Introduction to Psychology
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Lecture – 30
Genetic & Environmental Bases of Behaviour

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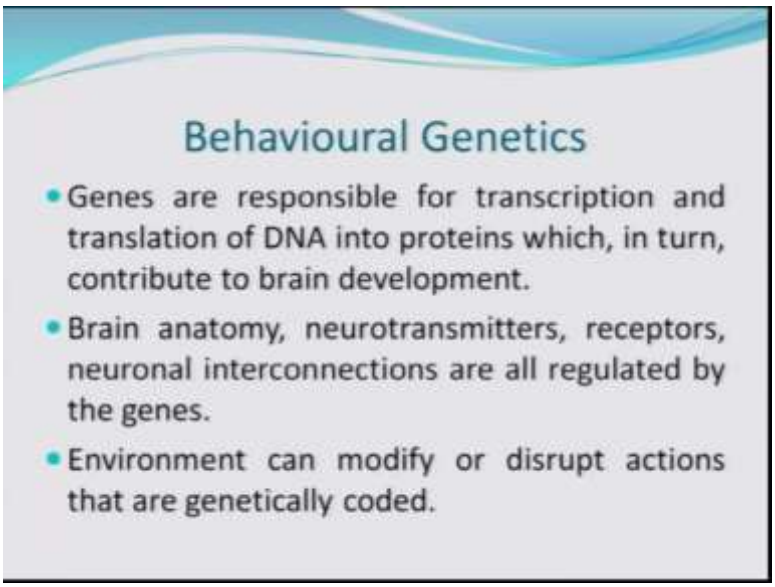

Now, we have understood that genes are basically responsible for transcription and translation of DNA into proteins and they in turn contribute to the development of the brain and it is the developed brain which is responsible for the cognitive viabilities that we displayed and it is this manifested behavior, the displayed behavior as well as the makeup that the back and support which is provided by the anatomy by the neurotransmitters and so forth. These are the subject matter of psychology.

Now, brain anatomy, neurotransmitters, the receptors, neuronal interconnections, they are all regulated by the genes, but what is very interesting is that the environment can, it may modify or it can even disrupt the actions that are genetically coded and we will see this with certain examples

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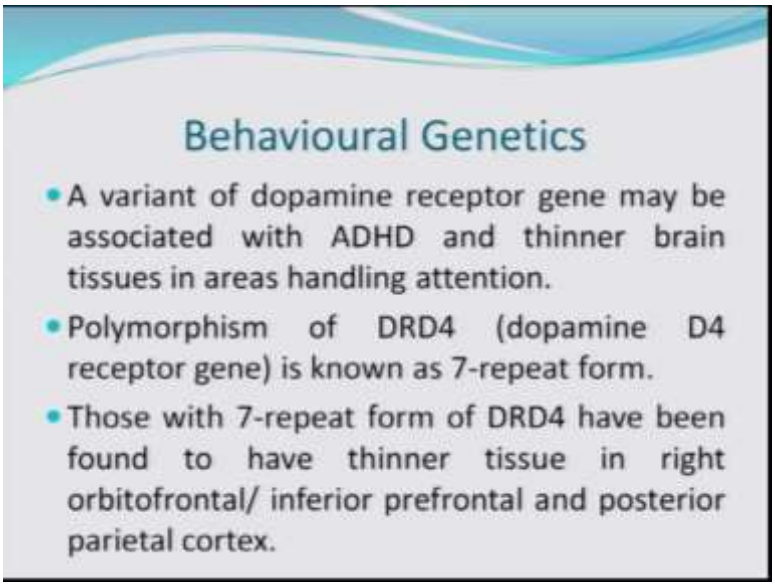

Let us take the example of ADHD. A variant of dopamine receptor gene may be associated with ADHD and the thinner brain tissues in the area that handles attention and ADHD, as we all know that it has to do with hyper activity and as well as attention deficit. Now polymorphism of DRD4 (dopamine D4 receptor gene), this is known as seven repeat form. Now those individuals who have 7-repeat form of DRD4, they have been found to have thinner tissue in their right orbitofrontal/inferior prefrontal and posterior parietal cortex.

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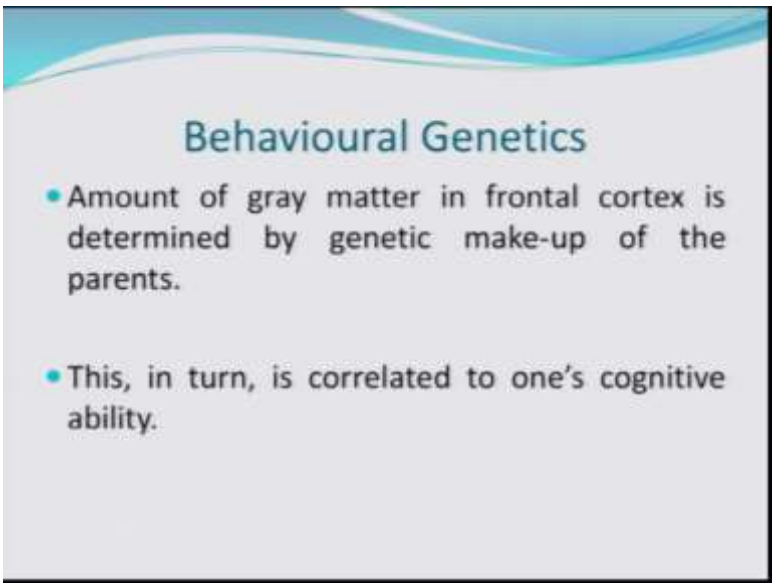

So, this is again the brain anatomy, how the genes regulate them and because there is something, the anatomy is based on the genetic make-up. This genetic make-up in turn finally makes you subject to ADHD one of the disorder say psychology looks at.

The amount of gray matter in pre frontal cortex that is also determined genetically by the make-up of the, genetic makeup of the parents now this, in turn, is correlated with the cognitive ability of the child.

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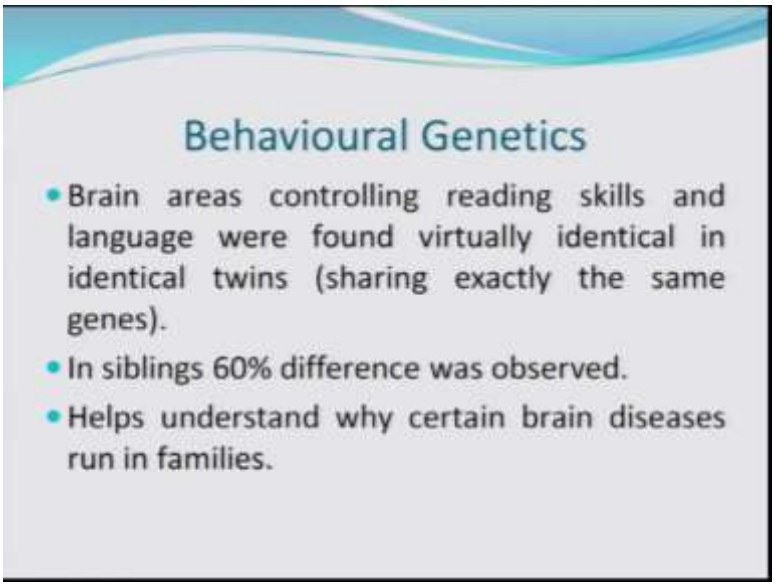

The brain areas that control reading skills and language they were found virtually identical in identical twins and as you know that identical twins are those who share exactly the same genes. Now in siblings sixty percent difference has been observed and this helps us understand why certain brain diseases they run in the family.

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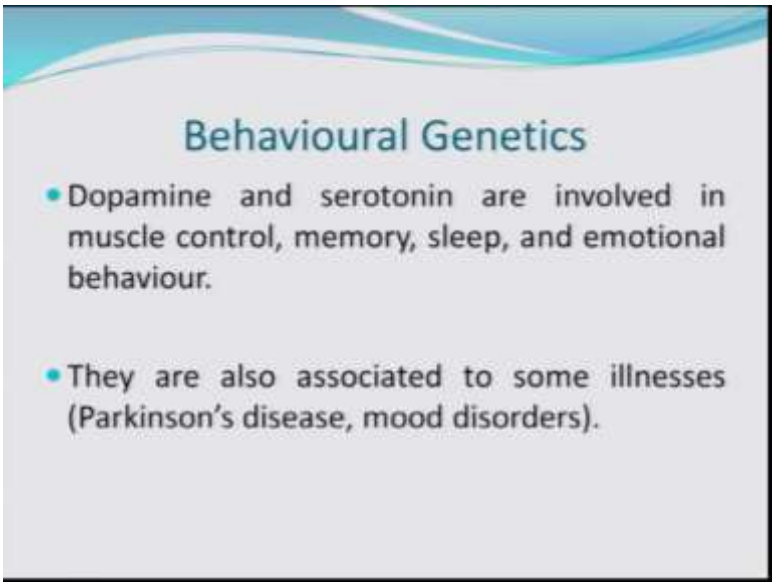

let us come to neurotransmitters dopamine and serotonin ,they are both involved in the muscle control, memory, sleep, and emotional behavior. Now fine muscular movements all types of motor activities memory, sleep, emotion all of them have know prime importance as for as a subject matter of psychology is concerned.

Now, they are also associated to some illnesses also say like Parkinson's disease, now Parkinson is a disease, where the motor movement gets compromised with mood disorders which has to do with the emotional balance or imbalance that you experience. Now let us come to mouse study, study conducted on rats and for instance.

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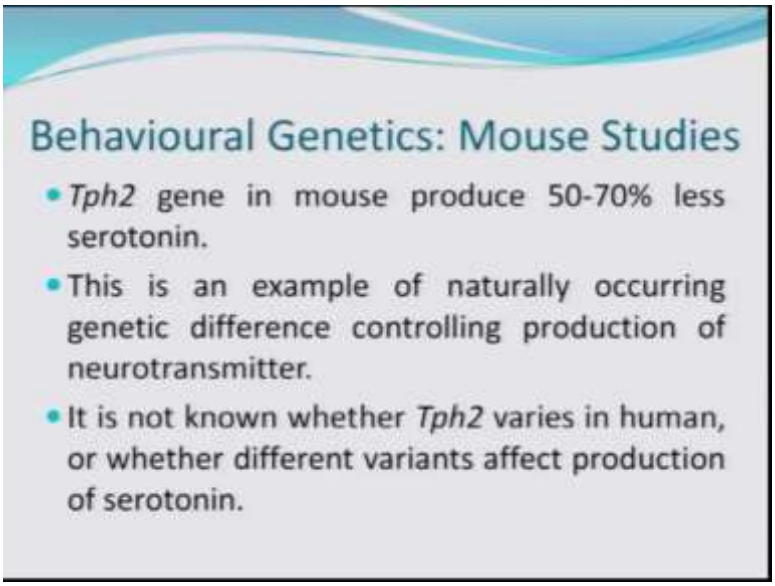

For there in then extended to humans now Tph2 gene in mouse it produces 50 to 70 percent less serotonin. Now this is an example of naturally occurring genetic difference that controls production of neurotransmitter. Now it is not known whether Tph2 varies in humans or whether different variants affect production of serotonins.

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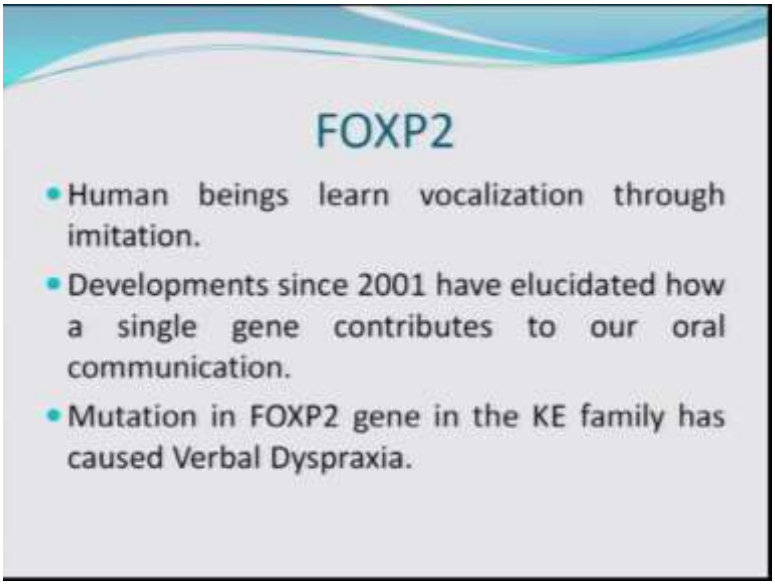

Let us take another example of FOXP2 gene which is basically responsible for oral communication that human being show, now human beings learn vocalization through imitation. So, you see others pronouncing you see the selection of words, how it is

pronounced the vocabulary you know gradually gather and then this is how vocalization and the entire communication develops in human beings.

Now, developments since 2001 have elucidated how a single gene contributes to our oral communication. We have talked about mutation, now mutation in FOXP2 gene in one of the families from United Kingdom named as KE family, this is the code used for them. Now this has caused verbal dyspraxia in the members of that very family.

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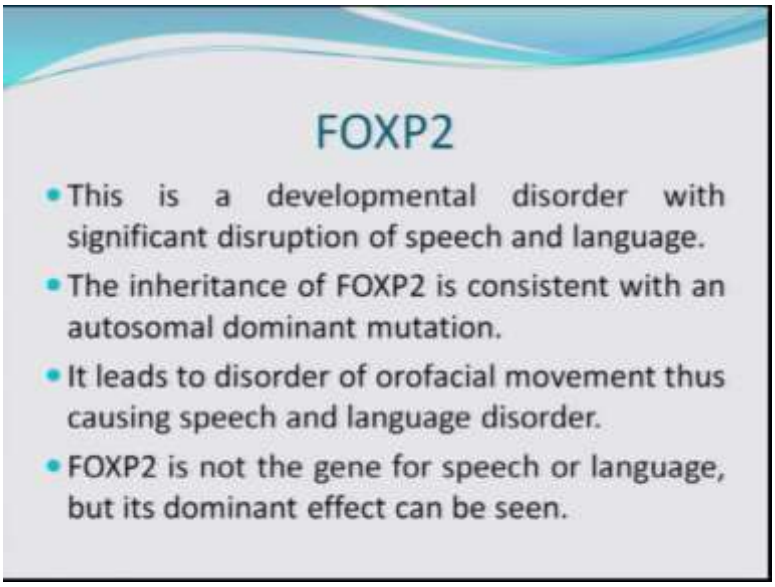

Now this is basically a developmental disorder with significant disruption of speech and language. The inheritance of FOXP2 is consistent with an autosomal dominant mutation. It leads to disorder of orofacial movement. Orofacial means that the facial muscle which is close to this which is also responsible for the oral movement. So, this very part now which helps you rotate your oral capital, the lips, tongue movement and so forth.

Now, this very disorder it leads to the disorder of knows orofacial movement thereby causing speech and language disorder. Now FOXP2 is not the gene responsible for a speech or language, but then it dominant effect can very easily be seen because the sufferer of this very problem suffers from verbal dyspraxia.

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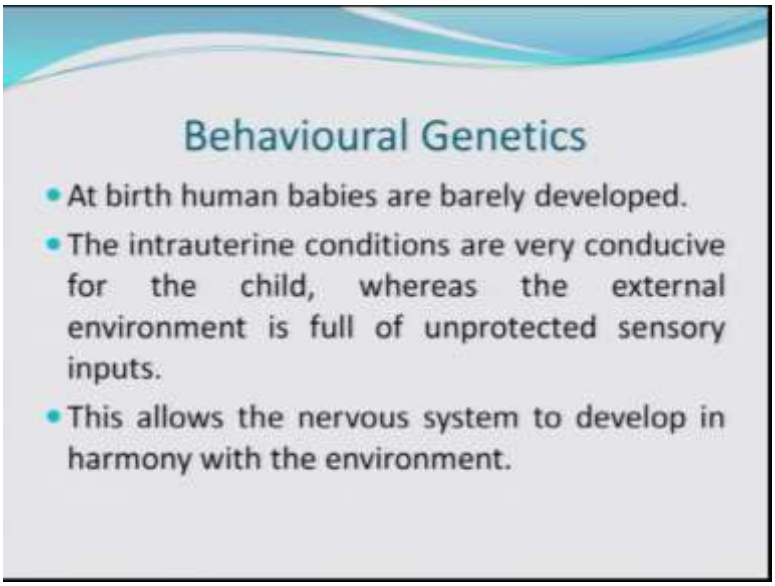

Let us take another example, at birth human babies are barely developed. The intrauterine conditions are very conducive for the child whereas the external environment is full of unprotected sensory inputs.

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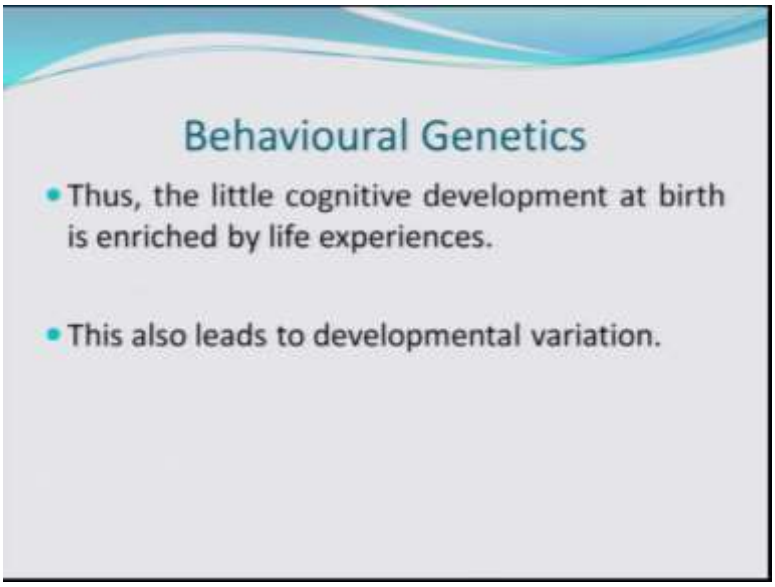

Now, this allows the nervous system to develop in harmony with the environment, but what happens when the child takes birth? The little cognitive development at birth it gets enriched by life experiences and this also leads to developmental variations among human beings.

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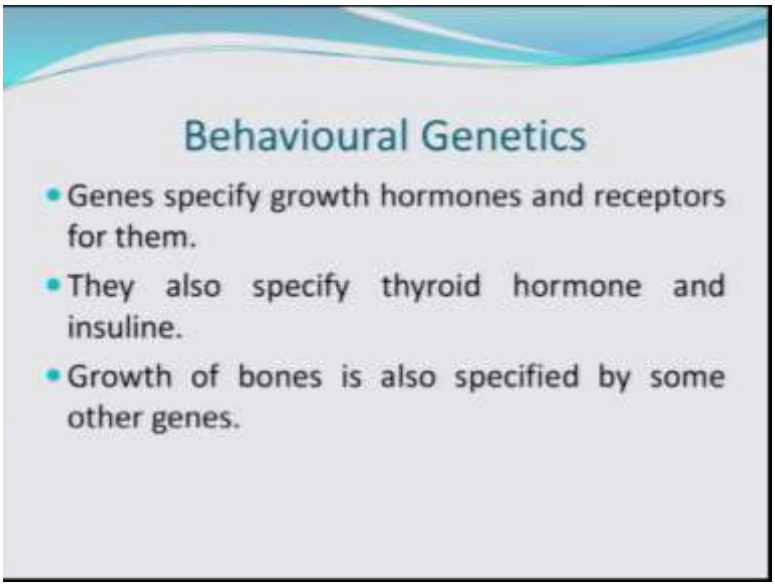

Now genes specify growth hormones and receptors for them they also specify thyroid hormone and insulin. Now growth of bone is also specified by some other genes with these genetic predispositions also, ones height might not achieve full genetic potential if he or she is experiencing starvation during puberty.

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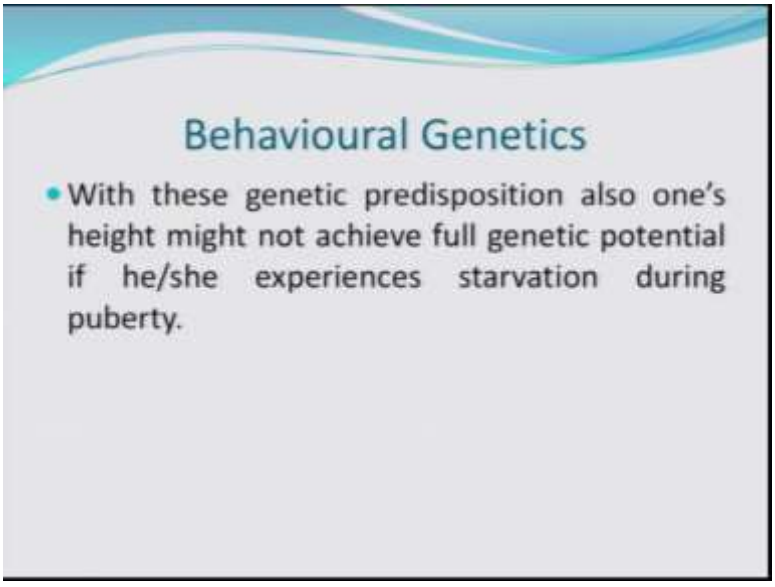

That means that you have the genetically upper disposed secretion of, rather balance secretion the desired secretion of the growth hormone and the insulin, but still if you have any environmental barrier of starvation, the situation in your life

that does not allow you nutritious intake of food starvation in turn despite having you know proper secretion of growth hormone and insulin still can stunt your fullest growth. So, all though you are genetically predisposed, but then you do not achieve your fullest potential.

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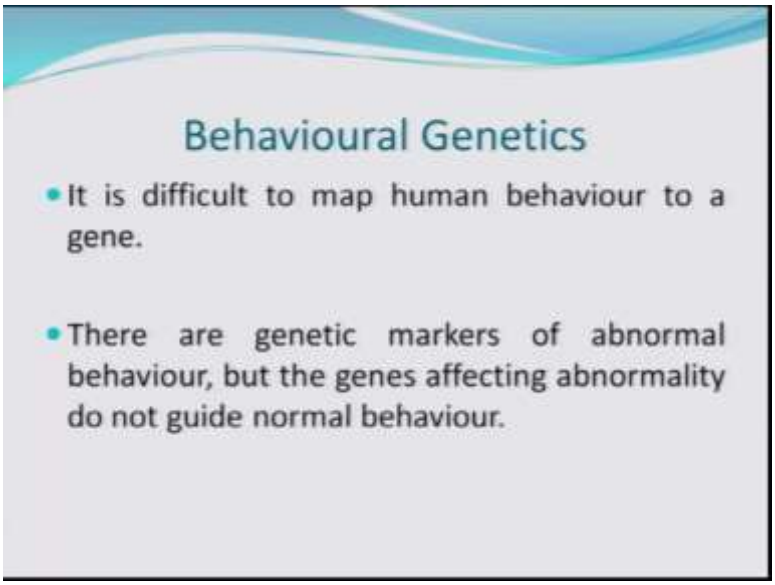

Now, it is difficult to map human behavior to genes, but there are genetic markers of abnormal behavior, but the genes affecting abnormality actually do not guide normal behavior. Basically genetic markers of abnormal behaviors are known, but if you go far a reverse cycle that, is it that now these genes that affects abnormality are they also responsible for guiding the normal behavior ? That is not true.

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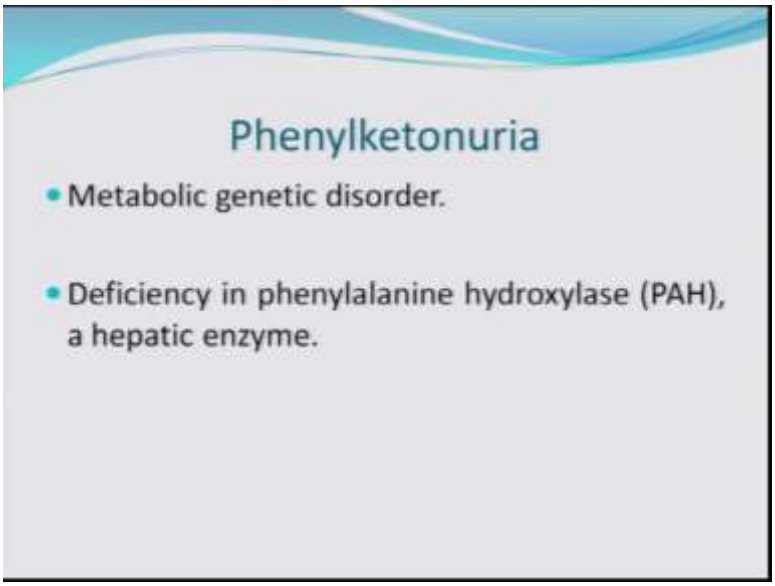

We will take some example let us take the example of phenylketonuria now phenylketonuria is a metabolic genetic disorder where the deficiency take place in phenylalanine hydroxylase the PAH, which is a hepatic enzyme.

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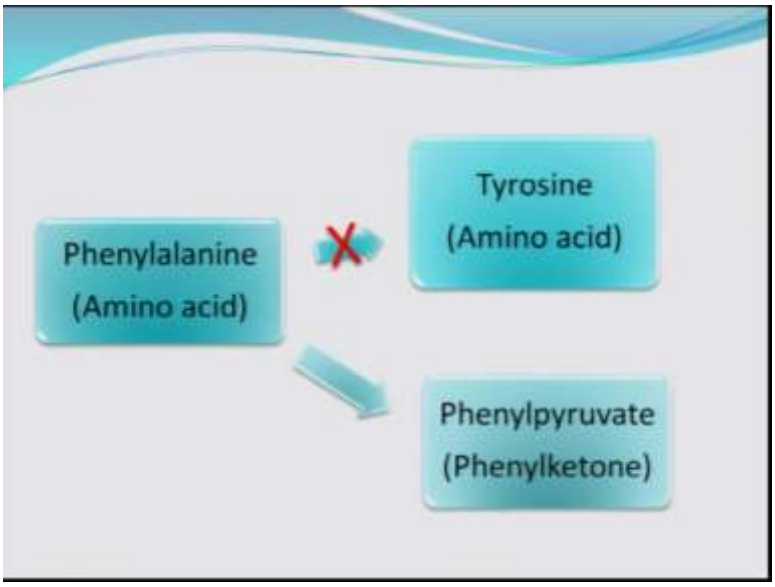

Now what happens actually in normal situation phenylalanine, which is an amino acid should actually convert into tyrosine. Now in the patients of phenylketonuria what happens this does not take place and in turn the phenylalanine it gets converted into Phenylpyruvate which is also called as Phenylketon.

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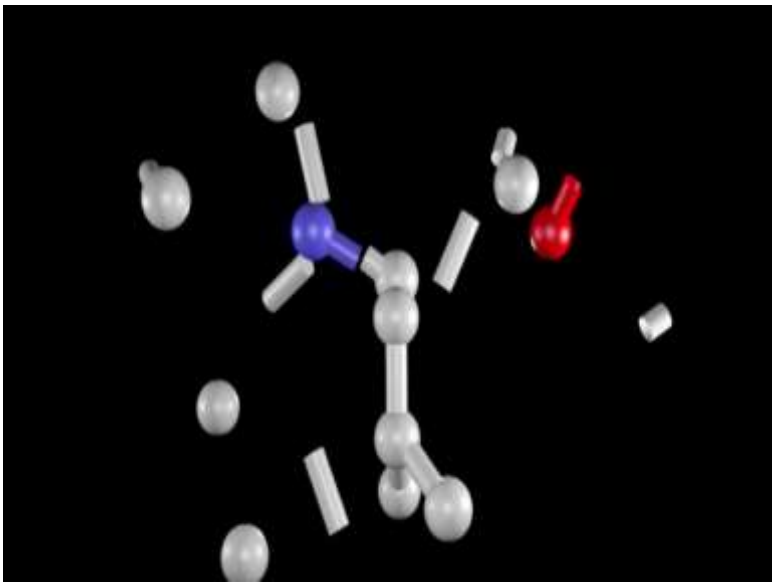

Formation of new cells and repairing of tissues require proteins, proteins are made up of amino acids. Unwanted amino acids are broken down and eliminated outer the body through urine, phenylalanine is one of amino acids. Individuals with phenylketonuria cannot process phenylalanine.

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And it is start building up in their blood this in turn adversely affects neurons. Children with untreated phenylketonuria tend to have a smaller than average head, this is known as Microcefalia.

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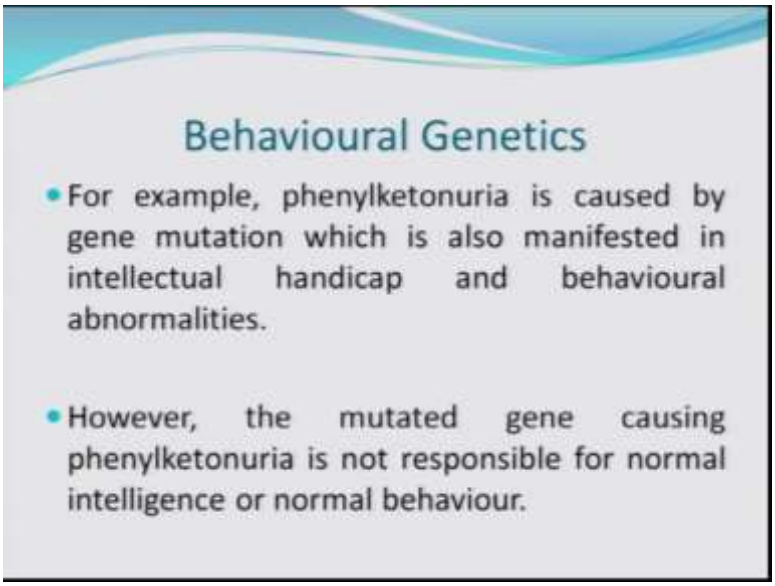

So, if you take this very example of phenylketonuria you realize that basically this is something that is caused by gene mutation which is also manifested in intellectual handicap and behavioral abnormalities. So, basically it has to do with metabolic disorder, mutation in the gene which does not allow the chemical to get broken down in the desired manner. So, phenylalanine which should have actually taken a different route suddenly now you realize that, now it gets converted into Pheenylketone and in turn this metabolic problem affects the intellectual ability of the individual which is also responsible for certain behavioral abnormalities. Now intellectual handicap one would ideally you know map it to the psychological aspect of the behavior, same is the case with behavior abnormality, but when you realize that basically it is the problem breaking down of one chemical in the body which is responsible for this then you realize that there is a genetic root to such type of intellectual inabilities and behavioral abnormalities.

Now, the mutated gene causing phenylketonuria is not responsible for normal intelligence in human beings. So, it is very interesting the mutated gene that we find responsible for phenylketonuria actually is not responsible for intellect. So, intelligence is not controlled, not moderated, not guided by this gene, but then you realized that this gene somewhere adversely affects the breakdown mechanism the metabolism and the metabolic problem actually results into intellectual impairment, there by adversely affecting the normality of the individual.

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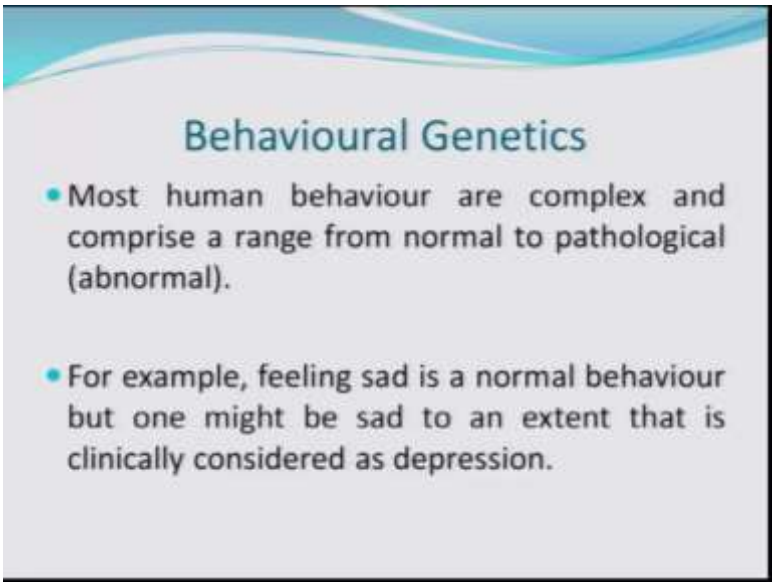

Now, most human behavior is complex and they comprise of a range from normal to pathological. If you remember we had talked about know the full range of possible reactions from normal to pathological in our introductory session let us recollected and then come to an example, now feeling sad is a normal behavior because of certain environmental condition you feel sad, but then your sadness is different when you compare it with depression. So, feeling sad is a normal behavior, but then if one might feel sad to the extend you are considered to be clinically depress then sadness no more remains sadness. The magnitude of it has change the whole nature of sadness is changed..

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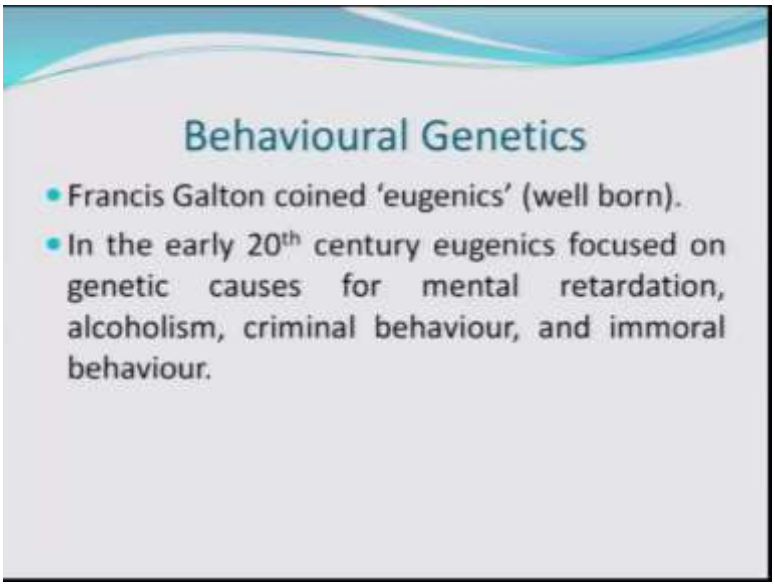

We have talked about Francis Galton when we were going through the schools of thoughts historical mile stones and he was the one who also talked about eugenics in the early twentieth century 'eugenics' focused on genetic causes for mental retardation alcoholism criminal behavior as well as immoral behavior.

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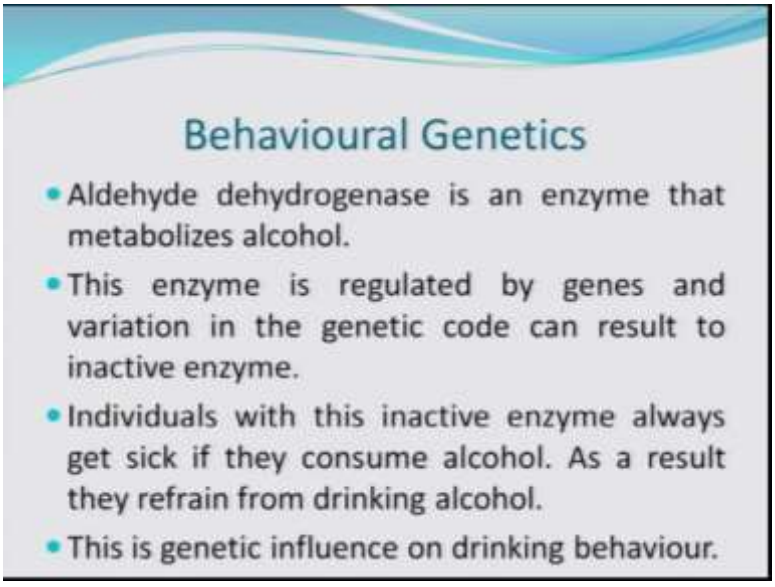

Let us take some examples from the modern study Aldehyde Dehydrogenase is an enzyme that metabolizes alcohol. Now this enzyme is regulated by genes and the variation in the genetic code can be result into inactive enzyme. So, if you have any

inactive enzyme in your body then such individuals will always get sick if they consume alcohol.

Now, if you fall sick because of the absence of this enzyme in your body then, the behavioral consequence of it would be that you will always try to refrain yourself from drinking. Now this is a basically a genetic control over your alcoholic behavior.

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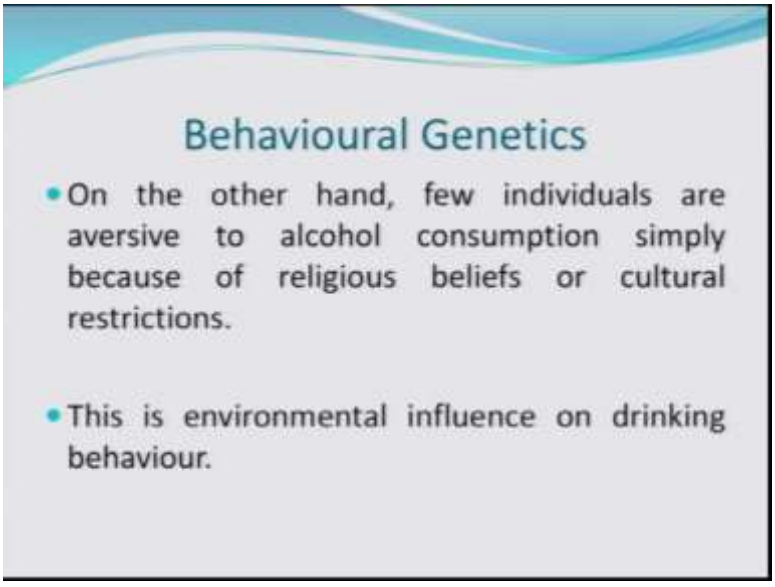

You would never prefer drinking you will never feel taking alcohol simply because you have a metabolic issue, you have the absence of the enzyme. Now think of the other side of this story, there are religious belief and there are cultural restrictions which basically prohibits you from consumption of alcohol. Now this is an environmental influence on the drinking behavior. So, in the previous case when we were looking at this enzyme which basically metabolizes alcohol you have a genetic control over alcoholism where as when we took the other case of religious belief and cultural restrictions this is an environmental control over your drinking behavior. So, drinking behavior remains the same, but the control at one and you find that there could be a genetic basis of control and there could also be environmental bases of control. So, this is an interesting you know example where you realize that drinking behavior gets moderated because of both the factors, genetic as well as environmental factors.

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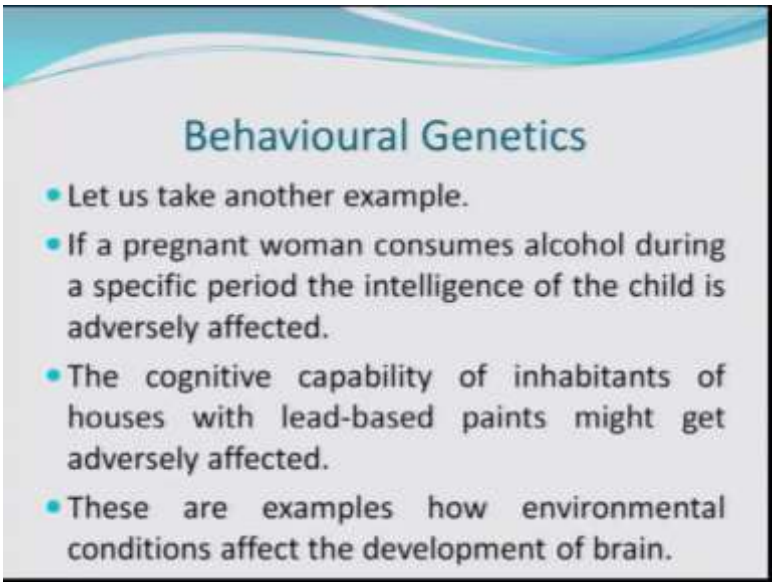

Let us take another example if a pregnant woman consumes alcohol during a specific period it has been found at the intellectual ability of the child gets adversely affected. Now the cognitive capability of inhabitants of houses where the walls are painted with lead based paints that might also adversely affect the intellectual ability of the child, cognitive capability of the child. Now this is an example of how environmental conditions they affect the development of our brain. So, in one case the mother consumes alcohol and then you realized that this has adversely affected the intellectual capability of the child in other case you will realize that the walls are painted you know with lead based paints and this in turn adversely affects the cognitive capability of the child. So, again both the conditions and you realize there is a intellectual capability is compromised only because of these factors. Let us come to another interesting psychological behavior a construct which is again well researched in psychology novelty seeking.

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Your search for novel experience .Now this trait is reportedly correlated to identical twins a studies have confirmed association between novelty seeking behavior and D4DR neuro-receptors.

Now, D4DR we have talking about it this is a dopamine D4 receptor and the neuro-receptors are also responsible in mood regulation.

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Let us take the example of dyslexia the image that you see here inverted h for example, mirror image of s. For example, now this is how dyslexic children they write

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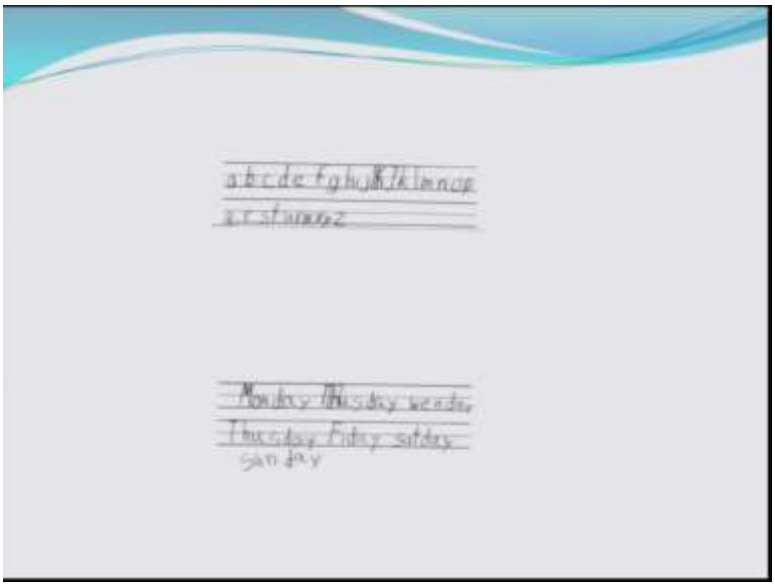

You can see here on your screen, on the top you have the English alphabets and on the lower side, you have the days of the week and you realize that there are certain alphabets where mirror images have been used and this is one of the problems in the dyslexic children.

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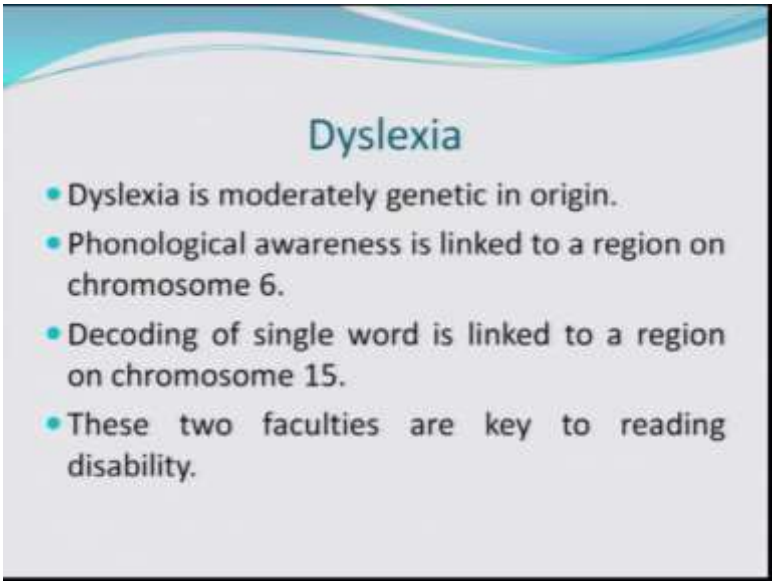

Now, dyslexia is a moderately genetic problem, the origin is moderately associated with the genes the phonological awareness is linked to the region on chromosome 6. Decoding of a single word is linked to a region on chromosome 15 and you know that

these two faculties are basically key to reading disability. So, simply because phonological awareness gets compromised because of chromosome 6 and then you realize that the word, how to decode a word, that has to do with chromosome 15 and because these two thinks have some problem. Therefore, you suffer from dyslexia. So, this is the genetic origin of dyslexia.

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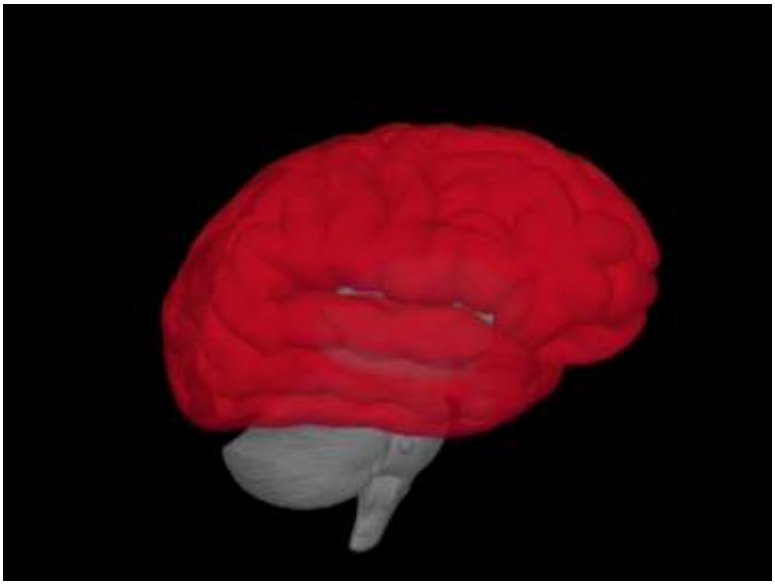

You are looking at two hemispheres of the human brain language is primarily considered a left hemisphere function this is the temporal lobe of the left hemisphere.

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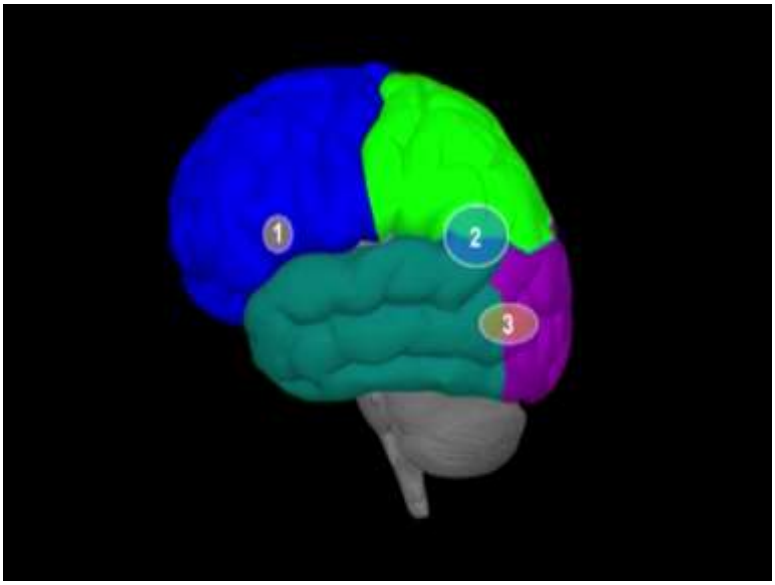

You now see the four lobes the frontal parietal temporal and the occipital lobes. During formal education we are trained for different reading and writing skills. The 3 neural systems for reading are located in the left hemisphere of the brain you see them as regions one two and three on this screen one is in the region of the inferior frontal gyros which is also known as Brocas area, 2 is in to Pareto tempore origin and three is in the occipital temporal origin or which is also known as visual word form area that is v w f a, this place significant role in fluent reading, it has been observed that compare to normal individuals, those with dyslexia have less gray matter in the left Pareto temporal area. Many of them also have less white matter in the same area and this white matter could adversely affect communication between the brain area the interior systems in those with dyslexia is slightly over activated compared to non impaired readers. In contrast the two posterior system are under activated the under activation of the left posterior reading system is the neural signature for dyslexia.

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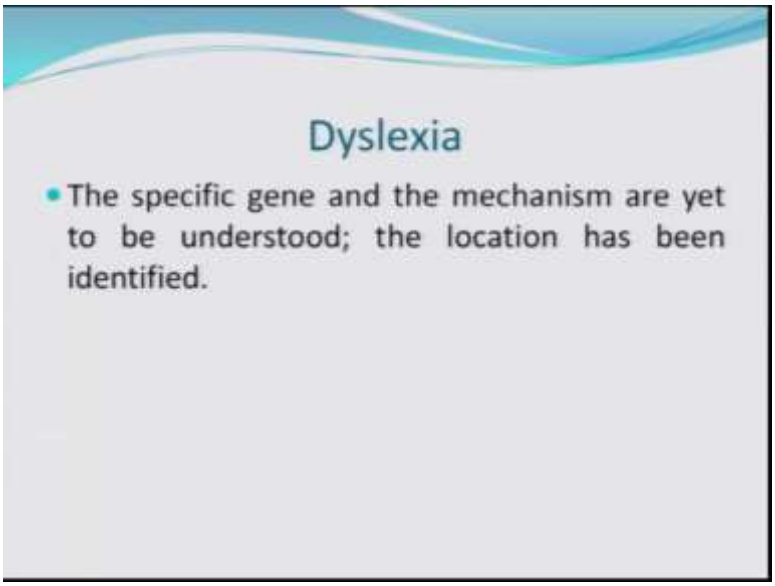

Now, the specific gene in the mechanism of dyslexia is yet to be understood, but at least the location is known. Now you know that chromosome six ,you know chromosome fifteen and both of them play an important role in this learning disability.

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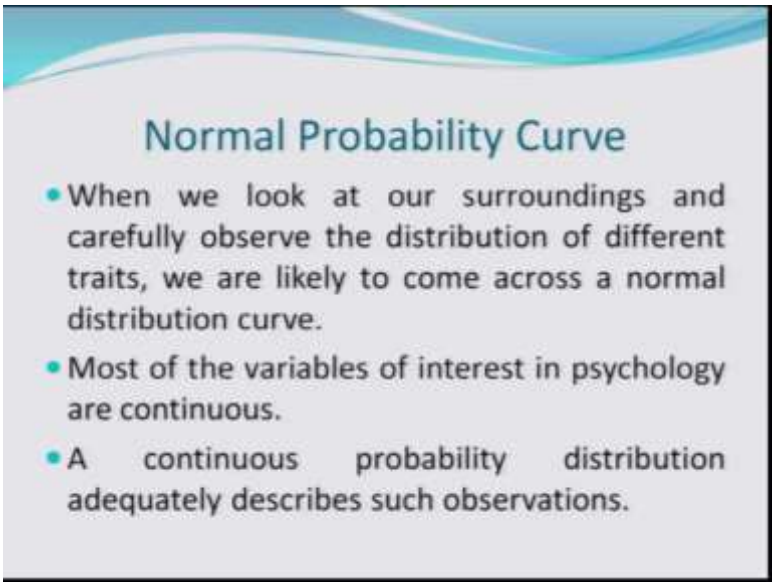

Now, if you look at literature in psychology, you have whole length of description on dyslexia. So, this an interesting way of looking at psychological phenomena, the constructs which are examined in psychology ,studied in psychology and then you realize that we have both the genetic control over all types of psychological construct as well as the environmental conditions that also affect the psychological constructs we will continue with this when we meet next.