**PSYCHOLOGY 612, fall 2018, DR. SHACKMAN**

**LEARNING OBJECTIVES FOR MODULES 2-19**

This document details the learning expectations for Modules 2-19. That is, what I especially want you to remember and to understand. In some cases, you will know the answer without study, in other cases you will need to review the slides. Please do not hesitate to reach out to me if you get stuck or have concerns that I can help you to address.

Good luck and warm wishes,

Dr. Shackman

**Module 2 – Impact**

* What did Walt Mischel suggest?
	+ Was he right? Are thoughts, feelings, and over behavior largely determined by the situation?
	+ What is meant by upper limit or ‘sound barrier’ for traits?
* How do you compute variance explained?
* What are some practically important ways in which dispositional traits are impactful?
	+ Academic performance
	+ Marital stability and satisfaction
	+ Mental health
	+ Physical illness/morbidity
	+ Premature death/mortality
	+ Economic impact
	+ Optional/Appendix Slides: Employment
* What is a meta-analysis?
	+ What are the advantages of a meta-analysis compared to an individual study?
* Did Big 5/OCEAN traits generally out-perform other kinds of important psychological factors, such as SES, IQ, age at marriage?
* What is C/SC/Conscientiousness? Representative items?
* What is N/NE/Neuroticism? Representative items?
* What are 3 ways in which contemporary culture potentially magnifies the impact of C/SC?
* Moffitt study
	+ Briefly describe
		- What’s the relationship between childhood C/SC and 3 key adult outcomes?
		- What are the 3 teen snares? What role did they play? Did teen snares account for all or only some of the impact of childhood self-control? Implications for intervention?
* What is a prospective longitudinal study?
	+ What are the key strengths and weaknesses of a prospective longitudinal study?
	+ Can the results of a prospective longitudinal study be used to ‘prove’ causation?

**Module 3 – Dimensions**

* How are temperament and personality similar vs. different?
	+ Are they categorically different or do they show continuity?
* Can the Big 5/OCEAN be discerned in elementary school aged children?
	+ What does this imply for those who conceptualize childhood temperament as categorically different than adult personality?
* What’s a hierarchy of traits?
* What are the Big 3?
* What are key characteristics of each of N/NE and E/PE in terms of emotion, appraisal, motivation, and cognition/attention/appraisal?
* What about C/SC, is it similar?
* What are the tradeoffs associated with using narrow vs. broadband traits?
* How universal are the Big 5/OCEAN across contemporary human cultures?
* Can we think of the Big 3 as purely cognitive or purely emotional?
	+ Briefly describe an example or two
* Do traits interact to produce important outcomes?
	+ What’s one example?
* Are particular traits good or evil?
	+ How is this related to “fit” with the environment (e.g. occupation, culture, special circumstances)?
* Are dispositional traits very fixed/immutable, very plastic/malleable, or somewhere in the middle?
* Which statistical test do we use to assess stability over time?
* Is there evidence that good (psychosocial therapy) or bad (marital conflict, trauma) things influence traits?
* Why are traits stable at all?
	+ What are some mechanisms that explain continuity over time?

**Module 4 – Measurement Issues**

* Are the Big 5/OCEAN real natural kinds that were just waiting to be discovered by scientists (like a new star in a telescope or a new cell under a microscope) or are they more like constellations (i.e., an organizational schema imposed by the human mind on the natural world)?
* What are some of the potential problems and limitations revealed in the YouTube video clip?
* What is the lexical hypothesis?
* In general terms, what are the Big 5 (OCEAN) and how did they come to be?
* In non-technical terms, what is factor analysis?
	+ Is it “lossy”? What is meant by “lossy”?
	+ Does it require subjective choices by the analyst or is it an objective tool for discovery (like a telescope)?
	+ How does the choice of input data determine the quality and breadth of the results?
		- If you include 50 items on fear/anxiety and 1 item on psychosis, how many factors will you extract?
	+ What is factor analysis good for?
* What is Block’s critique of the lexical hypothesis?
	+ What’s the potential problem of relying on untrained “lay” raters?
	+ What are some issues with everyday language? Does the same word always mean the same thing?
* Is the Big 5 superficial and descriptive or causal/mechanistic?
	+ Does it give us any clues about what to “fix” for people with psychological or psychiatric problems?
* What are 3 key limitations of self-report measures?
* What psychometric or statistical properties do we need to assess for biological or behavioral measures of T&P?
	+ What are the 2 key kinds of reliability?
		- Briefly describe each.
	+ What is construct validity? How is it related to sensitivity and specificity?

**Module 5 – Traits and States Part 1**

* What is Shackman’s definition of T&P (traits)?
* What are 3 models relating traits to states?
	+ Briefly describe each.
	+ What is the limitation of the traits = average of states model?
	+ What is an example of an item from the Eysenck Personality Questionnaire (EPQ) suggesting that traits, such as N/NE, can influence thoughts, feelings, and actions in the absence of clear-and-immediate reward or punishment?
	+ What is Fleeson’s probabilistic model?
		- Traditional measures of T&P suggest that each of us can be defined as a single, relatively fixed score (e.g. 5 on N/NE), whereas the probabilistic model claims that these traits are better conceptualized as a distribution of \_\_\_\_ that vary from moment to moment
		- According to this model, traits influence the \_\_\_\_\_\_\_ with which individuals engage in \_\_\_\_ behaviors.
		- What are 2 limitations of Fleeson’s probabilistic model? Does it address the role of the environment/situation?
* Describe the interaction or ‘reactive’ model.
* Allport, Eysenck, and other theorists have argued that the brain was somehow responsible for characteristic individual differences in the likelihood, magnitude, or persistence of emotional reactions.
	+ What did they suggest?
* What are 2 kinds of evidence supporting the reactive model?
	+ What is the limitation of the 1st kind of evidence?
* Briefly describe the limitations of the reactive (aka interaction) model? Does it address trait-like individual differences in 4 key processes that occur in the absence of immediate reward or punishment? Describe.
* These 3 models are \_\_\_\_ but \_\_\_\_ ?
* What are some ways In which trait-like biases and predispositions can occur in the absence of rewards and punishments (at ‘baseline’)?
	+ In what sense is this literally baked into widely used measures of N/NE? Example?
* Explain how the following figure provides evidence consistent with BOTH the reactive/interaction model and the endogenous/tonic model:

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* What are 3 ways in which traits can govern emotions in the absence of clear-and-immediate rewards and threat/punishments?

**Module 6 – Traits and States Part 2**

* Unconscious/Implicit Processes
	+ Is self-report data useful for measuring unconscious, pre-conscious, or implicit mental processes?
	+ Behavior is normally guided by both \_\_\_\_ and \_\_\_\_\_ processes that lie outside of \_\_\_\_\_.
	+ Briefly describe McNulty’s study
	+ Briefly describe Bechara’s study of conditioned emotional learning (Pavlovian fear conditioning)
		- Describe the ‘double dissociation’ (i.e., differential consequences of amygdala and hippocampal damage for implicit and explicit kinds of fear learning
		- What does this indicate?
			* Does this suggest separate neural substrates for conscious and unconscious mental processes?
		- Electrodermal activity (EDA aka GSR, SCR, SCL)
			* In brief what is it?
			* What can it be used to measure?
* What are 2 lines of self-report data that indicate that T&P is impactful in the absence of immediate reward or punishment (consistent with Watson & Clark’s endogenous/tonic model)?
	+ What are response biases?
	+ What is a potential limitation of this work in terms of possible response biases?
* How energetic is the brain at rest—a little or a lot?
	+ What does this suggest in terms of T&P?
* Briefly describe Kaczkurkin’s findings
* In a nutshell, how is ASL different than conventional fMRI techniques?
	+ What is conventional BOLD fMRI good for?
	+ When would you want to use ASL/perfusion fMRI?
	+ How is this relevant to testing the endogenous/tonic model?
* Abercrombie found a convergent pattern using different techniques. In a nutshell, what was her evidence that T&P is discernible in the brain’s spontaneous, on-going activity?
* What is a key limitation of the Kaczkurkin, Canli, and Abercrombie studies?
	+ Were the subjects really at emotional ‘baseline?’ Is the scanner a relaxing environment?
* Drew Fox extended this line of work by scanning monkeys in 4 conditions.
	+ Briefly describe the study.
		- What were the 4 conditions?
		- What did he observe in potentially threatening environments?
		- What about more secure environments? Is T&P evident when the monkeys were at home, chilling with their cagemate?
		- What is the limitation of this study?
* FDG-PET
	+ Signals measured in the PET scanner reflect those \_\_\_ of the brain that were \_\_\_\_\_ active during the preceding behavioral challenge (e.g., exposure to the human intruder’s profile).
* Shackman extended this line of work by assessing the functional connectivity of the amygdala under sedation?
	+ Did it work? Were stable individual differences in N/NE/Anxiety evident in the spontaneous, on-going connectivity of the brain in monkeys?
	+ In human children?
* Based on this body of work, using different species, populations, and measurement techniques, Is T&P embodied in the on-going activity of the brain or does it require a perturbation or challenge (reward, punishment)?
	+ How might this be related to the 4 key processes reviewed at the end of Module 5)?
	+ Is this consistent with the endogenous/tonic model of Watson and Clark?

**Module 7 – What do traits do?**

* What are 4 key parameters of the emotional response to challenges, according to Richie Davidson?
	+ How might these be related to dispositional traits?
	+ How would you test differences in threshold?
	+ Does the amygdala only show differences in peak response? If not, what are the other most relevant parameters?
* Describe Gray’s Big 2 (BIS/BAS) model
* How cleanly does the BAS map onto Caspi’s Big 3 model? What are some ways in which BAS does not map cleanly?
* BIS and BAS have been linked to frontal EEG asymmetry.
	+ Which pattern is associated with BAS, appetitive motivation, and reward sensitivity?
	+ Which pattern is associated with BIS, N/NE, avoidance motivation, and punishment sensitivity?
* How is Carver and White’s BIS/BAS questionnaire supposed to be different than extant trait measures?
	+ What is meant by sensitivity vs. typical experience?
* What approach did Gable and her colleagues take to assessing differences in emotional reactivity vs. exposure to rewards/punishments (positive/negative daily events)?
* What were the 3 key conclusions? (Take care to clearly describe the role of ‘exposure’)
* Do traits only influence reactivity? What are some ways in which they influence motivated behavior which, in turn, can influence mood?
* What does this imply about agency and free will?
	+ Are individuals necessarily the passive victims of chance encounters with rewards and punishments?
	+ If not, why not?

**Module 8 – EEG and MRI: Strengths and Limitations**

* T&P reflect trait-like individual differences in the \_\_\_\_ and \_\_\_\_\_\_\_ of the brain
* What are the key strengths and weaknesses of each technique?
* Are they mechanistic/causal or correlational?
* What kinds of things are hard to do with fMRI?
* Do they provide direct measures of neuronal firing?
* What is spatial resolution?
	+ If an image appears blocky or pixelated, it reflects low \_\_\_\_\_\_\_?
	+ Assays with high spatial resolution are on a \_\_\_\_\_ grid with \_\_\_\_ samples per unit space
* What is temporal resolution?
	+ Assays with high temporal resolution capture \_\_\_\_\_\_ samples per unit time
	+ What happens if you are trying to take a photograph of something that unfolds very quickly but you forget to switch to ‘sports mode’ (high temporal resolution)?
* If you’re interested in studying small subcortical structures, which technique should you use?
* If you want to measure psychological or neural processes that happen very rapidly, which technique should you use?
* Briefly describe the differences between structural and functional MRI.
* Does fMRI directly reflect blood oxygenation or neuronal firing?
* In very general terms, how do you compute an ERP? Why is it useful to average over many noisy trials to create a single ERP? What does averaging get rid of?
* Tomarken: What statistical properties do we need to assess for biological measures, like EEG and fMRI?

**Module 9 – Intermediate phenotypes, endophenotypes, and markers/biomarkers**

* What’s a general problem with trying to uncover the root causes of complex traits like C/SC (or psychiatric disorders, like substance abuse)?
* What might we do to circumvent this problem?
	+ What does it mean to ‘reverse engineer’ a system?
* What’s the problem with one-shot self-report measures of T&P?
* What are some concerns with reducing a trait to a single number?
	+ Are there different ways to arrive at the same number or does a “5” on some scale always indicate the same pattern of symptoms?
	+ Describe an example of how this problem of ‘heterogeneity’ applies to DSM 5 mental illnesses (e.g., PTSD or MDD)
* We can conceptualize the BART as a candidate intermediate phenotype for \_\_\_\_\_\_\_\_\_ ? We can conceptualize low C/SC as a candidate intermediate phenotype for \_\_\_\_\_\_\_\_\_\_\_ ?
* Intermediate phenotypes serve as a bridge between \_\_\_\_\_\_\_\_\_\_ and \_\_\_\_\_\_\_\_\_\_\_ ?
* What are some other benefits of using continuous/dimensional IPs, rather than categorical diagnoses or traits?
* How are markers different than intermediate phenotypes?
* What are 2 kinds of markers?
* Is there any value to markers? When might they be practically useful?
* What are 2 kinds of intermediate phenotypes?
	+ What is special about endophenotypes?
* Has the endophenotype strategy ever identified the links connecting molecules/genes to endophenotypes, or the links from endophenotypes to traits or psychopathology?
* What are some ways in which you might try to establish that a candidate intermediate phenotype is actually the cause of a trait or mental illness?
	+ In humans?
	+ In animals?
* Treatments and drug development
	+ Easy or hard to develop new treatments?
	+ Quick or slow?
	+ Cheap or expensive?
	+ Do treatments cure everyone or reduce symptoms in some?
* In very general terms, how might an intermediate phenotype help discover new treatments? How might it help improve or refine research in animals? What are some useful things you could do in the clinic with an intermediate phenotype (e.g. ‘anxiety brain fingerprint’ or ‘working memory deficit’ or ‘aberrant performance on the BART’)?
* Do intermediate phenotypes or markers have to be biological?
* Can a marker (e.g. tracks) cause a trait or disorder (e.g. injectable drug abuse)?

**Module 10 – Nature & Nurture Part 1: Heritability**

* What are the 4 lessons?
* Is T&P mostly nature/genes, mostly environment/experience/nurture, or a mix of the 2?
* Are specific behaviors and psychological processes hardwired into our genes?
* Can the ‘environment’ be heritable? What are 2 scenarios in which T&P and measures of the environment can be genetically correlated?
* Is nature (genetic influences / heritability) static or can it change over time? If I measure the heritability of a trait in 3 year olds, do I know the heritability in 30 year olds?
* What is heritability? What is the conceptual formula?
* In general how can you actually go about quantifying heritability?
* Standard techniques for computing heritability assume that \_\_\_\_ and \_\_\_\_ are uncorrelated (‘independent’) and do not interact with one another (technical term: have ‘additive’ rather than ‘multiplicative’ effects)
	+ Yet there is compelling evidence that these 2 simplifying assumptions are often violated in the real world.
	+ Moreover, there is evidence that genes interact with one another, violating yet a third simplifying assumption
	+ Hill Goldsmith: The bottom line is that estimates of heritability can be \_\_\_\_ing.
* Is heritability absolute or does it depend on the population and context in which it is measured?
	+ Describe an example (K-Pop vs Weasleys).
	+ How is heritability related to the amount of variation in a trait (e.g. hair color) in a particular sample or population?
	+ Having 2 arms or 10 fingers has a heritability of approximately \_\_\_\_\_\_\_\_\_, but that does not mean that \_\_\_\_ are not involved in the generation of arms or fingers. In other words, typical estimate of h2 and genetic influences are not the same thing.
	+ To social rules, taboos, or culture?
* What are 4 common misconceptions about heritability?
	+ Are genes or phenotypes/traits passed down?
	+ Does heritability describe individuals or variation in groups of people?
	+ Are genes our destiny?
	+ Are heritable traits potentially amenable to intervention or are we stuck?
		- Do we have free will in the face of apparent genetic destiny?
		- Provide an example (e.g. height, alcoholism, breast cancer).
	+ In short, “Heritability describes \_\_\_\_ ; it does not predict \_\_\_\_\_\_\_”
	+ Does heritability tell us something about the source of plasticity of group differences in a trait (e.g. race differences in height or IQ)?
	+ Are heritable traits deterministic or probabilistic?
* What do family twin and adoption studies teach us?

**Module 11 — Nature & Nurture (Part 2): Molecular Genetics**

* Provide a *brief*, non-technical description of:
	+ DNA
	+ chromosomes
	+ genes
	+ function of genes
	+ alleles
	+ SNP
	+ GWAS
	+ SNP chip
* Do most genes differ across individuals?
* What’s the vector of heredity?
* What is GWAS?
	+ What are the major strengths and weaknesses of GWAS approach?
	+ Why bother? In a nutshell, what is the premise or the promise of discovering the genetic variants that are associated with traits or disorders?
* Has there been any success using GWAS with psychiatric disorders?
	+ E/PE?
	+ N/NE?
* What is the 4th law of behavioral genetics?
	+ Do traits typically reflect few genes with big effects or many genes with small effects?
* What are the consequences of small statistical effects for replication? For sample size requirements?
* Is there any practical or biomedical value to weak genetic associations? Do weak genetic associations necessitate a lack of therapeutic value?
	+ Describe. Can GWAS be used to discover biomolecular pathways or prioritize ‘targets’ for drug development or the re-purposing of existing drugs?
* Do genes always have ‘main effects’ or do they sometimes confer risk that is conditional on the environment (G\*E interaction)?
	+ Describe an example of a G\*E interaction identified by Caspi and colleagues

**Module 12 — Nature & Nurture (Part 3): Neurogenetics and Epigenetics**

* What are the 3 key components of Caspi’s strategy for investigating G\*E interactions?
* In a nutshell, what is the neurogenetics strategy and what is the potential value?
	+ In principle, what can you potentially discover or address that would otherwise be difficult or impossible?
* Does the neurogenetics strategy rest on assumptions?
	+ Is it important to test these assumptions? In small samples or big samples (as with data shared across large international consortia)?
	+ Is the serotonin transporter polymorphism (5-HTTLPR) actually related to serotonin levels or receptors in the amygdala, as hypothesized by Caspi, Hariri, and the neurogeneticists? What do the most recent data teach us?
	+ Is the serotonin transporter polymorphism (5-HTTLPR) actually related to amygdala activation, as hypothesized by Caspi, Hariri and the neurogeneticists? What do the most recent data teach us?
	+ File-drawer problem: How might unpublished null results distort or bias meta-analyses of the literature?
	+ Do stress (negative life events, trauma) and the serotonin transporter polymorphism (5-HTTLPR) actually interact to predict the development of major depressive disorder (MDD), as expected based on Caspi’s highly cited *Science* paper? What do the most recent data teach us? Was Caspi right?
* What is the HPA axis? What does it do?
* In a nutshell, what kinds of life-long changes happen to rodents that have been handled by human experimenters?
* What drives these life-long changes to temperament?
	+ What is the relevance of maternal LG-ABN?
* What are the consequences for the amygdala?
* How are these psychological changes related to epigenetic regulation of glucocorticoid receptors in the hippocampus?
* What is epigenetics? Is the regulation of genes static or can it be influenced by experience and the environment?
* What are the 2 major flavors of epigenetics?
* How is epigenetics related to differentiation and development?
* Can epigenetic alterations be inherited?
	+ Yes! No! or Maybe?!

**Module 13 — Neuroticism/Negative Emotionality and Psychopathology (Part 1)**

* What’s an ‘emotional’ (sometimes termed ‘internalizing’) disorder?
* How is N/NE related to the emotional disorders?
	+ Cross-sectional data? Prospective longitudinal data? Both? Neither?
* What are key features of the anxiety disorders?
* Of major depressive disorder (MDD)?
* Are the emotional disorders
	+ Common or rare?
	+ Debilitating?
	+ Under or over-treated?
	+ Expensive or cheap?
	+ Major or minor burden on global public health?
* Does N/NE confer risk for a particular disorder or a whole family of closely related disorders?
* Briefly describe Barlow’s claim that N/NE and the emotional disorders reflect a common cause.
	+ Briefly describe the different lines of evidence
		- Which 3 lines of evidence show that emotional disorders and N/NE co-vary?
		- Which 2 lines of more mechanistic or causal evidence suggest that they reflect a common root cause?
		- Which 2 lines of research begin to address the biological substrates of the common cause?
* Are DSM diagnoses natural kinds waiting to be discovered or a convenient heuristic?
* What is the internalizing spectrum of disorders? Why do scientists and clinicians conceptualize the anxiety disorders and depression as a spectrum (name one piece of evidence)?
* What explains why individuals develop particular disorders, according to Barlow?
* Is N/NE a cause, a symptom, or ‘the same as’ the emotional disorders?
	+ What is the key piece of evidence?

**Module 14 — Behavioral Inhibition and Psychopathology (Part 2)**

* Briefly describe BI
* How is BI assessed
	+ in children?
	+ n monkeys?
	+ **i**n human adults?
		- What are some potential concerns with this approach?
* How is BI related
	+ to the Big 3 and N/NE?
	+ to Gray’s BIS?
	+ to anxious temperament (AT) in monkeys?
* How stable is BI across the lifespan?
* Do most kids grow out of it?
	+ Should we be fatalistic?
	+ How is this related to developmentally appropriate fears?
* How is BI related to psychopathology?
	+ Is there prospective longitudinal evidence?
	+ All kids who show extreme BI or only the subset of children who consistently show elevated BI across repeated assessments?
* Briefly describe SAD
* How might BI predict the normative acquisition of crucial social skills?
	+ How might this contribute to the development of social anxiety disorder?
* Is BI a strong candidate endophenotype for SAD?
	+ Is it heritable?
	+ What do we know at present about causation?

**Module 15 — Role of the Extended Amygdala in Negative Emotionality, Behavioral Inhibition, and Psychopathology (Part 3)**

* If you observe activation in the amygdala, is it safe to conclude that an individual (or group of individuals) is feeling scared, anxious, or fearful?
* Is amygdala activation sensitive AND specific to fear and anxiety? Or, is it sensitive AND non-specific?
* What is the relevance of the amygdala to fear conditioning or ‘learned’ fear?
	+ What are the consequences of amygdala damage?
* What is ‘fear potentiated startle’ (FPS)?
	+ How is FPS measured in rodents? In humans?
* What is the relevance of the amygdala to innate, intrinsic, or ‘unlearned’ fears (e.g., of predators or robo-predators)?
	+ Is there causal/mechanistic evidence? Imaging evidence?
* How is the amygdala related to freezing and BI in monkeys?
	+ Is there causal/mechanistic evidence? Imaging evidence?
* What’s special about excitotoxic lesions?
	+ What do they spare?
	+ What’s the problem with classic lesion techniques (e.g., gross dissection)?
* What is the relevance of individual differences in amygdala activity/reactivity to individual differences in N/NE, including trait anxiety?
* To adults with a childhood history of extreme BI?
* Patient SM
	+ What is the nature of Patient SM’s neurological damage?
	+ What are the consequences for the experience and expression of fear and anxiety in naturalistic settings (e.g., exotic pet store, haunted house)?
	+ Does Patient SM show deficits in her ability to learn from scary experiences in the real world? Does she avoid people and situations that were associated with trauma?
	+ How does she look when you ask her to complete paper-and-pencil measures of trait anxiety (N/NE)? High, low, normative?
* Amygdala Stimulation/Video Clip
	+ What are the consequences of amygdala stimulation (specifically the Ce) in humans?
* What evidence suggests that the amygdala exerts ‘bi-directional’ control over fear and anxiety?
* Does this evidence mean that you can conclude that the amygdala is a ‘fear/anxiety’ center? Or is it possible that the amygdala is sensitive to a wide range of stimuli and participates in a broad spectrum of psychological processes?
* What is the relevance of the amygdala to mood and anxiety disorders (sometimes termed ‘emotional’ or ‘internalizing’ disorders)?
	+ Is it more or less reactive in patients?
* Do individual differences in amygdala reactivity in the scanner prospectively predict the onset of anxiety disorders in the future?
	+ What is the relevance of exposure to stress or negative life events?
* Do anxiety reducing drugs alter amygdala reactivity?
* What about cognitive behavioral therapy for anxiety?
* What kinds of regions does the amygdala project to?
	+ How is this related to triggering or orchestrating states of fear and anxiety?
* Is the amygdala only related to fear and anxiety?
	+ Does it contribute to trust?
	+ To personal distancing?
	+ Optional/Appendix: To economic and behavioral choice?
* Optional (Appendix Slides): Is the amygdala necessary for all kinds of fear, anxiety, and panic? Is there any evidence that Patient SM can experience panic? What are the implications for our understanding of the amygdala?

**Module 16 — Splitting Negative Emotionality into Its Constituents, Part 1**

* Is N/NE simple or complex? Messy or straightforward? Does it encompass a heterogeneous set of signs and symptoms?
* What’s the problem with reducing N/NE/BI to a single number, if your goal is to discover the underlying neural bases?
* What is one strategy for circumventing this kind of complexity and heterogeneity?
* How are uncertainty and anxiety related, according to Grupe & Nitschke?
* What are the 5 key components of the anxious phenotype, according to Grupe & Nitschke?
* Are the five components *specific* to particular anxiety disorders, such as Generalized Anxiety Disorder, or are they non-specific (‘transdiagnostic’)?
* Briefly describe the dot probe task
* What have dot-probe studies taught us about anxiety and vigilance?
* ABM
	+ Can hyper-vigilance be retrained, can attentional biases be modified?
		- What are the consequences of ABM for stress reactivity in unselected (non-clinical) samples?
			* Why are the behavioral results so compelling? What is the potential limitation of relying exclusively on self-reported changes in anxiety or distress following ABM training?
		- For patients with anxiety disorders?
		- What does this imply in relation to causation or ‘active ingredient’?
* Briefly describe 2 pathways through which the amygdala can influence or prioritize the processing of incoming sensory information
* What do amygdala lesions do to face processing in the visual cortex (fusiform face area)?
* Briefly describe the amygdala 🡪 Nucleus Basalis of Meynert / Basal Forebrain 🡪 sensory cortex circuit.
	+ What is the relevant neurotransmitter?
	+ What does the transmitter do to sensory processing in the cortex? To cortical arousal?
* Why are safety signals important?
	+ How do they normally allow individuals to tune, titrate, or optimize their anxiety?
* Briefly describe the evidence for over-generalization and the maladaptive persistence of anxiety in objectively safe contexts.
	+ <Brief description>
	+ Why is this a big deal? What was the older model?
	+ What is the relevance for the development of future anxiety disorders? For childhood BI?
* Which brain regions support the maladaptive persistence of contextually inappropriate anxiety?

**Module 17 — Splitting Negative Emotionality into Its Constituents, Part 2**

***Note: If you open the slide deck in PowerPoint, you can see the notes for each of the slides. These will be useful for answering the following questions and mastering the Learning Objectives for this module***

* Why is avoidance maladaptive? What are the consequences of avoidance for learning to extinguish one’s fears and anxieties?
* Contemporary models suggest that avoidance reflects hyper-sensitivity to threat or punishment. More recent work has expanded this idea, suggesting that anxious individuals are also more sensitive to \_\_\_\_ ?
* Briefly describe the ERN.
	+ What is it?
	+ What elicits or evokes it?
* Is the ERN increased or decreased by anxiolytic drugs, such as benzodiazepines? [Slides dropped from Lecture: It is decreased]
* Which region of the brain is thought to help generate the ERN and other frontal-midline ERP’s (e.g., FRN, N2)?
* Is the ERN bigger or smaller in individuals with a more anxious temperament (i.e., those with higher levels of N/NE/BI)? What about other frontal-midline ERP’s?
* Are these signals (ERP’s) clinically relevant?
	+ Do individual differences in the amplitude of the ERN predict the future development of anxiety disorders?
	+ Is stress exposure important?
	+ Does it predict symptom severity?
* What do these data imply about the potential relevance of the MCC—the region that generates these scalp-recorded signals—to trait-like individual differences in Anxiety/N/NE/BI?
* What do we know about the MCC’s function more generally: What 3 kinds of tasks (or psychological processes) are co-localized in the MCC? (Put another way: what 3 kinds of tasks consistently activate an overlapping region of the MCC, as indexed by either fMRI or neuronal recordings?)
* Are anxiety/negative affect, pain, and cognitive control associated with similar patterns of activity in the MCC, suggesting that they are functionally integrated there?
* The adaptive control hypothesis (TACH) suggests that the MCC uses information about threat, punishment, errors, and pain to bias, tune, and optimize behavior when there is uncertainty about \_\_\_\_\_ and their potentially \_\_\_\_\_\_\_\_ .
* How are ERP signals generated in the MCC related to actual, overt behavior?
	+ Do individual differences in the size of the ERN and FRN actually predict behavior on future trials?
	+ Do they predict increased or decreased caution, inhibition, and avoidance?
* Slides Dropped: Pharmacology: Do anxiety-reducing drugs, such as alcohol and benzodiazepines influence this process? Yes!
	+ Do they increase/decrease anxiety and negative affect? Decrease!
	+ Do they increase/decrease ERP signals generated by the MCC? Decrease/Disrupt!
	+ Do they increase/decrease behavioral signs of avoidance, caution, and inhibition? Decrease/Disrupt
* Are individuals with an anxious, neurotic disposition more or less sensitive to uncertainty?
* The elevated plus-maze is a widely used behavioral measure of anxiety in rodents…
	+ Briefly describe it.
	+ What behaviors would distinguish bold from anxious rodents?
* Is hyper-reactivity to uncertainty altered by anxiety-reducing compounds, such as alcohol and benzodiazepines?
* What is the implication of knowing that the very drugs used to treat and ameliorate pathological anxiety in the clinic selectively and significantly reduce anxiety elicited by uncertain threat?
	+ What does it mean in terms of causation and ‘active ingredient’ in the anxious phenotype?
* Which brain region(s) is(are) especially sensitive to uncertainty?
* Is there evidence that the 5 constituents are co-vary or interact in ways that can reinforce and promote chronic, pervasive distress and anxiety?
	+ Do they seem to hang together in anxious individuals? (Hint: you can respond on the basis of either the material at the end of the module focused on work in marmosets or based on Pete and Paul)

**Module 18 — Positive Emotionality, Self-Control, and Dopamine (Part 1): Depression and Anhedonia**

* Briefly describe E/PE, BAS, and how they are conceptually related
* Is MDD a major or minor burden on global public health?
	+ ‘Common and costly’ or ‘rare and comparatively cheap?’
* Briefly describe the relationship between T&P and MDD according to recent meta-analyses.
	+ Does E/PE prospectively predict the future emergence of depression?
	+ What’s the implication?
	+ What does this not prove, in terms of causation?
* Briefly describe Shackman’s ‘too cold’ hypothesis
* Briefly describe anhedonia/liking vs. abulia/avolition/wanting
	+ How are they normally related?
		- Briefly describe the normal cycle of approach/appetitive/wanting 🡪 consumption/liking 🡪 satiety
* Describe key diagnostic criteria for depression
* How do patients with depression respond to positive and negative stimuli in the lab?
* Treadway/Pizzagalli:
	+ Briefly describe 2 major ways to behaviorally assess reducing wanting/appetitive motivation in the lab
	+ Briefly describe Mike’s EefRT task
	+ Briefly describe Diego’s RR task
	+ Are behavioral measures of RR stable? Heritable? Candidate endophenotypes for MDD?
* Are these kinds of lab results consistent with daily diary and experience sampling data collected in the real world? Briefly describe recent diary and experience sampling data.
	+ Depressed individuals appear to \_\_\_\_\_invest in high effort/high-reward activities in daily life.
* Briefly describe behavioral activation therapy.
	+ Does BAT reduce depression?
	+ What does this suggest for causation (see the *Key Take Home Points* slide)?
* What is the MFB?
	+ Which regions does it connect?
	+ Which transmitter system is it associated with?
* Briefly describe the behavior of rodents given the opportunity to SS the MFB
* Briefly describe the evidence that dopamine is a kind of common neural currency for reward. How is dopamine release related to drugs of abuse?
* Briefly describe Roy Wise’s Dopamine=Pleasure/Yumminess hypothesis
* Briefly describe the results of human MFB SS work (e.g., The Tulane Studies / Subject “B-19”).
	+ Changes in liking, wanting, both?
* How did Berridge and Robinson behaviorally tease apart wanting from liking in rodents?
* Briefly describe what they learned about dopamine using this kind of behavioral ‘read out.’
	+ Wanting/effort or liking/pleasure?
	+ Is dopamine necessary and/or sufficient for hedonic pleasure?
* Based on their research (and more recent work in humans by Christian Buchel’s group), which regions and transmitters mediate hedonic pleasure and liking?
* The ventral striatum is part of the nucleus \_\_\_\_\_\_\_\_\_ .
* What about humans?
	+ What is the Monetary Incentive Delay Task (MIDT)?
	+ Based on recent meta-analyses of brain imaging studies, is the VS/NAcc generally responsive to reward?
	+ Does activation in the VS assayed using fMRI predict positive arousal/excitement?
	+ Does it predict trait-like individual differences in E/PE?
	+ Is VS activation trait-like?
* What about mechanistic evidence?
	+ Do neurofeedback and/or drug studies suggest a causal role? Briefly describe.
* Is this system altered in patients with MDD?
* Do individual differences in VS/NAcc activation (during performance of the MIDT) prospectively predict the future onset of depressive symptoms?
* Do drug and/or DBS interventions targeting the VS dopaminergic system ameliorate depressive symptoms? What does this suggest for causation?
* Is reward better conceptualized as a single process –or- as a family of closely related processes that normally work together to guide survival and everyday motivated behavior?

**Module 19 — Positive Emotionality, Self-Control, and Dopamine (Part 2): Substance Abuse, Impulse Control Disorders, and Everyday Temptation**

* Is addiction simple or complex?
	+ Key features of the addiction cycle
* Is addiction a big deal, in terms of public health burden?
* What are key diagnostic features of SUD’s.
* Is the VS/Nacc hyper or hypo-responsive to drug cues in addicts? What about everyday rewards and appetitive stimuli, such as food? This suggests that addiction \_\_\_\_\_\_\_\_\_ brain circuits involved in ordinary wanting and approach, like the VS/NAcc.
* How are trait-like individual differences in E/PE related to SUDs? Strong effect or weak?
* Contrary to Shackman’s naïve hypothesis, there is a \_\_\_\_\_\_\_ of systems involved in reward and positive emotion.
* How are trait-like individual differences in C/SC related to SUDs?
* Why might addicts show reduced E/PE? Is there a blunting or tuning?
* Among addicts, approach/wanting/go circuits appear to be \_\_\_\_\_ sensitive to \_\_\_\_\_\_\_\_\_\_\_ substances.
* What’s the go/no-go task?
* How is the IFG related to performance on inhibitory tasks (e.g. Go/No-Go or Stop Signal tasks)?
* Is activity in the IFG related to resisting temptation and self-control in daily life? Briefly describe.
* Why do some patients with PD develop impulse control disorders when they take pramipexol? What does this suggest about the mesolimbic dopaminergic system and self-control/impulsivity? Briefly describe.
* How is this related to the material we discussed in the last module (e.g. work by Berridge and Robinson, MFB -SS)?
* Does MFB-SS (or other manipulations that alter dopamine or activity in the VS/Nacc) increase liking, wanting, both?
* How does incentive sensitization theory address these 2 questions:
	+ Why do addicts crave drugs?
	+ Why does vulnerability to relapse last so long (long after acute withdrawal is over)?
	+ Molecular changes in the mesolimbic dopamine system are \_\_\_\_\_ persistent; none of us knows if we’re \_\_\_\_\_\_\_\_\_\_\_ to these kinds of long-lasting changes when first experimenting. Are you Philip Seymour Hoffman or are you Keith Richards?
* Does activation in this VS/NAcc circuit predict impulse control in the real world? Briefly describe.
* Does activation in the VS/NAcc predict craving in addicts?
	+ What are the consequences of anti-craving drugs?
	+ What does this suggest, in terms of craving causation?
* The IFG plays a key role in \_\_\_\_\_\_\_\_ behavior and \_\_\_\_\_\_\_\_\_ temptation.
* Alterations in multiple brain systems, some involved in \_\_\_\_\_\_\_\_, others in \_\_\_\_\_\_ can create a similar phenotype, one which is \_\_\_\_\_\_\_\_\_\_\_\_\_\_ .
* Which brain regions play a role in choosing between healthy and unhealthy options? Between tasty and un-tasty options?
	+ What is the role of the lateral PFC?
	+ Of the OFC?
	+ Which region seems more closely tied to ‘Stop’ and long-term declarative goals (I will stick to my diet)?
	+ Which region seems to integrate information about different kinds of values, such as yumminess and healthiness?
* Briefly describe the Phineas Gage and Patient “EVR” (Elliot) case histories
	+ What does this suggest in terms of the OFC and the adaptive self-control of behavior in the face of immediate temptation?
* Which brain region plays a role in telling OFC about long-term, declarative goals (stick to the diet, skip the pie, pick the healthy option)?
* Does self-control reflect a single brain region –or- does it reflect the coordinated activity of a widely distributed circuit in the brain? Can you achieve similar behavioral/phenotypic effects (e.g. decreased self-control or increased impulsivity) via manipulations/lesions of different nodes in that circuit?
* Dropped Slides: Is the amygdala only sensitive to threat and punishment? Or does it seem to be sensitive to a broad spectrum of emotionally and motivationally significant stimuli (e.g. food, drugs, faces, threats)? Answer: Broad spectrum; amygdala shows enhanced reactivity to drug cues among addicts.