SCHIZOPHRENIA & OTHER PSYCHOTIC DISORDERS

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SCHIZOPHRENIA & OTHER PSYCHOTIC DISORDERS

WELCOME MESSAGE

Pawan Kumar / Associate Professor

Hello everyone !
I welcome you to this online presentation during COVID-19 pandemic as we are all staying at our homes. I hope you will find this lecture engaging and helpful in your studies. Stay home , stay healthy and keep learning.
Best wishes!

PRESENTATION MAP

My presentation will include following slides:

- What is psychosis?
- How do you classify and diagnose psychosis?
- Definition of schizophrenia.
- Symptoms and diagnosis of schizophrenia.
- Risk factors of schizophrenia
- Basic neurobiology of schizophrenia
- Course and prognosis of schizophrenia
- Management of schizophrenia
- My Contact details and study resources
- Self-assessment

WHAT IS PSYCHOSIS?

WHAT IS PSYCHOSIS?

Concepts and definitions of Psychosis.

- Psychosis is a common symptom of many psychiatric, neurodevelopmental, neurologic, and medical conditions and is an important target of evaluation and treatment in neurologic and psychiatric practice.

- Psychosis is also identified as only one of several dimensions of neuropsychiatric disturbance in these disorders, with others encompassing abnormal psychomotor behaviors, negative symptoms, cognitive impairments, and emotional disturbances.

References:

Arciniegas D. B. (2015). Psychosis. *Continuum (Minneapolis, Minn.)*, *21*(3 Behavioral Neurology and Neuropsychiatry), 715–736. https://doi.org/10.1212/01.CON.0000466662.89908.e7

WHAT IS PSYCHOSIS?

Concepts and definitions of Psychosis.

ICD-10 clinical description & diagnostic guidelines

"Psychotic" has been retained as a convenient descriptive term, particularly in F23, Acute and transient psychotic disorders. Its use does not involve assumptions about psychodynamic mechanisms, but simply indicates the presence of hallucinations, delusions, or a limited number of severe abnormalities of behaviour, such as gross excitement and overactivity, marked psychomotor retardation, and catatonic behaviour.

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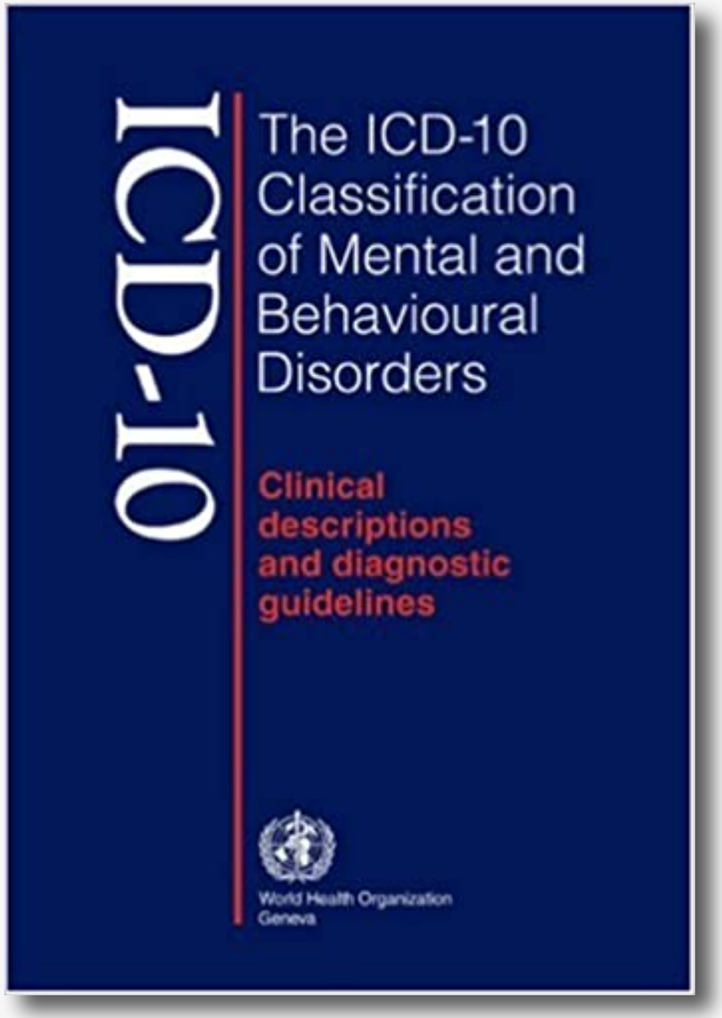


- Presence of hallucinations,
- Delusions,
- or a limited number of severe abnormalities of behaviour, such as :
  - gross excitement and overactivity,
  - marked psychomotor retardation, and
  - catatonic behaviour ”

WHAT IS PSYCHOSIS?

Concepts and definitions of Psychosis.

Schizophrenia spectrum and other psychotic disorders

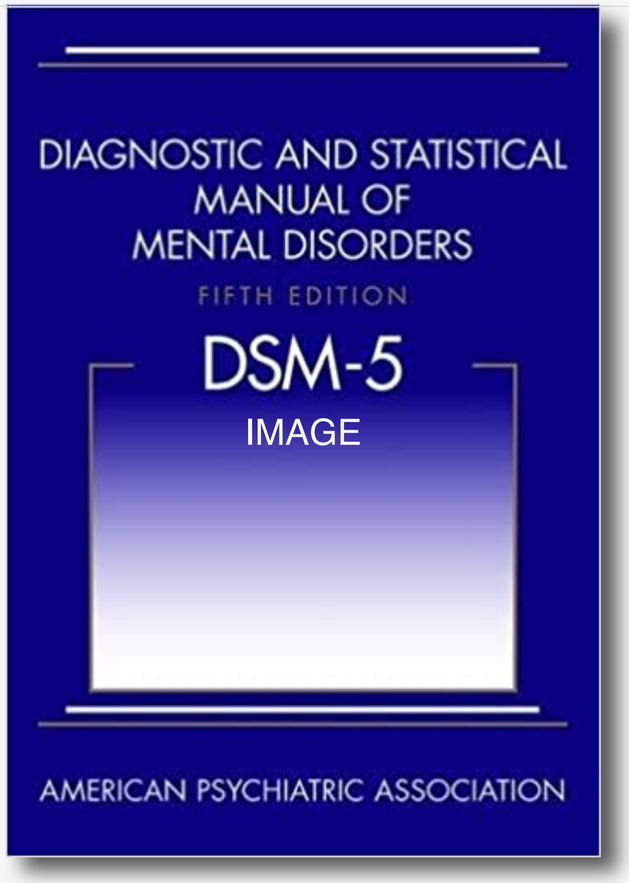
comprises schizophrenia and related disorders, other major psychoses, and disorders with sub threshold psychoses.

All are unified by the presence of one or more of the following five domains of psychopathology:

"delusions, hallucinations, disorganised thinking, grossly disorganised or catatonic behaviour and negative symptoms."

The **first four domains are examples of psychosis,**

negative symptoms are characterised by the absence of something that should be present, such as fluency and spontaneity of verbal expression.



American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). https://doi.org/10.1176/appi.books.9780890425596

WHAT IS PSYCHOSIS?

Concepts and definitions of Psychosis.

- In both of these current diagnostic classification systems, impaired reality testing remains central conceptually to psychosis.

- In their current conceptualization of psychosis, both the APA and the World Health Organization define psychosis narrowly by requiring the presence of hallucinations (without insight into their pathologic nature), delusions, or both hallucinations without insight and delusions.
- This dimensional approach regards hallucinations and delusions as arising from neural systems subserving perception and information processing, thereby aligning the neurobiological framework used to describe and study such symptoms in primary psychotic disorders with those used to study psychosis associated with other neurologic conditions.

References:

Arciniegas D. B. (2015). Psychosis. *Continuum (Minneapolis, Minn.)*, *21*(3 Behavioral Neurology and Neuropsychiatry), 715–736. https://doi.org/10.1212/01.CON.0000466662.89908.e7

DIAGNOSIS AND CLASSIFICATION

Diagnosis is done by clinical evaluation and it it either based on ICD-10 or DSM-5.

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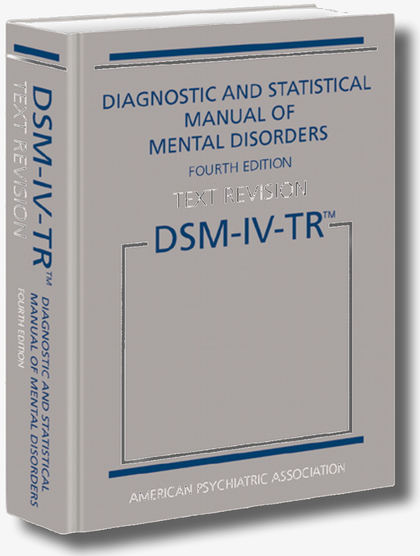
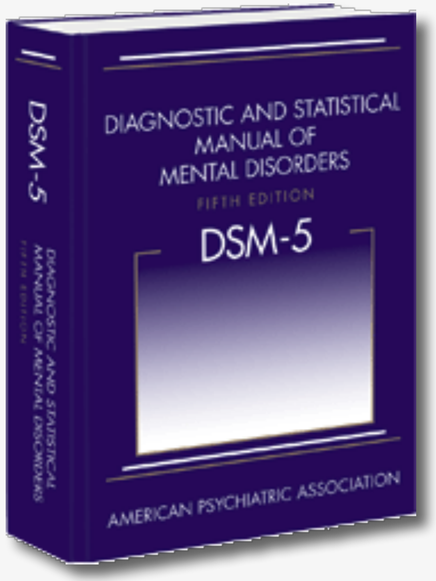
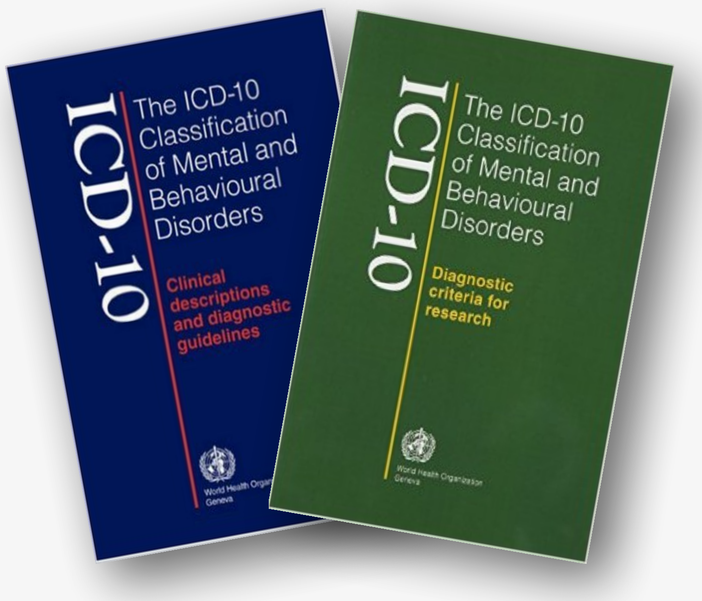


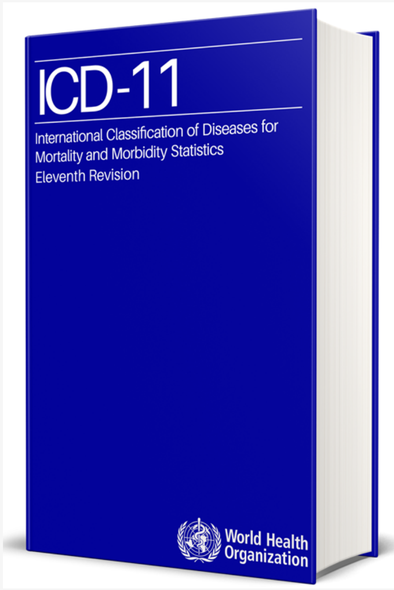
DSM-IV, Diagnostic and Statistical Manual of Mental Disorders, 4th Edition[1]



DSM-5, Diagnostic and Statistical Manual of Mental Disorders, 5th Edition[2]



ICD-10, Classification of Mental and Behavioral Disorders 1993[3]



ICD-11, International Classification of Diseases for Mortality and Morbidity Statistics, 11th revision[4*]

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). https://doi.org/10.1176/appi.books.9780890425596

World Health Organization (2019). International statistical classification of diseases and related health problems (11th ed.). https://icd.who.int/

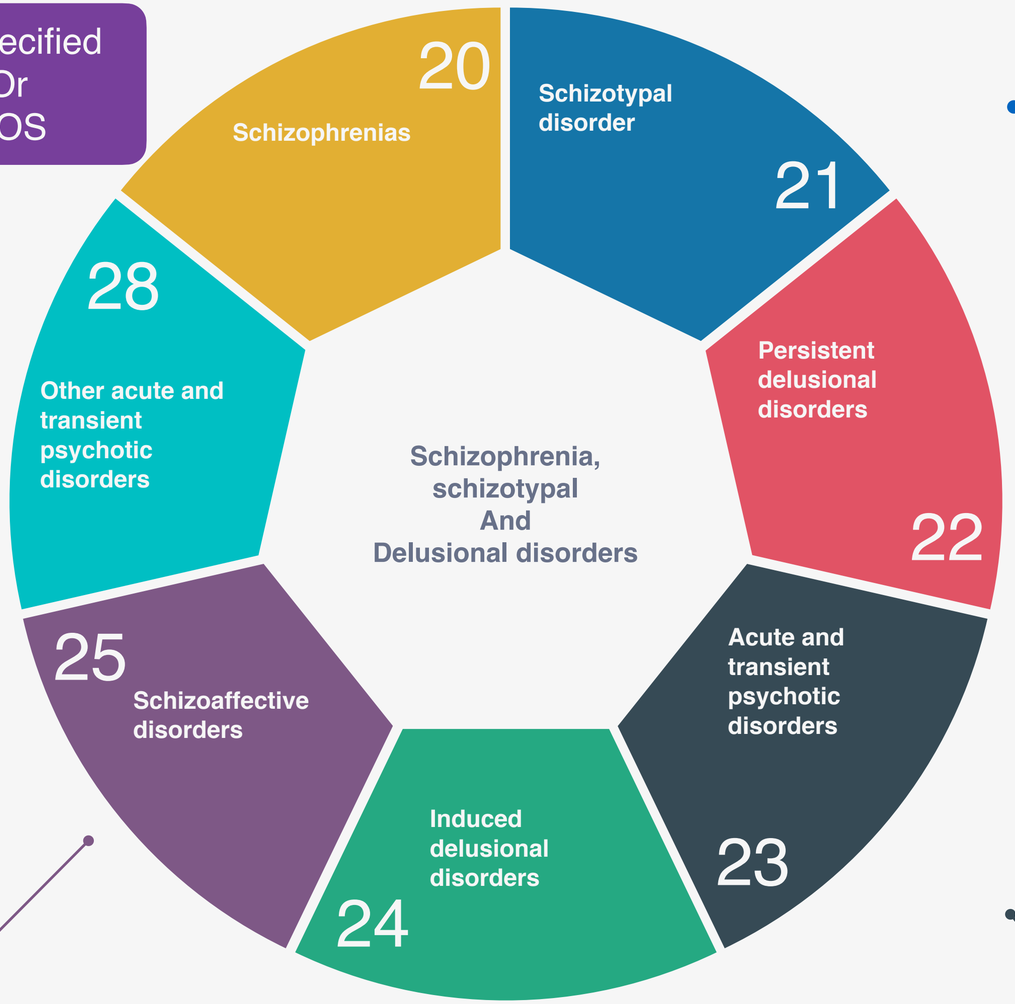
*Pending full release

ICD-10 CLASSIFICATION OF PSYCHOTIC DISORDERS

Coded from F20-F29.

Psychosis with Unknown etiology

Unspecified Or NOS



Other acute and transient psychotic

This diagnosis is made when the criteria of F20-25 are not met.

Schizoaffective Disorders

The subdivisions listed here should be regarded as provisional. Schizoaffective disorders have been retained in this section in spite of their controversial nature.

Schizotypal disorder

Schizotypal disorder possesses many of the characteristic features of schizophrenic disorders and is probably genetically related to them; however, the hallucinations, delusions, and gross behavioural disturbances of schizophrenia itself are absent and so this disorder does not always come to medical attention.

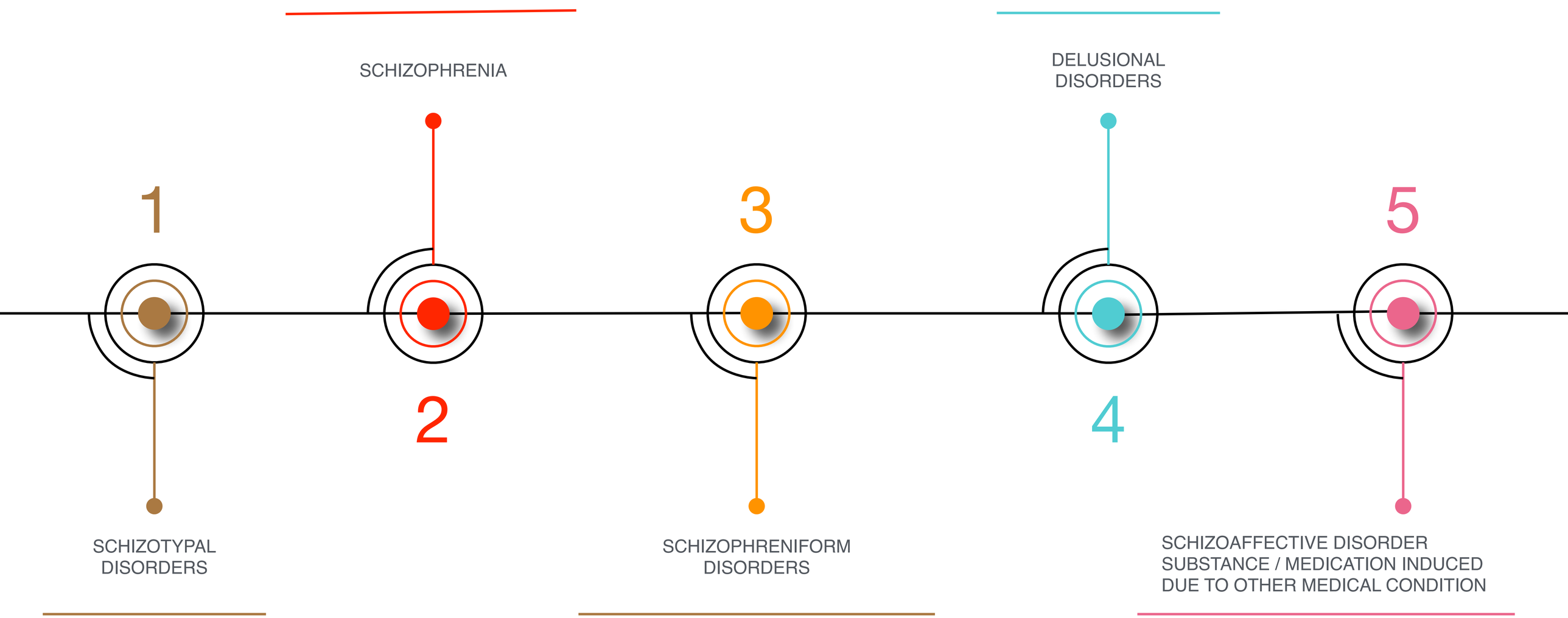
Persistent delusional disorders

Most of the delusional disorders are probably unrelated to schizophrenia, although they may be difficult to distinguish clinically, particularly in their early stages. They form a heterogeneous and poorly understood collection of disorders, which can conveniently be divided according to their typical duration into a group of persistent delusional disorders and a larger group of acute and transient psychotic disorders

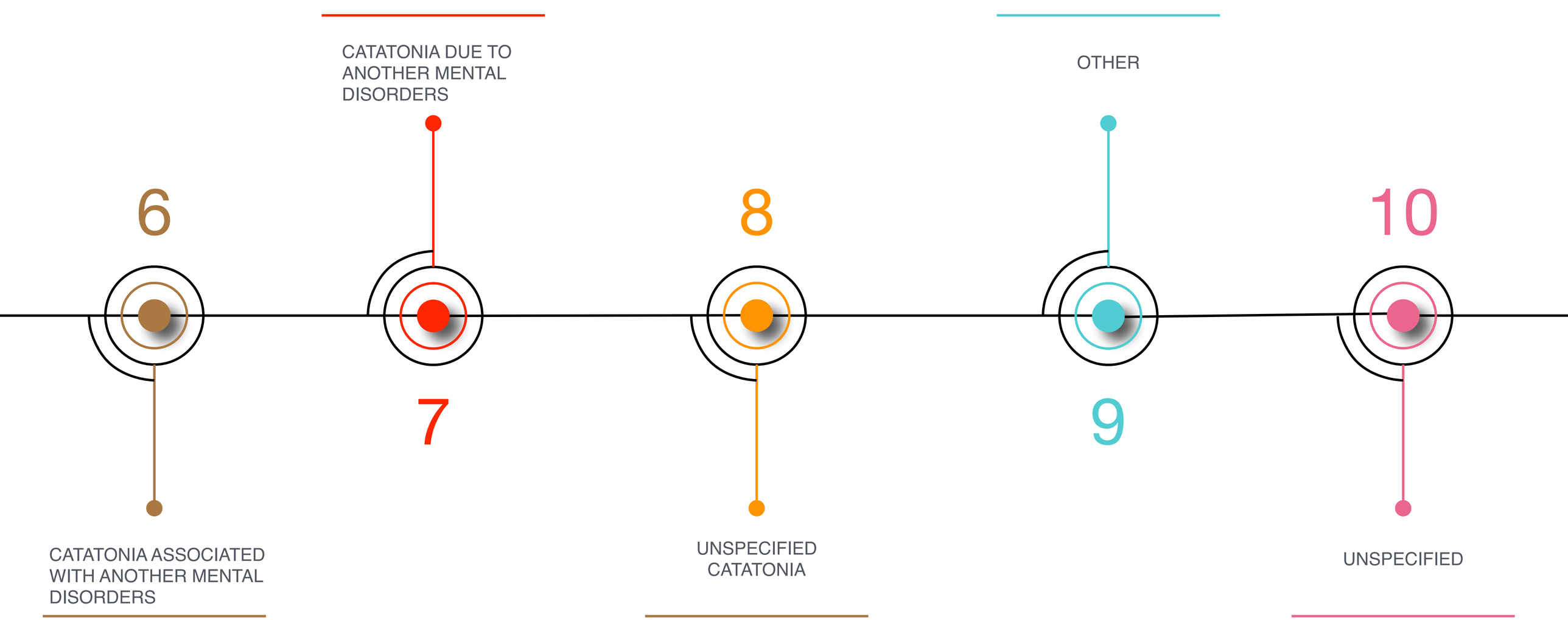
Acute and Transient psychotic disorders

This category is very common in developing countries

DSM-5 CLASSIFICATION OF PSYCHOTIC DISORDERS



DSM-5 CLASSIFICATION OF PSYCHOTIC DISORDERS



WHAT IS SCHIZOPHRENIA?

Heterogenous group of symptoms diagnosed based on ICD-10 or DSM-5.

WHAT IS SCHIZOPHRENIA?

Symptoms dimensions of schizophrenia

DELUSIONS

- Themes: persecutory, referential, somatic, religious, grandiose, erotomanic and nihilistic delusions
- Bizarre delusions are clearly implausible and not understandable to same culture peers and do not derive from ordinary life experiences
- Thought insertion, thought withdrawal, delusions of control are considered bizarre delusions

THINKING

HALLUCINATIONS

- Vivid and clear, with the full force and impact of normal perceptions, and not under voluntary control
- Occur in clear sensorium
- Auditory hallucinations are experienced as voices heard distinct from one's thoughts

PERCEPTION

DISORGANISED THINKING AND SPEECH

- **Formal thought disorde**r includes
- Derailment or loose associations,
- Tangentiality,
- Incoherence or word salad

THINKING AND SPEECH

DISORGANISED BEHAVIOUR

- Grossly disorganized or abnormal motor behavior (including catatonia)
  - Problems in goal directed behavior
  - **Catatonia**

CONTACT US

WHAT IS SCHIZOPHRENIA?

Symptoms dimensions of schizophrenia

NEGATIVE SYMPTOMS

- **Affective blunting**: inability to understand and express emotions
- **Alogia**: decrease in verbal communication e.g. poverty of speech, blocking
- **Anhedonia**: loss of ability to find pleasure from relationships and/or activities
- **Avolition**: loss of will or drive e.g. hygiene, school
- **Asociality:** social withdrawal

THINKING

COGNITIVE SYMPTOMS

- Attention
- Episodic memory
- Executive functions (including language function)
- Working memory
- Processing speed
- Inappropriate Affect
- Inhibitory capacity

PSYCHOMETRIC

AFFECTIVE SYMPTOMS

- **D**epression
- Anxiety
- Anger
- Hostility
- Aggression

THINKING AND SPEECH

CATATONIC BEHAVIOUR

- Motor abnormalities
- Repetitive
- Complex gestures
  - Usually of the fingers or hands
- Excitable
- Wild flailing of limbs.

BEHAVIOURS

IMPORTANT PERSONALITIES

WHO CONTRIBUTED IN EVOLUTION OF THE CONCEPT.

IMPORTANT PERSONALITIES

WHO CONTRIBUTED IN EVOLUTION OF THE CONCEPT.

EMIL KRAEPELIN
(1856-1926)
Dementia Praecox

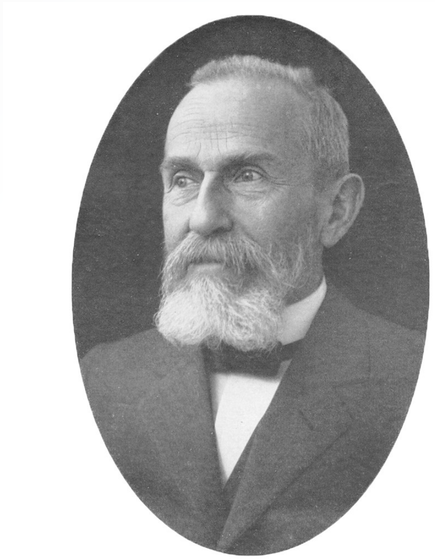


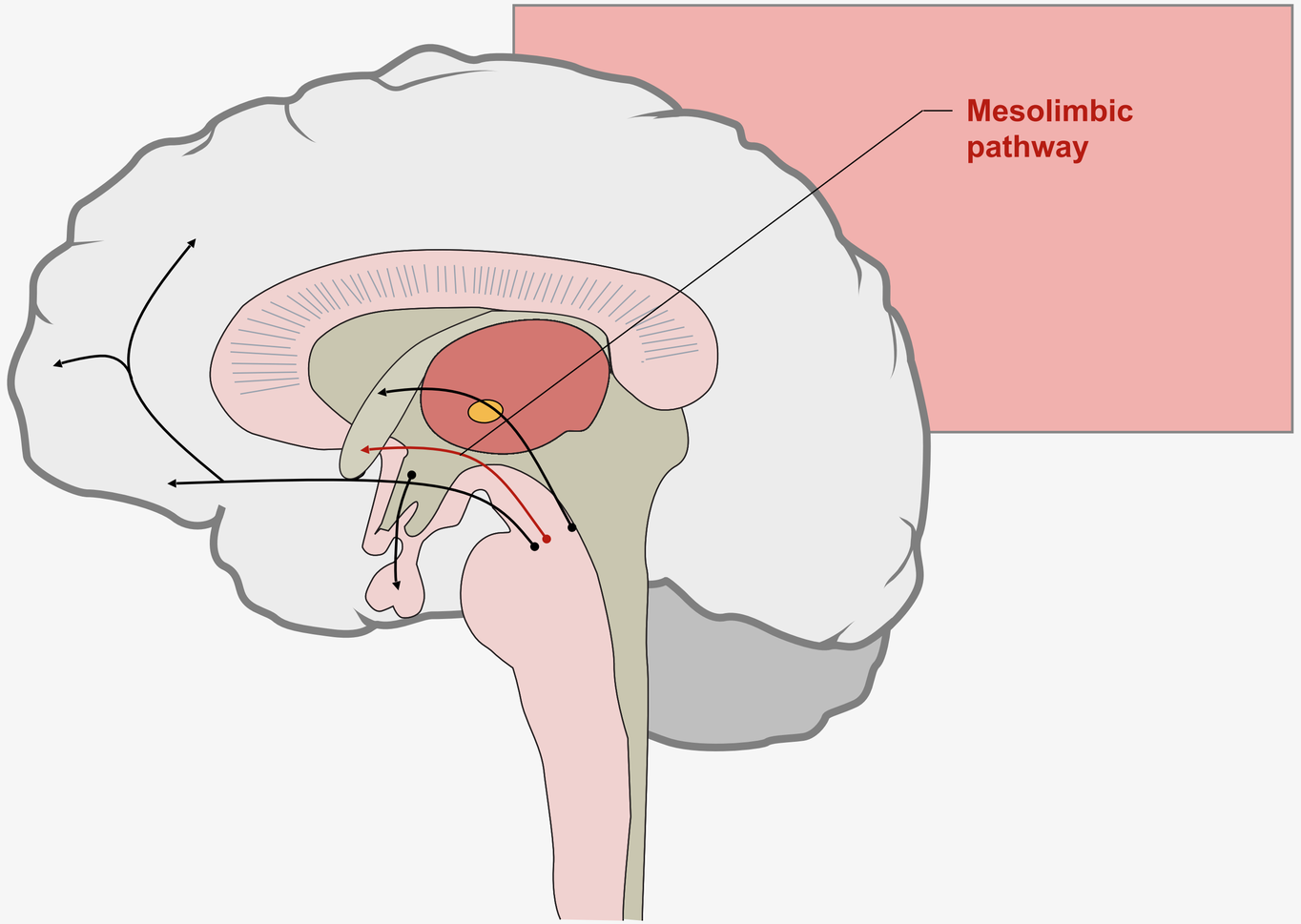
IMAGE EUGENE BLEULAR
(1857-1939)
4 A's

KURT SCHNEIDER
(1887-1967)
First Rank Symptoms

NEUROBIOLOGY

THE POSITIVE SYMPTOMS OF SCHIZOPHRENIA

- The positive symptoms of schizophrenia are thought to be caused by an excess of dopamine in the mesolimbic pathway, although the reasons for this increase are not known
- Positive symptoms include hallucinations and delusions
- Theoretically, decreasing dopamine in this pathway would be therapeutic



Adapted from: Stahl. Stahl's Essential Psychopharmacology. 2013; Owen et al. Lancet 2016;388(10039):86–97

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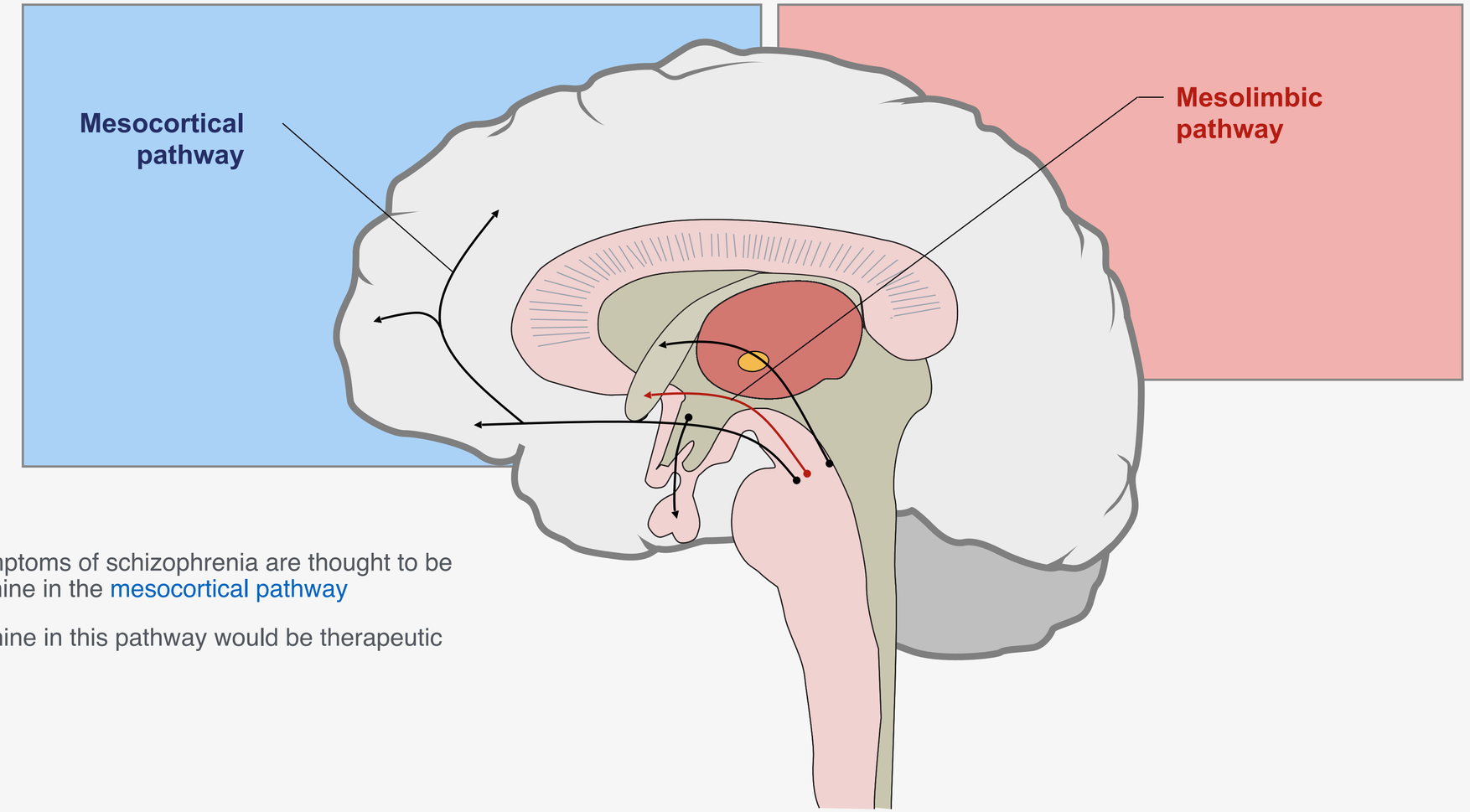
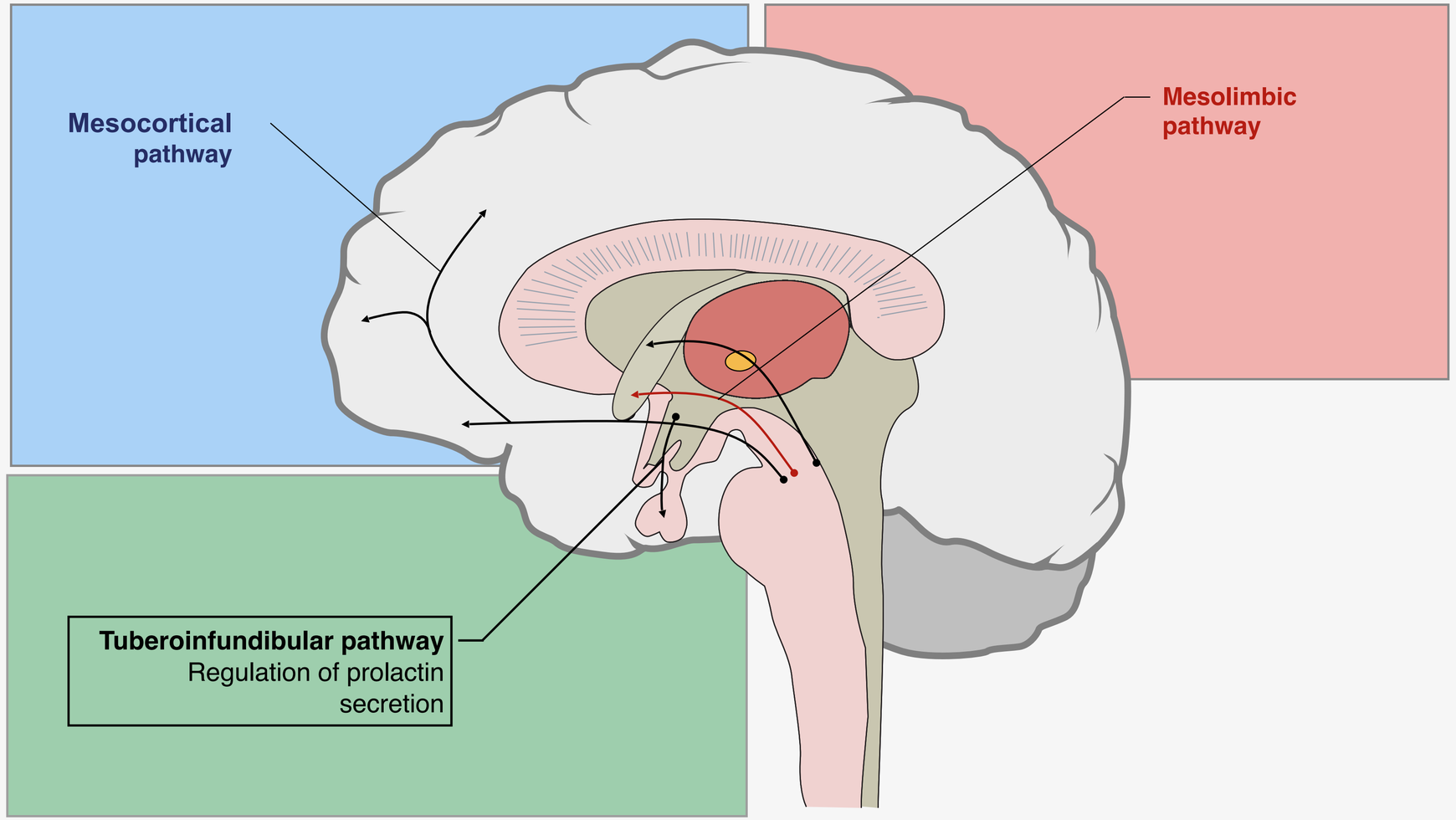


- The negative and cognitive symptoms of schizophrenia are thought to be caused by a shortage of dopamine in the mesocortical pathway
- Theoretically, increasing dopamine in this pathway would be therapeutic

Adapted from: Stahl. Stahl's Essential Psychopharmacology. 2013; Owen et al. Lancet 2016;388(10039):86–97

THE TUBEROINFUNDIBULAR PATHWAY



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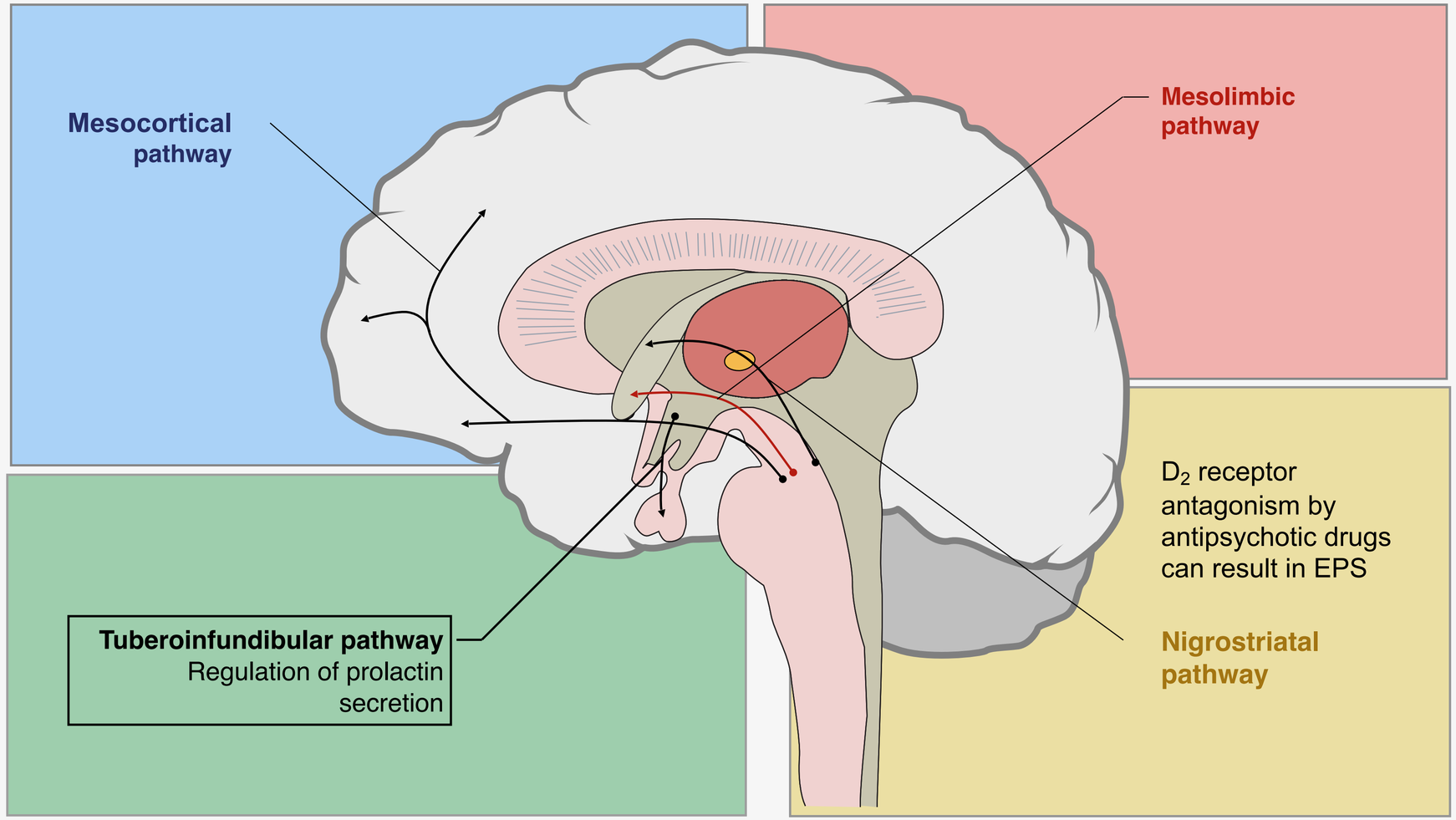
THE NIGROSTRIATAL PATHWAY CAUSING EPS



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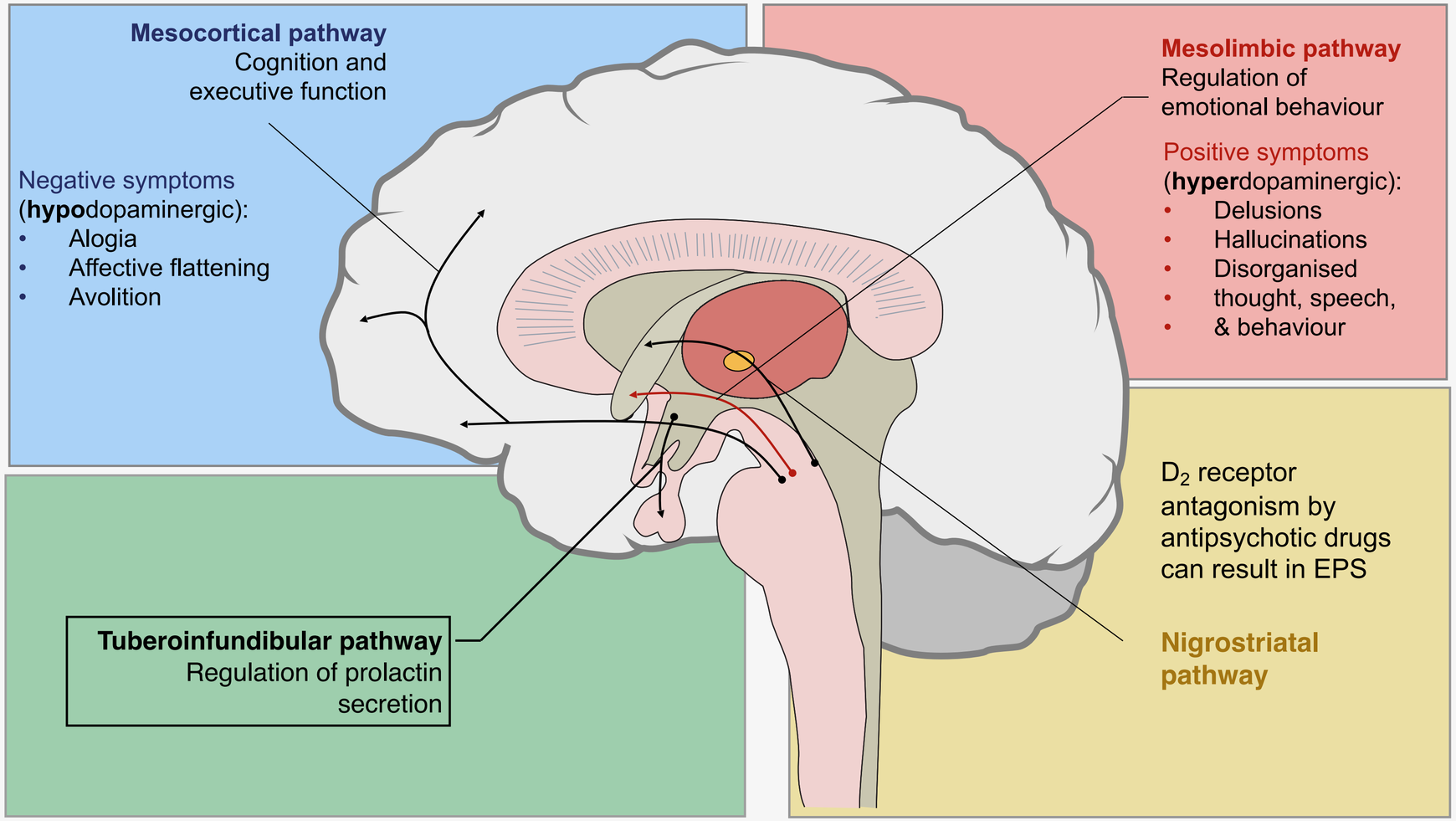

Adapted from: Stahl. Stahl's Essential Psychopharmacology. 2013; Owen et al. Lancet 2016;388(10039):86–97

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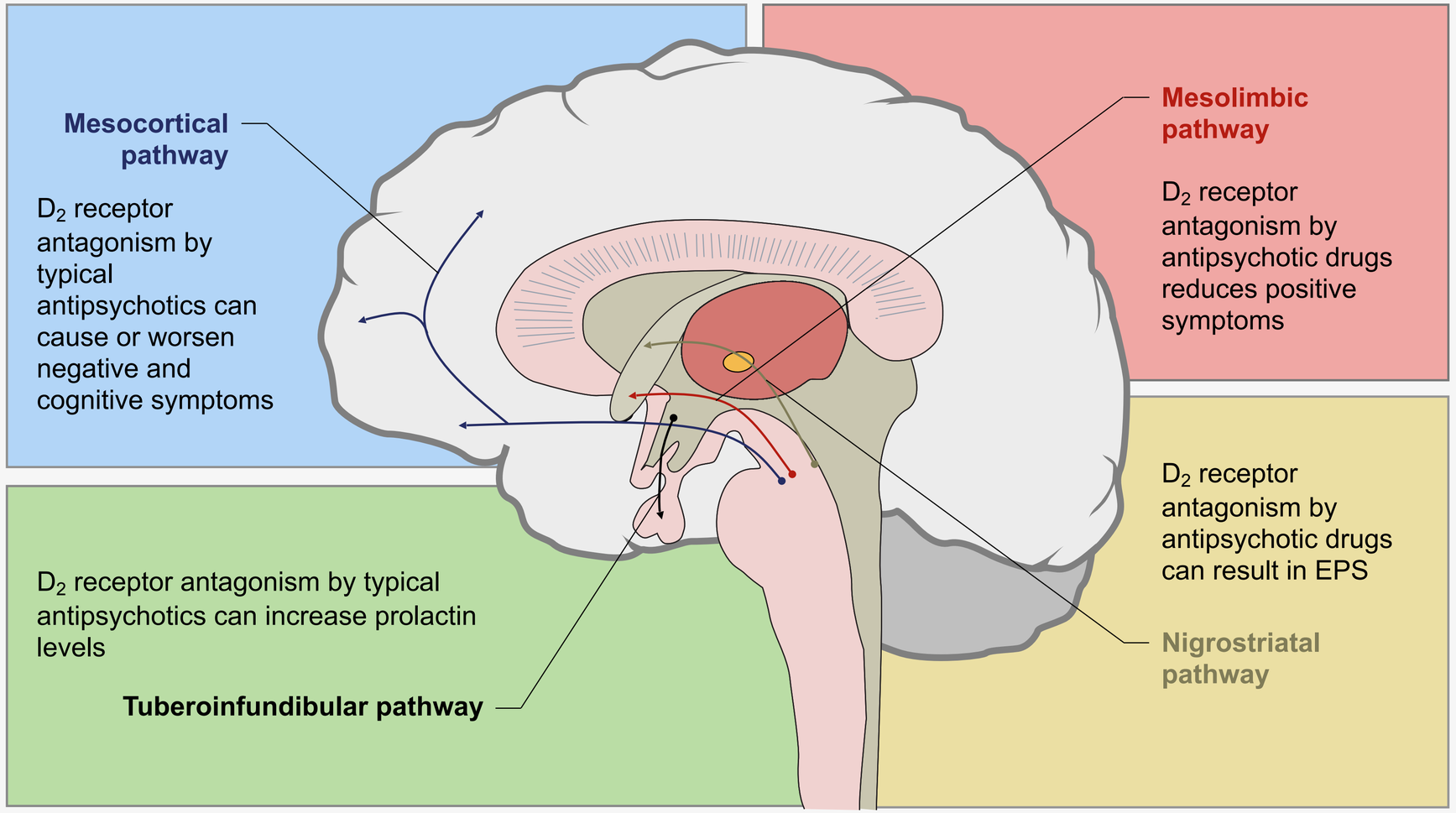
- The therapeutic actions of typical antipsychotic drugs are due to antagonism of D_2 receptors, specifically in the mesolimbic dopamine pathway.
- This has the effect of reducing the excess release of dopamine in this pathway that is thought to cause the positive symptoms of psychosis
- However, typical antipsychotics block D_2 receptors throughout the brain and not just those in the mesolimbic dopamine pathway;
- this extensive blockade of D_2 receptors is responsible for many undesirable adverse effects. Atypical antipsychotics are more discriminating



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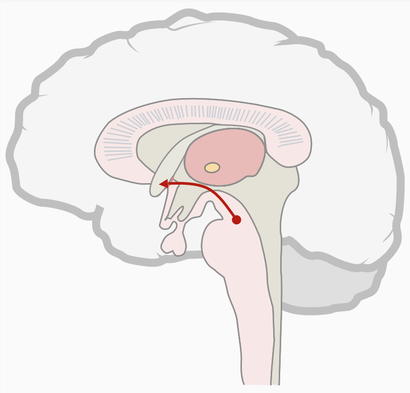
In the ‘normal’ brain

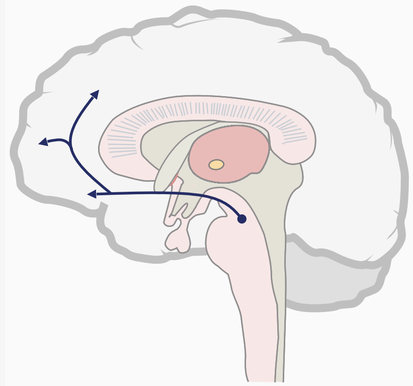
- Dopamine levels within both the mesolimbic and the mesocortical dopamine pathways are at normal levels, therefore no symptoms of schizophrenia are experienced



‘Normal’ dopamine level

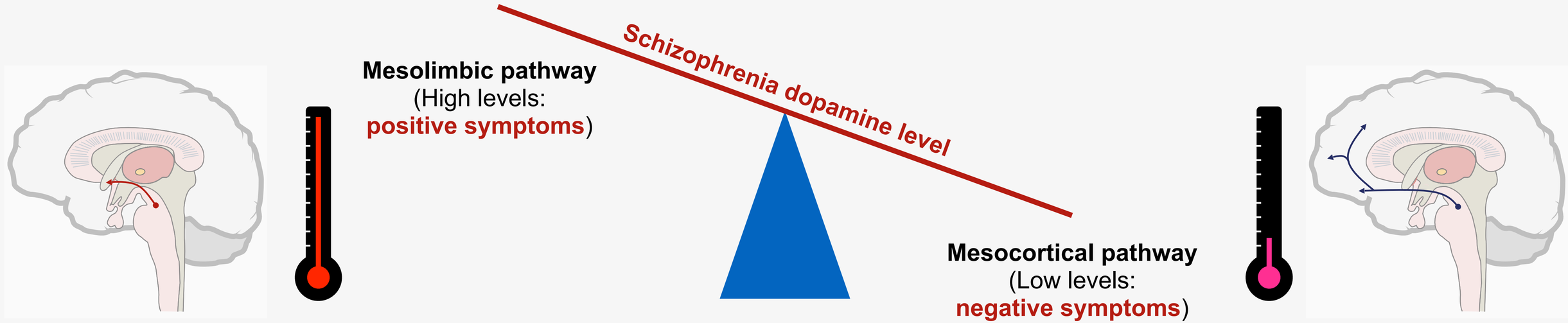
Mesolimbic pathway
(Normal levels:
no positive symptoms)

Mesocortical pathway
(Normal levels:
no negative symptoms)



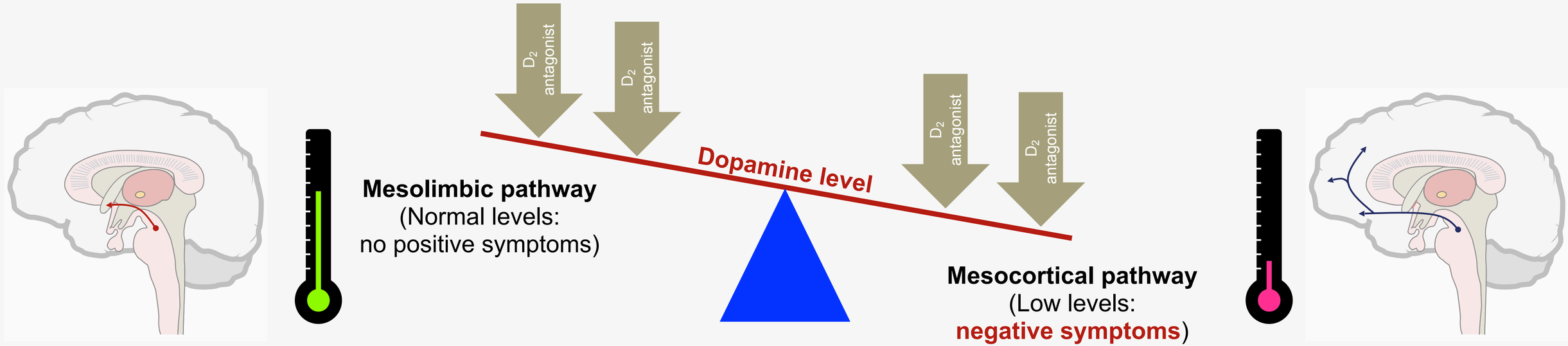
In the schizophrenia brain

- Dopamine levels in the mesolimbic pathway are increased, causing the positive symptoms of schizophrenia
- Simultaneously, the dopamine levels in the mesocortical pathway are decreased, leading to negative and cognitive symptoms



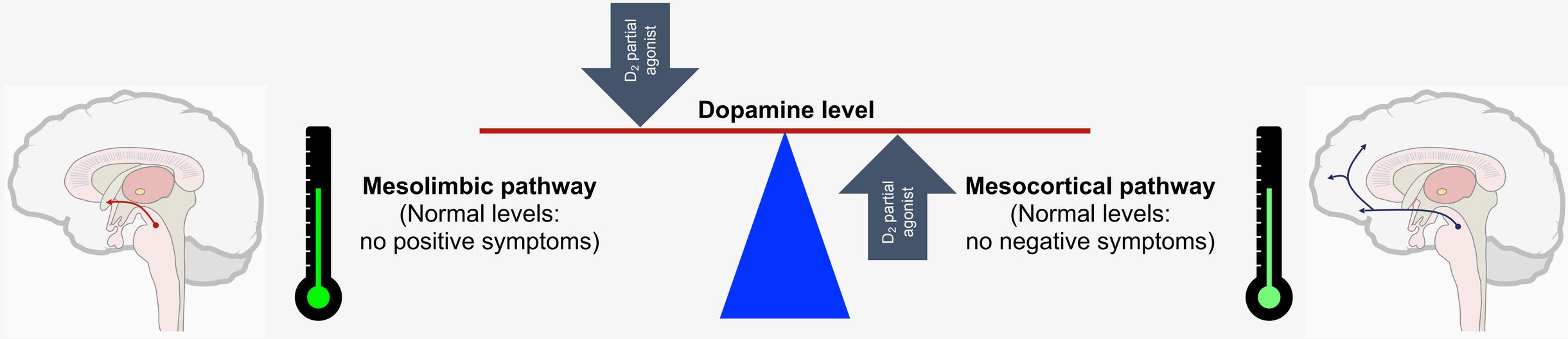
Schizophrenia treated with D_2 antagonist (TYPICAL) antipsychotic

- Treating a patient with schizophrenia with a dopamine antagonist can successfully treat their positive symptoms by reducing dopamine signalling in the mesolimbic pathway
- However, the dopamine antagonist also reduces signalling in the mesocortical pathway, meaning that the negative and cognitive symptoms are not addressed, and in some cases can be worsened



Schizophrenia treated with an atypical, D_2 partial agonist antipsychotic

- A dopamine partial agonist works to reduce the excess dopamine in the mesolimbic pathway, treating the positive symptoms of schizophrenia
- Simultaneously, within the mesocortical pathway a dopamine partial agonist will act to enhance dopamine signalling, meaning that the negative and cognitive symptoms of schizophrenia could be improved as well

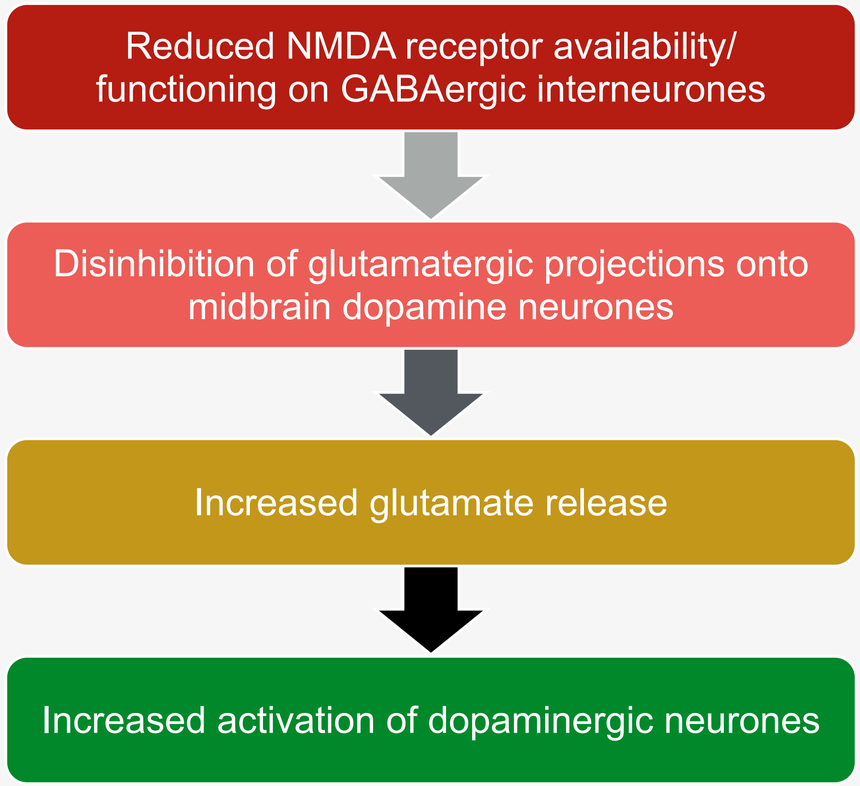


THE ROLE OF GLUTAMATE IN THE PATHOLOGY OF SCHIZOPHRENIA

- It seems clear that changes in dopamine signalling in the brains of patients with schizophrenia underlie the symptoms of psychosis, but what causes these changes?

The glutamate hypothesis

- The predominant ‘go’ neurotransmitter in the brain is glutamate[1,2]
- There are many lines of evidence implicating glutamate NMDA receptors in schizophrenia:[1]
  - Post mortem changes in NMDA receptors in the brains of patients with schizophrenia
  - NMDA-receptor antagonists can cause psychotic symptoms in humans
  - Some glutamatergic drugs have shown promise in treating schizophrenia



GABA=gamma-aminobutyric acid; NMDA=N-methyl-D-aspartic acid

1. Howes et al. J Psychopharmacol 2015;29(2):97–115; 2. Purves. Neuroscience. 2008

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HYPOTHESES FOR THE UNDERLYING CAUSES OF SCHIZOPHRENIA

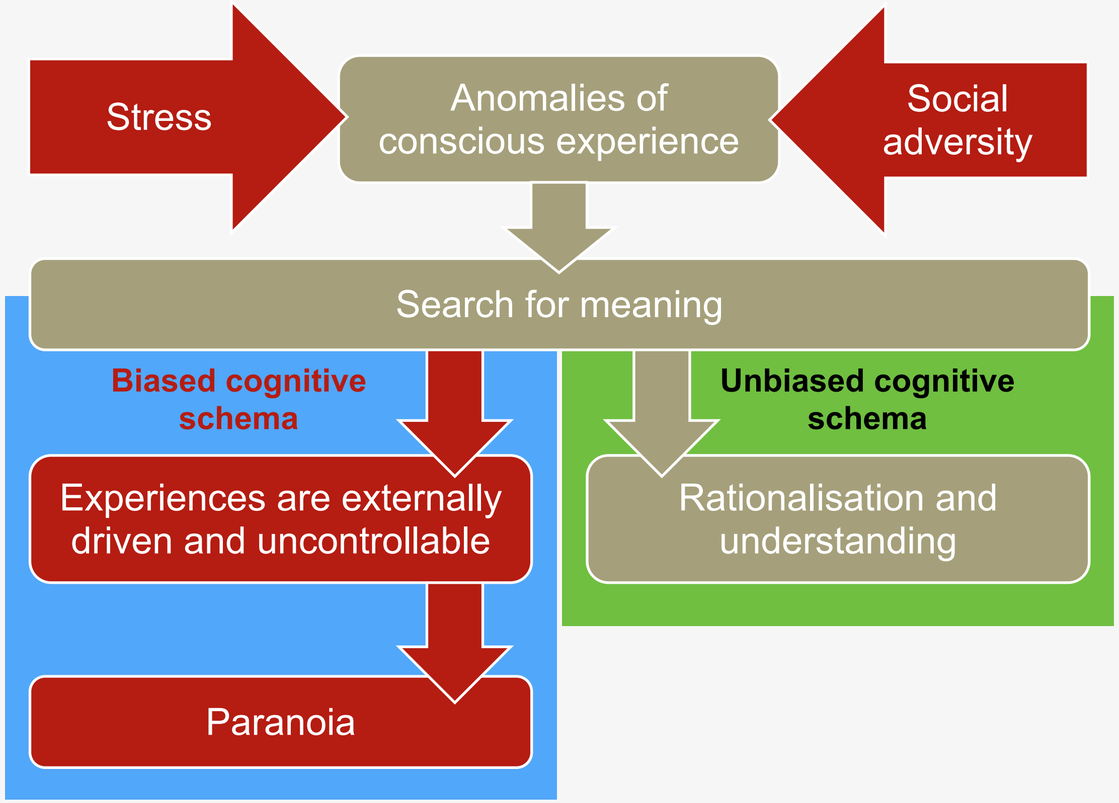
THE NEURODEVELOPMENTAL MODEL OF SCHIZOPHRENIA

- Normal cortical development involves proliferation, migration of cells, dendritic arborisation (circuit formation), and myelination, with the first two processes occurring mostly during prenatal life and the latter two continuing through the first two post-natal decades[1]
- A progressive reduction of grey-matter volume with age is observed with longitudinal neuro-imaging. [1,2] The combined effects of pruning of the neuronal arbor and myelin deposition are thought to account for this[1]
- Psychosis nearly always emerges in late adolescence or early adulthood, with a peak between the ages of 18 and 25, when the prefrontal cortex is still developing[1]
- The neurodevelopmental trajectory in children developing schizophrenia could include reduced elaboration of inhibitory pathways, and excessive pruning of excitatory pathways, leading to altered excitatory–inhibitory balance in the prefrontal cortex[1]

1. Insel. Nature 2010;468(7321):187–193; 2. Paus et al. Nat Rev Neurosci 2008;9(12):947–957

SOCIO–DEVELOPMENTAL–COGNITIVE MODELS OF SCHIZOPHRENIA

- There have been attempts to explain schizophrenia using cognitive models[1,2]
- Cognitive models of schizophrenia suggest that the interpretation of social adversity (e.g., child abuse) through biased cognitive schema and appraisal processes, results in the individual judging the adversities as being externally driven, giving rise to paranoid delusions[1,2]
- Attempts have been made to integrate these cognitive models with the known patho-physiology of schizophrenia, postulating that genetic predisposition and neurodevelopmental insults disrupt the dopamine system, alongside social adversity leading to biased cognitive schema – these forces act in concert to hardwire the individual in favour of the psychotic interpretation of the world around them[1]



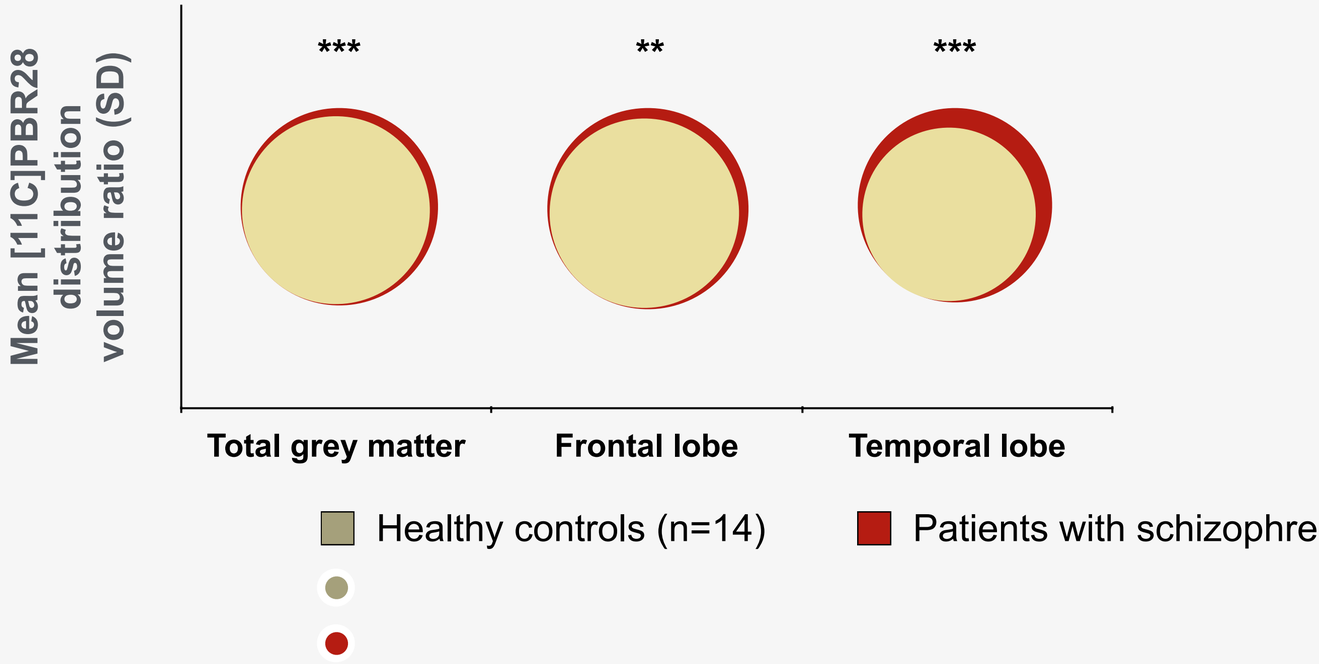
1. Howes & Murray. Lancet 2014;383(9929):1677–1687; 2. Bentall et al. Arch Gen Psychiatry 2009;66(3):236–247

Evidence for involvement in the pathophysiology of schizophrenia	
Dopamine	• Drugs that prevent the activity of dopamine in the brain, by blocking D_2 receptors, can reduce positive symptoms[1] • Amphetamines, which increase the levels of dopamine in the brain, can increase psychotic symptoms[1]
Glutamate	• NMDA receptor antagonists, such as phencyclidine and ketamine, produce psychosis-like features indistinct from schizophrenia[1]
GABA	• Reduced synthesis and reuptake of GABA has been demonstrated in the prefrontal cortex in patients with schizophrenia[1]
Acetylcholine	• Decreased levels of cholinergic receptors are observed in the hippocampus, thalamus, and striatum in patients with schizophrenia[1]
Serotonin	• Prefrontal 5-HT_{2A} receptors have been linked to the pathogenesis of schizophrenia[2,3] • Activation of 5-HT_{2A} receptors induces a schizophrenia-like psychosis in humans[2,3]

INFLAMMATION AND SCHIZOPHRENIA

- The immune system is linked to the pathology of schizophrenia, with evidence including elevated cytokines and microglial activation[1,2]
- PET imaging has been used to examine immune system activity in patients with schizophrenia[1]
- One study found elevated microglial activity in unmedicated patients with sub-clinical symptoms who were at ultra high risk of psychosis, and found a significant positive correlation with symptom severity[1]
- These data indicate that neuroinflammation is linked to the risk of psychosis and related disorders, and the expression of sub-clinical symptoms[1]

Microglial activity measured with PET in patients with schizophrenia and matched controls[1]



**p<0.01, ***p≤0.001

PET=positron emission tomography; SD=standard deviation

1. Bloomfield et al. Am J Psychiatry 2016;173(1):44–52; 2. Melborne et al. Curr Treat Options Psychiatry 2017;4(2):139–151

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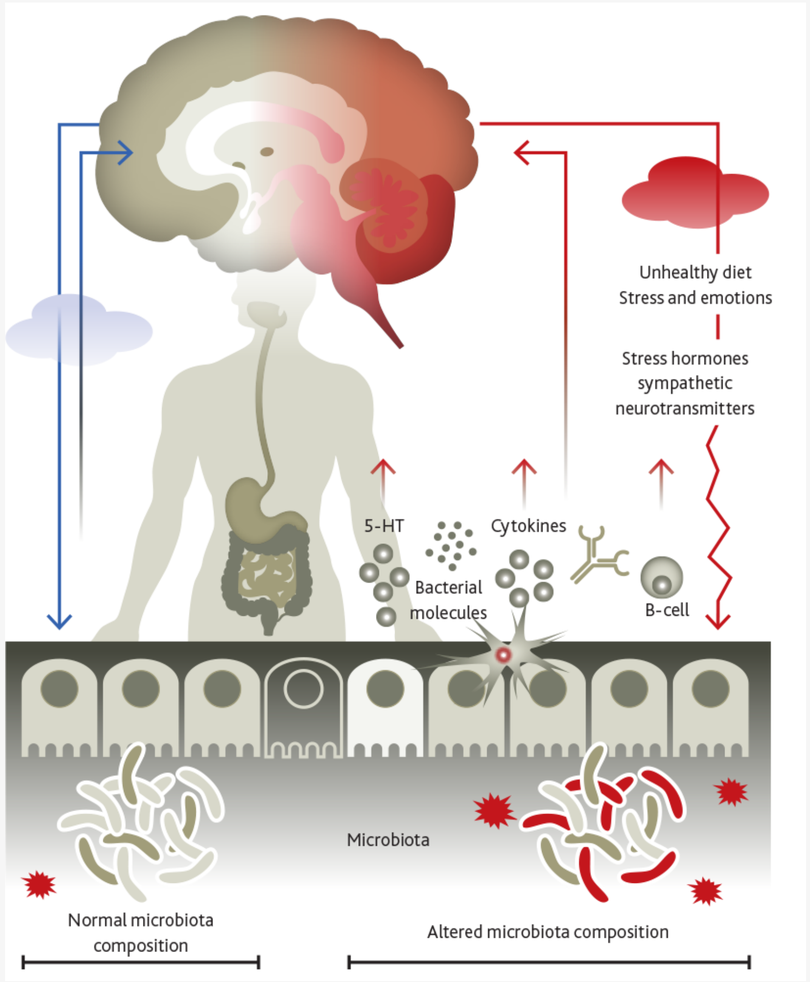
THE MICROBIOME AND SCHIZOPHRENIA

- The makeup and function of the gut microbiota is increasingly being linked to the pathology of neurological disorders, including schizophrenia, depression, and bipolar disorder[1-3]
- The normal flora of the gut is made up of several species of bacteria, and also of viruses and fungi, which colonise the gut at birth[4]
- In a comparison of 16 patients with schizophrenia and 16 controls, differences in oropharynx flora were:[5]
  - Patients with schizophrenia were dominated by a greater number of microbiome species
  - Patients with schizophrenia had greater abundance of lactic acid bacteria
  - There were differences in the metabolic pathways controlling glutamate and B12 transport (increased in schizophrenia) and carbohydrate and lipid metabolism (decreased in schizophrenia)

5-HT=serotonin

1. Nguyen et al. J Psychiatr Res 2018;99:50–61; 2. Chrobak et al. Arch Psychiatr Psychother 2016;2:5–11;
4. Rodrigues-Amorim et al. World J Biol Psychiatry 2018;19(8):571–585; 5. Castro-Nallar et al. PeerJ 2015;3:e1140

The gut microbiota–brain route



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ENVIRONMENTAL FACTORS

THE ENVIRONMENT AND SCHIZOPHRENIA

- Factors pre- and post-natal have been linked to an increased risk of schizophrenia[1,2]
- Epidemiological studies and twin studies have identified many environmental factors that are linked to the development of schizophrenia, for example:[1,2]
  - **Prenatal exposure to viral infections**
  - **Poor pre-natal nutrition**
  - **Adverse obstetric events**
  - **Cannabis smoking during adolescence**

1. Sadock et al. Kaplan & Sadock's Comprehensive Textbook of Psychiatry. 2009;
2. Lakhan & Vieira. Ann Gen Psychiatry 2009;8:12

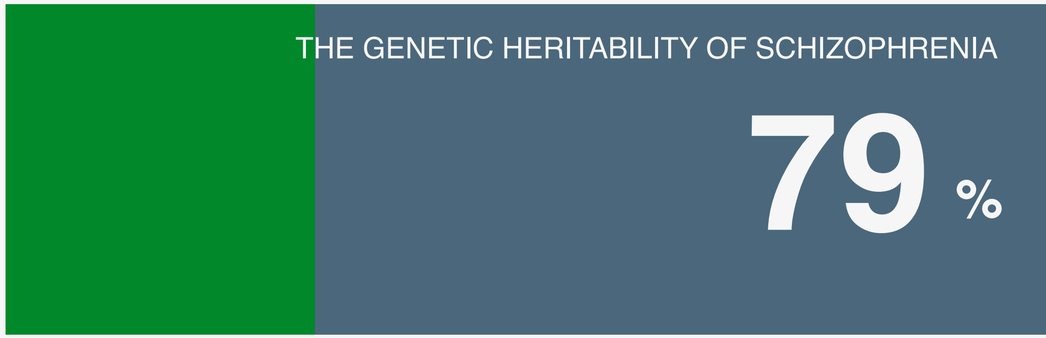
GENETIC FACTORS

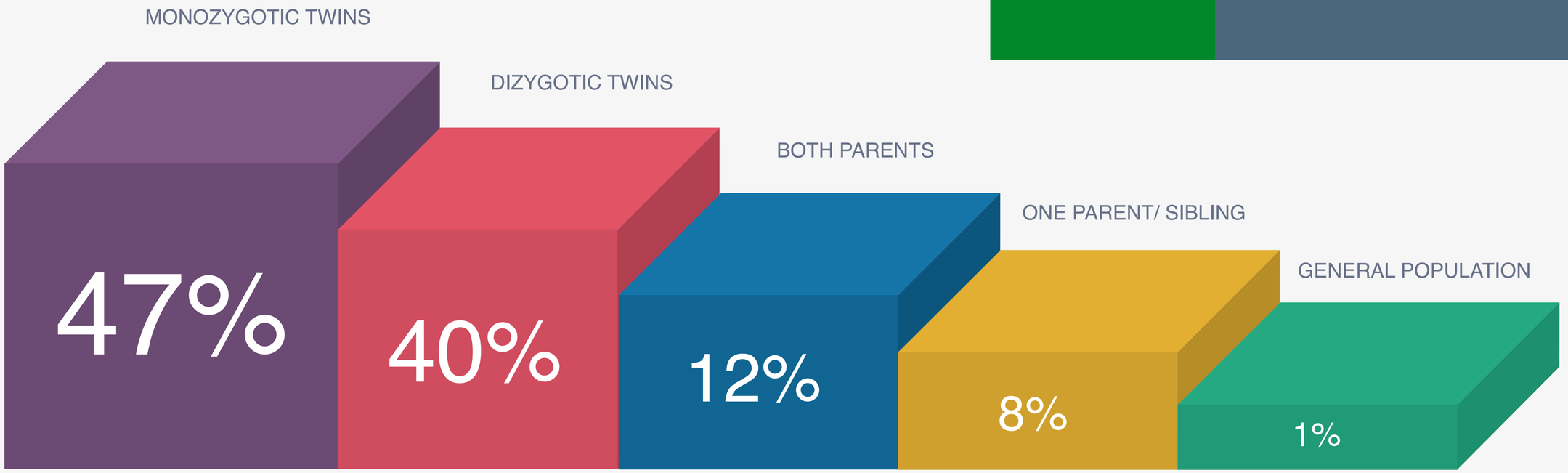
THE HERITABILITY OF SCHIZOPHRENIA – TWIN AND ADOPTION STUDIES

- Numerous studies have shown that the risk of developing schizophrenia is greater in the relatives of patients with schizophrenia[1-3]
- Data from twin studies and adoption studies support the significant role of genetic factors in schizophrenia[1]
- Research conducted more recently has identified susceptibility genes that may result in an increased risk of developing schizophrenia[2,4,5]
- It was found that early age of schizophrenia onset in the first twin was a risk factor for the second twin developing schizophrenia – this suggests that early-onset schizophrenia may have a stronger genetic component of risk than other subtypes of schizophrenia[6]
- These results demonstrate that there is a high genetic component to the risk of developing schizophrenia, however, vulnerability to the illness is not solely genetic[6,7]

1. Sadock et al. Kaplan & Sadock's Comprehensive Textbook of Psychiatry. 2009; 2. Gejman et al. Psychiatr Clin North Am 2010;33(1):35–66;
3. McGue & Gottesman. Schizophr Bull 1989;15(3):453–464; 4. Keshavan et al. Schizophr Res 2011;127(1–3):3–13;
5. Lakhan & Vieira. Ann Gen Psychiatry 2009;8:12–19; 6 Hilker et al. EBioMedicine 2017;18:320–326; 2. Hilker et al. Biol Psychiatry 2018;83(6):492–498

THE GENETIC HERITABILITY OF SCHIZOPHRENIA





1. Hilker et al. EBioMedicine 2017;18:320–326; 2. Hilker et al. Biol Psychiatry 2018;83(6):492–498

THE GENETICS OF SCHIZOPHRENIA- GWAS FINDINGS

- An ambitious genome-wide association study (GWAS) was conducted by the Schizophrenia Working Group of the Psychiatric Genomics Consortium, analysing genetic data from >35,000 individuals with schizophrenia and >110,000 controls[1]
- This GWAS analysis identified 108 distinct loci – 83 of which had not been previously implicated in schizophrenia[1]
- Noteworthy gene locations included:[1]
  - **The dopamine receptor D_2 gene** – highlighting the known importance of dopamine neurotransmission in the pathology of schizophrenia
  - Several **genes encoding proteins involved in glutamatergic neurotransmission,** and several **voltage-gated calcium channel component proteins** – providing an aetiologically relevant foundation for treatment development
  - **Genes expressed in tissues with important roles in immunity** – supporting the hypothesised link between schizophrenia and the immune system
- In an analysis of data from several different GWAS studies, attempting to integrate the data, six crucial genes have been identified as being linked to an increased risk of developing schizophrenia – five of which are related to neurodevelopment[2]

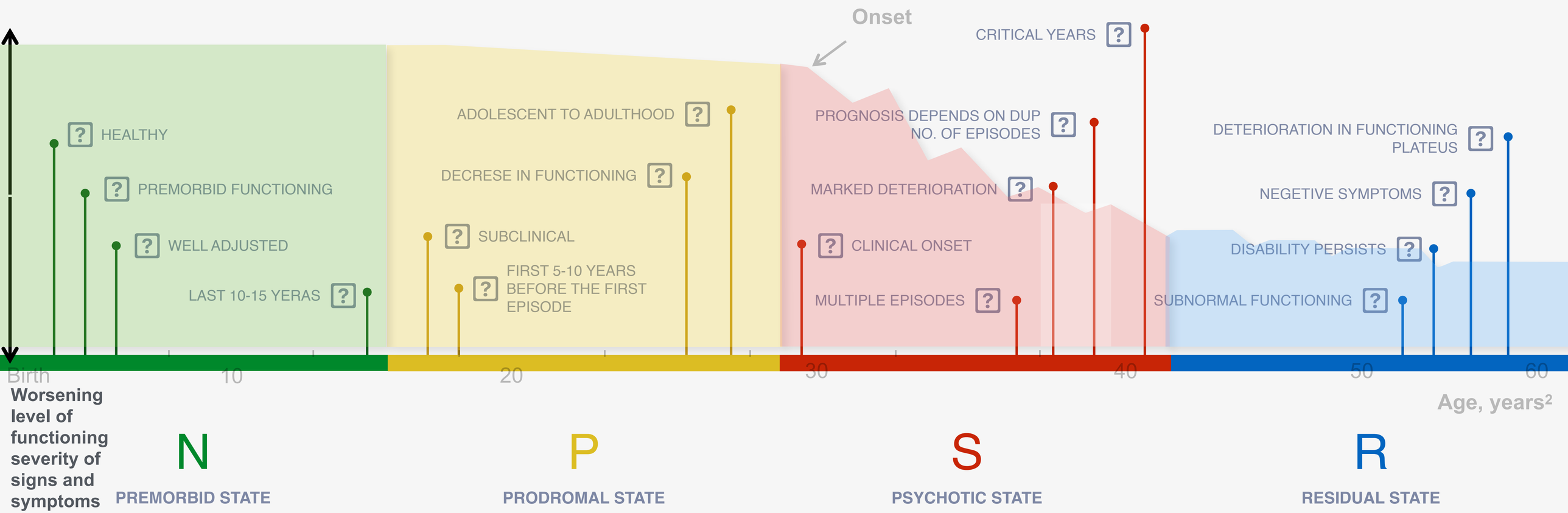
GWAS=genome-wide association study

1. Schizophrenia Working Group of the Psychiatric Genomics Consortium. Nature 2014;511(7510):421–427;
2. Ma et al. Transl Psychiatry 2018;8(1):67

COURSE AND PROGNOSIS

PROGRESSION OF SCHIZOPHRENIA

Schizophrenia progression may lead to functional decline



American Psychiatric Association. Practice Guideline for the Treatment of Patients With Schizophrenia. 2nd ed. Arlington, VA: American Psychiatric Association; 2004.

GOOD PROGNOSTIC FACTORS

LATE ONSET

PRESENCE OF PRECIPITATING FACTOR

ACUTE ONSET

POSITIVE AND AFFECTIVE

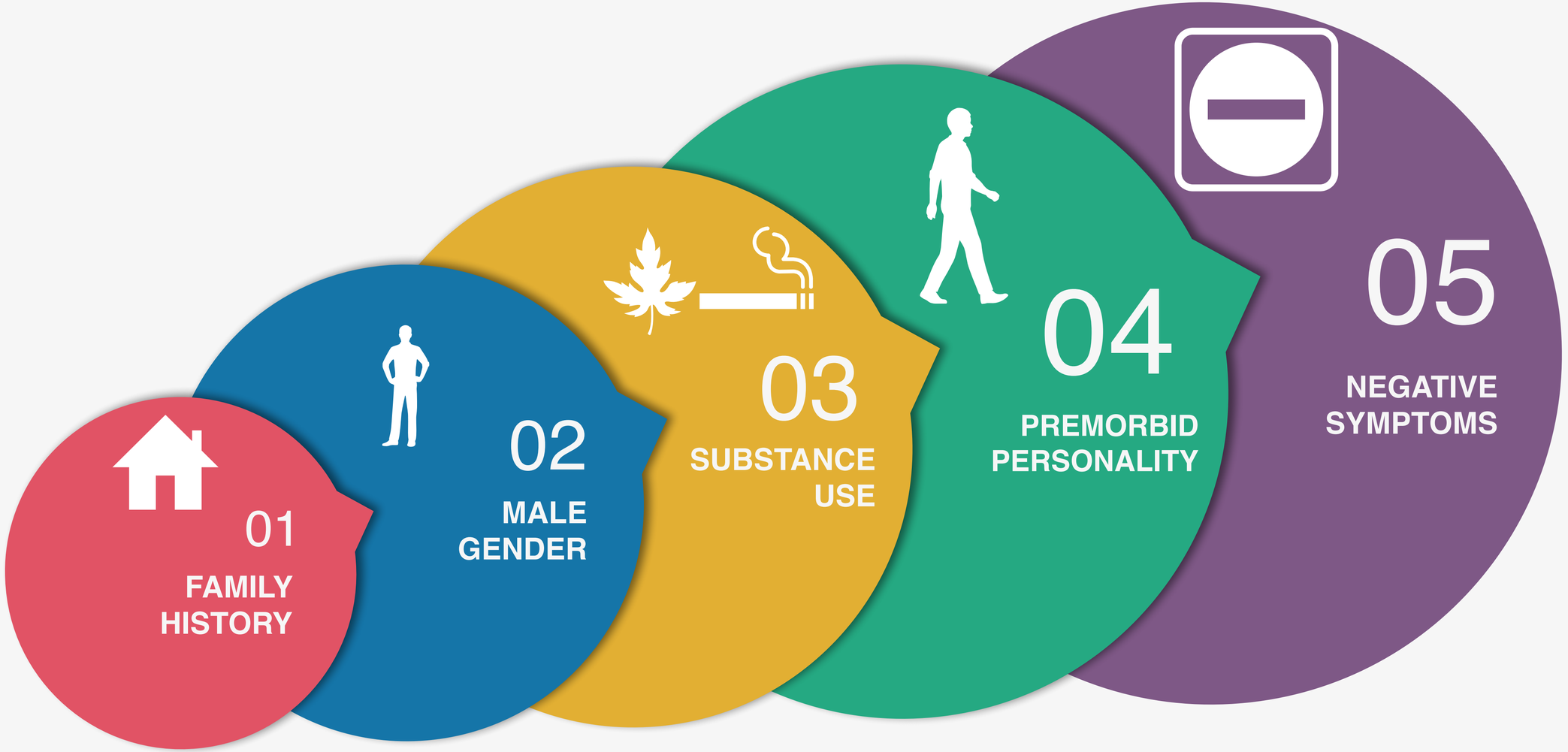
GOOD FAMILY SUPPORT

GOOD COMPLIANCE/ TOLERANCE

FEMALE GENDER

CATIE=Clinical Antipsychotic Trials of Intervention Effectiveness.
Glick ID, et al. J Clin Psychopharmacol. 2011;31(1):82-85.

BAD PROGNOSTIC FACTORS



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Pawan Kumar Gupta
Associate Professor
Department Of Psychiatry

APPROACH TOWARDS MANAGEMENT

IN NEXT FEW SLIDES YOU WILL UNDERSTAND HOW TO INCORPORATE
ALL THE PREVIOUS INFORMATION IN YOUR CLINICAL ASSESSMENT

ASSESSMENT OF A CASE WITH PSYCHOTIC SYMPTOMS

HISTORY TAKING

CHIEF COMPLAINTS

EXAMPLES: HEARING VOICES, ODD BEHAVIOUR, DECRESED SLEEP,

HISTORY OF PRESENT ILLNESS

EXPLORE IN DETAIL ABOUT EACH COMPLAINTS, ASSOCIATED SYMPLTOMS
ONSET
COURSE
DURATION
EPISODES
PRECIPITATING FACTORS
TREATMENT TAKEN

NEGATIVE HISTORY

TO RULE OF DIFFERENTIAL DIAGNOSIS
SUICIDE, HOMICIDE,
SUBSTANCE USE
MEDICAL HX.

HISTORY TAKING

PAST HISTORY

OF SIMILAR EPISODES
OTHER EPISODES
MEDICAL
SUBSTANCE USE

FAMILY HISTORY

REMEMBER ABOUT THE FAMILIAL RISK FACTORS
ATTITUDE OF FAMILY
HOUSING CONDITION
HX OF PSYCHIATRIC ILLNESS

HISTORY TAKING

PERSONAL HISTORY

PERINATAL, CHILDHOOD, ADOLESCENTS, EDUCATION, OCCUPATION, MERITAL, SUBSTANCE USE
PREMORBID PERSONALITY


NEVER FORGET TO PERFORM COMPLETE PHYSICAL EXAMINATION
PHYSICAL EXAMINATION
FINDINGS OF PHYSICAL EXAMINATION CAN AFFECT THE WHOLE DIAGNOSIS AND MANAGEMENT

MENTAL STATUS EXAMINATION

GENERAL APPEARANCE AND BEHAVIOUR

DISORGANISATION, ODDITY OF BEHAVIOUR, SPEECH, RAPPORT, THERAPEUTIC ALLIANCE

AFFECT

INAPPROPRIATE AFFECT, IRRITABILITY, PERPLEXITY

THINKING.

FLOW (IRRELEVANCE, INCOHERENCE)
FORM-FORMAL THOUGHT DISORDERS
CONTENT- IDEAS OR DELUSIONS,

PERCEPTION

HALLUCINATIONS
AUDITORY (2 OR 3 PERSON)

MEMORY

IMPAIRMENT IN MEMORY IS NOT USUAL FINDING BUT MAY OCCURE

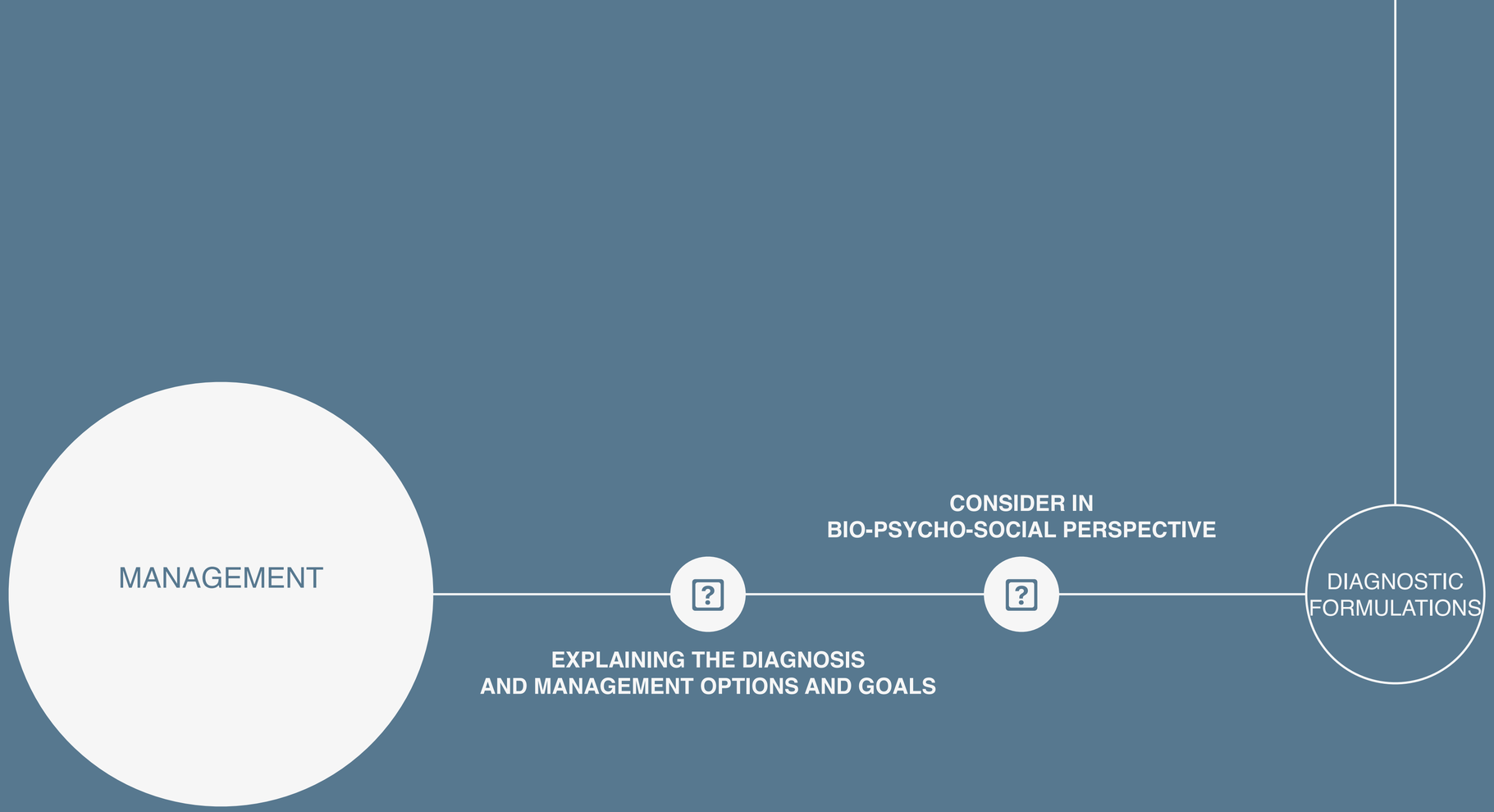
HIGHER FUNCTIONS & INTELLIGENCE

IMPAIRMENTS NOT APPARENT ON MSE BUT MAY PRESENT

JUDGEMENT & INSIGHT

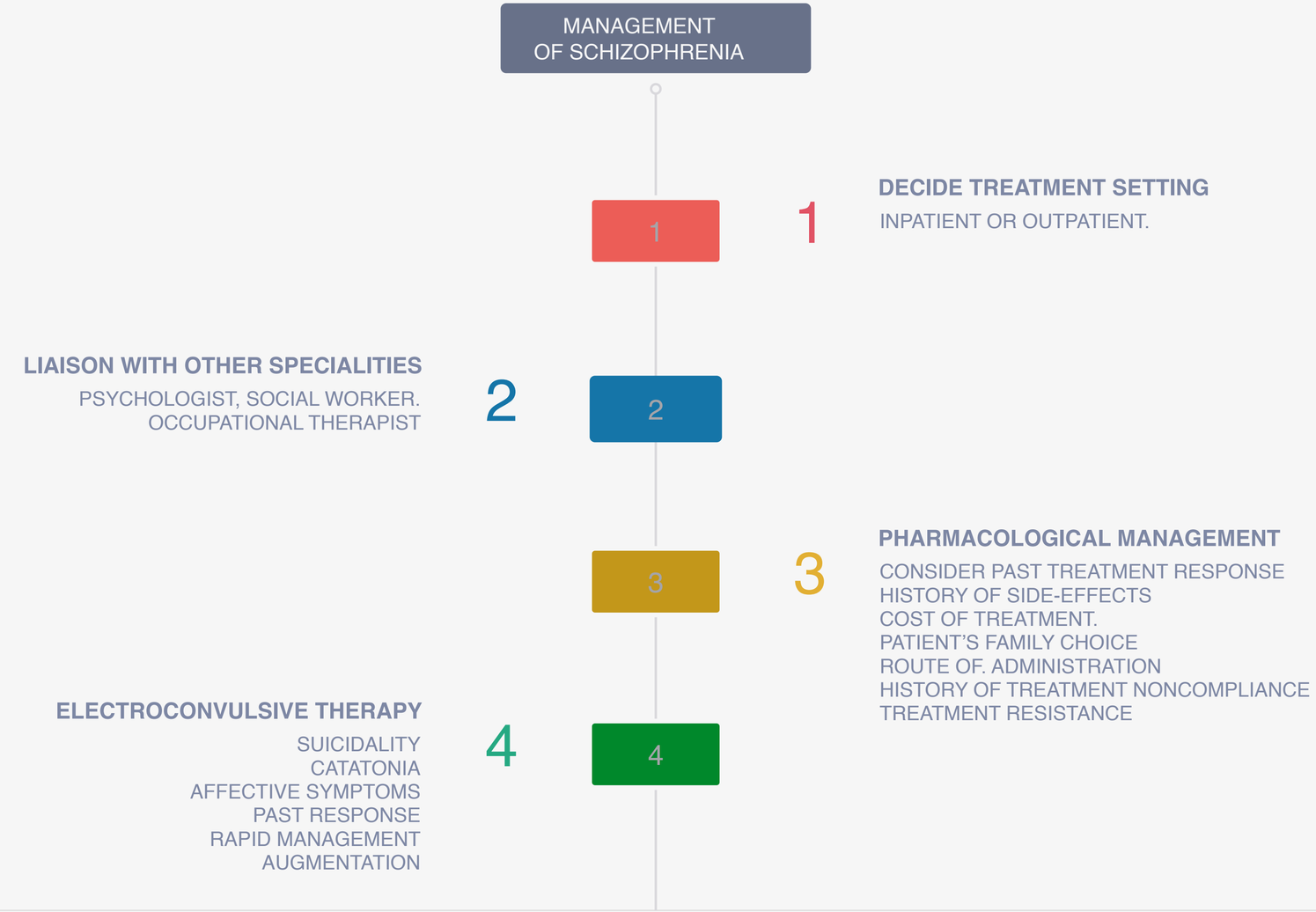
USUALLY IMPAIRED IN PSYCHOSIS
INDICATES LOST TOUCH WITH REALITY

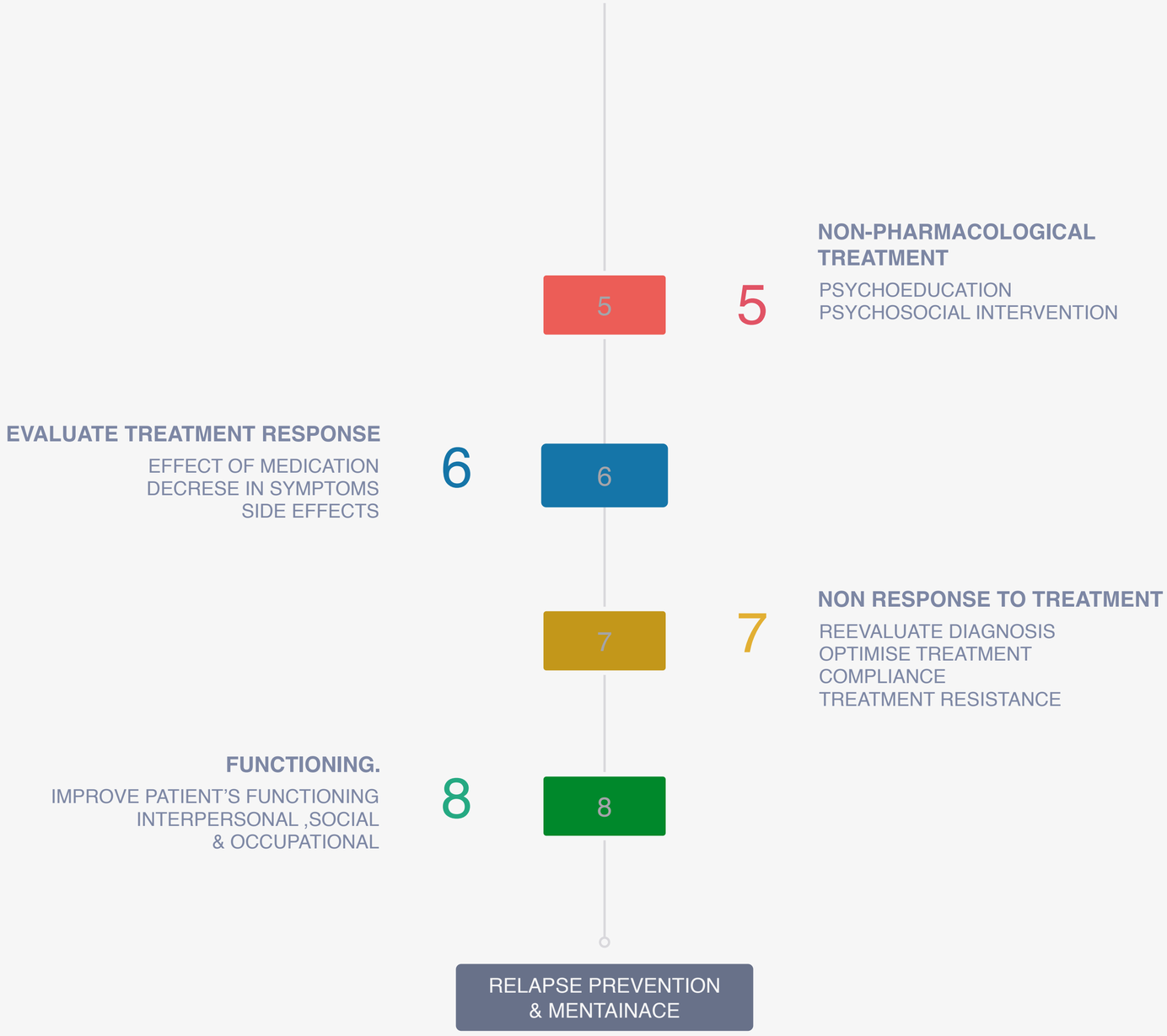
SUMMARIZE


MANAGEMENT
EXPLAINING THE DIAGNOSIS
AND MANAGEMENT OPTIONS AND GOALS
CONSIDER IN
BIO-PSYCHO-SOCIAL PERSPECTIVE
DIAGNOSTIC
FORMULATIONS

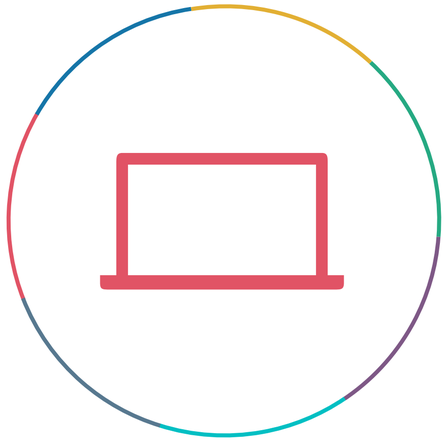
MANAGEMENT OF SCHIZOPHRENIA

Grover S, Chakrabarti S, Kulhara P, Avasthi A. Clinical Practice Guidelines for Management of Schizophrenia. Indian J Psychiatry [serial online] 2017 [cited 2020 May 16];59, Suppl S1:19-33. Available from: http://www.indianjpsychiatry.org/text.asp?2017/59/5/19/196972





Grover S, Chakrabarti S, Kulhara P, Avasthi A. Clinical Practice Guidelines for Management of Schizophrenia. Indian J Psychiatry [serial online] 2017 [cited 2020 May 16];59, Suppl S1:19-33. Available from: http://www.indianjpsychiatry.org/text.asp?2017/59/5/19/196972



e-mail me @

gpawan2008@gmail.com

Thank you!!

Improvement begins with "I"

Click here

Self-assessment

EPIDEMIOLOGY

ONLY 1%

THIS MEANS ABOUT 1 PERSON IN 100 WILL DEVELOP SCHIZOPHRENIA DURING THEIR LIFE TIME

GENDER & AGE