Lecture 30
Dyslexia

Hello and welcome to the course, introduction of the psychology of language. I am Ark Verma, from IIT Kanpur. And we are running in the sixth week of the course. Today is the last lecture of the week. And in this lecture we will be talking about, dyslexia. Now dyslexia, as you might know is a difficulty in reading, irrespective of say, for example, you know other cognitive attributes being. Alright? Individuals might face difficulty in reading. So, say for example if the IQ is alright if other you know, developmental aspects are completely working fine, intellectually and other you know, motor another cognitive function,

what you find. And still the person is sort of, finding it difficult to read that is usually where dyslexia kind of you know can be diagnosed. And we will kind of talk about, certain aspects, of dyslexia not really there disordered in so, much detail. But we'll probably talk a little bit about how the specific deficits in dyslexia can be handled by various of the models of word recognition that we've talked about so let us not waste,

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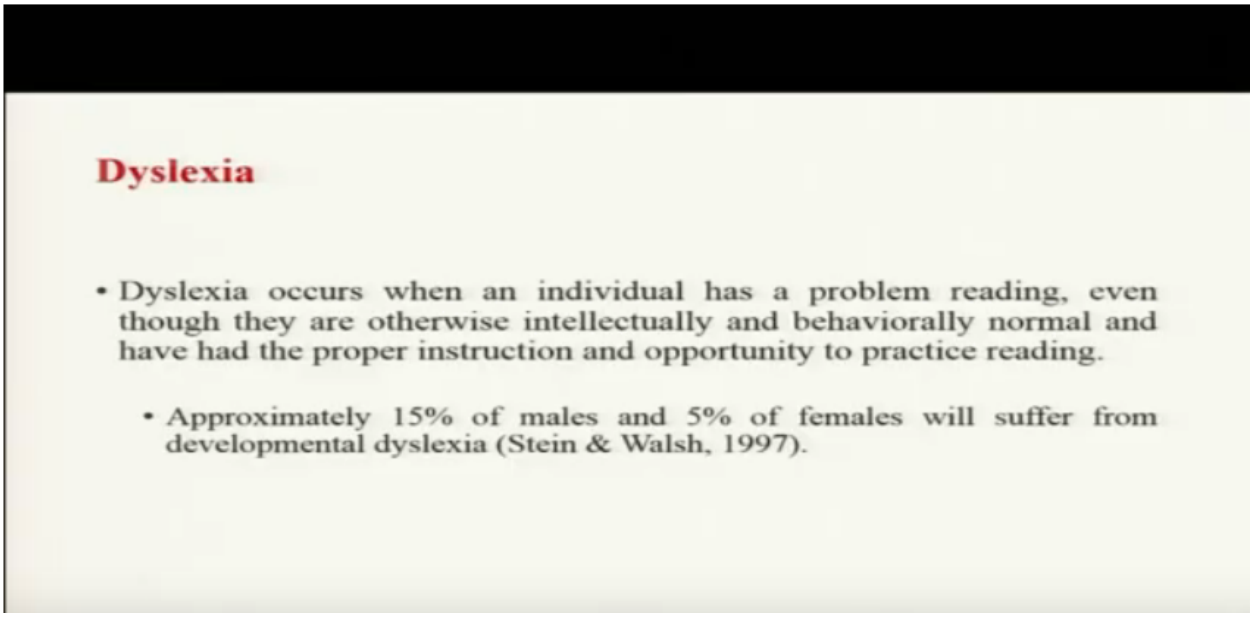

a lot of time and start, with this now dyslexia basically, occurs when an individual has a problem with reading, even though they are otherwise intellectually and behaviorally normal. And have had proper instruction and opportunity to practice reading. So, the last part is also very, very important now sometimes, people would have difficulty reading, because they have not got the best of the instruction, because they have not practiced, hard enough because they have not been exposed to correct practices of reading, however dyslexia can only be diagnosed if both, their you know the behavior and the intellectual parts are normal and also in after enough practice, after enough exposure to reading, the individual is still finding it difficult, to learn to read, approximately, say for example as far as the Western data is concerned, approximately about 15 percent of males and around 5 percent of females, will of what is called developmental dyslexia, basically they are not able to learn to read. Okay?

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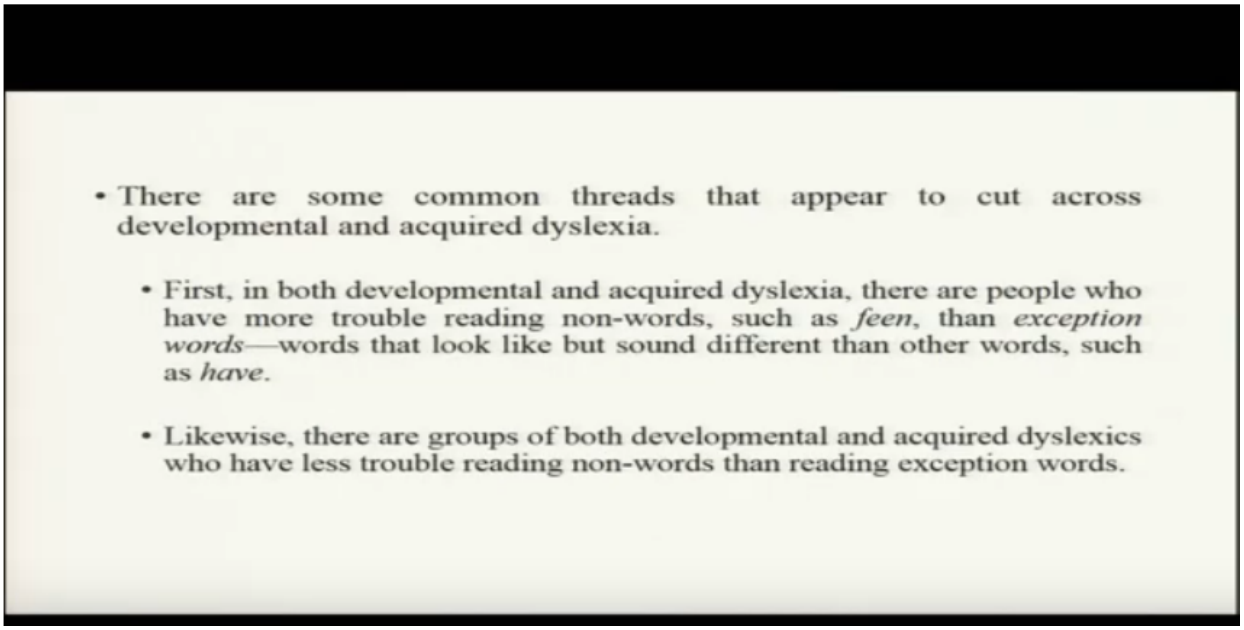

There are also say for example, common you know traits between developmental dyslexia. And acquired dyslexia, acquired dyslexia could be say for example, the ability to learning, to read ability of learning to read is lost on the way, but say for example, in developmental dyslexia, it is that the individual could not acquire the skill of reading anyways. Now the commonalities that we are talking about, basically could be say for example, in both developmental and acquired dyslexia there are people, who have trouble reading non words, such as fene and then exception words, words that look like but sound different than other words such as have you know, we compared to have and save. So, non words basically you know so people in both developmental, dyslexia and acquire dyslexia might, have trouble in reading non words. Okay? Basically telling us that you know the assembled phonology, route might not really be working fine. Now let's really talk about that in more detail. Now in both, cases in the groups of both developmental and acquired dyslexics, who have sometimes less, trouble reading non words, than reading exception, verses sort of a dissociation can be seen, in some patients find it more difficult to reach non words. But find it you know easier, to read exception words, whereas some patients will find it easier to read exception words, but have more difficulty to read non words or maybe I just mix it. So, exception words, non words some people are good at exception or at non words, some people are poor at exception good at non words so, something like that.

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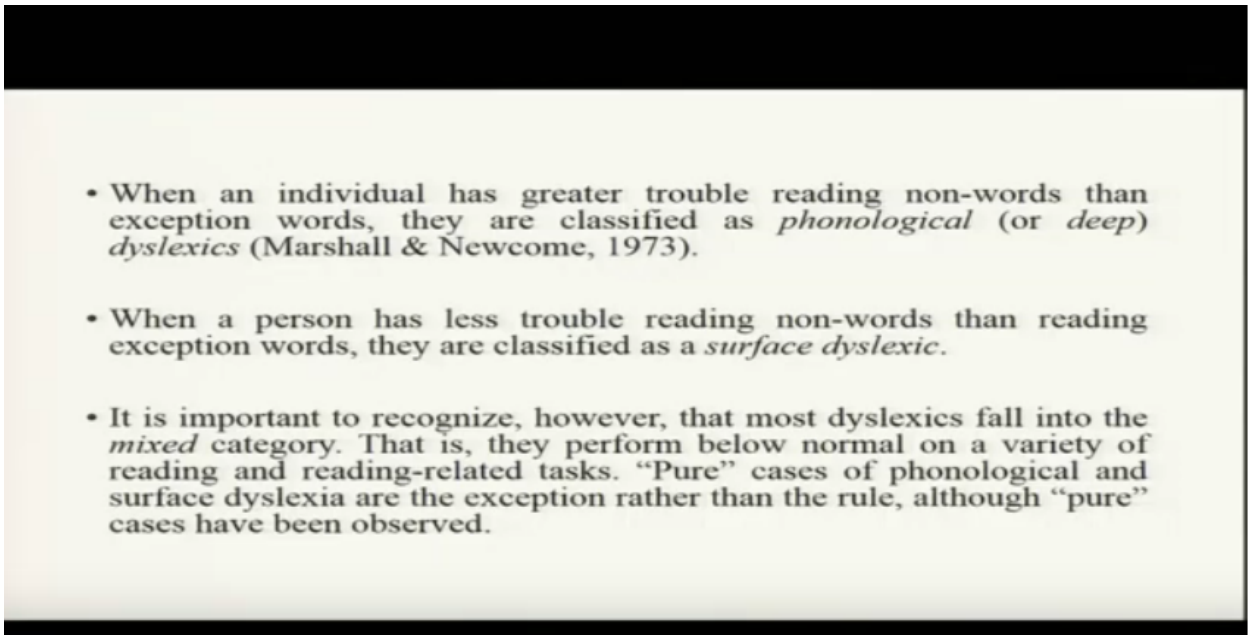

Now when an individual has greater trouble reading non words, than exception words, they are classified as phonological or deep dyslexics .Basically, because they are assembled for analogical route is not really working fine, they are not being able to create grapheme to phoneme conversions and then concatenate, basically sound level representations of each of the letters, to create the sound level representation, of the entire word. When a person has less trouble reading non words that they're you know, phonological route are working fine, but they have difficulty reading exception or it's like you know, pint and have and tio2 and geo go etc. Then basically they are referred to as surface dyslexics basically, the directory route is probably you know at problem here. Because while they can still do the phonological grapheme to phoneme conversion, they cannot really come up, with the direct pronunciations, of these words. Now in this sense, it is important to recognize that most dyslexics, fall into mixed category, most dyslexic will have some problems with the exception words. And some problems with non words it's not really a very neatly categorizable, you know deficiency that somebody would only and only, have problems with non words and somebody else would have only and only problems with exception words. Okay? So, pure cases that's why as I was saying pure cases of either phonological dyslexia or surface dyslexia are more like exceptions, rather than being the norm now.

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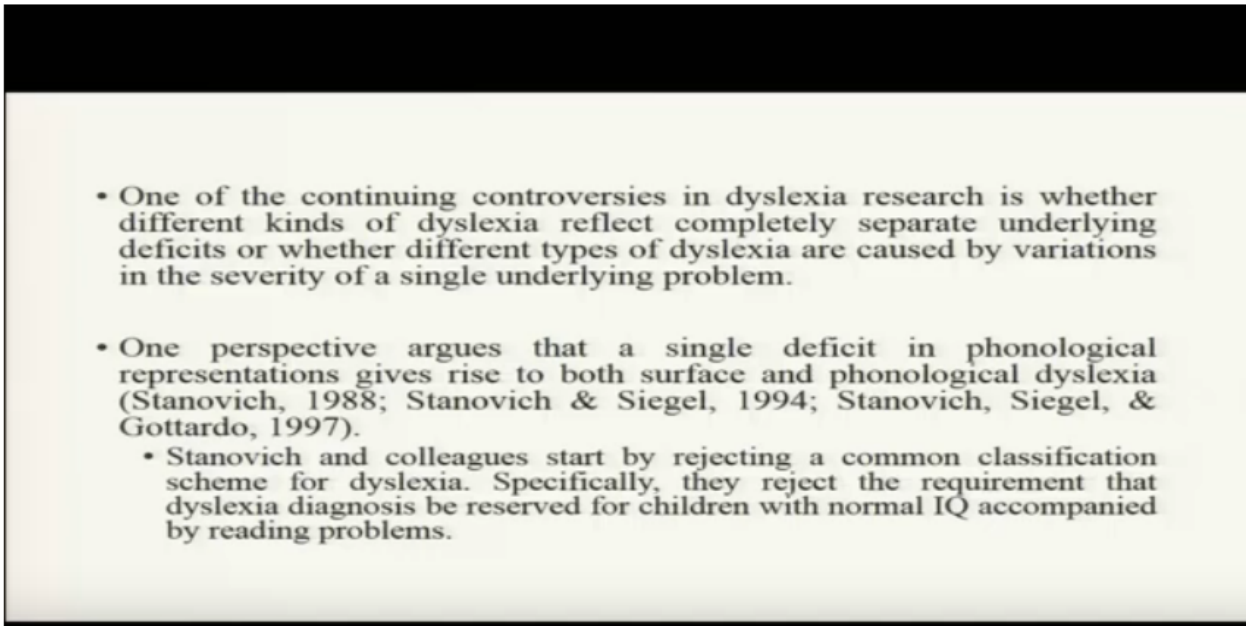

One of the continuing, controversies in this lecture research, is whether different kinds of dyslexia actually, reflect the deficiency in different kinds of you know underlying deficits or underlying processes or whether dyslexia is caused by a single underlying problem, which affects both the processing, of non words and processing of exception words. Now one of the, one of these kind of you know perspectives that have sought to understand dyslexia, basically argues that a single deficit in phonological representations, gives rise to problems, with both the phonological dyslexics and surface dyslexics. This was put forward by Stanovich colleagues and Stanovich; basically start by rejecting a common classification scheme for dyslexia. They basically say, they reject the requirement of dyslexia diagnosis, can be reserved for children with normal IQ accompanied by reading problems they say, say for example it cannot be seen like that, they note that this definition implicitly assumes that reading problem problems of people who had higher, the intelligence have a different you know, locusts or have a different source, as compared to the reading problems of people with lower intelligence they say, intelligence has nothing to do with it and should not really, you know factor in, in the definition of dyslexia as such. Okay? Because also because there is no direct evidence that they found that supports this assumption. And the distinct they kind of reject this distinction altogether this dyslexia is this problem with reading, irrespective of whether you have a higher in normal IQ or a lower IQ.

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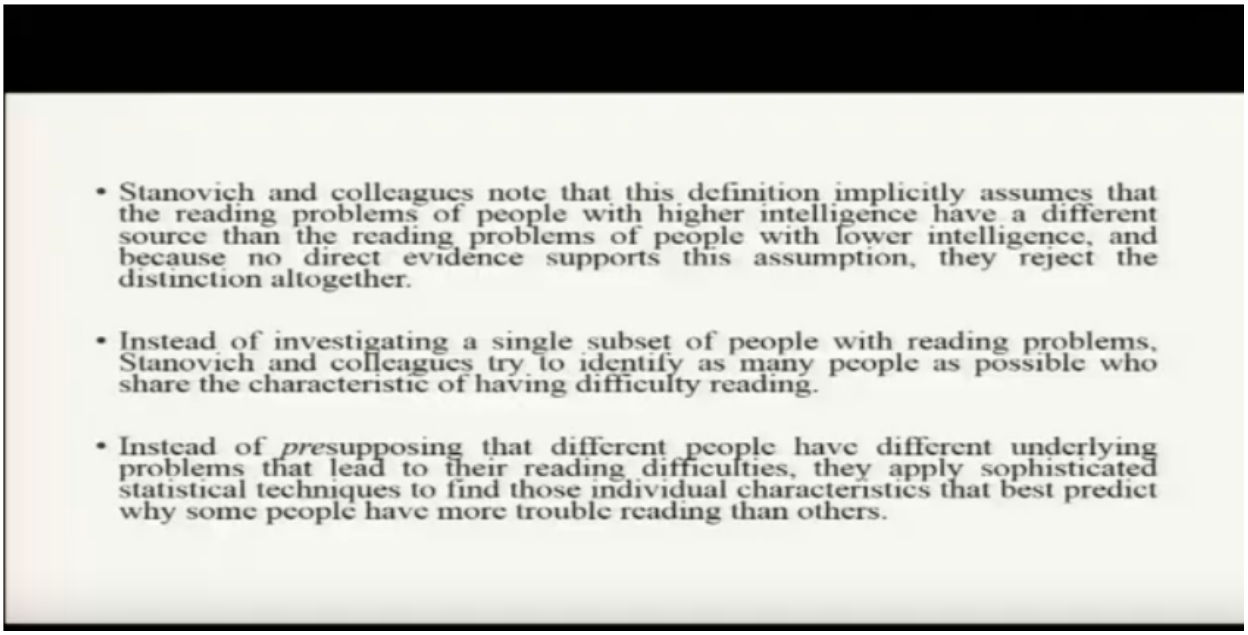

Now what they do is that instead of investigating, a single subset of people with reading problems, Stanovich colleagues try to identify as many people, as possible who shared the characteristic, of having difficulty reading. So, for example you will not really only go to you know normally intellectually functioning people and see their, reading problems you will go to everybody and you will see what kind of reading problems, exists in the population. And then you'll try to understand that instead of presupposing that different people, have different underlying problems that lead to reading difficulties, they basically Stanovich apply sophisticated statistical techniques, to find those, individual characteristics that best predict their reading problems that people might face.

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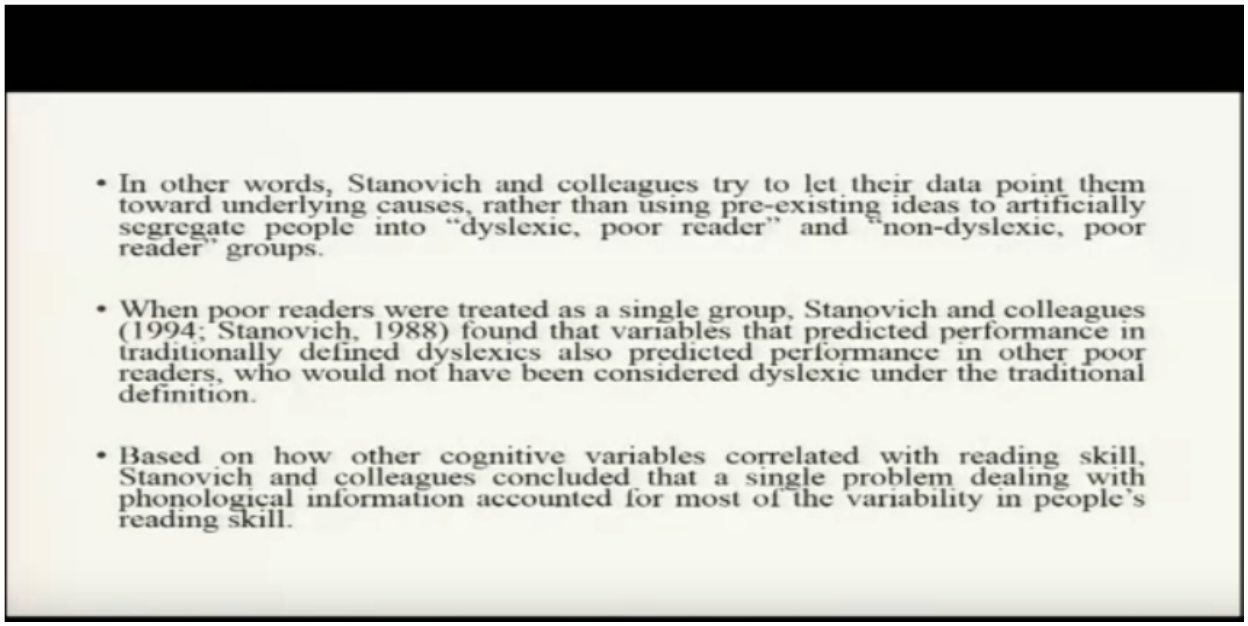

In other words what they're trying to do? Is they're trying to let their data do the talking they're trying to. Let that data, point towards the underlying causes and then they can use this data to, basically classify, the people into dyslexic, poor reader, non dyslexic, poor reader, kind of groups. Okay? Now when poor readers in this kind of a classification, are treated as a single group, what happens is that in a sandwich and click find variables that critic performance, traditionally you know define dyslexics. And also predict and the same variables per have found to predict performance in other poor readers, who are traditionally not classified as dyslexics. Now also say for example, you can kind of in this sort of method find other cognitive variables that could be correlated, with reading, a Stanovich increase basically concluded that that is why there could be just a single problem, dealing with, the you know dealing with the specification of the phonological information, which kind of accounts for people's variability in reading performance. If you remember in the last lecture I was talking to you about phonological awareness and I said that, phonological awareness could be you know, a basic predictor of how well a person is going to be able to read eventually, it is very similar to what the Stanovich and clicks have found and they say that basically a single problem, dealing with phonological information's about. How well you understand that a large sound can be composed of so, many different sounds. And you can kind of you know delete sounds or add sounds or blend sounds together. That kind of knowledge, could be able to you know explain, people speeding these difficulties, the irrespective of the fact that they are high IQ or low IQ, irrespective of the fact that whether they are classified, as actually select 6 or not as dyslexic but poor readers.

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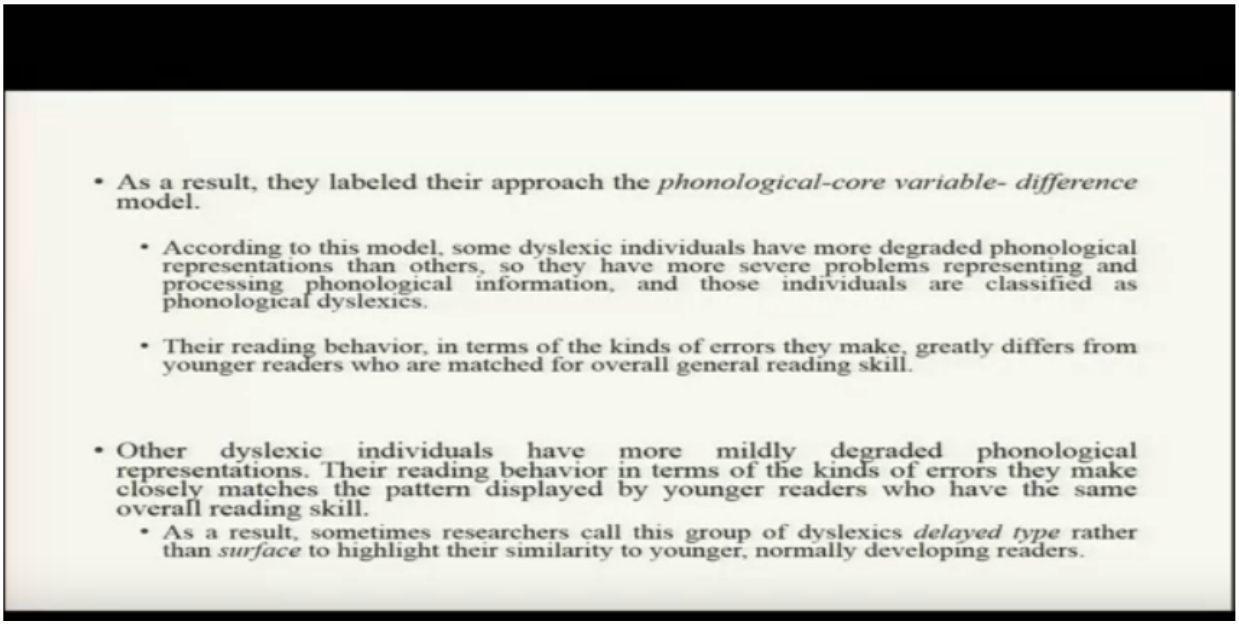

As a result basically they call this as the, phonological core variable difference model. So, they say the basic difference is between the phonological cores, according to this kind of model, some dyslexic individuals have degraded phonological representations, as compared to others and that is why they have more severe problems, by representing the N proper processing the phonological information. And then these individuals are classified as phonological dyslexics. The, the reading patterns, of the phonological dyslexics and the kinds of errors, they make basically differs a lot from younger readers, who are matched? Who are generally measured overall you know other kinds of skills, general reading skills for

example, other dyslexic individuals, which have slightly mildly degraded phonological representations, their reading patterns and the kinds of errors they make would, be would basically, closely match the pattern displaced by younger readers, who have the same overall reading skill. Okay? As a result, sometimes the researchers call you know basically call this group, as delayed type dyslexics rather than surface dyslexics, to highlight their similarity to younger, you know normally developing the readers.

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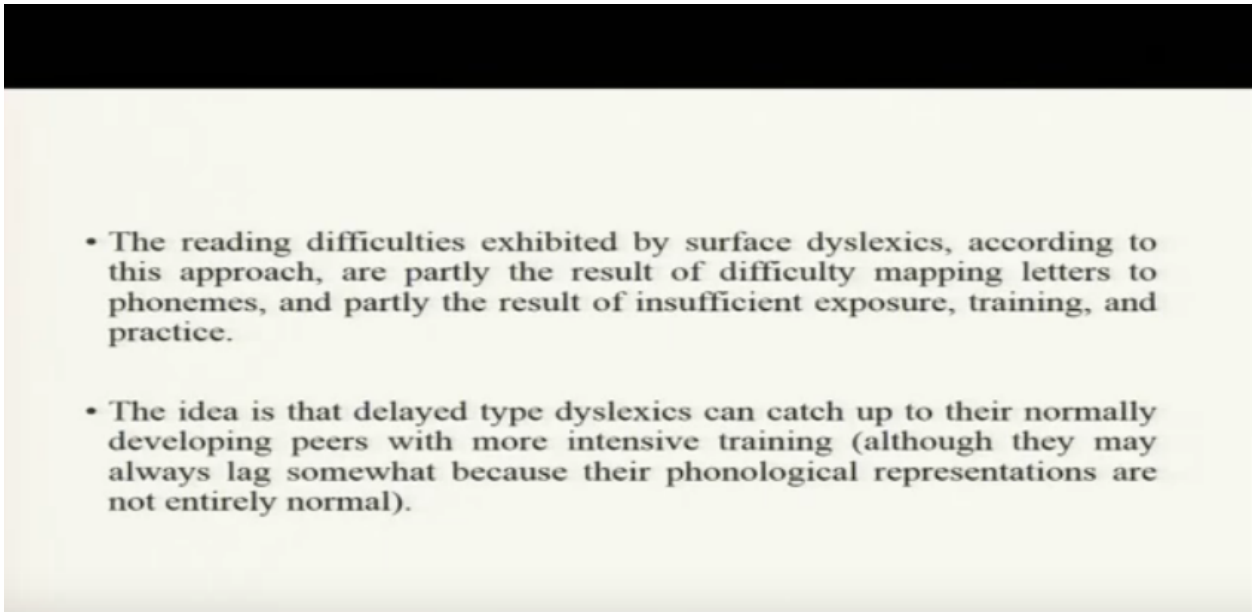

Now basically the reading difficulty is exhibited surface dyslexics according to this kind of approach, the phonological variance, variable model are partly the result, of different difficulty in mapping, letters to phonemes. So, this is the basic problem that is happening with these individuals. And partly the result of insufficient exposure and training and practice. So, these people basically have not really got, sufficient in for information sufficient opportunity to train sufficient opportunity to practice, how you know these skills need to be, learned now either idea, basically here, is that these delayed types like six can catch, up to they're normally reading pairs, with more intensive training with the say for example training of on tasks of phonological awareness and so, on so, forth.

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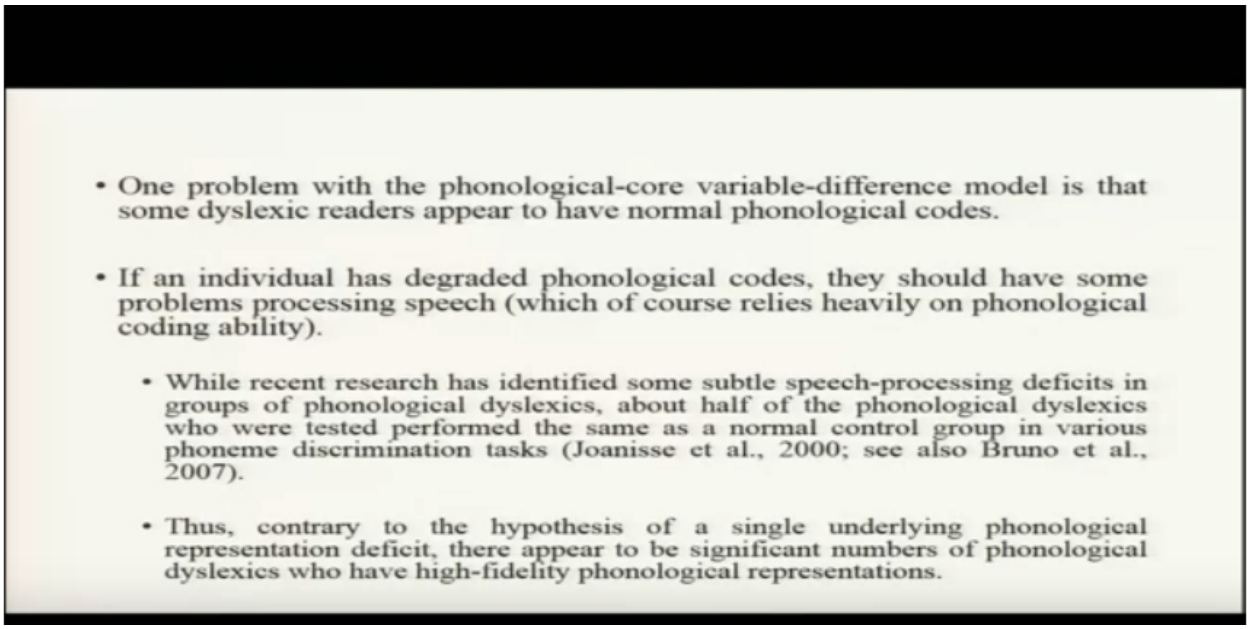

Now one of the problems aware with this phonological code, variable difference model, is that some dyslexic readers appear to have normal phonological codes, whereas other dyslexic readers have degraded severely or mildly degraded phonological codes. Now if you kind of look at it and if you say that if an individual has degraded phonological codes, they should have at least some problems processing speech. Okay? They should have some problems in understanding and listening perceiving speech as well. But recent research kind of identifies, there's some subtle speech processing, deficits and groups of phonological dyslexics, happen about half of the phonological dyslexics were tested, for performance or you know tests basically, tested who were tested performed about the same as a normal control group, in various phoneme discrimination tasks. So, this is sort of a you know problem, with the for logical code difference model, which basically says that contrary to the hypothesis of a single, underlying phonologically deficit there appears to be significant number of follow logically dyslexics, who have high fidelity for logical representations. So, therefore this idea of ,a single variable you know, single phonological core variable, that kind of you know defines everybody's reading problems, might not really be very correct after all, even though it kind of does a decent job, of you know offering diagnostics and training.

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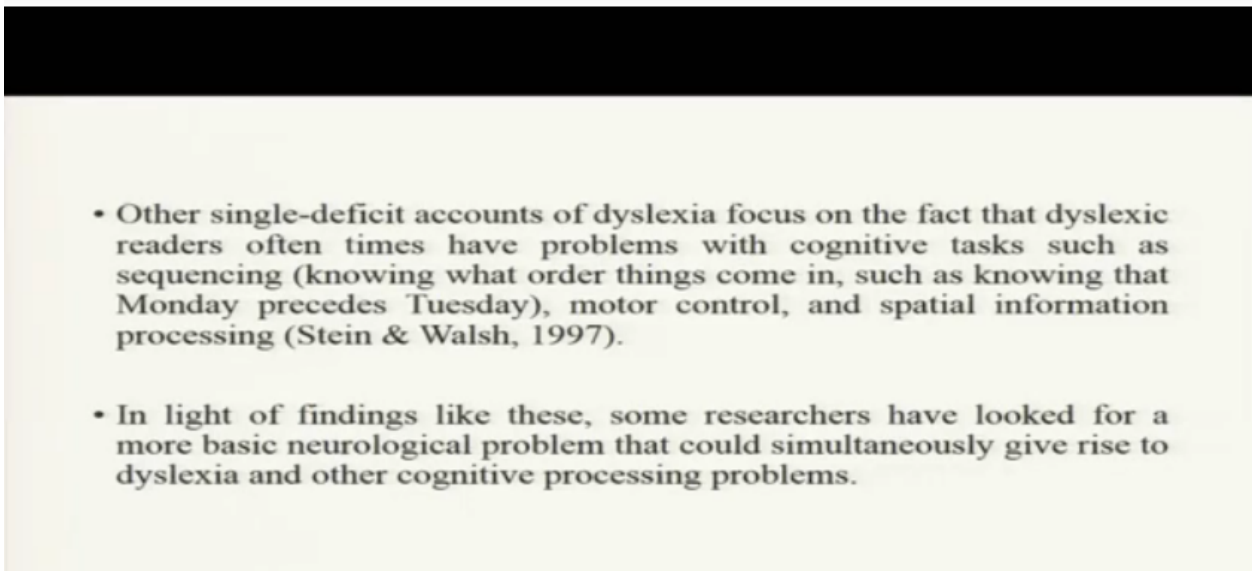

Now other types of des deficit accounts, basically of dyslexia, focus on the fact that dyslexic readers often also, have problems with other cognitive tasks such as sequencing, motor control and spatial information processing. So, basically the idea is that they kind of have data, which shows that there are other problems happening as well. So, what is the deficit that could be common, to all of these from those reading problems, racial problems, motor problems, sequencing problems that is dyslexic patients are kind of facing? Now in light of these kinds of findings, some researchers have looked for a slightly more, basic slightly more deeper neurological problem that could simultaneously give rise, to dyslexia and also the other kind of cognitive deficits.

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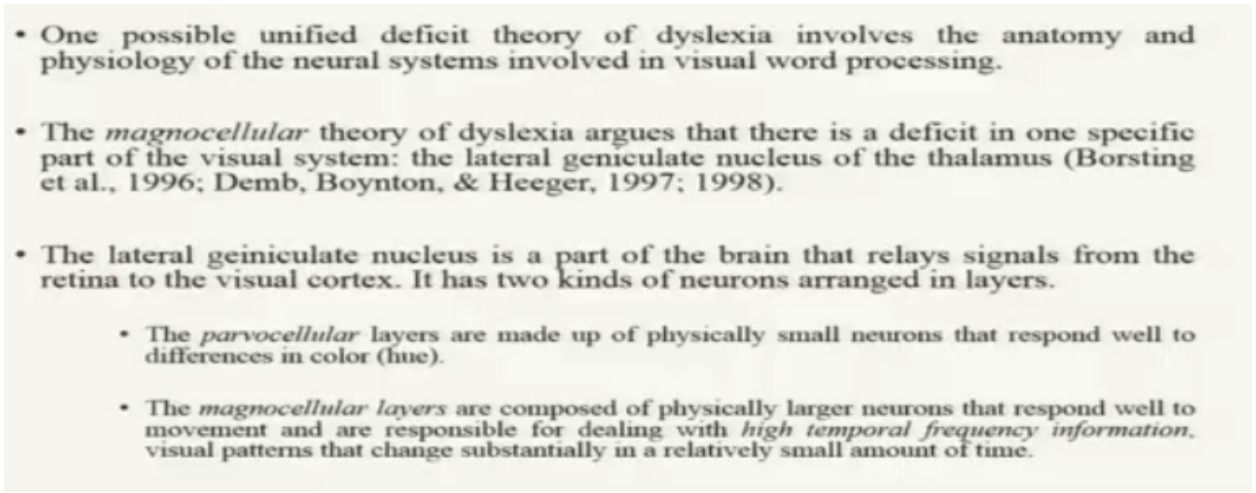

One of these, theories basically, one of the possible unified deficit theory of dyslexia, therefore in was the anatomy and the physiology, of the neural systems, involving visual, word processing. So, the eyes and the and those kind of systems. A similar theory is the magnocellular theory, of dyslexia, the magnocellular theory of dyslexia basically argues: that there is one deficit, in that in the specific part of the visual system, basically the later algeniculate nucleus, now if you remember the organization of the visual

system. I've talked about it, in a different course basic cognitive process, which is also going out, you, can kind of go and look at that chapter. But, if you look at how, the visual system is organized, basically you have the eyes and the eyes kind of from the eyes, there is the system of optical nerves and the optical nerves kind of go and they kind of cross, somewhere which is called a you know, optical chiasm and then go to into the cortex. Somewhere before it kind of reaches there, it kind of passes through this set of cells, called the, 'Lateral Geniculate Nuclei' and this is literally Geniculate nucleus, is basically there at the thalamus, it's kind of you know, a part of the brain: that relieves the signals from the retina, to the visual, cortex and it has two kinds of neurons, arranging these layers, there are parvocellular layers and them magnocellular neurons. The parvocellular layers are basically made up of physically small neurons: that respond well to differences in colors and the magnocellular layers are composed of physically, slightly larger neurons, they respond well to movement and in and also, are responsible for dealing with high, temporal frequency information you have, to quickly, read you know, the information changing in time. Visual patterns that change, substantially in a relative amount of time: that is the you know, job of this magnocellular layers.

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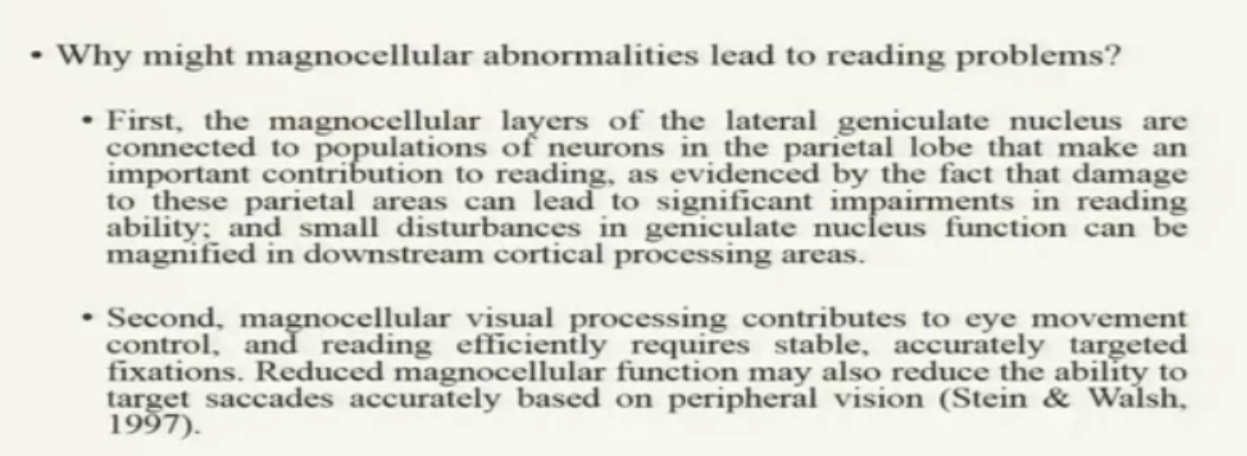

Now, why should it be and you might ask, why should it be that the magnocellular layer or the deficits in the magnocellular layer, lead to problems with reading. There are two answers. First, is that the magnocellular layer, of the lateral Geniculate nucleus, are connected to populations of neurons in the parietal lobe: that make an important contribution to reading: that is one, as evidenced by the fact that damage to these parietal areas can also, lead to significant, impairment in reading ability and small disturbances in this lateral Geniculate nucleus, function can be magnified downstream, towards the cortical because this is the relay area. So, if the information is not really handled very well here, it will not really go in a very, vertical fashion to the in a very, correct fashion to the higher cortical area. So, it will kind of magnify the problem, with the system is you know, slightly a problem problematic here. Second is, the magnocellular visual, processing layers, basically also contributes to eye movement, control and reading efficiency and this basically I mean, it's known and we've been talking and I think, in the first and the second lectures that, you know, reading efficiently requires, you to have very good control, on your eye movement, you know, it requires you to have very stable, accurately targeted fixations. So, there are these two reasons, where you can kind of think: that the okay, the definitely the magnocellular layers

might be involved here and that is why? The magnocellular layers, deficit in the magnocellular layer, might lead to problems here.

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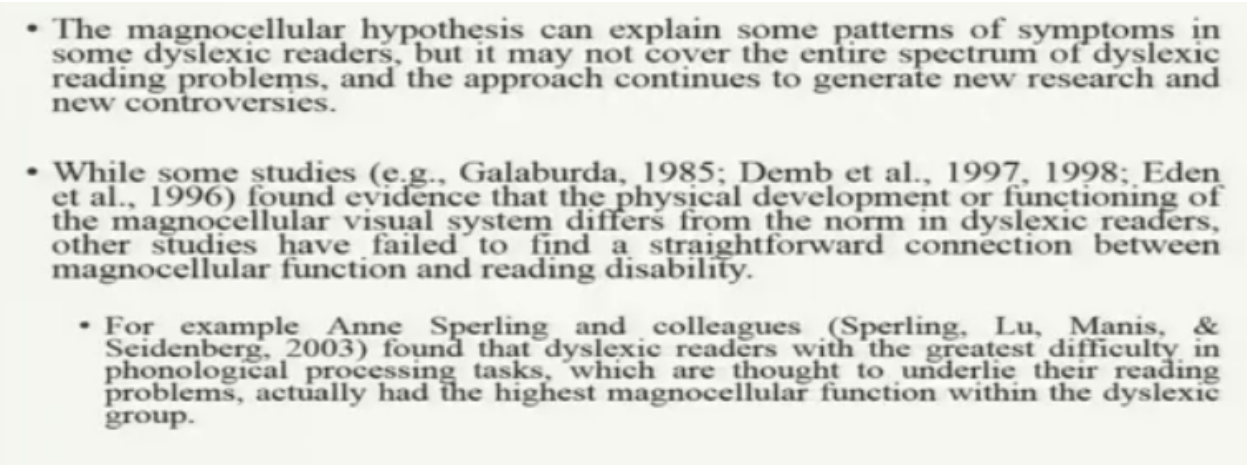

Now, the magnocellular cellular hypothesis basically can explain some patterns of symptoms in some dyslexic readers, but it does not really, cover the entire spectrum of dyslexic reading problems and therefore the approach continues, to generate new research and new controversies. While some studies say for example, you know, Galaburda in 1985and Eden and colleagues in 1996, found evidence that the physical development or functioning, of the magnocellular visual system, differs, from the normal in dyslexic leaders, other studies have really, failed to find a straightforward connection, between deficits in the magnocellular layer and people's reading problems. For example, Anne sperling and colleagues, they in 2003, founded dyslexic readers with greatest difficulty in phonological processing tasks, were thought to, you know, which are thought to, underlie their reading problems, actually had the best magnocellular function, within the dyslexic groups.

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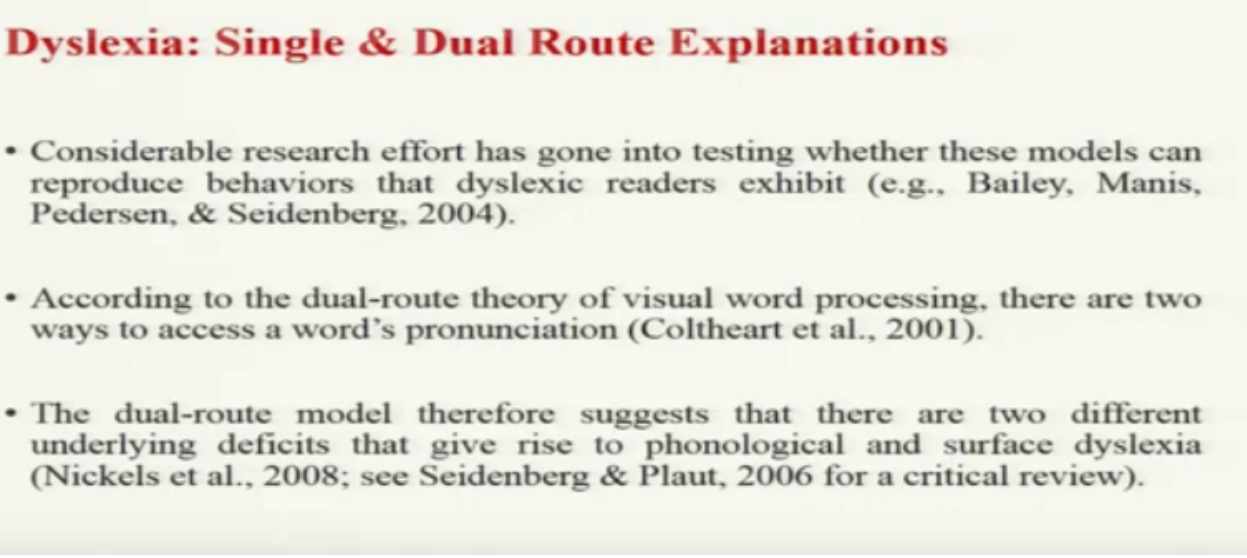

So that kind of you know, does not really follow very well. Now, this was some of the single route theories, single deficit theories of dyslexia. Let us move on, to how dyslexia can be explained by the single route models of word reading versus dual route models of word reading. Now, also considerable research has gone into testing whether, these models the dual route cascaded model or the single route models, can reproduce behaviors that dyslexic readers exhibit. So for example, if these models reproduce similar patterns of deficits, as actual dyslexic readers, you can kind of figure out, what pattern of difficulties, led to this kind of performance and it might be a good clue, to check as to, what really has happened with the dyslexic readers in the first place. Now, according to the dual route theory, of visual word processing, there are two ways to access our pronunciation, as we said, the assembled phonological route and the direct orthography route. Now the dual route model, therefore suggests: that there are two different underlying deficits: that could give rise to phonological dyslexia and surface dyslexia. Let's look at them, look at that in a bit more detail.

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- Damage to the *assembled phonology* route leads to phonological dyslexia, as individuals lose the ability to "sound out" words that they have not seen before.
- Damage to the direct route leads to surface dyslexia, as individuals are compelled to "sound out" all words, even words like *have*, *pint*, and *yacht* that cannot be "sounded out."

Now, damage to the assembled phonological route, would lead to phonological dyslexia and the individuals will you know, lose the ability to sound out, the words that they have not seen before. I was saying, this earlier as well a brief, damage to the direct route, basically would read leads to surface dyslexia, as individuals will find difficult to come up, directly with the pronunciations, of the exception words like bouquet, psychology and so on.

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- Single-route models of reading view word reading as resulting from the operation of a unified neural network, with different kinds of impairment resulting from different types of damage within the system (Harm & Seidenberg, 2004; Woollams et al., 2007).
- Phonological dyslexia can be modeled within this framework as resulting from damage to units that represent the phonological (sound) codes needed to pronounce words.
- Surface dyslexia can result from changes to other aspects of the model, such as the number of processing units available to the system (which can be thought of as the amount of processing resources that the system can dedicate to the task), or the rate at which the system can learn from feedback.

Now, the single route models of reading, view word reading as resulting from the difficulty, reading from the operation of unified neural networks. They say, this is a single neural network and this is basically,

what will be damaged in order to lead to the different kinds of impairment in dyslexia. Phonological dyslexia and these kind of models can be you know, seen as resulting from damage to units that represent the phonological information: that is the phonological units, surface dyslexia can result from changes to other aspects of the model, such as the number of processing units available, to the system basically, you know, going from phonology to meaning and phonology to orthography and so on.

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- So, while single-route models view non-word and exception-word reading as being governed by the same sets of representational units running the same processes, different kinds of dyslexia reflect different underlying deficits, as different kinds of damage to the system produce different patterns of behavior.

So, while single group two models, view on-word and exception word reading as being governed by the same sets of representational units. And running the same processes, different kinds of dyslexia, could reflect different underlying deficits. So, as different kinds of damage to the system, can produce different kinds of behavior. So, what is really happening here is? In the single route models, because they view non word an exception word reading as being governed by the same, sets of representational units, running the same kind of processes, different kinds of dyslexia basically, can come up, if there are different patterns of damage to these same units.

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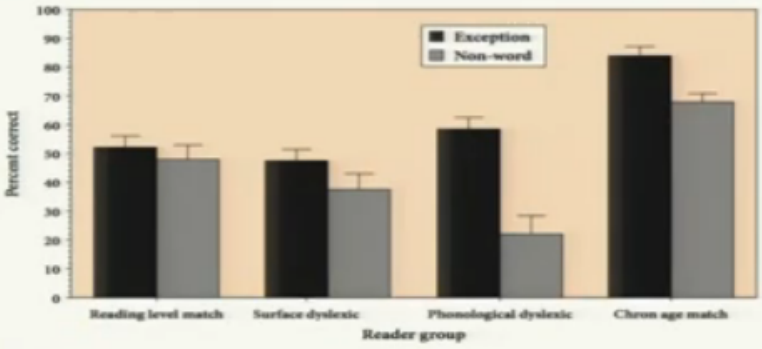

Figure 10.10 Accuracy at reading exception words (e.g., *have, pint*) and non-words (e.g., *bint, tade*) for surface and phonological dyslexics compared to reading level (left) and age-matched controls (right) (from Bailey et al., 2004, p. 141). Notice that surface dyslexics are about equally impaired on exception and non-words, while phonological (deep) dyslexics are far more impaired at reading non-words than familiar exception words. Both groups of dyslexics are impaired on both kinds of targets compared to age-matched controls

Image: Traxler (2011). Introduction to Psycholinguistics. Wiley Blackwell. Fig. 10.10. Page. 401.

We look at that, in, in a bit more detail here. If for example, this is a particular graph from Bailey's study and you'll see, the prediction so, what kind of deficits are there? There's reading level match control, so reading on exception words and non-words, their surface dyslexics and for logical dyslexia. So, you can see, the surface dyslexics are still alright and the difference between exception word reading and onward

reading is a little bit, but in the phonological dyslexic you see: that they are highly, deficient in reading the non-words. Okay?

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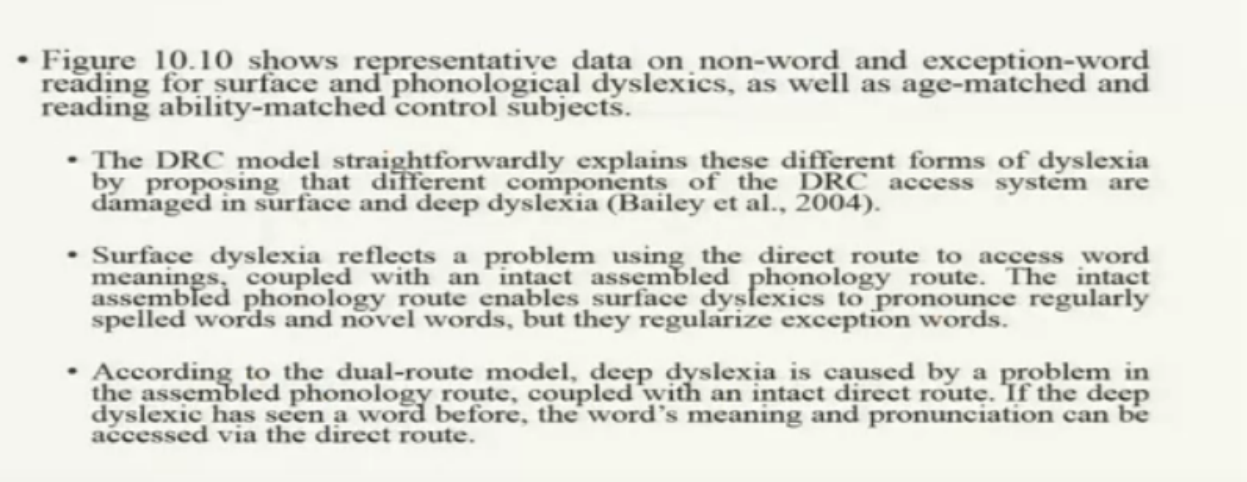

Now, if you kind of look at that figure. The DRC you know, it shows data, on non word and exception about reading for surface dyslexics, phonological dyslexics, as well as, age mass, reading ability match control subjects. What do you find there? If you look at the data, the DRC model straightforwardly explain, these difference, in four different forms of dyslexia by proposing: that different components or routes, of the dual route models have been, broken down. The surface dyslexia in, in that sense, will reflect a problem using the direct route to access word meanings, coupled with an intact, assembled phonology route, the intact assemble phonology route basically, enables the surfaces dyslexia, to pronounce the non-words, completely fine, but they, obviously will lead to in a regularization errors, with respect to exception words. According to the dual route model, deep dyslexia phonological dyslexia shall be caused by a problem in the assembled phonology route, coupled with an index, intact direct route. So that is why? These people will be able to read the exception words completely fine, but obviously they will not be able to read the, the normal words, from the assemble phonology route. So, now word reading will also be suffering here. Now, if the deep dyslexic has not, seen the word before, the assembled phonology route, is not able to compile the pronunciation and the resulting pronunciation is actually, going to be,

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- If the deep dyslexic has *not* seen the word before, the assembled phonology route is not able to compile a pronunciation, and the resulting pronunciation is usually unrelated to the correct pronunciation.

- When the DRC model is used to simulate dyslexic symptoms, separate lesions in the direct route and the assembled phonology route are necessary to capture the kinds of behavior exhibited by patients with different types of dyslexia (Nickels et al., 2008).

unrelated to the correct pronunciation, it'll lead to a pattern of errors like the regularization errors. Now, when the DRC model was used to simulate dyslexic symptoms, different lesions in the direct route and the assemble phonology route, were found necessary, to capture the pattern of performance that is shown by the dyslexic individuals.

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- Single-route models have also been used to capture aspects of dyslexia.
  - For example, the original Seidenberg and McClelland model could simulate some aspects of poor word reading.
  - When half of the hidden units in the model were removed, its error scores for high-frequency words were at about the same level as the error scores for low-frequency words in the full model.
  - This reduction in the number of hidden units had a greater effect for irregular than for regular words, which suggests that a greater number of hidden units is necessary for the model to represent the item-specific information that is required to pronounce exception words accurately.

If you talk about the single route models, single route models have also been tweaked, in order to capture aspects of dyslexia. So, what happened was that, in the original Seidenberg and McClelland model, it could also simulate some aspects of poor word reading. So, when half of the model, basically in half of the hidden units in the model were removed, so the processing power is kind of you know reduced, its error scores for high-frequency words were at about the same level, while the error scores for the low frequency words kind of, it was basically the error scores for high low frequency words, we resort of in the same range. Now, however this reduction in the number of hidden units had a greater effect for irregular, words than regular words. We suggest: that a greater number of hidden units is necessary for the processing of in regular words, as compared to the processing of regular words.

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- A subsequent model adopted a more complex system of phonological representations and was trained on phonology before it was trained to recognize words from print (Harm & Seidenberg, 1999).
  - When the phonological representations in this model were degraded in different ways, different patterns of reading performance emerged.
    - *Mild* damage to the network was imposed by limiting the degree to which representations of different phonemes differed from one another.
    - *Moderate* damage was simulated by eliminating one subcomponent of the phonological system and reducing the number of connections by half in the remaining units, on top of the mild form of damage. In the mild damage condition, the model had no trouble with exception words, but did have mild problems with non- words (the pattern observed in deep dyslexia).

Now, a subsequent model you know, later than the Seidenberg in McClelland model adopted as lightly more complex system of representing phonology and it was trained on phonology and phonology, before it was trained to recognize words. So, a different kind of model we're talking about, when the phonological representations, in this model, this is a different model, were degraded indifferent ways, different patterns of deficit emerged, when mild damage was basically made to the network, by imposing the limit by imposing or limiting the degree to which memory representation, it imposed, it kind of limited the degree to which different you know, phonemes could be, differed from one another. When moderate damage was simulated by eliminating one sub, component of the phonological system and reducing the number of connections by half, in the remaining units on top of the mild form of damage, in the mild damage condition, in this condition, the model really, had no trouble with dealing with the exception words, but it kind of broke down with respect to normal words and you know, it started having problems, with non words readings.

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- *Severe* damage was simulated by adding random noise to the pattern of activity within the phonological units. Severe damage to the network produced deficits in both exception-word and non-word reading, which is the pattern observed in surface dyslexia.
- These simulations provide an existence proof for the single-route architecture:
  - Different patterns of word-reading deficits that are observed in different kinds of dyslexia can be produced by a computational mechanism that has a single set of connections between visual input and word pronunciations.

When severe damage was simulated, by adding random noise to the pattern of activity within the phonological unit. Severe damage to the network you know, it kind of produced, deficits in both exception word reading and non word reading, which is sort of the pattern: that is, observed in surface dyslexia. So, you see that this model, kind of presents to us that there might be, different degrees, of damage in the phonological you know, processing system: that could lead to, different kinds of performance by these readers, mild damage could lead to problem with the reading on words, but not so much, in exception words, but severe damage can lead to problems with reading both non words and

exception words at the same time. These simulations sort of provided an existence for proof of single, route kind of architecture. That it is the same deficit, which kind of you know, indifferent degrees, leads to different degrees of deficits in reading performance. So, different patterns of reading word, word reading deficits: that are observed in different kinds of dyslexia, can therefore be produced, by a single come by computational mechanism: that has a single set of connections, between the input units and the pronunciation units. And that is where; again it's very, similar to the phonological code difference explanation: that is what is kind of playing a part?

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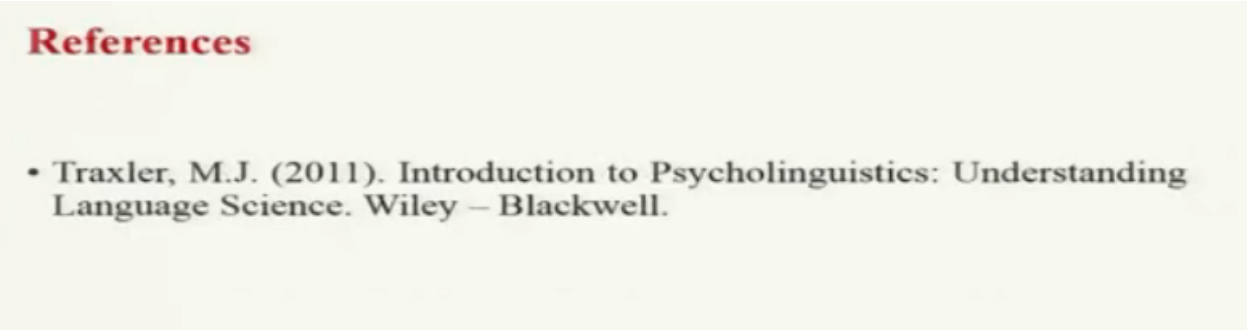

That is all from me about, reading we've sort of gotten a little bit of a clue, about how dyslexia really happens and what are the theoretical underpinnings of different kinds of dyslexia. Thank you.