiety levels as an adult can be impacted by the prenatal environment (in rats). the more stressed the mother, the higher the glucocorticoid levels in the bloodstream, resulting in smaller brain, thinner cortex, more glucocorticoid receptor, fewer benzodiazepine receptor more of a decline in cognitive ability as you age.

this leads to harder time bouncing back from stress - meaning more cumulative exposure to glucocorticoids & therefore more damage.

This can be referred to as an non-Mendelian inheritance of traits since the thinking is that it's not a genetic thing.

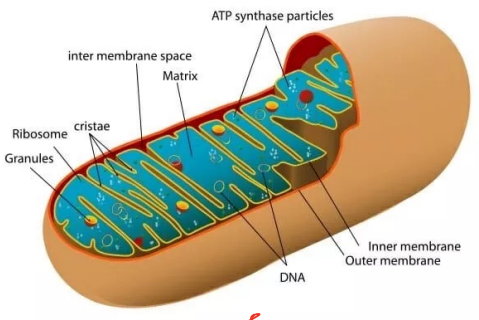
Fetal Origins of Adult Disease (FOAD)

- Holland 1944 and the Dutch Hunger Winter. The Nazis divert all the food in Holland to Germany. The Dutch diet thus goes from normal to starvation level. 3rd trimester fetuses develop super thrifty metabolisms due to nutrient deficiency and thus become much more likely (1 9 fold increase in risk) to develop metabolic diseases such as diabetes, obesity, high blood pressure, etc.
- because their bodies keep a greater than normal percentage of nutrients - sugar, sodium, fat - all stored. They in turn have offspring who are at a greater risk because the mothers' thrifty metabolisms don't share as freely with their offspring.

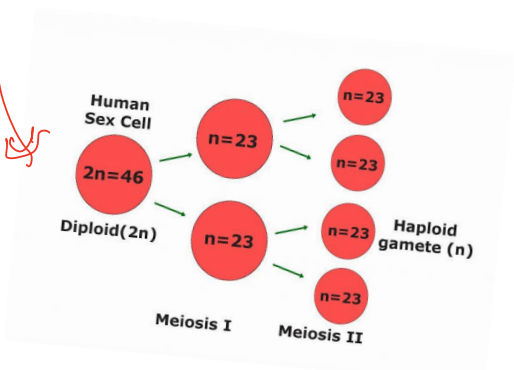
- Interestingly, the poor Russians at Stalingrad did not demonstrate a similar pattern because their starvation went on much longer and showed a pattern of slow but steady decrease followed by a slow rise.

- Incidentally, Antony Beevor's Stalingrad is an excellent book.

A study demonstrated that the fetus took on the characteristics of the placental mom. when she was high anxity, the rat was also in high anxity, regardless of the genes of the true mom.



Mitochondria, the powerhouse of the cell have their own DNA & along with other junk in the cell split somewhat randomly during gamete formation. Mitochondria only come from the mother's side. (cause eggs have it & sperms don't) so all the genes which come from mitocondia you get from mom) So it's not 50/50 split. a disproportionate amount from your mother.

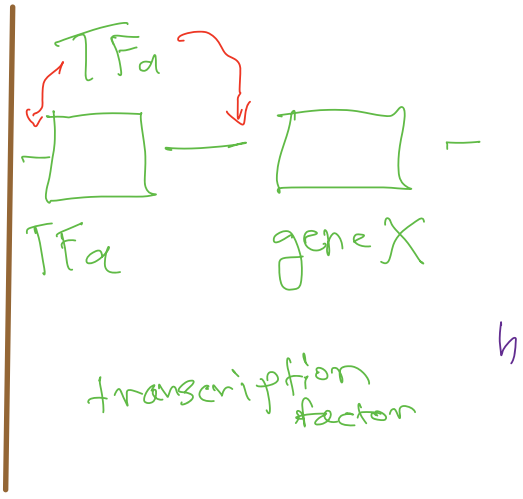


- Indirect genetic traits. Judith Rich Harris and The Nurture Assumption. Here the question is to what extent the environment acts on genetic traits in order to reify them. To wit, where you are on the extroversion/introversion scale is as much a result of how the world interacts with you as it is your genes.

- Thinking in terms of a good looking baby and an ugly baby - both have extrovert genes but only one of them gets a lot of smiles back in response to extroverted behavior. Other genetic factors will mediate the impact of the gene in question as well the world at large.

human hight is a heritable trait to some extent & that endless studies have shown that taller people are treated better & considerd more attractive, "comma, he says bitterly".

not surprisingly, people who are treated better during the developmental periods end up being more extroverted. Thus with have heritability of a trait that inturn causes to be treated differently in the world whic brings about changes in personality.



Again, the genes are having a hard time winning out on their own.

The pecking order - you inherit the colour & iridescence of your feathers. Get bad feathers, you get packed at more often & head to bottom of the social ladder.

- Studies have suggested 70% heritability of political preferences in the US. However, this is actually mediated by personal characteristics, especially comfort with ambiguity. Conservatives tend to not like ambiguity, preferring black and white analyses of situations.

then he transitions into the Kohlberg Scale of moral development & the notion that there is a theory that ties to

link up political preference with one's stage of moral development. & in both conversation ended up looking preety bad- Simplelistic world view & under-developed morals.

- **This is one area where Professor Sapolsky may be presenting a biased view. While your author agrees with him in many ways,**

the conservative viewpoint has it's nuances & there are issues for which adding in ambiguity may be possible but not necessarily smart (crime, for example, which endless studies have shown is significantly impacted by SES & all kinds of developmental elements. yet & still, there is a crime that's been committed & the why doesn't undo it- is ambiguity correct here? I'm not taking a stance. but am nothing that it's an area of ethical debate in which ambiguity isn't necessarily the winner.)

curiously, studies showing heritability of aggression in rats actually have an underlying mediating factor - pain sensitivity. The more agressive rats are less able to tolerate pain & thus more likely to lash out aggressively when they

feel it. Again the surface behaviour is not the one that's being passed along.

Nurturing also has effect on mothering.

- Mothering styles of rats impact the robustness of the rat as an adult. Better mothering leads to a healthier rat that's likely to be a good mother when grown. This is accomplished through epigenetic changes in transcription factors.

Psychology also works between pshiology & biology.

LECTURE 7

the lecture opens up with an interesting note- sibariyan foxes he mentioned in previous lecture, you breed them for tameness, purely on behavioral trait, & come back 30 generation later & they look like puppies.

Sibariyaan fox

Moral of the story —

(1) evolution can be fast

(11) In some mystrious way if you choose some behavioural traits in this case on where you like being around human & all cuddly with them. but you're also gonna select a whole buch of traits that are associated with baby wolves in terms of physical appearance.

Now there is a flip side going on right now —

- He opens on an interesting note - in Russia they have what are called Metro Dogs that are essentially feral dogs that roam the city (allegedly riding the subway) and that are moving away from the domesticated dog that we are used to. As the generations pass, the dogs look and behave less like puppyish dogs and more like wolves. They are becoming less cute and cuddly.

he indicates that the concept is looking for patterns in behaviour that increase with relatedness.

A constant confound is the presence of enviormental factor. that could also influence behaviour.

(it's worth noticing, that there's a significant gap between genetically controlled & genetically influenced behaviours. any study that demands a controled & genetically influenced behaviours, any study that demands a control behaviour in order to admit any influence will skew the genetic influence result in the direction of suggesting less influence.

a person who is really concerned about precision

pendantic

However, there is a risk of being pendantic when setting up scientific rules for study, especially when the results fly in the face of everyday knowledge, including the common notion that kid ends up like their parents. The catch is this isn't an absolute necessity, but that doesn't mean it's common)

- In addition to difficulties pinpointing the genes that cause defects and illness (as he puts it, you think you know how the universe works in regard to this disease), there are also huge ethical issues -

advising people against having kids, notifying people that a fetus may have a terrible disease, telling someone they may have a terrible disease are all dubious actions. He points out there's a big gap here between possibly having a disease and having it for sure. Is it really ok to guess here? Especially when the DNA suggests a possibility,not a certainty?

in this case the problem will be the result of network of genes, promoters, transcription factors, environmental effects & more. In addition to that when you have an idea of where to look. they're couldbe some areas you're not looking.

this is fine when you're in kitchen looking for a snack but may not be so hot of an idea when dealing with genetics.

You need a genetic difference that has a functional difference. For example, bdnf genes in rats impact the activity & growth of amygdala, which in turn impact behavior in them (fear & anxity). This can risk your anxiety disorders.

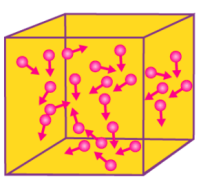
He runs through a series of these examples that pinpoint a particular gene, receptor or hormone that differ and have been found to impact behavior. It's been my experience that these types of examples sound a whole lot more convincing coming from Professor Sapolsky than your average college student, which may point to an underlying flaw. We've all encountered a psych or med student who explains the manner in which dopamine "causes" this or that, an explanation that usually falls flat due to its overly simplistic nature. Here Sapolsky is venturing into the same area. , albeit with multiple cautions

As we have seen in previous lecture the DNA's control over human behaviour & cellular development is far from total among our neurons especially the neurons in the cortex.

So if neurons that run our executive functions have a surprisingly high amount of freedom,

How seriously can you take a suggestion that a hormone can overpower them? I've leave that to you to judge since it's an old philosophical issue returning in a new form.

Brownian motion : molecules oscillate in ways that can be completely random. So two cells that begin genetically identical will be different after just one split. Other elements, such as transcription factor will also experience random distribution when cells split.



https://youtu.be/4m5JnJBq2AU

Heriability doesn't only mean it's genetic what heritability means is that the impact persists in different enviornments & is independent of those environments. ~~will produce a change in the behavioral genetics.~~ He runs through a variety of examples that demonstrate how little can be considerd truly heritable since changes in the environ-ment will produce a change in behaviour.

when it comes to human behav-ioral genetics, very little is deterministic because enviornmental changes carry so much weight in how the person develops.

He states it's not about the trait itself but rather the amount of variability around the trait. This sounds complicated until you pause and realize it's really the same thing.

If you have a heritable trait for brown hair, it can't bloody well be heritable if your kids have black and red hair (too much variability). It can only be heritable if they have brown hair and the shades of brown very close to the original. Once environment is able to push the range too wide, the trait is no longer strictly heritable but rather reflects the interaction of genes and environment.

The hay maker is that the vast majority of scientific studies demand that "you control the environment". thus, heritability has wobbly knees. It is baised towards the genetic influence appearing more important that it is.

powerful punch

The counter to this is that environment doesn't usually vary that drastically (think niche) and it's more realistic to control for it. This is as weak an argument as can be proposed when you recognize that if the environment has to be controlled for so that its effects don't throw off the results, you've pretty much already lost the genetically determined hypothesis.

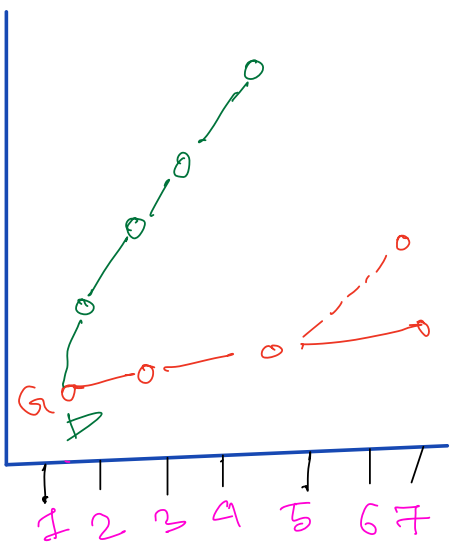
the conclusion simply & unalterably this: It is impossible to say what a gene does. you can only say what a gene does within the environments that's been studied in to date.

Because heritability is a measure of variations the fact that nearly everyone has 10 fingers to start with creates no variability in the number of fingers you have, & thus no heritability of the trait. (which is 100% from your genes).

However, wearing earrings in the 1950's in the US was universally common among women & verboten (forbidden) among men, so the heritability ends up being 100% since the one genetic factor, female or male, accounts for all of the variation.

Another example is PKU, which relates to a genetic disorder in which the body cannot break down phenylaline. It builds up to toxic levels and there you are. On the face of it this would be a 100% heritable disease since the initial comparison question "Would you rather know where this person lives or if they have a genetic mutation?" points to the gene side.

But these days foods are labeled when they have phenylaline, and thus knowing where someone is living can be as powerful of an indicator as genes. Again heritability is only heritability within an environment. Remove phenylaline and the person doesn't have the problem. Remove racism, social distinctions, abuse, nutrient deficiency and you may also not have the problem.



& it's been tested in 99 different enviornment.

He next puts up a chart that we'll see often - it's the bad gene + bad experiences graph.

Have a bad gene and your odds go up a bit, but have both the bad gene and the bad experiences and the rate goes through the roof.

Stress hormones, including glucocorticoids, play a big role in this process as they are activated and interact with the genes in question, thus providing a synergistic effect.

a creation of a whole that is greater than the simple sum of it parts

the conclusion is this is a "it depends study". the more environments you study something in, the lower heritability is going to be.

Math, "at which men are better than women", when actually studied in the context of gender equality within the society, does not demonstrate an inequality on the average.

Instead, the greater the level of gender inequality, the greater the difference in math skills on the whole. The worst profile went to Turkey, Tunisia and South Korea. The US was in the middle. Our utopian Scandanavians were the best. In Iceland, the girls bested the boys.

In the US the gap at the high end has narrowed from 13:1 to 3:1 in last 20 years.

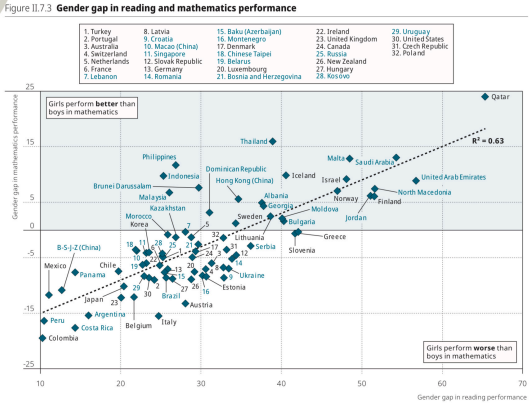
Women contineue to hold an edge in the verbal side, both in the worst places & the best, with the advantage increasing as the social equality level goes up.

He closes by noting (warning) caveats about behavioral genetics.

Environmental effects & modulating effects, intermediaries & what not. In the end, he suggest that a lot of what we see in neural feedom suggest that what's coded for is freedom from the constraints of controlled genetic behaviours more so than coding for genetically determined traits.

7 Girls' and boys' performance in PISA

Figure II.7.3 **Gender gap in reading and mathematics performance**



Note: Gender gap refers to the difference between girls and boys (girls minus boys).
Source: OECD, PISA 2018 Database, Tables II.B1.7.1 and II.B1.7.3.
StatLink https://doi.org/10.1787/888934037887

Most of my genetic trait will be expressed differently when the enviornment changes.

LECTURE 8

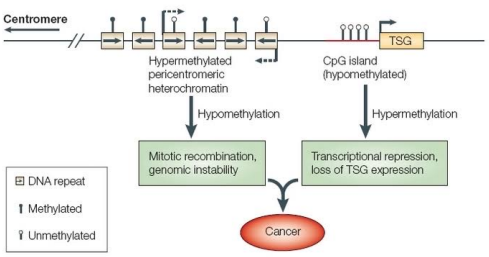
Quick recap - inherited has to do with items passed down that are consistent, while heritability is about the independence of genes relative to environment.

Professor Sapolsky selects a terrible example with the whole five fingers thing since the polydactyls in the crowd are well aware that it's actually a heritable trait given that having five or six fingers will be based on genetics whether you're in Brooklyn or Yemen - know that family's history and you've got more predictive power than knowing geography. Heritability is really about the environment's influence, not the gene's influence (technically the definition runs the other way, but the manner in which heritability is established is all about where the genes are expressed.

If they are what they are no matter where, then it's heritable. If variation pops up based on environment, then it's not heritable.) In the end the point he is hammering home is that very little of what comes to define us will turn out to be truly heritable - most everything will be about the gene-environment interaction and this opens up a Pandora's box when it comes to human behavioral biology and what it means to have this or that behavior, disease, or even achievement, a topic he will sum up in grand fashion in the closing lecture.

first starts with epigenetics! what is Epigenetics?

⇒ Epigenetics is the way the culture, environment all of that affects biology. another explanation is epigenetics is the regulation of cromatine remodeling & methylation of genes all of that.



next chutes & ladders example. there was a study funded by WHO said people in Nepal are good with chutes & ladders than people in Belgium. then he asked question about—

what do you wanna know about study?

check (audio)

---

neotenize = to cause to become neotenic

(delaying or slowing of the physiological development) juvenilization

---

a study done in Norway & published in Science that demonstrated that first born children had higher IQ scores than second born children. In the end, though, the point that was missed was that the difference was miniscule & not statistically significant.

Some side points —

(1) not all first born kids had higher IQ's than second born kid. So this shit isn't deterministic &

(2) by definition one of these is going to be higher than the other whether there's any meaning to it or not.

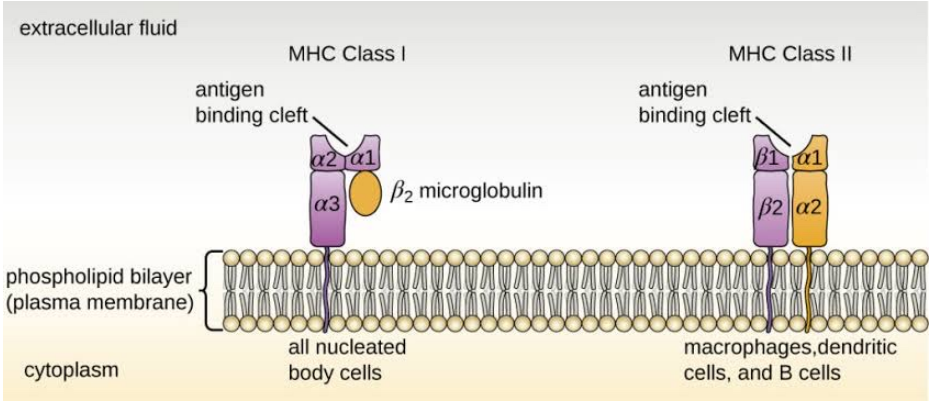
(3) at the age of 12 the latter born kids tend to have higher IQ's. Ultimately there will always be some difference but it doesn't mean it matters.

Rat studies demonstrate that they recognize relatives & are able to do so based on urine markers. - for this to happen there must be at least 2 things.

(I) qualitative differences & (II) reception area within the brain that can identify these.

(Just as the immune system is an example of juggling around protine combinations, so too will the body create it's own protine markers)

This stretch of genes is called the major histocompatability complex (MHC) & it is crucial in understanding this topic, but more importantly, auto-immune disorders.



The major histocompatibility complex is a large locus on vertebrate DNA containing a set of closely linked polymorphic genes that code for cell surface proteins essential for the adaptive immune system. These cell surface proteins are called MHC molecules.The major histocompatibility complex is a large locus on vertebrate DNA containing a set of closely linked polymorphic genes that code for cell surface proteins essential for the adaptive immune system. These cell surface proteins are called MHC molecules.

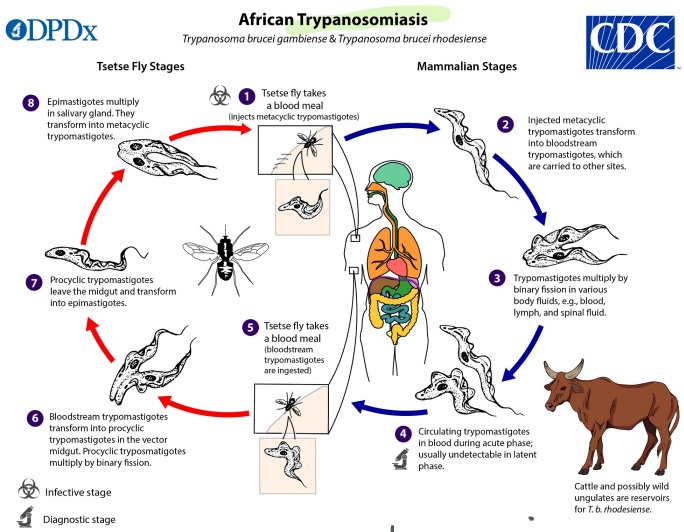
https://youtu.be/R69M7NuBNBA

the protein signature is slapped onto all of the organism's cells to identify them as "US" "Cells that don't have this are them."

One of the roles of the thymus is to screen immune cells to insure they can tell the difference. when they can't, you get nasty results when the immune system attacks itself. The MHC is also a major issue when organ is "them" & accordingly the immune system gets geared up to attack it.

This is why recipients ends up on immunosuppressant drugs. It is also why stem cell research is likely critical to our capacity to regenerate damaged cells & organs; in addition to having the right DNA, it's also "us".

a nasty trick hat can be played by invaders, such as trypanosoma brucei involves changing the protine coat that it displays. Although the immune system will figure out there's an issue, by the time antibodies are formed to attack the original form. the Shield has been changed & the invader continues to reproduce "itself".



Trypanosomes were!

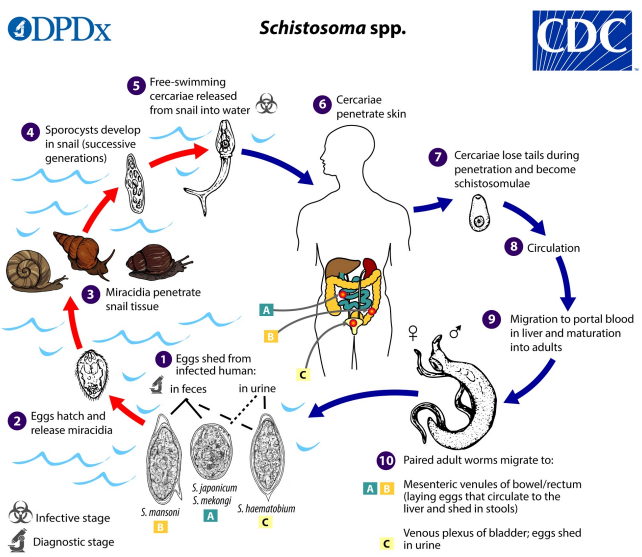
there was a false info that you can't make nurons after age 3. but from last 2 decades nuroscience has discoverd it's not true. adult "nurogenesis" this happen only 2 area of the brain

① hippocampus :

you learn something new you make new nurons there. you get stress you make less nurons there.

(check audio)

Even worse is schistosomiasis - what these buggers do is cloak themselves in your MHC, grabbing the protein sheath from your cells and hiding out as if they were your own cells.



& the most pathathic ones are cancerous cells which can be cloaked in your MHC protein & thus evade attack. if apoptosis

doesn't get them, you end up in treatment.

The MHC protein can become soluble & are thus exuded in saliva, perspiration urine etc. & can become genetic markers at some level. Oxytocin & vasopressin (AHC) tuneup the cells that recognize MHC signal especially around the time of pregnancy & giving birth. This marks the child & leads to nursing & resource investment.

How mother recognise her babies?
=> who smell like my, vaginal fluids./ saliva/ mouth smell like my milk / amniotic fluid / someone i've mated with my past

Research suggests a possible mutation in the genes coding for oxytocin and vasopressin in families with autism, where there are major deficits (or differences) in social communication, bonding, etc.

Oxytocin & vasopressin (AHC) make you more likely to make those receptors or increase the number of those receptors.

Much to our delight, it turns out that new neurons are generated, primarily in the hippocampus (learning) and the olfactory system (scent, during pregnancy and post natal).

Here he throws out an interesting hypothesis. During the time you're pregnant you're restructuring your olfactory system, taste is driven by olfactory cues, and it's no wonder that stuff smells weird, foods taste weird and you get odd cravings. Which makes more sense than the notion that changes in diet and morning sickness are caused by evolutionary attempts to protect the fetus from fetid meat - were women eating fetid meat before the pregnancy?

an interesting study with baboons found that dominance reversal cries were very interesting to the group if the two were not relatives but not interesting if they were.

A human parallel would be employees not getting all worked up over the intern/mail clerk son of CEO arguing with dad & getting a concession but being very confused.

To wit, if #4 and #27 had a squabble and were relatives, no one cared that much if #27 gave a dominant howl and #4 a submissive one. But if they were not relatives, the baboons were instantly tuned in to figure out why #4 was submitting to #27. Crazy relatives get no attention.

Imprinting is another method for recognizing relatives. the learning is innate but the process is experiential.

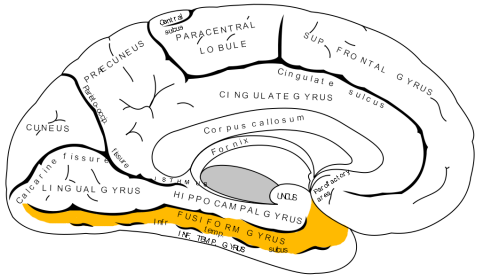
Now the question is how do our brain recognise this kind of info or subtle sign ?

=> it's the fusiform cortex.

(check audio)

The fusiform cortex (or fusiform face area) is a section of the brain within the cortex that appears to be centrally involved in facial recognition. Show someone a portrait, picture or even a good cartoon of a known face and this part lights up.

However, show an autistic person a portrait, picture or even a good cartoon and it doesn't. This brain area may be centrally involved in cognitive understanding of what's a relative, or at least a known person. For autistics, mother=armchair=stranger.



fusiform cortex

LECTURE 9

today's topic is ethology, aka process of interviewing an animal in it's own langage.

We start at the turn of the century. Freud and William James have established psychology as an introspective field of study, more philosophy than science.

this in between standing occasioned a revolution with in the field. resulting in a transformation that placed behaviorism on top. the behaviorsts offered a quantitative method that made phychology seem like a science than a field of rumination. Thus it became an experimental, data driven field that distrusted any behaviour that could not be seen or measured.

Thus there was no interest in what was going on inside - all that mattered were what happened in the environment right before the behavior and what behavior was produced (stimulus-response). Everything else was dismissed as speculation. The field reached its peak influence with B.F. Skinner (whose work was really just a repackaging of concepts introduced earlier by Thorndike).

Key features:

① Radical environmentalism ~ we are blank state whose behaviour is determined by the environment.

(2) Reinforcement theory: with control of positive & negative reinforcement along with control of punishment you can produce whatever behaviour you want in an organism.

(Quick note: positive reinforcement is a reward for a behavior; negative reinforcement is a reward via the removal of something bad - think pain medication; positive punishment is actual punishment - think pain; negative punishment is removal of something good - think of Mom taking away your iPhone.)

(3) notion of universality: it works the same for everyone.

Skinner penned Walden Two, an ode to the use of operant conditioning to build a better society. A cheeky title, too since it's hard to imagine a historical figure who would dig the concept of behaviorism less than

he with his stupid pigeons :

Henry David Thoreau

As simplistic as the theories sound, they still hold considerable sway in the field today. One need only consider the medical model of treatment to see that the notion of stimulus-response hasn't died down all that much.

the founding father of ethology are Nikolas Tinbergen, Konrad Lorenz & Karl von Frisch

As a side note I see a commentator on YouTube has suggested that Sapolsky misrepresents Skinner's ideas. I'm no fan of Skinner and won't be coming to his defense.

That said, some elements of behavioral theory have validity and anyone who's interested in this area may find it worth exploring. In my opinion what they got right were the obvious points that anyone could see - such as you're likely to engage in rewarding behaviors more than unrewarding ones - but they fall off the tracks completely at the more complex and subtle levels, those involving motivation, self-destructive behaviors and psychological hang-ups - you know, the raison d'etre of the field of psychology...)

He notes studies on enriched environments done in the 1960's that demonstrated that -" a rat's cortex was thickened by being placed in an enriched environment".
But another study showed that - "when rats from the wild were captured and their cortical thickness was checked, their cortex was thicker than the rats from the enriched environment."

Point

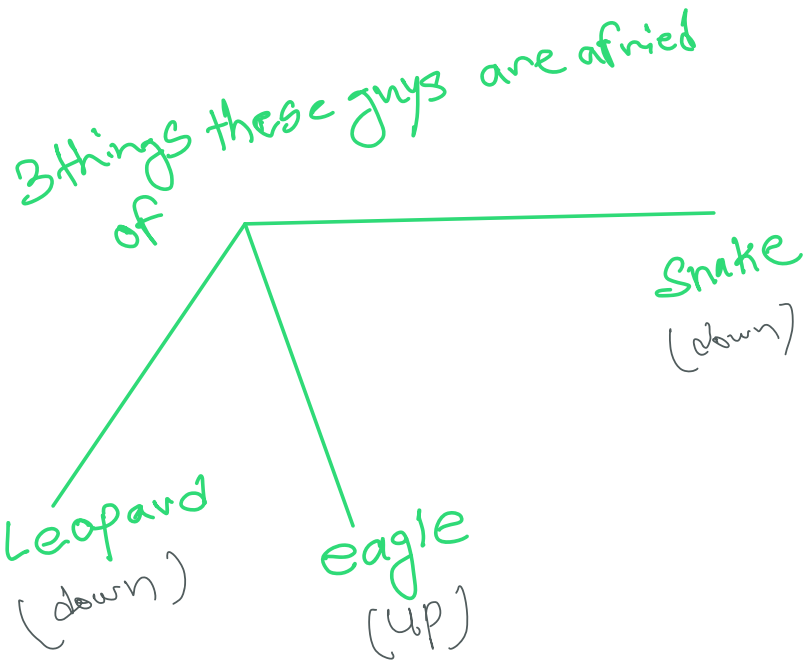
In other words, you've got to check animals out in their real environment - no lab setup will ever give you the same results.

Now talks about fixed action patterns. these behaviors are linked in with instinct/gene in subtle ways. They are triggered by an environmental release stimulus. So how?
=> for instance vervet moneky in east

africa.

vervet monkey

(check audio)



Vervet monkeys have fixed action patterns for alarm calls (scary thing below, scary thing above) but they have to learn to use them correctly. An infant may shriek out an alarm call but no one's moving until an adult confirms. Sometimes they have the basic idea right (Yikes, predator) but they panic and call out the wrong instructions. With experience the fixed action pattern grows into a reliable behavior. Thus they move when the adult calls out the play.

infant smiling is also a classic example of a fixed action pattern in human. Fetuses smile, Blind baby smile, Nursing is also a fixed action pattern.

Von Frisch performed interesting studies on bees, deciphering that they do their little figure 8 dance in the hive to establish the location and quality of food.

One of his experiments included creating a food source in the middle of a lake. The bee then heads back and tells everyone about it.

They laugh, since it's in the middle of the lake. In another one he rotated the hive so the directions were wrong. The studies were suggested by Jack Handey.

the Harlow monkey studies featured two wire monkeys, one with milk & other with soft fabric. baby monkeys were seperated from their mother & given a choice between the two, while the behavioral model suggest that they'd go for the milk (nurishment being reinforced), the baby monkeys actually prefer the psychological comfort of the soft wire monkey.

Studies demonstrate that female rehesus monkey have to learn how to be effective mother. the behaviour aren't instinctual. later offspring have a better chance of surviving. having an older "sister" also leads to better outcome modeling.

A human parallel was seen with premature/at risk babies that were kept in special care at hospitals. Reasoning that nourishment and warmth were the keys to care, hospital staff curbed parental visits to 30 minutes a week and, with the introduction of incubators, began limiting all kinds of touch.

Sadly this resulted in shorter lifespans and worse outcomes wherever incubators were found. Fortunately the radical idea of actually touching the babies was reintroduced and outcomes improved.

---

ovulation — the process of which mature egg is released from ovary.

meerkats

meerkats learn how to kill scropions step by step. Mom brings a dead scorpion first to teach. then live scorpion without the stinger. Finally, once those lessons have been mastered she presents with a ragular one.

Apes make tools. The more experience watching and learning by experience, the better they are. Female chimps learn more quickly than males because the females actually pay attention to Mom.

one trial learning, Example of birds imprinting on mom.

First big thing is the thing to follow. Some sort of neurological wiring to guide you.

Are humans innately scared of spiders & snakes?

⇒ No but we have a very strong prepared learning for it. Cultural factor overcome this but the amygdala is ready to be frightened. it takes a much smaller stimulus to get us going in that direction.

Sauce Bearnaise syndrome - get nauseous and food correlated in time with the experience will trigger the same response the next time you smell it. Prepared learning.

next a sad section — do animals have self awareness? Studies focusing on whether animals examine themselves in the mirror or not. Bit of human arrogance here.

also an example of the limited boundaries of science — measuring only what it's disigned to measure & ruling out what it doesn't have the capacity to measure as unreal.

Good juxtaposition by Professor Sapolsky since the section before was on echolocation. What science doesn't know how to measure is unreal until science catches up and then science gets a little arrogant.

this is the epistemological function of knowlage — we think the thinkable thoughts but not the unthinkable ones.

mamosets don't stare into other marmosets eyes. thus they failed the forehead spot test until, it was placed on their throat instead.

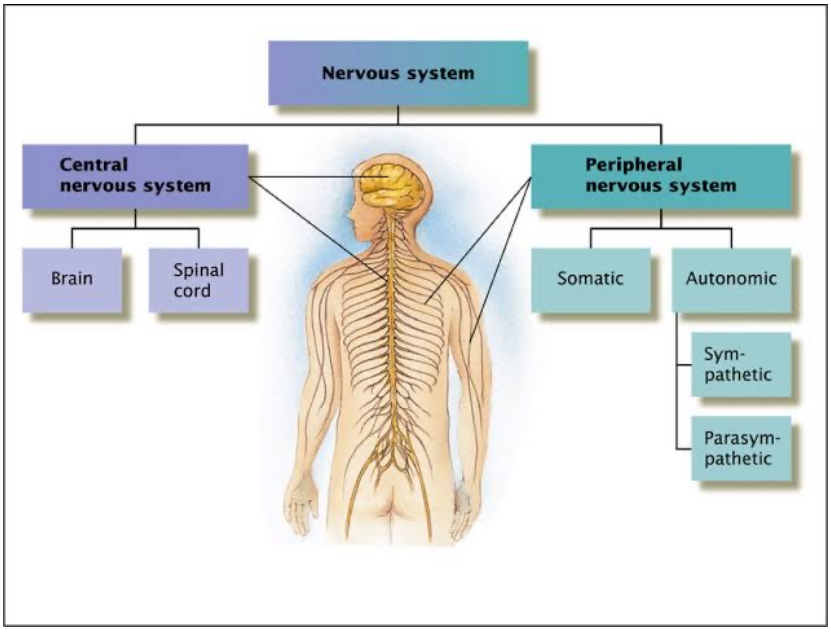
theory of mind — not everyone sees the world the way you do

LECTURE 10

Why does chiken cross the road?

lymbic system

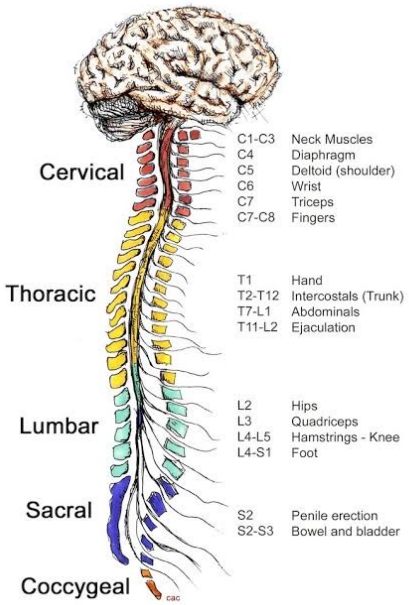
He begins by noting that his students that move on to medical school will hears tons about the spinal cord and cerebellum, but little about the upcoming topic, the limbic system, because therapeutic interventions are possible with the former, but difficult with the latter. Nevertheless, the limbic system is involved in the production of emotions and personality and is core to who we are.



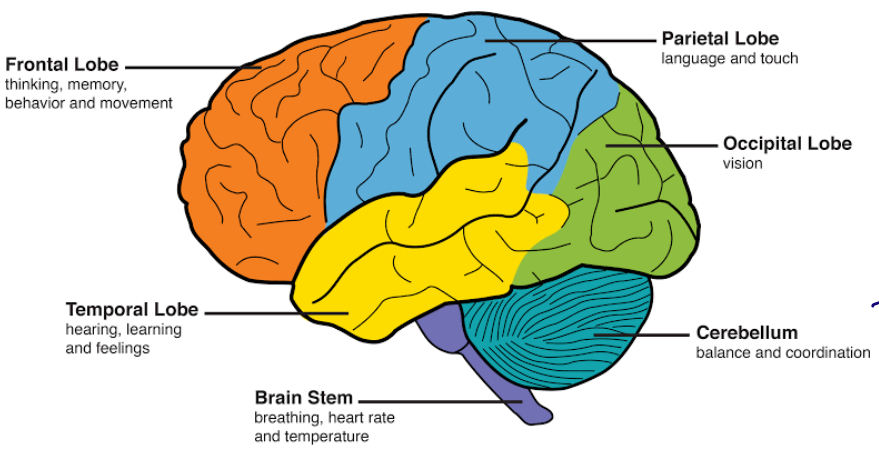
2 Parts of nervous system.
(I) Central
(II) Peripheral

Dale's Laws. Dale's second law begins with a neuron with the axon and axon terminal and states that each neuron has one characteristic neuron and releases only that type from its axon terminals. (This is not the same as stating it only has receptors for one type of neurotransmitter - it would still accept many.)

CNS contains Brain & Spinal cords

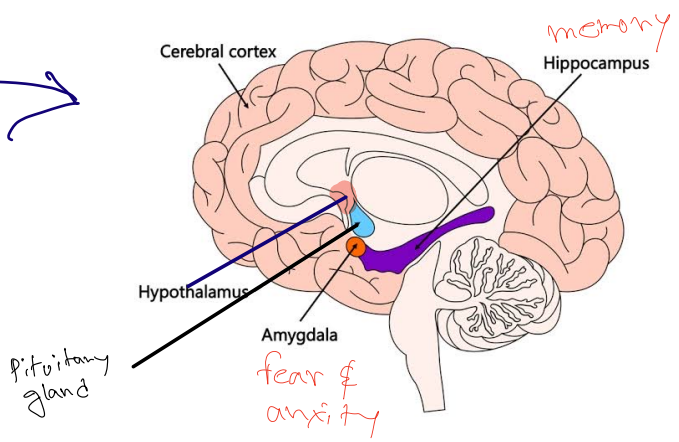


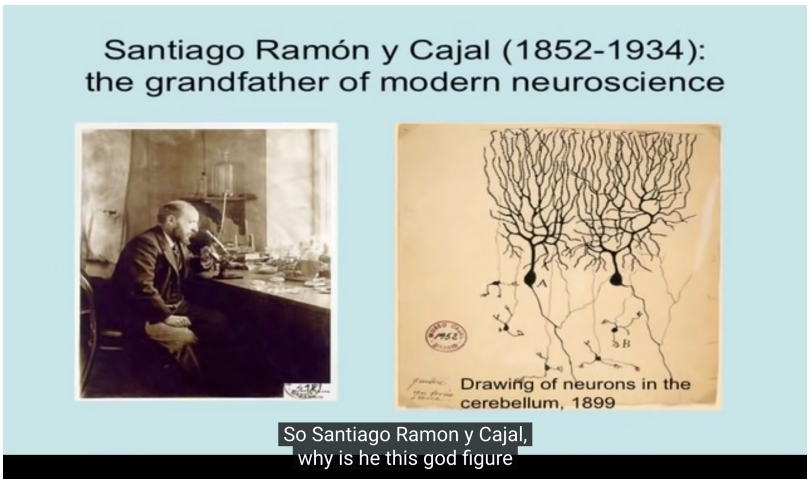
Research in the 1980's showed Dale#2 was incorrect. Researchers discovered that not only would the neuron itself have more than one neurotransmitter, but the vesicles themselves would have two types. A few even have three types. Generally the types are structurally very different, perhaps a single amino acid and a complicated protein structure. This impacts speed of action. One of the neurotransmitters will have receptors for it on the neuron itself (bookkeeping).



Different parts & different function of the brain.

Hippocampus means Seahorse it form new memory.

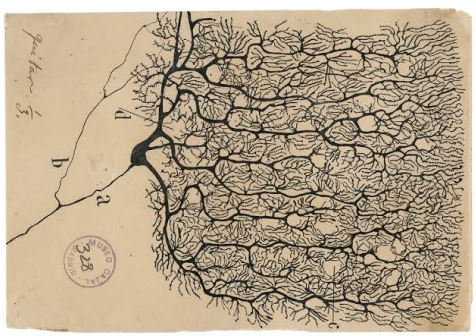




90% of your cells in your Spinal cords isn't actually neurons.

they're called glia.

you've got a quadrillion Sinapses in your brain when we have 300 billion Stars in milky way Gallxy.



He then sidesteps into his favorite topic - glucocorticoids. Why Zebras Don't Get Ulcers is mainly about these guys. In short they are stress hormones (hydrocortisone is the human equivalent - it's a steroid that is used for its anti-inflammatory and immuno-suppressant effects. These steroids are different than anabolic steroids that weightlifters use for increased strength). He cites the example of the stimulation of ACTH by the pituitary stimulating release of epinephrine and epinephrine (adrenaline and noradrenaline).

These are activating hormones that tell your body to get ready for action, whether it be running, fighting, killing a squirrel or fretting about the mortgage. In the short term they redirect energy to your muscles, enhance your focus (mostly) and put you in a stimulated state. In the long term they burn you out and leave you vulnerable to cell damage and death (heart disease, stroke, Alzheimer's). It's a fight or flight stimulus mechanism that ignites under stress and, as such, is great for handling real stress but can be disastrous if turned on too often.

receive the info

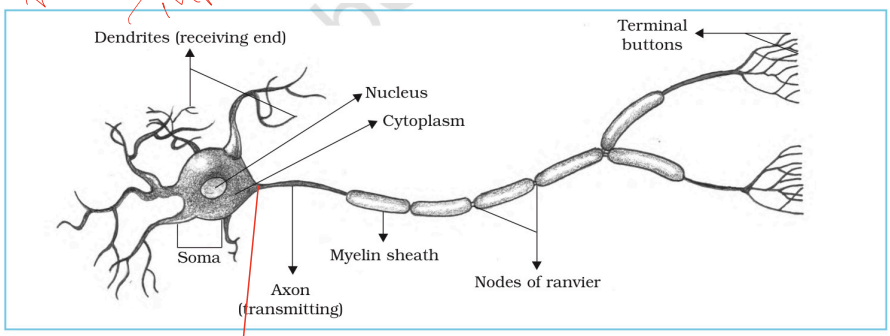
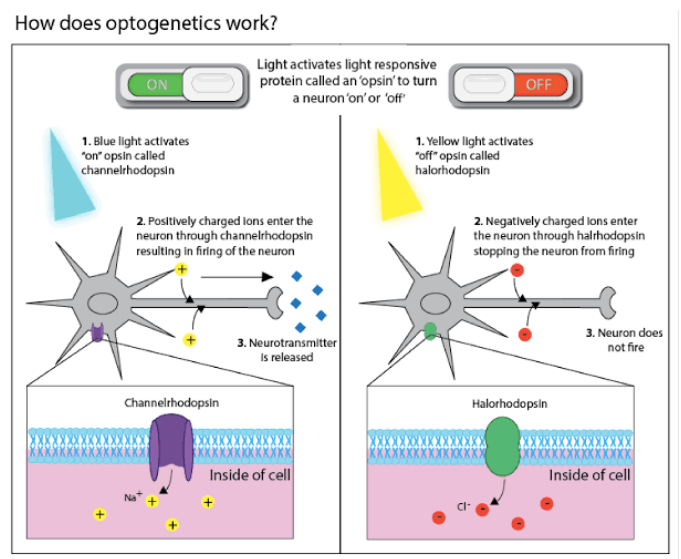


Fig.3.1 : The Structure of Neuron

axon hillrock

Corticotropin inhibiting factors contribute by inhibiting the release of ACTH by the pituitary, instead releasing, possibly, Delta 6 sleep inducing hormone (this is not known for sure). He points out that this makes sense because sleep time is a good time to turn off the stress response and do some repairs.

How neurons keep it on/off



Dale's Law#1 states that once the action potential is reached and the neuron is turned on, it will result in the release of the neurotransmitter from all the axon terminals. (Action potentials work as all or none deals, so once the threshold is reached, it's off to the races.)

In the 1970's (probably) Jerry Letvin published a paper that provided examples of some exceptions to Dale's first law, with some of the action potentials being blocked at the axon terminal site.

The pituitary excretes seven major hormones that can be organized under the acronym FLATPeG. Why this is the best word is not at all clear. The hormones are follicle-stimulating hormone (FSH), luteinizing hormone (LH, ICSH), adrenocorticotropic hormone (ACTH), thyroid stimulating hormone (TSH), prolactin (PRL), beta-endorphin and growth hormone (GH, STH).

this is how →

There are specialized cells within the pituitary that release their specific type of hormone.

Within the hypothalamus, depending on the neighborhood that a cell lives in, the effects of the hormones will vary. There is a lot of communication between the cells and the hormones.

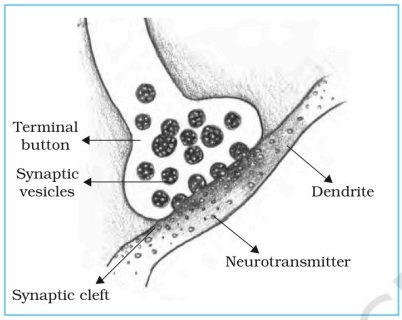


Fig.3.2 : Transmission of Nerve Impulse through Synapse

- How do you understand & identify any neuro-transmitters ?

  ⇒ first you gotta know where it's located. they're not located just anywhere in the brain. they're located in axon terminals.

- Now what triggers the action of a neurotransmitter ?

  ⇒

- what is the effect of a neurotransmitter?

  ⇒

most of the hormones we talked about are neurotransmitters.