Exam 3 Questions: **NOTE: the best way to do well on this exam is to study the lectures notes thoroughly! I will write my questions based on the lectures and the answers will be found in the lectures! However, I will take a majority of questions from below so know these questions thoroughly!**

**Lecture 9 Psychoactive Drugs**

1. What is useful about psychoactive drugs (think in terms of research approaches)?
2. What is the difference between exogenous substances and endogenous substances?
3. If I synthetically make dopamine or Acetylcholine and inject it into a subject, would these substances be considered endogenous substance?
4. If I synthetically make a drug that acts like dopamine, but is structurally different and inject it into a subject, would this substance be considered and endogenous substance?
5. Two basic ways that exogenous and endogenous substances can be used to answer research and clinical questions about brain and behavior, what are they? (think in terms of manipulation and measurement [i.e. Dependent and independent variables]).
6. There are many routes of administration of a drug, which is typically the most rapid while also delivering the highest concentration?
7. What are 5 things that ultimately influence the fate of any substance regardless of delivery method?
8. Absorption is different from binding, both which are different from inactivation and excretion, how so?
9. Agonists do what in terms of neurotransmitters?
10. Antagonist do what in terms of neurotransmitters?
11. There are 6 general steps that can be modulated by substances (drugs) what are they?
12. If you inhibit an auto-receptor what end effect would this have on NT’s or neurotransmission? What if you activated auto-receptors?
13. If you inhibit an hetero-receptor what end effect would this have on NT’s or neurotransmission? What if you activated hetero-receptor?
14. What is the margin of safety and why is it important? How do you determine the margin of safety?
15. What is the difference between LD and ED?
16. What is the difference of efficacy and affinity?
17. In the figure below what is represented by each line? Think it terms of efficacy and affinity and also sensitization and tolerance of substances?

18. In the Figure below, what does each line demonstrate? What is unique about the green line?

19. What is a Competitive antagonist? What is an example?

20. What is a non-Competitive antagonist?

21. Drugs or substances can produce 3 basic effects called Antagonism, Additive and Potentiation, how are these 3 effects different? Can you think of any examples in which these effects could be dangerous or helpful?

22. The Acetylcholine (Ach) terminal can be influenced by many different substances! Know these particular ones (Choline, Botox, Spider venom and Nicotine)?
23. The dopamine (DA) terminal can be influenced by many different substances! Know these particular ones (Amphetamine, cocaine and Haldol)?
24. What is unique about amphetamine compared to cocaine?
25. Can both amphetamine and cocaine produce states of schizophrenia? If so how is this related to Haldol treatment (i.e. What receptors do they work on and are the agonists or antagonists)?
26. The Serotonin (5HT) terminal can be influenced by many different substances! Know these particular ones (MAOIs and SSRIs and Tyrosine)
27. In simple terms what is serotonin syndrome?
28. Can a Rapid (within 1 hour) depletion of tyrosine Tryptophan produce a relapse in depressive symptoms? If so, what does this imply about molecular players and mechanism and depressive behavior?
29. If 28 is true, who was tryptophan was depleted in what type of subjects? (i.e. Normal control subjects or Depressed patients)
30. Norepinephrine (also called Noradrenaline or NE for short) is responsible for eating and awake states, how did I demonstrate this? What was injected for rats to eat (synthetic exogenous or endogenous substances?) what was injected for producing excitatory states? And how were these substances injected (systemic or microinfusion). What is unique about this injection method, i.e. What does it allow for you to specifically manipulate?
31. What is the definition of psychoactive drugs?
32. Sedatives are class of what? What are some examples?
33. What is a receptor action of the 3 sedatives we talked about? These drugs can produce a cross tolerance how so? The can also produce an additive effect how so?
34. Antipsychotics are an example of what? Psychotherapeutics?
35. The only drug for antipsychotics I want you to know is Haldol, how does this drug work? Can Cocaine and Amphetamine use produce psychotic like behaviors?
36. What are antidepressants? What are the two that I want you to know and how do they work? In lecture 10 we will talk in depth about the action of these drugs and One other.
37. I don’t care about tricyclics, because they are horrible drugs that suck, SO do not memorize them.
38. Can anti depressants like SSRIs and MAOIs affect 5HT/DA/NE?
39. Opiates do what by acting on what receptor? Heroin is different from Morphine how?
40. What is the amygdala pain pathway (slide 22 number 3)?
41. How do pain killers or opiate like drugs work in terms of neural system (what connects to what and what inhibits/activates what)?
42. What is the general stimulant definition that I provided?
43. What is a behavioral stimulant? Again how does amphetamines and cocaine work?
44. What are stereotyped behaviors? What are some examples? Could you uses these behaviors as dependent variables in a research study?
45. What is the exogenous and endogenous substances that bind to CB1 receptors, and how do these receptors work (where are they found what do they do?)?
46. I consider marijuana to be a psychoactive substance why?
47. I demonstrated two learning and memory paradigms used to suggest two different actions of CB1 receptors what are they and what did they demonstrate? Hint they are in red type!
48. Does CB-1 receptor activation (i.e. Use of marijuana) blunt acquisition of memory?
49. Can CB-1 receptor activation be used to treat pain, glaucoma or wasting syndrome of Aids patients?
50. What is substance abuse? Drug Dependences?
51. What is Drug Tolerance and how does this relate to efficacy and affinity?
52. Three types of tolerance mechanism exist what are they?
53. What is drug sensitization? What is physical dependence and how does it relate to withdrawal syndrome?
54. What is psychological dependence what does it produce?
55. What is conditioned drug tolerance? This phenomena is responsible for to different effects talked about in class one with morphine use in two different environments and another example with Vietnam vets addicted to heroin returning home. Both have to do with associated cues?
56. What are the three stages of addiction?
57. What is incentive-sensitization refer to?
58. What Brain area is strongly associated with rewarding experiences?
59. What is the basic neural system responsible for Reinforcement behaviors that produce reward properties associated with behavior? What is an experiment that demonstrates this (we talked about two)?
60. What two connected brain area maybe involved in Stereotyped behavior or habits?
61. Do not stress about Treatments in this lecture!

Lecture 10 Brain disorders

1. What are the 5 overlapping neural systems involved in behavior and that also responsible for clinical disorders of the brain.
2. What Brain areas are responsible for Dopamine (DA), serotonin (5HT), noradrenaline/Norepinephrine (NE) pathways (think in terms of starting
points and end points)? What behaviors are this neural pathways associated with?

3. Basically GABA and Glutamate neural pathways are everywhere in the brain! Again these two pathways are the major neural communicator of the CNS and are the most abundant.

4. What is the HPA axis? What brain disorders is associated with HPA axis dysfunction? What is the major endpoint hormone that is ultimately responsible for these disorders?

5. How is Cortisol (CORT) in humans released? There is two fundamentally different ways this can be accomplished?

6. Two hormones (NT’s) from the adrenals are released by stress one through the HPA axis (Adrenal cortex) and the other through the SAM system (adrenal medulla). What are they?

7. In a typical phobia/anxiety response involves activation of what ANS, this system produces a panic response in the PNS?

8. What is GAD? And what brain areas/projections (in red) are responsible/associated with this disorder? Hyperactivity/hypofunction/Overactivity!

9. What is PTSD? What initiates its onset? What areas of the CNS are possible associated with PTSD? What is a treatment for PTSD (name and is it an antagonist of what receptor, and thus is a inhibitor of what neural system?) Down regulation of this system likely leads to down regulation of the hyperactive CORT/amygdala.

10. Interestingly PTSD is associated with low circulating CORT and High circulating what?

11. What is Anxiety PD? What are the major neural systems associated with PD?

12. In PD what happens with increased activity to the periaqueductal grey produce?

13. Hint for anxiety disorders in general the CTRX-Amygdala-hypothalamus are overactive, and have a lack of modulation by both GABA projections and 5HT projections (from the Raphe). Also over activity of the CTRX-Amygdala-hypothalamus is likely a result of hyperactive NE system (projections from the Locus Coeruleus)!

14. What is OCD, what brain areas/projects/neural systems are associated with this condition? Note OCD is in particular has a hyperactive basal ganglion (likely produces the repetitive behaviors/habits with lack of PFC control)

15. In server OCD cases what surgical procedure can be used to treat this condition?

16. What enzyme(s) are possible genetic variations in OCD patients?

17. What class of Drugs maybe used to treat anxiety, what effect over the CNS does this DRUG produce, because it mainly binds to what receptor, which is found everywhere in the brain?
18. What is Depression? What are the 3 brain regions associated with this disease (hint in red-type)?
19. Basal CORT in depressed patients is different form controls how? This leads to a disturbance in NE and 5HT how?
20. How is stress and CORT associated with depression? (involves BDNF and neuronal atrophy)
21. What receptors are downregulated by MAOIs and SSRIs, is this a form of tolerance?
22. Ultimately antidepressants lead to changes in the neuron via increased BDNF, how is this accomplished? (I will tell you, signal transduction which involves metabotroic receptors, second messengers (kinases [proteins]) which lead to increased gene transcription and protein translation of BDNF!
23. Is BDNF responsible for synaptogenesis? What is synaptogenesis?
24. What is Ketamine? What was/is it typically used for, what receptors does it bind to and is it an antagonist or agonist?
25. What does ketamine lead to? Synaptogenesis?
26. How does Ketamine work to rapidly treat depression? (slide 22!) Steps 1-3 produce the rapid effects (SSRIs cannot lead to an emendate release of stored BDNF)! Also, Ketamine s effects are sustained by steps 1,2,3,4 and 5 What are these steps? Know these steps and not the other intermediate players (so know the order of activation and location of NMDA, GABA interneuron, AMPA and glutamate, VDCC/CA++ and BDNF release, mTOR activation and production of BDNF)! Steps 1-5!
27. What is Schizophrenia? What are positive and negative symptoms?
28. What are neural abnormalities of schizophrenia?
29. What areas of the brain are affected in schizophrenia patients?
30. What is the neural development model of schizophrenia?
31. What is AD? What major brain region is particularly affected by this disease?
32. What is Tau and AD? What does Tau mutations produces that ultimately lead to neuronal dysfunction and death?
33. To help keep it simple all Anxiety disorders have a similar dysfunctional neural system: Hyperactivity: Locus Coeruleus (NE neurons) leads to hyperactivity of the Cortex (usually the PFC), Amygdala, thalamus, hypothalamus. Hypo-activity: Dorsal Raphe Nuclei (5HT neurons) leads to a lack of modulation of Cortex (usually the PFC), Amygdala, thalamus, hypothalamus! Remember that the hypothalamus leads to Autonomic changes (i.e.anxiety symptoms, increased heart rate etc.).
34. GAD Specifically has increased activation of the whole cortex not just the PFC and seems to in particular have a lack of GABA inhibition of the cortex.
35. More than other Anxiety disorders PTSD patients have Atrophy of neurons in the hippocampus.
36. PD disorders have increased Limbic (also the cingulate cortex) activity. And increased activity in the PAG (periaqaductal grey), which produces defensive behaviors like postural freezing.
37. OCD disorders are characterized by increased Basal ganglia activity from increased Substance Nigra activity (DA neurons) and lack of modulation by the Raphe projects (5HT neurons).

**Lecture 11 Emotions and Stress**

1. What are some basic emotions?
2. What is the difference between Emotional Experience and Emotional expression?
3. How does Emotional Expression help use study the neurobiology of emotions in animals.
4. Can we measure emotional experiences in Animals, and if so How?
5. Can animals communicate subjective feelings (i.e. Can they verbally rate an a scale their feelings in a given experience?)
6. What are some similar behaviors that Humans and animals share in regrade to emotional expression?
7. What do emotions arise from (3 basic things)?
8. What two emotions have clear behavioral manifestations?
9. 3 theories of emotions are? (hint named after people)?
10. Darwins theory of emotion states what
11. What are examples of Darwin Emotions?
12. James lang theory of emotion is different form CannonBard theory how?
13. What is unique about Cannon Bards Emotional theory.
14. Antonio Damasio's hypothesis of emotions states what? This notion indicates that emotions must have a neural system.
15. What is the Limbic system? What are the major brian areas in this system and what particular function do these areas serve to process i.e. Fear, memory, emotions, which area does what particular response).
16. Destruction of the Amygdala in animals results in what changes?
17. What is fear conditioning? How is the Amygdala and hippocampus important for these learned fear behaviors?
18. What was the fear conditioning paradigm I talked about in class?
19. What are some similar effects that the amygdala in humans?
20. What doe polygraph detect?
21. Note that stimulus, is a thing or event that evokes a specific functional reaction in the CNS or PNS.
22. Stimuli that are perceived as stressful are not stressful by nature, stimuli do not posses a physical dimension of stress, and thus there can be not
receptor that detects stress like there are for light, sound, taste, touch and smell!

23. Stress like emotions do not have receptors that detect eliciting and perceived stimuli, so then how do we detect stressfully perceived stimuli?

24. Stress like emotions are states, in that they are discriminable from other states like anger or joy. Thus they require a neural system.

25. What is the difference between physiological and psychological stress?

26. What is a current definition of psychological stress proposed in class?

27. What is the ANS and Endocrine response to stress? What two Adrenal Hormones are important for these responses. And What major brain area is responsible for activation or secretion of these 2 adrenal hormones?

28. Acute stress is good for what?

29. Chronic Stress is bad why?

30. Chronic CORT secretion and Chronically high CORT are associated with several brain disorders and generalized physical disorders what are some?

31. Acute stress leads to acute secretion of CORT which is facilitates what aspects of learning and memory? And which are disrupted?

32. In question 31, what are the underling cellular/molecular changes that facilitate stress/CORT induced memory retrieval (recall) deficits and stress/CORT induced facilitation of memory storage? Keep it simple.

33. Stress involves the limbic system (PFC, Amygdala, hippocampus and hypothalamus).

34. What are 3 mechanisms in class that help deal with stress?

35. What is escapable and inescapable stress? What is the neural brain areas required for control over stress?

36. Is the ability to control stress adaptive?

37. Does voluntary exercise helps reduce stress? How?

38. What is stress habituation (this not drug habituation!)?

39. What brain area is required for Brain Habituation? How do we know this?

40. Does stress habituation decrease or increase CORT secretion?

41. How does Habituation typical occur?

42. The PFC through indirect connections modulates the Hypothalamus which in turn modulates CORT secretion!