


Psychiatry an Overview
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Module-03
Psychiatric Disorders and their treatment-1
Lecture-10
Schizophrenia

Welcome again so in the last lecture I briefly introduce you to.

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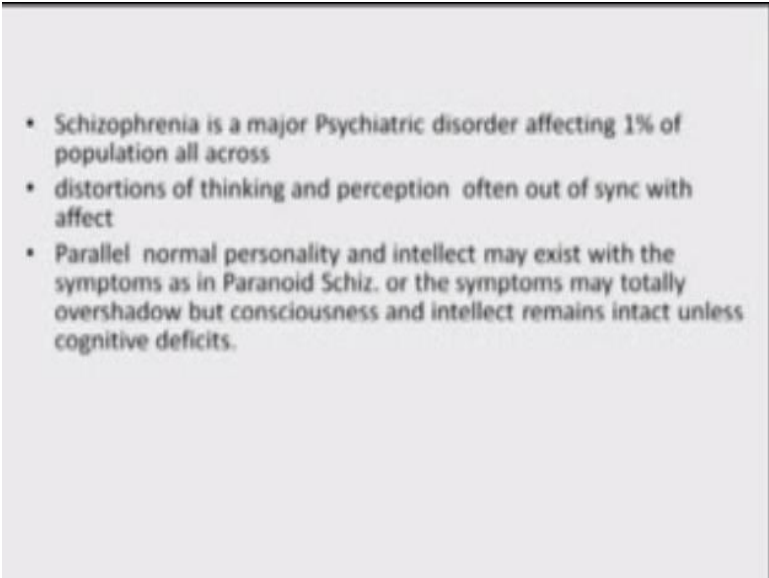
Schizophrenia

What we call organic brain disorders or organic mental disorders and in this lecture I will talk to you about something which you would have heard the word in common parlance and this often said that and the lot of method around it also people are having schizophrenic mind or Vienna schizophrenic state so what does this schizophrenia means, whatever people mean when they are talking about schizophrenia and socio-cultural and cognitive language.

Probably most people mean it is I split personality that is the common myth. But yeah, the way it was understood previously it has given an impression that split personality that people live in two personalities at different times. But it's much more than that it is actually one of the most serious psychiatric illness and obviously over last more than hundred years since it was defined and discovered.

It has changed their scores and the weight has presented so we just try to in the next half an hour we will try to delve into what schizophrenia is all about. It's a major psychotic disorder and it affects.

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- Schizophrenia is a major Psychiatric disorder affecting 1% of population all across
 - distortions of thinking and perception often out of sync with affect
 - Parallel normal personality and intellect may exist with the symptoms as in Paranoid Schiz. or the symptoms may totally overshadow but consciousness and intellect remains intact unless cognitive deficits.

One percent of population all across the world. The hallmark is there is the distortion of thinking and perception is often out of sync with mood often I mean it's not necessarily in the mood and the thought can be in sync times in schizophrenia. But at in most of the schizophrenic patients all three or more or less affected the thought the perception and the mood.

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That normal personality and intellect make exist with the symptoms as in paranoid schizophrenia or other symptoms may totally over power than on normal personality as in say happy freakin schizophrenic or undifferentiated not but bothered do not get bothered about this two heavy words. But they are just the type of schizophrenia which we classify but just to make you understand the paranoid or schizophrenic patient may have schizophrenia and still present you as a very, very well-preserved person.

Whereas the disorganized schizophrenia or a person with undifferentiated may really present you with a lot of total disorganized they say disturb behavior which is not very difficult to notice.

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The hallmark is one of the major one of the two major set psychotic illnesses, psychosis as you remember from the previous lectures loss of touch with reality lack of inside presence of delusions and hallucinations. So delusion is falls into thought and hallucination to perception so what are the symptoms how do you know the common symptoms.

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symptoms

– Positive

- Hallucinations
1st, 2nd, 3rd, running commentary
- delusions
- Formal thought disorders
- Thought Phenomenon
insertion, withdrawal
- thought broadcasting
- Somatic passivity
- Made phenomenon

• Negative

- -avolition
- Apathy
- Asociality
- Alogia
- amotivation

Which are more or less very robust our hallucinations which can happen in any morality. The Communists are in auditory like from the year first person, second person or third person it can be is it is like somebody talking directly to the person first person is very rare me talking to myself doesn't happen normally but cannot be rule out, second person is some voices are telling me something to do criticizing me but directly mentioning like you.

The third person hallucination that two or three voices discussing me. The extension of which is running commentary like was that actually when I am doing something they are giving a commentary delusions as I said this is, so this is a disturbance in perception delusion is a disturbance in thought formal thought disorder we discussed like breaking up of the link between the thoughts the losing of residence there is no co-ordinates in the thought.

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symptoms	
<ul style="list-style-type: none">- <u>Positive</u>- Hallucinations 1st, 2nd, 3rd, running commentary- delusions- Formal thought disorders- Thought Phenomenon insertion, withdrawal- thought broadcasting- Somatic passivity- Made phenomenon	<ul style="list-style-type: none">• <u>Negative</u>• -avolition• Apathy• Asociality• Alogia• amotivation

Thought phenomena like some sometimes they speak the patient feel that somebody's inserting thoughts against their will in their thought or somebody's withdrawing the thought against their will are the thoughts are being broadcasted so might take passivity's

the feelings with the undergo are controlled by some external event or external object. Whatever they are doing is being controlled by some something else.

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But these form a group of positive psychotic symptoms like something new is coming up. Whereas in negative symptoms this evolution there is no will to do anything a patty not interested in objects outside, not willing to talk a logia is poverty of content or poverty of speech. The whole world output may be less or even if they are speaking there will be no significant content in that and A motivation.

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symptoms

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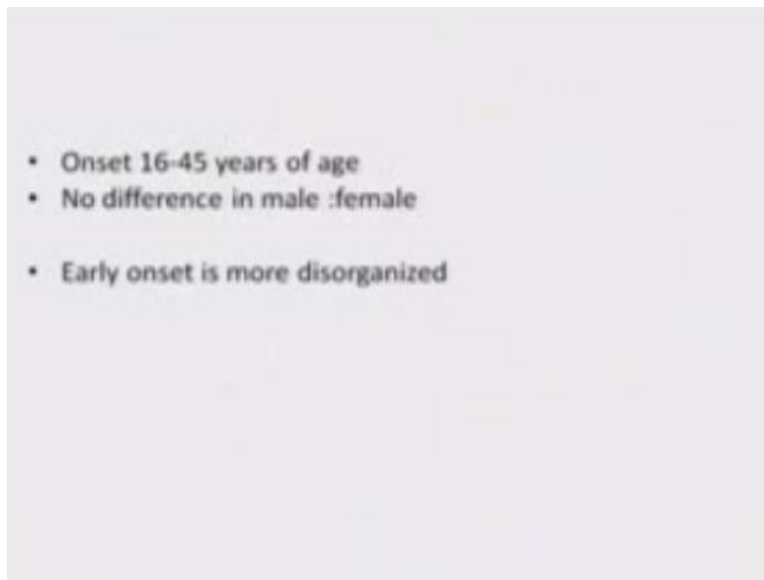
Now all these symptoms barring Alogia a little bit here and there everybody knows what they mean but in schizophrenia that becomes a mental state which is almost continuous and are worsening onset of schizophrenia.

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- Onset 16-45 years of age
- No difference in male :female
- Early onset is more disorganized

Is between 16 to 45 years of age largely, with people can have late-onset schizophrenia right time is schizophrenia like symptoms can appear as we discovered because the organic reasons or because of some other radiology there is no difference in.

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And whereas on said before 16 years we call early-onset schizophrenia. If you remember from the previous lectures we talked about that the brain overgrowth in the initial yes, in there is a pruning of neurons and synapses which starts this age almost corresponds to that in fact one of the theories that you will discuss later is associated and has been linked to this pruning which starts there is no difference in male or female.

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- Onset 16-45 years of age
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And early onset is more disorganized.

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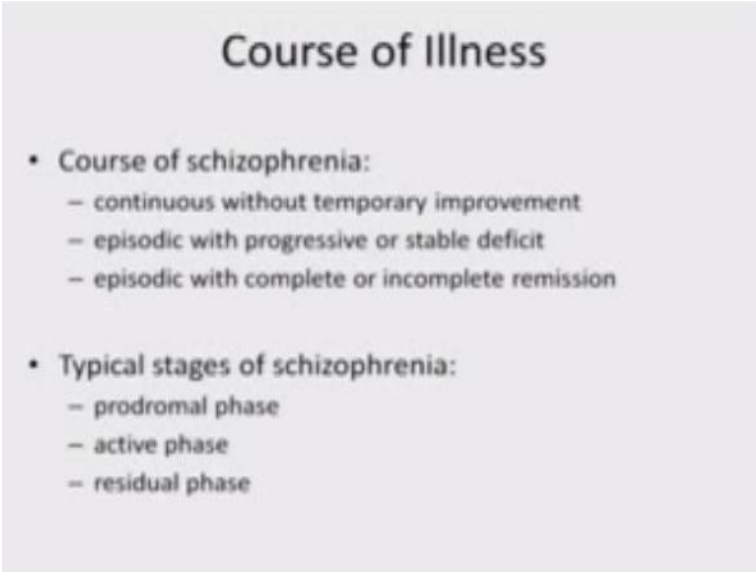
History

- **Emil Kraepelin:** This illness develops relatively early in life, and its course is likely deteriorating and chronic; deterioration reminded dementia („Dementia praecox”), but was not followed by any organic changes of the brain, detectable at that time.
- **Eugen Bleuler:** He renamed Kraepelin's dementia praecox as schizophrenia (1911); he recognized the cognitive impairment in this illness, which he named as a „splitting” of mind.
- **Kurt Schneider:** He emphasized the role of psychotic symptoms, as hallucinations, delusions and gave them the privilege of „the first rank symptoms” even in the concept of the diagnosis of schizophrenia.

So let us briefly look at history this Emil Kraepelin was the first person who called it Dementia praecox it develops early life and course deteriorates chronic but was not followed by any organic changes. Bleuler what game renamed the Dementia praecox schizophrenia in 1911 and he was the person who actually give the concept of splitting of mind.

Kurt Schneider actually organized it better and the role of psychotic symptoms the hallucinations delusions that gave them the privilege of what we call is Narian first rank symptoms. Now the symptoms which I talked about previously are the first rank symptoms. So they formulated the concept of that the experiential mental state which the patient is undergoing so they can be.

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Course of Illness

- Course of schizophrenia:
 - continuous without temporary improvement
 - episodic with progressive or stable deficit
 - episodic with complete or incomplete remission
- Typical stages of schizophrenia:
 - prodromal phase
 - active phase
 - residual phase

It used to be a run a very, very continuous progressively declining course if you 50 years back. But with medication or maybe the spontaneous change in course what we see now that Serena schizophrenia is showing some better course like.

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Course of Illness

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It's continuous it could be continuous without temporary improvement it could be episodic with progressive or stable deficit. So their episodes which happened there episode which happened and then.

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Course of Illness

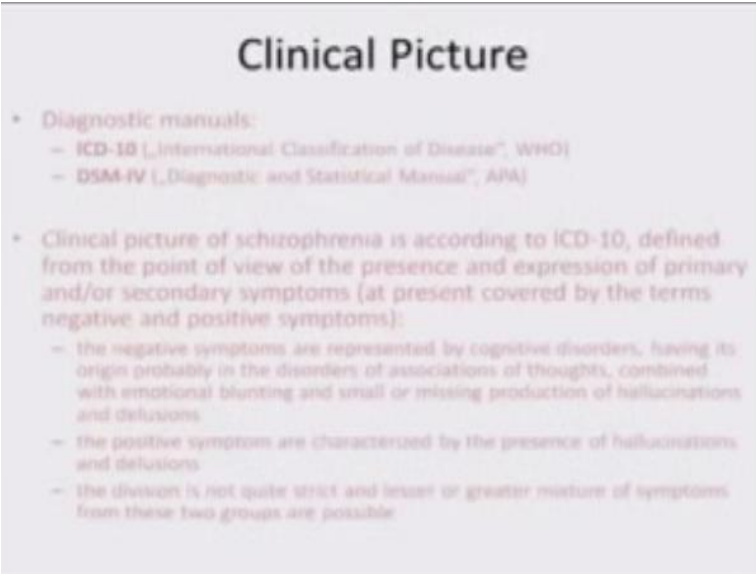
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With every episode there are some deficits which appear and then more or less it goes on like that or it could be episode with complete remission they could be episodic

schizophrenia the person improved then you can keep having many episodes in the life so but more or less schizophrenia has a continuous course. Now these if you look at the stages not everybody has these stages but there are people who have a prodromal like small soft symptoms which are going on for months to years.

There is active phase or either positive or negative symptoms which more or less stabilizers within 4, 5 years and then the person remains in residual phase for the rest of their life. These are the type of course which we which we have seen or evolving over time but we medication lot of things have changed.

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Clinical Picture

- * Diagnostic manuals:
 - ICD-10 („International Classification of Disease“, WHO)
 - DSM-IV („Diagnostic and Statistical Manual“, APA)
- * Clinical picture of schizophrenia is according to ICD-10, defined from the point of view of the presence and expression of primary and/or secondary symptoms (at present covered by the terms negative and positive symptoms):
 - the negative symptoms are represented by cognitive disorders, having its origin probably in the disorders of associations of thoughts, combined with emotional blunting and small or missing production of hallucinations and delusions
 - the positive symptoms are characterized by the presence of hallucinations and delusions
 - the division is not quite strict and lesser or greater mixture of symptoms from these two groups are possible

So ICD-10 has international classification of disease and we said DSM-IV not DSM-V is there. This is according to ICD-10 the negative symptoms are represented by cognitively disorder having its origin in the disorders of association of thought as they mentioned with emotional blunting. The positive symptoms the characterized by the presence of hallucinations and delusions.

We have talked about it the division is not quite strict and they can be a great mix of symptoms. So obviously, the patient has not read the book so when the person comes and they will not be acting according to the book and creating those symptoms what normally happens is a huge mix and overlap of symptom.

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F20-F29 Schizophrenia, Schizotypal and Delusional Disorders	
F20	Schizophrenia
F20.0	Paranoid schizophrenia
F20.1	Hebephrenic schizophrenia
F20.2	Catatonic schizophrenia
F20.3	Undifferentiated schizophrenia
F20.4	Post-schizophrenic depression
F20.5	Residual schizophrenia
F20.6	Simple schizophrenia
F20.8	Other schizophrenia
F20.9	Schizophrenia, unspecified

This is the diagnosis according to the classificatory system ICD, you see schizophrenia is the F20 chapter 5 of ICD-10 and the type like we said paranoid, hebephrenic, catatonic undifferentiated there is something called post- schizophrenic depression, residual the third phase which we talked about the prodromal active and residual simple schizophrenia which is very difficult to diagnose and unspecified.

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F20-F29 Schizophrenia, Schizotypal and Delusional Disorders

F21	Schizotypal disorder
F22	Persistent delusional disorders
F22.0	Delusional disorder
F22.8	Other persistent delusional disorders
F22.9	Persistent delusional disorder, unspecified
F23	Acute and transient psychotic disorders
F23.1	Acute polymorphic psychotic disorder with symptoms of schizophrenia
F23.2	Acute schizophrenia-like psychotic disorder
F23.3	Other acute predominantly delusional psychotic disorders
F23.8	Other acute and transient psychotic disorders
F23.9	Acute and transient psychotic disorder, unspecified

Now the other syndromes which almost are at fringe or related to schizophrenia like schizotypal disorder something called delusional disorder or acute and transient psychotic disorder we will just discuss it briefly as we move on.

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F20-F29 Schizophrenia, Schizotypal and Delusional Disorders

F24	Induced delusional disorder
F25	Schizoaffective disorders
F25.0	Schizoaffective disorder, manic type
F25.1	Schizoaffective disorder, depressive type
F25.2	Schizoaffective disorder, mixed type
F25.8	Other schizoaffective disorders
F25.9	Schizoaffective disorder, unspecified
F28	Other nonorganic psychotic disorders
F29	Unspecified nonorganic psychosis

Schizoaffective disorder we have not talked about more disordered till now but when we will talk about it later. What will you see there is a group of disorder which has the

presence of both the schizophrenic symptoms and depressive symptoms obviously there are criteria to diagnosed schizophrenic disorder manic type or depressive type.

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But largely it is a because it but that is the dilemma of understanding the cause. But dilemma is whether schizophrenia the different illness and more disordered differently that's what it is understood. Schizophrenia is a disorder of thought and perception with some more disorder where the mood disorder where many and depression are diagnosed are essentially a disturbance of mood. But there is a small group of patients who have both symptoms so they are normally fit in.

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Schizoaffective disorder, wherein the absence of mood disorder schizophrenic symptoms should be present that is one of the basic criteria. For a substantial period of time in the absence of mood disorder if the psychotic symptoms continued your Schizoaffective.

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F22 Persistent Delusional Disorders

- Includes a variety of disorders in which long-standing delusions constitute the only, or the most conspicuous, clinical characteristic and which cannot be classified as organic, schizophrenic or affective.
- Their origin is probably heterogeneous, but it seems, that there is some relation to schizophrenia.

Delusional disorder is where normally there is if you recall the definition of delusion it is a false fixed moderate belief. Whether they are related to schizophrenia or

independent disorder we still do not know through the cause but for functional classificatory purpose it is understood as a part of this is psychos. The only symptom there is what you call a delusion the personally functioning normally in other areas of life have a normal mood not having any perception problem having insight and judgment in other things.

But where it comes to this fall straight belief that is one of the major occupation of the mind. If I give you an example, there is some it something used to be called what is called mono symptomatic hypocritical psychosis a lot of old people who will come and tell you that I always feel their aunts are small organ of organisms which are crawling under my skin.

Are there is no proof for that obviously it cannot they cannot crawl they cannot be this not a biological possibility. But they are forming that or they may be a person who, who has a firm form believe that about suspicions about somebody that this person is planting something against me. Now this delusion the most common is something called illusion of infidelity where I supposed to be suspecting of the other person of.

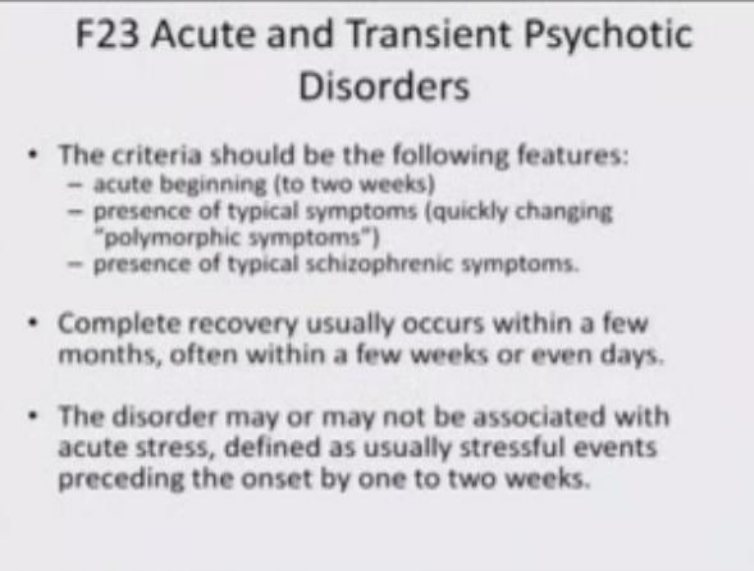
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Having some affair and being invited in delusion of persecution people feel that somebody's proteins up against them. But if they have hallucinations and other be able this organization along with it then probably it's not a delusional disorder but they can maintain this delusion and keep moving in life as they go along.

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F23 Acute and Transient Psychotic Disorders

- The criteria should be the following features:
 - acute beginning (to two weeks)
 - presence of typical symptoms (quickly changing "polymorphic symptoms")
 - presence of typical schizophrenic symptoms.
- Complete recovery usually occurs within a few months, often within a few weeks or even days.
- The disorder may or may not be associated with acute stress, defined as usually stressful events preceding the onset by one to two weeks.

The other thing called acute psychotic disorder so if suddenly somebody get psychotic within two weeks and typical fleeting symptoms, polymorphic symptoms what we call but this schizophrenic symptoms like in schizophrenia they are more or less persistent and go beyond just one month or two weeks it used to be six months before that but now the criteria is one month.

If it is less than that and it is not as well systematized elaborate symptoms like for example delusion, now this is one hallmark when people are having acute psychotics of the symptoms are more fleeting there is schizophrenia like or they may be typical schizophrenic symptoms but the recovery usually occurs within a few.

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Few months and it can make or may not be associated with this stress although stress can precipitate schizophrenia also. But if the symptoms have begun within one or two weeks it is normally acute psychosis symptoms are polymorphic they are typical but they are quickly changing at times. Where the schizophrenia if you look at delusion.

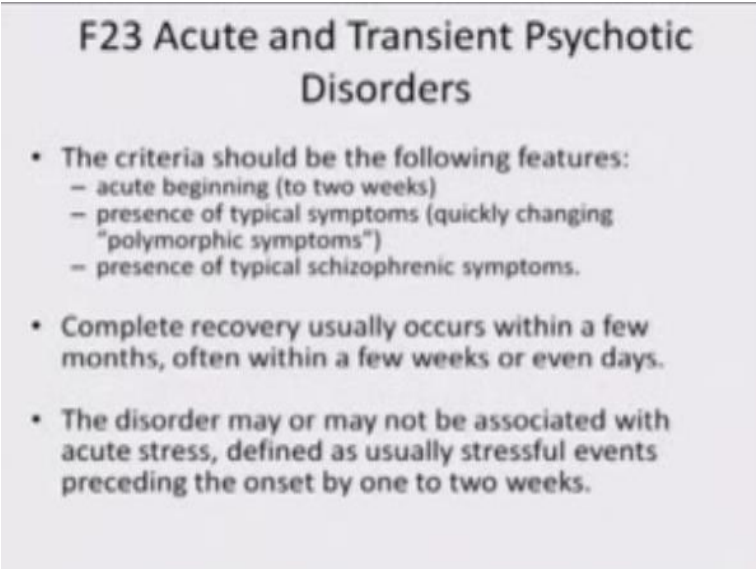
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The delusional disorder when people having delusions the system the delusions very well systematized and very elaborate and over years the person would have built a whole logic of this delusion. So it is like a whole thought process story of that delusion which is goes on to the head when it comes to delusion in schizophrenia of short duration it may not be that well elaborated or well systematize. But it still it would have been persisting four months along with other disturbances. So it's a it is a sometimes the fine distinction and the lot of mental state examination and interactions of a lot of sessions are required.

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But largely they form a group of.

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F24 Induced Delusional Disorder

- A delusional disorder shared by two or more people with close emotional links. Only one of the people suffers from a genuine psychotic disorder; the delusions are induced in the other(s) and usually disappear when the people are separated.
- The psychotic disorder of the dominant member of this dyad is mainly, but not necessarily, of schizophrenic type. The original delusions of dominant member and his partner are usually chronic, either persecutory or megalomaniac.

Psychotic symptoms they induce delusional disorder is a rare thing where one person is suffering in a family and other person also starts believing.

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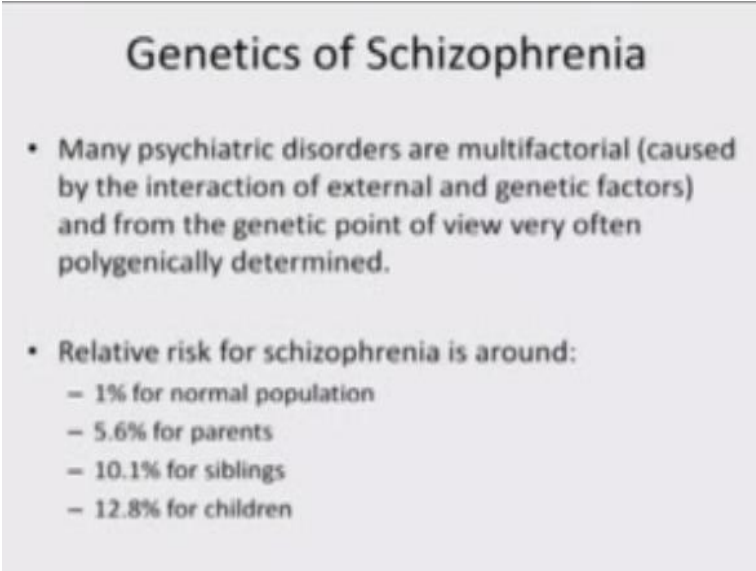
F25 Schizoaffective Disorders

- Episodic disorders in which both affective and schizophrenic symptoms are prominent (during the same episode of the illness or at least during few days) but which do not justify a diagnosis of either schizophrenia or depressive or manic episodes.
- Patients suffering from periodic schizoaffective disorders, especially with manic symptoms, have usually good prognosis with full remissions without any remaining defects.
- They are divided in different subgroups:
 - F25.0 Schizoaffective disorder, manic type
 - F25.1 Schizoaffective disorder, depressive type
 - F25.2 Schizoaffective disorder, mixed type
 - F25.8 Other schizoaffective disorders
 - F25.9 Schizoaffective disorder, unspecified

Schizoaffective disorder they said the episodic disorder with both effective and simple schizophrenic symptoms are prominent. But you cannot says its schizophrenia and either

can you say depressive or manic episode. They have periodic schizoaffective disorder with manic symptoms and normally they're fully.

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Genetics of Schizophrenia

- Many psychiatric disorders are multifactorial (caused by the interaction of external and genetic factors) and from the genetic point of view very often polygenically determined.
- Relative risk for schizophrenia is around:
 - 1% for normal population
 - 5.6% for parents
 - 10.1% for siblings
 - 12.8% for children

So what causes schizophrenia the most common theory they used to be a lot of psychological theory of having a cold schizophrenic son is Christopher energetic mother where the mother was emotionally blind and not attached to the child. So this was one of the psychological theories of it there is another social drift theory that because of the, the people living in poor socio-economic extract predisposes them to developing schizophrenia like symptoms.

But a lot of this multi factorial caution is established not, not one thing because even when you talk about genetics.

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You all we all know monozygotic twins which come from the same embryo and either dichotic where the two embryos there is a high concordance rate when you study people with so the genetic studies normally have been done either by their adoption studies like keep kids from the family where there is a history of any illness have been adopted into other few minutes other family and have developed illnesses.

Or twin a studies 100% concordance is not found in two mysteries. So even if you find the 60, 70% monozygotic twins having if one has the other also has ideally they should have because they share the same set of genes. If it they were 100 out of 100 monozygotic twins develops schizophrenia if one has the other also has then total genetic inheritance can be considered.

But it is not so, so where does the 30% come from and now with the genetic and epigenetic debate more or less settling down it is understood that you may have a propensity of vulnerability to develop schizophrenia you have the genes and with adequate amount of stress it gets precipitated which is the current understanding how the so they lot of gene candidate but we still have not found the real one gene which is.

So probably it is a polygenic thing multiple genes.

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So it is caused by interaction of external and genetic factors very often polygenically. So the relative risk for schizophrenia is around one person for normal population all of us me you everybody has a risk of developing 1% and that is because I don't get surprised by it because we all share the common genetic pool at least the modern man or a lack of year or two lack of years or maybe less than that.

And with the migration genetics telling us that we all come from single-family or single mother in Africa the genetic pool is the same. So as the genes for color of eyes, skin hair, height all of us have come down in us the genes of illnesses they have also come down.

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Genetics of Schizophrenia

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So that gives us that 1% risk for normal population. It increases to 5.6 or 5 to 6 % if the parents have it almost 10% is the sibling have it and almost 12 to 12 or 13 for the children of a percent who has schizophrenia.

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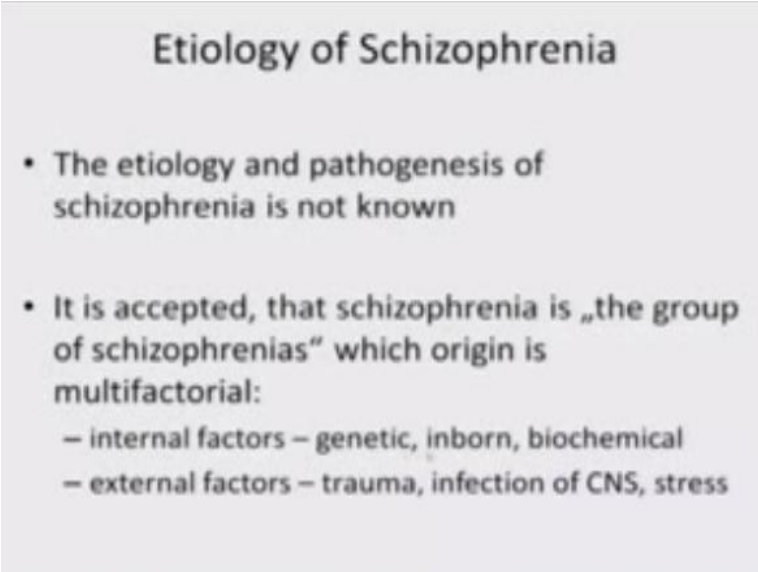
Etiology of Schizophrenia

- The etiology and pathogenesis of schizophrenia is not known
- It is accepted, that schizophrenia is „the group of schizophrenias“ which origin is multifactorial:
 - internal factors – genetic, inborn, biochemical
 - external factors – trauma, infection of CNS, stress

So as I said the etiology and the pathogenesis not known. It is accepted is a group whether, whether all schizophrenia are the same and we are calling them paranoid and

differentiated her friend is disorganized because we, we try to look at the collection of symptoms in a different way or there is there are different schizophrenias is still a debate so internal factors.

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I said genetic inborn biochemical the external factors are trauma infection of CNS, stress. Now these factors modulate the genetics so you do not develop schizophrenia if your genes if you do not have schizophrenia and he go. The external stress when you are old can trigger but you would because, because if external factors are causing then everybody.

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Will undergoes say a trauma or say a tumor will develop schizophrenia. But then that is not schizophrenia that is maybe schizophrenia like symptoms with you talk in the organic think because of EEG factor with the stress you may have a schizophrenic like symptoms which may go off into three weeks that is acute psychosis but when

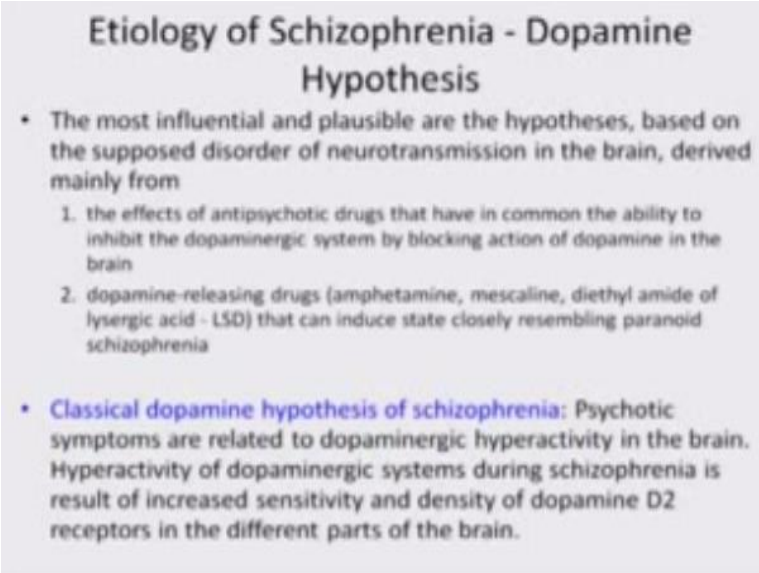
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The genes are modulated with this external epigenetic factors that may be the trigger of schizophrenia.

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Etiology of Schizophrenia - Dopamine Hypothesis

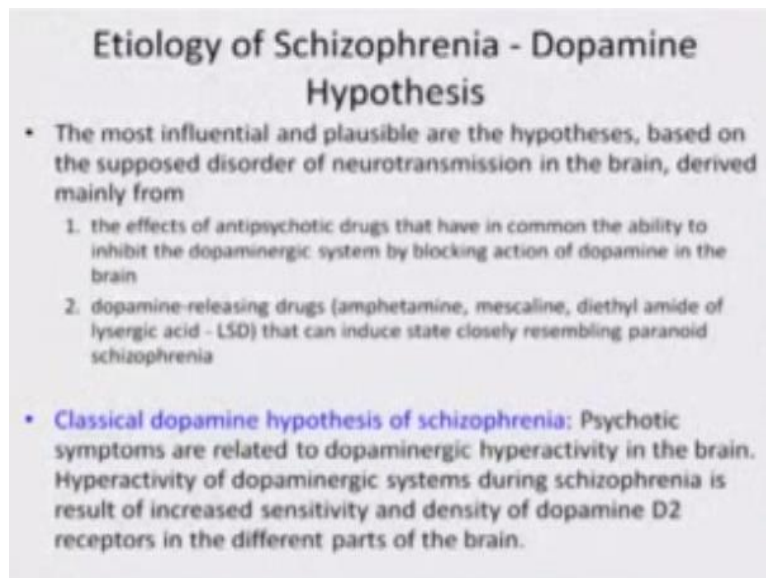
- The most influential and plausible are the hypotheses, based on the supposed disorder of neurotransmission in the brain, derived mainly from
 1. the effects of antipsychotic drugs that have in common the ability to inhibit the dopaminergic system by blocking action of dopamine in the brain
 2. dopamine-releasing drugs (amphetamine, mescaline, diethyl amide of lysergic acid - LSD) that can induce state closely resembling paranoid schizophrenia
- **Classical dopamine hypothesis of schizophrenia:** Psychotic symptoms are related to dopaminergic hyperactivity in the brain. Hyperactivity of dopaminergic systems during schizophrenia is result of increased sensitivity and density of dopamine D2 receptors in the different parts of the brain.

So the most, so whether we know the cause or we do not know what we have definitely found out two large extent is the disturbance in the neurotransmission this is largely the most influential possible is based on the disorder of neurotransmission. What we found out which is the river sort of actually it's a reverse learning which went through. When we founded that they are group of drugs which block dopamine.

The action of dopamine if you remember I showed you the dopamine pathways in the brain in certain areas those areas we have learned over time with imaging and rest of the biochemical studies. But what we found out that the drug which blocks dopamine improves the schizophrenic symptoms. And the drugs some of them are of drug of abuse like dopamine releasing drugs amphetamine, mescaline LSD they increase the dopamine level and they almost induced state closely resembling paranoid schizophrenia. But this is a two well learning which went on.

One the drugs which are blocking dopamine improved and the drugs which increased dopamine which are drug overviews create symptoms like LSD creates a hallucinatory State.

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So the classical dopamine hypothesis which is actually got the guiding principle of all the treatments which have developed till now is basically you give drugs to block dopamine, block dopamine in a certain areas block the receptors of it which in turn to a feedback mechanism degrees the dopamine the related to.

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Dopaminergic hyperactivity in the brain. So the hyperactivity during schizophrenias is the result of increased sensitivity and density of dopamine d2 and other receptors.

The lot of receptors with just them label them d2 and d3 and d4 the dopamine if you understand broadly is the increased levels of dopamine cause the psychotic symptoms, and we treat them by decreasing dopamine or blocking the receptors. So the other models are.

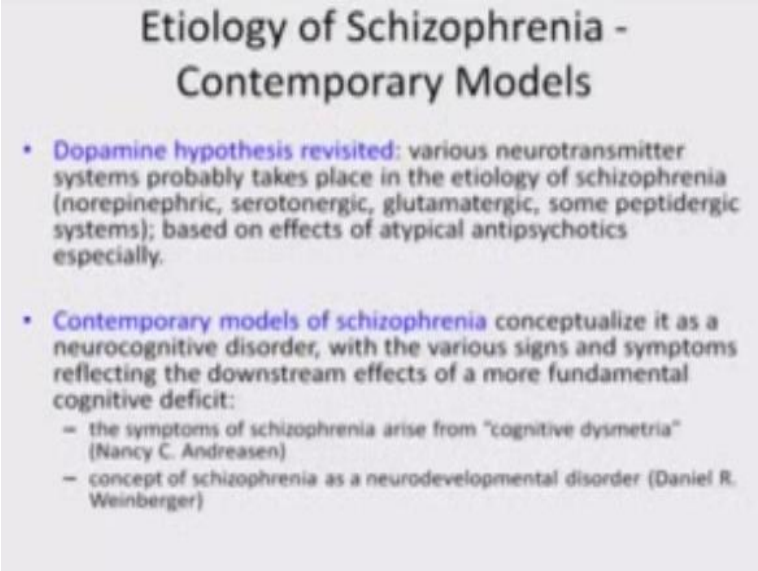
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Etiology of Schizophrenia - Contemporary Models

- **Dopamine hypothesis revisited:** various neurotransmitter systems probably takes place in the etiology of schizophrenia (norepinephric, serotonergic, glutamatergic, some peptidergic systems); based on effects of atypical antipsychotics especially.
- **Contemporary models of schizophrenia** conceptualize it as a neurocognitive disorder, with the various signs and symptoms reflecting the downstream effects of a more fundamental cognitive deficit:
 - the symptoms of schizophrenia arise from "cognitive dysmetria" (Nancy C. Andreasen)
 - concept of schizophrenia as a neurodevelopmental disorder (Daniel R. Weinberger)

Various other neurotransmitters overtime have been implicated norepinephric, serotonergic excitedly gluten glutamatergic if you remember we talked about now these have been implicated as more refinement of development of medicines has gone in especially with the atypical antipsychotics which I will tell you.

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The other models which talk about that it may be a possibility of disorder of cognition neuro cognitive disorder where we look at the whole disturbance the larger scale disturbance in the network. The large-scale disturbance in the way information is taken into this is the mind of this schizophrenic patient and integrated and the decisions on action or emotion is taken.

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So they arrived from a cognitive dysmetria like Nancy Anderson says or a new development we talked about the neuro developmental thing previously. When we are talking about that the brain actually is over growing and the synaptic pruning and that is a time and schizophrenia triggers.

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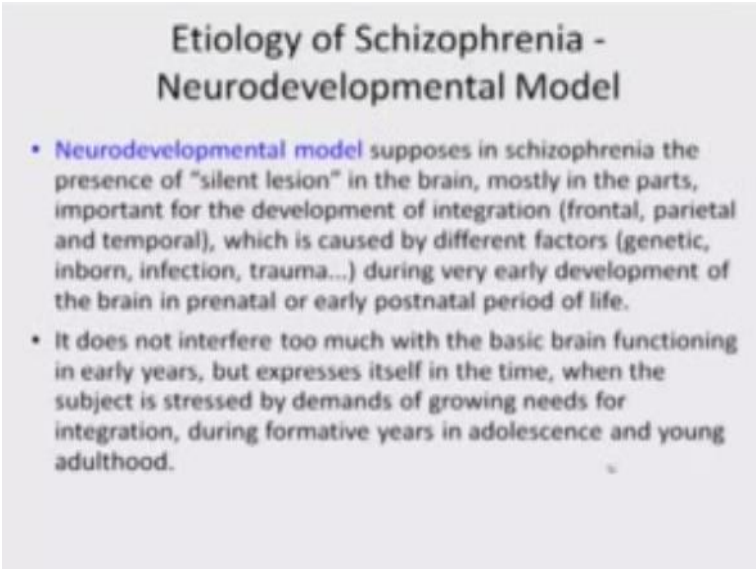
Etiology of Schizophrenia - Neurodevelopmental Model

- **Neurodevelopmental model** supposes in schizophrenia the presence of "silent lesion" in the brain, mostly in the parts, important for the development of integration (frontal, parietal and temporal), which is caused by different factors (genetic, inborn, infection, trauma...) during very early development of the brain in prenatal or early postnatal period of life.
- It does not interfere too much with the basic brain functioning in early years, but expresses itself in the time, when the subject is stressed by demands of growing needs for integration, during formative years in adolescence and young adulthood.

That the presence of silent lesion in the brain, mostly important for the development of integration for of information. Genetic inborn infection this can affect all this but during very early development so it is possible when the brain is developing in the first two years of life or in the womb there maybe mother had under gone an infection or maybe as simple thing as a mother being in stress.

But this is the, the basis of what we talked of well-being clinic that mother during pregnancy should be stress free because all that stress.

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And the subsequent disturbance cortisol another hormones it really plays a epigenetic effect on the genes and which triggers of schizophrenia in a liege because it affects the vase and if is the form it affects the way the network is being integrated and when the Sun epic pruning starts and the neurons are removed are the sin epics removed. It actually trigger of a deficit in certain area.

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So it does not interfere too much with the basic brain functioning but expresses herself at the time and the subject is stressed by demands of growing need for integration. During formative years.

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antipsychotics

- Chlorpromazine,, clopenthixole,, , thioridazine
- flupenthixol, fluphenazine, haloperidol,,, penfluridol, pimozide, trifluoperazine
- amisulpiride, clozapine, olanzapine, quetiapine, risperidone,

So

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This is one of the most comprehensive and most plausible theories of schizophrenia now that it all can be integrated so you get exposed to trauma toxins infection or some epigenetic thing which can uncover the gene of schizophrenia not one single gene maybe it maybe it is apologeneic and it is a lot of genes can get triggered of so as you are growing the brain already is over growing at that time like a normal process.

But the formations which are happening maybe getting affected why these epigenetic factors and when the brain starts pruning it does not go on the network does not remain like the other person was not having a schizophrenia is more deficit which arises in certain areas like the, the imaging has found out that imaging and EG have found it definite or we awoke to a potential they have found definite.

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Changes in schizophrenic the patients with schizophrenia right in the central executive network like, like in the insula like an anterior singulate gurious. The network which dig the information from outside and integrate and then the processing of information is different a lot of this patients with schizophrenia especially in say hello conditions they keep responding to the internally generated image which are normally suppressed in a normal person like you mind maybe generating a image.

And at the same time external stimulus is also going forming an image in the brain which response to the external stimulus. So what is important for survival is that you respond to the external image. So the thinking brain the Central Executive network or the higher center what do you call all the sailors network takes out features from both the internal image as well as the external image.

Now the emotional centers will decide which is important that normally naturally they will decide so that you keep in connect with the external thing it will be suppressed the internal image but this magnetic fields in schizophrenia. So there is a there may be a deficit in the central executive networks connection with the areas which are sending

image to it. So the brain is not reading what is internal, what is external and internal image overpowers the externally image leading to hallucination.

So this deficiencies network is because of probably the epigenetic damage which has which has happened and some neurons are some part of the network may be missing so broadly this is what.

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So I will briefly about the treatment the minister of treatment is called antipsychotic drugs are the ministry of treatment. So these are just the name of the drug you don't have to really go into it this group is called atypical and this is called typical just to sum up the treatment of choice in schizophrenia is medication and it takes a long time it may continue from months to years earlier the treatment started the faster it is stopped.

Within if the list goes beyond six mother treatment can go on for very long and was the person improves the application is important in the person has to return back to normal life there in clinical psychology and rehabilitation the steps in they can lead a normal life John Nash even won a Nobel prize at the North schizophrenia. Thank you and in the next lecture we will talk about more disordered something which you already had. Thanks.