NURS 660 PSYCHOPHARM EXAM 1 2023-2024 MARYVILLE UNIVERSITY COMPLETE 200 QUESTIONS AND CORRECT DETAILED ANSWERS WITH RATIONALES|ALREADY GRADED A+

1. Know the positive and negative symptoms of schizophrenia, where they originate from, and what medications treat these symptoms.

Positive symptoms:

- psychosis, hallucinations, delusions
- Mesolimbic origin
- Medications: D2 antagonists

Negative symptoms:

- more related to mood disturbance, apathy, anhedonia, neuroleptic dysphoria, cognitive blunting
- Mesocortical/prefrontal cortex, nucleus accumbens, reward circuit origins
- Medications: None FDA indicated. Least likely to help are first generations r/t lack of 5HT2A action.

Cognitive symptoms:

- Attention issues, learning, problem solving, modulating behavior based on social cues, verbal fluency
- Dorsolateral prefrontal cortex origin
- Medications:

2. Identify the emerging neurobiological hypothesis of schizophrenia.

Dopamine Hypothesis:

- Hyperactivity of dopamine in mesolimbic pathway;
- Mesocortical pathway hypoactivity of dopamine

Glutamate Hypothesis:

- NMDA receptor hypofunction/dysconnectivity on GABA interneuron
  - NMDA receptor needs glycine and glutamate

- Possible issue with neurodevelopment/degeneration
- Glutamate Hypothesis leads to downstream overactivity of dopamine in mesolimbic, underactive mesocortical

Serotonin Hypothesis

- Serotonin hyperfunction
  - Hallucinogen psychosis
  - Parkinson's Psychosis = over-production of 5HT2A receptors
  - Psychosis in dementia = over-excitation/unclear-inhibition
- 5HT2A receptor hyperfunction in cortex
  - Agonism of 5HT2A, some 5HT2C
  - Visual hallucinations, mystical delusions, insightfulness
- Serotonin production
  - Tryptophan transporter
- Serotonin destroyed via MAO enzyme
- Neurodegeneration process that destroys GABA interneurons creating imbalance between serotonin and BABA neurotransmission at glutamate neurons in cerebral cortex
  - May lead to excessive activation of glutamate neurons via 5HT binding at 5HT2A receptors
- 5HT2a receptors are targets for drugs to treat psychosis in Parkinson's and dementia

3. Identify the different dopamine pathways and what role each pathway plays in relation to schizophrenia/psychosis and treatment.

Dopaminergic Pathways

- **Mesolimbic** – (Ventral tegmental area to nucleus accumbens) – overactive, too much dopamine activation of dopaminergic neurons = positive symptoms
- **Mesocortical** – (VTA to Cortex) – underactive dopamine from VTA to cortex = negative symptoms
- **Nigrostriatal** – [substantia nigra to striatum (including caudate + putamen)] – decreased dopamine causes drug-induced Parkinsonism, EPS
- **Tuberoinfundibular** – (dopaminergic neurons to hypothalamus inhibits prolactin release from pituitary gland) – decreased dopamine disinhibits the hypothalamus causing increased prolactin resulting in galactorrhea and amenorrhea (decrease FSH)

4. Identify potential medical emergencies associated with antipsychotic medications and how to manage these emergencies.

Agranulocytosis/severe neutropenia (<500): discontinue clozapine, daily CBC, heme consult if not resolved

Neuroleptic malignant syndrome: fever, tachycardia, muscle rigidity, confusion

➔ Rhabdomyolysis (high CK, K, WBCs)