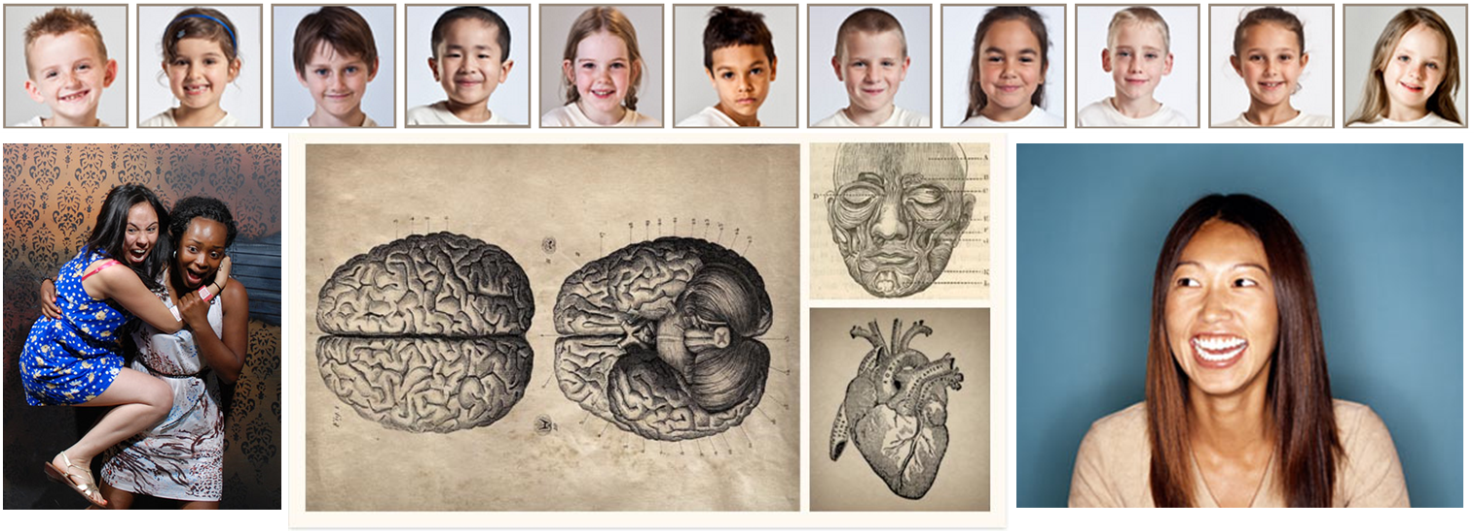
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**PSYCHOLOGY 210: INTRODUCTION TO TEMPERAMENT AND PERSONALITY (T&P)**

**PROFESSOR ALEX SHACKMAN, UNIVERSITY OF MARYLAND**

**SPRING 2016**

*What makes each of us unique? Where do these differences come from? How do they contribute to enduring differences in health and wellness?*

*We will selectively review cutting-edge research in humans and non-human animal models aimed at understanding the mechanisms underlying lasting differences in personality and their implications for risk and resilience.*

*We will discuss the developmental origins of temperament, measurement issues, fundamental dimensions, mechanisms contributing to stability/plasticity, heritability, implications for psychopathology and therapeutic intervention, as well as broader implications for public policy.*

1. *A major focus of the course will be the neurobiology of trait-like differences in fear and anxiety, including neural circuits, molecular genetic pathways, and epigenetics.*
2. *A secondary focus will be on individual differences in behavior and biology that confer elevated risk for the development of depression and impulse control disorders (e.g., substance abuse), including neural circuits involved in hedonic pleasure, reward motivated-behavior, and the regulation of impulses in the face of temptation.*

*An extensive background in biology, genetics, neuroscience, statistics, or other ‘STEM’ fields is not necessary to enjoy and benefit from this course.*

***Continued…***

**ADMINISTRATIVE INFORMATION**

* **Tue/Thu 11:00-12:15 AM in Biology-Psychology (BPS) 1243**
* **Instructor:** Dr.Alex Shackman ([shackman@umd.edu](mailto:shackman@umd.edu); 3123G BPS)
* **Teaching Assistant:** Rachael Tillman ([rmtillma@umd.edu](mailto:rmtillma@umd.edu); 0124 BPS)
* **Required Materials**
  + **Textbooks**: n/a
  + **Technology**: n/a
  + **Readings:** Available in .pdf format via Canvas ([www.elms.umd.edu](http://www.elms.umd.edu))
* **Class cancellation, room change, or other time-sensitive announcements:** Will be directed to the email account listed in Canvas
* **Academic Calendar:** <http://www.provost.umd.edu/calendar/> and <http://faculty.umd.edu/teach/dates.htm>
* **Office Hours**
  + Dr. Shackman: By appointment
  + Ms. Tillman: Tuesday 2:30 pm – 3:30 pm (0124 BPS). Other times by appointment.

***Continued…***

**general learning objectives: *Course overview***

Welcome!

This course will introduce students to a diverse array of theoretical and empirical issues related to the study of stable individual differences in temperament and personality (T&P). We will discuss recent research in humans, monkeys, and rodents that helps to clarify

* *The childhood origins of temperament*
* *The fundamental dimensions of T&P*
* *The psychological and neurobiological mechanisms that underlie trait-like differences in T&P*
* *The mechanisms that contribute to stability and plasticity in T&P across the lifespan and across generations*
* *The nature and nurture of T&P. We will delve into…*
  + *behavioral genetics (i.e., heritability)*
  + *molecular genetics and ‘imaging genetics’*
  + *recent advances in epigenetics*
* *The complementary strengths and limitations of different tools and approaches for assaying T&P*
* *The nature of temptation and self-control*
* *Implications for mental health and physical wellbeing, public policy, and public safety*
* *Implications for understanding ourselves and our loved ones (our parents, our children or children-to-be) and becoming more thoughtful and informed tax payers, voters, and citizens*

The information in this document is designed to help you understand how the course works and to get you started. If you have any questions, please contact the instructor. We’re excited to have you aboard and want you to get the most out of this opportunity to learn more about the science of individual differences!

**Note:** This is an introductory course and an extensive background in biology, genetics, neuroscience, statistics, or other “STEM” fields is not assumed.

***Continued…***

**detailed learning objectives: *can you be more specific about the CONTENT covered in the class?***

Sure! Here are the key concepts that students will learn in this course.

**Structural Models**

BIS/BAS; Behavioral Inhibition; Big 2; Big 3; Big 5 (OCEAN)

**Scientific Concepts**

Affective chronometry; Appetitive motivation; Approach/Withdrawal; Biomarkers, Endophenotypes & Intermediate Phenotypes; Epigenetics and Non-genomic transmission of acquired traits; Fear vs. Anxiety; Frontal EEG asymmetry; G \* E interactions; Hedonic hotspots; Heritability (common misconceptions); Incentive sensitization model; Liking vs. Wanting; Natural language hypothesis; Pavlovian fear conditioning; Scientific skepticism; Self-stimulation; Sensitivity, Specificity, and Reliability (e.g., test-retest); Serotonin transporter polymorphism; Spatial and temporal resolution; SNP; Strengths and weaknesses of prospective longitudinal studies

**Psychometric Concepts (Non-Technical Overview)**

Correlation (vs. causation); Construct validity; Factor analysis; Internal-consistency reliability; Meta-analysis (classical and ALE); Test-retest reliability;

**Brain Regions**

Basal forebrain cholinergic system; Extended amygdala, Hippocampus, HPA axis, Lateral prefrontal cortex, Medial forebrain bundle, Mesocorticolimbic dopamine system, Midcingulate cortex, Nucleus accumbens, Orbitofrontal cortex, Ventral striatum

**Methods (Non-Technical Introduction Focused on Strengths and Weaknesses)**

ASL MRI; BART; Cortisol; Daily diary; Deep brain stimulation (DBS); EDA/SCR/GSR; EEG/ERP (including N2, ERN, FRN, and P3b); Eriksen flanker; Excitotoxic lesions; Experience sampling; FDG-PET; Fear-potentiated startle; fMRI (task-related and resting-state functional connectivity); GWAS; Limitations of introspective measures and self-report (e.g., peak-end rule); NeuroSynth; Pharmacological methods (e.g., benzodiazepines); Stop-signal task

**Famous and Not-So-Famous Neuropsychological Patients**

B-19, EVR, SM, and Phineas Gage

**Neuropsychiatric Disorders (Epidemiology/Prevalence, Burden, Symptoms)**

Anxiety; Depression; Substance Abuse/Addiction; Impulse Control Disorders (e.g., gambling); Parkinsons

**Investigators**

Ralph Adolphs; Yair Bar-Haim; David Barlow; Kent Berridge; Jenni Blackford; Jack Block; Ryan Bogdan; Niall Bolger; Turhan Canli; Avshalom Caspi & Temi Moffitt; Lee Anna Clark; Michelle Craske; Tony and Hannah Damasio; Richie Davidson; Mike Davis; Hans and Mike Eysenck; Drew Fox; Nathan Fox; Jeffrey Gray; Christian Grillon; Dan Grupe; Amad Hariri; Jerry Kagan; Ken Kendler; Roman Kotov; Will Fleeson; Carl Lejuez; Joe Ledoux; Schmuel Lissek; Jerry Kagan; Ned Kalin; Ken Kendler; Roman Kotov; Seymour ‘Gig’ Levine; Colin Macleod; Michael Meaney; Walt Mischel; Jack Nitschke; Danny Pine; Diego Pizzagalli; Tony Rangel and Todd Hare; Terry Robinson; Kerry Ressler; Alex Shackman; Jerry Suls; Andy Tomarken; Mike Treadway; Peter Visscher; Nora Volkow; David Walker; David Watson; Paul Whalen; Tal Yarkoni; David Zald and many others

If this sounds interesting, you’re in the right place!

**a multi-disciplinary perspective on the contemporary science of t&p**

As we begin our adventure, it’s helpful to keep the following idea firmly in mind:

*When a scientist doesn’t know the answer to a problem, he is ignorant. When he has a hunch as to what the result is, he is uncertain. And when he is pretty damn sure of what the result is going to be, he is still in some doubt…Scientific knowledge is a body of statements of varying degrees of certainty—some most unsure, some nearly sure, but none absolutely certain.*

—Richard Feynman (1955), Nobel Laureate

Science is not a body of facts established by experts, but a set of methods for estimating and reducing uncertainty; a process, at times messy or tedious, of grappling with nature and our preconceived notions about how it works. There are many, many fundamental questions about T&P that remain unresolved. That’s one of the things that make this class so enjoyable. We haven’t figured it out and there are many challenges that remain for future research.

Accordingly, in this class you will learn about the current state of our scientific knowledge about facets of T&P, their organization in the brain, and the implications for understanding psychopathology and other important outcomes. You will also learn about some of the key behavioral and physiological techniques used for measuring and understanding facets of T&P. But we will not systematically review the history of personality research (e.g., Galen, Freud, Jung — a.k.a. the *Hall of Fame or Graveyard Tour* approach). As several leading researchers recently noted,

*Personality psychology has long been identified in the minds of many people with the first (and perhaps only) course in the subject that they took in college. Too often, this was (and sometimes still is) the classic “tour of the graveyard” that focuses on brilliant but long-deceased theorists and leads students to end the semester thinking the burning concern of the field is the disagreement between Freud and Jung…A course that is restricted to theorists like these is an unforgivable misrepresentation of the field, a failure in one’s duty to educate students, and a slap in the face to every contemporary personality researcher*

*It is unacceptable that personality psychology remains, generally, a side trip through the history of psychology while the rest of the science of psychology is presented to students through the lens of the most cutting-edge research.*

—Benet-Martínez, Donnellan, Fleeson, Fraley, Gosling, King, Robins, & Funder (APA Handbook of Personality and Social Psychol, 2015)

In general, my emphasis will be on a multi-disciplinary perspective, in which research at different levels of analysis, using different tools, samples, or species, is viewed as complementary and mutually informative. Put another way, the class will not be organized around “biological theories,” “psychoanalytic theories,” and so on.

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# course structure

1. Classroom Lectures on the Scientific Study of T&P

You are *strongly* encouraged to attend all course meetings. Each lecture or “module” will last approximately 75 minutes and will include the following components:

1. Conceptual roadmap outlining the new topics to be covered
2. The science of T&P drawn from your readings and other sources. The lectures will incorporate occasional multimedia elements, such as film clips. There will be plenty of time for questions and discussion. The lectures are designed to provide a broad overview of the core conceptual themes, methodological issues, and highlights from the recent empirical record.
3. Recap of the most important take-home points
4. Critical take-home questions (detailed below)
5. Summary of Learning Objectives

On occasion, we may have special invited guest lectures.

**It is critical that you regularly attend class in order to do well in this course.** I encourage you take notes during class to ensure comprehension of the material. It is important to emphasize that there are many opportunities for us to learn from one another in the classroom. Learning can stem from sharing knowledge or from asking questions.

**The summary of Learning Objectives appended to the end of this Syllabus provide a powerful tool to guide your independent study and review. I strongly recommend using the Learning Objectives to guide your test preparation. Please review them before you dive into the PowerPoint to forage for crucial pieces of information.**

2. Background Readings

Readings for this course have been hand-picked by the instructor; many are empirical papers or reviews by leading scientists in the field. What better way is there to learn about T&P then straight from the most exciting researchers working in the field today?

To get the most out of this course, it is important that you understand the key take-home points from the readings. **Please read the assigned papers before class.** This will allow for a better understanding of the lecture and also give you the opportunity to ask questions. Please do not hesitate to ask questions about anything you found confusing or challenging! Readings will be available for download via the course website on Canvas. Again, there is no text book.

While many of the readings were written for a general scientific audience, some of the empirical reports employ complex or unfamiliar methods. My expectation is that you will be able to discern the larger take-home points and implications, even if some of the techniques are unclear. Throughout the Readings section (below), I have identified papers where I do not expect you to invest the time required to fully understand the more technical aspects of the methods.

**My aim is to avoid overburdening students with reading.** But in some cases, you may find yourself hungry to learn more. The optional readings that accompany most of the learning modules are a great place to start. The source material for the lectures is also cited within my slides and I am happy to provide the papers upon request.

# course REQUIREMENTS & GRADING

### 1. Four Cumulative Examinations (Collectively worth 75%; lowest of the 3 midterm grades dropped)

**4 cumulative exams: 3 mid-terms and 1 final examination.**

Exams will consist of multiple-choice questions that involve critical thinking about concepts drawn from the readings and lectures.

Exams will take place in class on the assigned date in the syllabus.

**The lowest of the 3 mid-term grades will be dropped** (cf. http://www.sfcollege.edu/cat/?section=techTips/ExcelLowVal).

**You are welcome to bring a double-sided (8.5” x 11”) sheet of notes to the exam.** No other notes, notebooks, materials, or devices will be permitted.

The purpose of the exams is two-fold. First, you should be able to demonstrate that you have read the material and understand the factual points and arguments. Second, you should be able to synthesize and integrate the material such that this knowledge can be applied in a broader context.

Because the exams are cumulative and occur on a regular basis, you will need to continuously study in order to be successful. On the other hand, you probably will not need to cram for any particular exam.

**There will be a review session before each exam during our regularly scheduled meeting time.** The review sessions are designed to provide a fun, painless way to test your understanding of the material and address any questions that crop up before each exam.

**Make-up exams will only be considered in exceptional circumstances.** Make-up exams will involve different questions than the standard exam (Advice: you want to avoid having to take a make-up exam).

It is important to emphasize that much of what is covered in the exams is not contained verbatim in the lecture slides, so attendance and attention during class is absolutely critical to your success in the course.

### 2. Take-Home Written Assignments (Collectively worth 25%; two lowest grades dropped)

For each lecture, you will receive two or more critical thinking take-home questions (CTQs).

You will be required to respond to any two of the CTQs that are assigned for a particular Module.

One aim of the CTQ’s is to encourage students to complete the assigned readings for each module prior to the associated lecture. This will make the lectures easier to follow. Accordingly, the CTQ’s for a particular module will be assigned at the end of the prior module (e.g. the CTQs relevant to Module #2 will be assigned by the time I am done presenting Module #1 in class).

Another aim of the written homework is to encourage on-going review and learning (and minimize the need for cramming). To encourage this, you are welcome to substitute the Learning Objectives for the CTQ’s. What do I mean? For any particular lecture, ignore the CTQ’s altogether and simply provide short written responses to all of the “prompts” for the current Module, which are conveniently provided later in this Syllabus and are also available on Canvas. You must respond to every prompt to receive full credit. Note that if you choose to do this, I still expect you to complete the assigned readings before coming to the relevant lecture.

Each assignment (pair of CTQ’s or the Learning Objectives) will be assigned one of the following grades: 1 (full credit), 1⁄2 (half-credit), 0 (no credit). Grades will be made available in Canvas. Unexcused late responses will be assigned a score of 0.

At the end of the semester, your two lowest response grades will be dropped (cf. http://www.sfcollege.edu/cat/?section=techTips/ExcelLowVal).

For the CTQ’s, your response should be approximately 1 paragraph per question (i.e., total of 2 separate paragraphs). **Responses are due by 11:00 am one week later (i.e., CTQ’s provided in class Tuesday are due by 11:00 am the following Tuesday; questions provided in class Tuesday are due by 11:00 am the following Thursday). If you choose to substitute the Learning Objectives, you still have one week to upload the assignment.**

You will submit your responses using the “assignment” tab in Canvas. Responses should include 2 components (with each component clearly labeled using headers). Length should range between ½ to 1 page single-spaced for the 2 questions.

### 3. Extra Credit

Four points of extra credit will be available to students who complete the Department Mass Survey using the SONA system (see below for details). These points will be added directly to those that you earned based on the exams and critical thinking assignments. For example, if a student earned a total of 89 points and completed the extra credit, his or her final letter grade would be based on 89 + 4 = 93 / 100 points. Final grades will not be curved or otherwise transformed.

Final grades will not be curved or otherwise transformed.

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### 4. Optional Snack with Shack, Man

Large, lecture-format courses can be very impersonal. To help address this, on Thursdays I will take 6 lucky students out for a snack at The Coffee Bar (Stamp Union). Your choice of coffee, tea, juice and a pastry on me!

My hope is that this will provide a an opportunity to get to know one another a little better and a relaxed, informal setting for chatting about the material covered in the class, other aspects of psychological science, your experiences as students on campus, and professional development (e.g. advice about working in a lab or applying to graduate programs).

Most of the time, we will meet up after class and walk over to the Union together. On those days where an exam is scheduled, we can meet in the hallway outside of The Coffee Bar. ***Additional details are provided in the Schedule on the next page. Please let me know as soon as possible if your assigned date and time does not work for you and we can determine a suitable alternative. Please note that ‘Snack with Shack, Man’ is completely optional!***

***Continued…***

**PSYC 435 SCHEDULE**

Please check the course website for the most up-to-date information.

|  |  |
| --- | --- |
| Date | Activity |
| January 26 | Snow Day |
| January 28 | Module 1: Introductions, Course Mechanics, and Fundamental Questions Roundtable  Optional Snack with Shack, Man: Abraham, Anthony, Argyle, Barnard, Barrios, Bart |
| SECTION I: FOUNDATIONAL ISSUES IN THE SCIENTIFIC STUDY OF TEMPERAMENT AND PERSONALITY | |
| February 2 | Module 2: Is T&P impactful? |
| February 4 | Module 3: How is T&P defined? What are the fundamental dimensions of T&P? (note: 1st critical thinking assignment) |
| February 9 | Module 4: How should we measure T&P? |
| February 11 | No Class / Dr. Shackman in Wisconsin |
| February 16 | Module 5: How Are Traits and States Related? (Part 1) |
| February 18 | Module 6: How Are Traits and States Related? (Part 2)  Optional Snack with Shack, Man: Bird, Blume, Byrne, Chestney, Cholvibul, Chung |
| February 23 | No Class / Dr. Shackman at Johns Hopkins |
| February 25 | Module 7: What Do Traits Do? (Part 3) |
| March 1 | In-Class Review Session  Optional Snack with Shack, Man: Dean, Dillard, Dixon, Duncan, Elfant, Etemadi |
| March 3 | **Cumulative Multiple-Choice Exam #1** (Class Led by the TA/Proctor)  Optional Snack with Shack, Man: Fontaine, Glime, Goldsmith, Heatley, Herlihy, Huang [MEET AT THE COFFEE BAR IN THE STAMP @ 12:20] |
| SECTION II: THE NATURE AND NURTURE OF TEMPERAMENT AND PERSONALITY | |
| March 8 | Module 9: Intermediate Phenotypes and Brain Imaging Tools |
| March 10 | Module 10: Nature & Nurture (Part 1): Behavioral Genetics and Heritability |
| March 15/17 | Spring Break: Owing to the Snow Day, students complete *Module 8: Intermediate Phenotypes and Brain Imaging Tools* on their own by the end of Break |
| March 22 | Module 11: Nature & Nurture (Part 2): Molecular Genetics |
| March 24 | Module 12: Nature & Nurture (Part 3): Neurogenetics and Epigenetics  Optional Snack with Shack, Man: Ladep-Nandang, Maney, Mei, Mudd, Naeem, Nagarajan |
| March 29 | In-Class Review Session  Optional Snack with Shack, Man: Hudson, Jamei, Karp, Kaye, Kelman, Khizder |
| March 31 | **Cumulative Multiple-Choice Exam #2** (Class Led by the TA/Proctor)  Optional Snack with Shack, Man: Obiyor, Oluwafemi, Pifer, Prem, Roffeld, Rowland [MEET AT THE COFFEE BAR IN THE STAMP @ 12:20] |
| SECTION III: NEUROTICISM / NEGATIVE EMOTIONALITY | |
| April 5 | Module 13: Neuroticism/Negative Emotionality and Psychopathology (Part 1) |
| April 7 | Module 14: Behavioral Inhibition and Psychopathology (Part 2)  Optional Snack with Shack, Man: Sidhu, Skibniewska, SmithA, SmithS, Sultan-Reisler, Swaminathan |
| April 12 | Module 15: Role of the Extended Amygdala in Negative Emotionality, Behavioral Inhibition, and Psychopathology (Part 3) |
| April 14 | Guest Lecture: Rachael Tillman. Emotion Regulation, T&P, and Psychopathology |
| April 19 | Module 16: Splitting Negative Emotionality into Its Constituents, Part 1 |
| April 21 | Module 17: Splitting Negative Emotionality into Its Constituents, Part 2  Optional Snack with Shack, Man: Tan, Tandoi, Truong, Tuttle, Walsh, Wiedefeld |
| April 26 | In-Class Review Session |
| April 28 | **Cumulative Multiple-Choice Exam #3** (Class Led by the TA/Proctor) |
| SECTION IV: EXTRAVERSION / POSITIVE EMOTIONALITY & CONSTRAINT / SELF-CONTROL | |
| May 3 | Module 18: Positive Emotionality, Self-Control, and Dopamine (Part 1): Depression and Anhedonia |
| May 5 | Module 19: Positive Emotionality, Self-Control, and Dopamine (Part 2): Substance Abuse, Impulse Control Disorders, and Everyday Temptation  Optional Snack with Shack, Man: Wilson, Woller, Yan, and Lingo |
| May 10 | Module 20: Semester Recap | In-Class Review Session |
| TBA | **Final Exam** (Class Led by the TA/Proctor) |

Please note: This schedule is subject to change. Any required updates will be announced in class and posted on the course website. All readings will be available on the course website. Examinations may be proctored by the TA or another member of the Department staff.

**readings**

**SECTION I: FOUNDATIONAL ISSUES IN THE SCIENTIFIC STUDY OF TEMPERAMENT & PERSONALITY**

**Module 1: Introductions, course mechanics, and fundamental questions roundtable**

Required

* Spotting Bad Science
* Spotting Logical Fallacies
* Carl Sagan’s ‘Baloney Detection Kit’ – Popova Brain Pickings 2015

Optional

* Lillienfeld et al. Frontiers in Psychol 2015 [50 psychological terms to avoid]

**Module 2: Is T&P impactful?**

Required

* Moffitt et al. PNAS 2011 [do not worry about the technical details of the analyses]
* Duckworth PNAS 2011 [brief scientific commentary on Moffitt]
* Lahey Amer Psychol 2009 [review detailing the myriad consequences of neuroticism; highlights are described in lecture]
* Kelly Psych Today 2010 [brief popular press summary of work linking neuroticism to divorce]
* Barker Time 2014 [brief popular press summary of work linking conscientiousness and neuroticism to diverse outcomes]

Optional

* Moffitt et al. Amer Sci 2013 [popular scientific press summary of Moffitt et al. PNAS 2011; reviewed in lecture]
* Duckworth et al. Perspectives on Psychol Sci 2016 [accessible review focused on strategies for enhancing and situations that can undermine self-control]

**Module 3: How is T&P defined? What are the fundamental dimensions of T&P?**

Required

* Caspi et al Ann Rev Psychol 2005 [you are welcome to skip the sections on Behavioral Genetics & Social Development]
* Srivastava 2016

Optional

* Goldsmith et al Child Dev 1987
* Shiner et al Child Dev 2012
* Fox & Walker 2015
* Shiner chapter 3 *in press*
* Shiner chapter 14 *in press*
* Clark & Watson chapter 2008
* Zentner & Shiner chapter 2012
* Soto & John J Personality & Soc Psychol 2016

**Module 4: How should we measure T&P?**

Required

* Block Psychol Bull 1995a
* Tomarken Psychol Assessment 1995

Optional

* Stromberg & Caswell Vox 2015 [on-line magazine article on why the popular Meyers-Briggs test is worthless]
* Funder Psychol Inquiry 1994 [entertaining essay on the strengths and weaknesses of trait theory]
* Epstein Psychol Inquiry 1994 [short, entertaining essay on the limitations of the Big 5 and similar descriptive models of T&P]
* McRae Psychol Inquiry 2010 [Updated rebuttal of Block; I found this to be very compelling]
* John, Naumann & Soto Handbook of Personality 2008 [definitive defense of the Big 5 and FFM]

**Module 5: How are traits and states related? (Part 1)**

Required

* Chap 4 in Matthews, Deary & Whiteman 2009 [pp. 85-89 as well as pp. 107-end]
* Suls & Martin J Personality 2005

Optional

* Watson & Clark Psychol Bull 1984
* Fleeson JPSP 2001
* Fleeson JPSP 2009

**Module 6: How are traits and states related? (Part 2)**

Required

* Fox et al PlosOne 2008 [please do not worry about the technical aspects of FDG-PET imaging]
* Canli et al PNAS 2006 [please do not worry about the technical details; focus on the description of phasic vs. tonic models]

Optional

* Bolger & Schilling J Personality 1991

**Module 7: What do traits do? (Part 3)**

Required

* Davidson Cog and Emo 1998 [please read Sections I and II only]
* Gable, Reis & Elliot JPSP 2000 [please do not worry about technical details of the analytic strategy]

Optional

* None

**SECTION II: THE NATURE AND NURTURE OF TEMPERAMENT AND PERSONALITY**

**Module 8: Intermediate phenotypes and brain imaging tools, Part 1**

Required

* Ariely & Berns Nature Rev Neurosci 2010 [you only need to read Box 2 on page 288; feel free to read more!]
* Schwartz et al. Amer Psychol 2016 [you only need to read pp. 59-61; feel free to read more!]

Optional

* Lillienfeld Behav Res Ther 2014 [cautionary note on the use of biological measures and the search for biomarkers]
* Logothetis Nature 2008 [please do not worry about the finer details; for those interested in delving more deeply into brain imaging techniques]

**Module 9: Intermediate phenotypes and brain imaging tools, Part 2**

Required

* Miller Ann Rev Clin Psychol 2013 [detailed analysis of endophenotypes]
* Patrick Psychophysiol 2014 [brief non-technical commentary on ‘the end of endophenotypes’]
* The Neuroskeptic 2014, *Psychiatry: End of the Road for “Endophenotypes”?*

Optional

* Iacono et al Psychophysiol 2014c [summary of a large-scale effort at Minnesota to link psychophysiological and electrophysiological endophenotypes to genetic variants; please do not worry about any of the technical details]
* Roiser The Psychol 2015 [brief, entertaining piece on the value of neuroscience for developing novel intervention strategies]

**Module 10: The Nature & Nurture of T&P (Part 1): Behavioral Genetics and Heritability**

Required

* Visscher et al Nat Rev Genetics 2008 [please do not worry about the finer details]
* Plomin et al. Perspectives on Psychol Sci 2016

Optional

* Miller Perspectives on Psychol Sci 2010 pp 18-23 [critical perspective on genetic reductionism]
* Dar-Nimrod & Heine Psychol Bull 2011 [review focused on how misunderstandings about genetics facilitate stereotyping and prejudice, influence morality, and can mis-lead decision-making about interventions for the self (e.g. dieting) and others (e.g prison vs. rehab/treatment)]

**Module 11: The Nature & Nurture of T&P (Part 2): Molecular Genetics**

Required

* Caspi & Moffitt Nat Rev Neuro 2006
* Hyman Nature 2014 [brief non-technical commentary by the former director of the NIMH]
* Couzin-Frankel Science 2014 [science writer’s personal story about getting genetic testing for familial breast cancer]
* Pinker NY Times Magazine 2009 [science writer’s personal story about getting genetic testing]

Optional

* Mukherjee New Yorker 2016b [science writer’s story about his family and psychiatric genetics]
* Chabris et al. Curr Dir Psychol Sci 2015 [very accessible overview of GWAS]
* Smoller Neuropsychopharm 2016 [comprehensive, but approachable review of the genetics of mood and anxiety disorders]
* Iacono et al Psychophysiol 2014 [accessible overview of molecular techniques with a glossary]
* Topol Cell 2014 [very readable discussion of personal genomics]
* Moffitt et al Perspectives Psychol Sci 2006 [a wonderful introduction to G\*E interactions that also provides a very useful tutorial on study design]
* Caspi Amer J Psychiatry 2010 [for those interested in G \* E interactions and the serotonin transporter polymorphism]
* Monroe Psychol Sci 2008 [for those interested in G \* E interactions and the serotonin transporter polymorphism]

**Module 12: The Nature & Nurture of T&P (Part 3): Neurogenetics and Epigenetics**

Required

* Hughes Nature 2014 [brief non-technical commentary on Dias & Ressler Nature Neurosci 2014]
* Meaney Ann Rev Neurosci 2001 [please do not worry about the finer technical details; seminal review paper by one of the key pioneers]
* Mukherjee New Yorker 2016b [science writer’s story about his family, twins, and epigenetics]

Optional

* Bogdan et al Neuropsychopharm 2016 [sobering updated discussion of neurogenetics]
* Dias & Ressler Nature Neurosci 2014 [please do not worry about the finer technical details]

**SECTION III: NEUROTICISM / NEGATIVE EMOTIONALITY**

**Module 13: Neuroticism/Negative Emotionality and Psychopathology**

Required

* Barlow et al Clin Psychol Sci 2013
* Ormel et al Clin Psychol Rev 2013
* Smith Nature 2014 [infographic on the global burden of neuropsychiatric disease]
* Morrison Vox 2014 [short essay describing one patient’s experience living with generalized anxiety]
* Orlando et al. Houstonia 2015
* ACHA-National College Health Assessment 2015

Optional

* Watson & Naragon-Gainey Clin Psychol Sci 2014 [do not worry about the technical details of the analysis]

**Module 14: Behavioral Inhibition and Psychopathology**

Required

* NY Times Magazine article on behavioral inhibition
* Fox et al Ann Rev Psychol 2005

Optional

* Fox & Walker 2015 [for those hungry to learn more about BI]
* Kagan et al. Science 1988 [for those interested in delving more deeply into BI; seminal study]
* Schwartz et al. Science 2003 [please do not worry about technical aspects of fMRI; for those interested in delving more deeply into BI; seminal study]
* Clauss & Blackford J Amer Acad Child & Adol Psychiatry 2013 [please do not worry about technical aspects of the meta-analysis; for those interested in delving more deeply into BI]
* Mihalopoulos et al. J Child Psychol & Psychiatry 2015 [detailed analysis of what makes for a cost-effective targeted prevention program]

**Module 15: Role of the Extended Amygdala in Negative Emotionality, Behavioral Inhibition, and Psychopathology**

Required

* Davis et al Neuropsychopharm 2010
* Fox & Kalin Amer J Psychiatry 2014 [please do not worry about the technical details]
* Feinstein et al Curr Biol 2011
* Adolphs Ann NY Acad Sci 2010 [addresses the contribution of the amygdala to social cognition]

Optional

* Oler, Fox, Shackman & Kalin in press [lesions in monkeys, relevance to BI and social anxiety disorder]
* Shackman et al PNAS 2013 [please do not worry about the technical details]
* Fox et al Trends in Neurosci 2015
* Fox et al PNAS 2015 [please do not worry about the technical details]
* Swartz et al Neuron 2015 [please do not worry about the technical details]
* Etkin & Wager Amer J Psychiatry 2007 [please do not worry about the technical details; seminal meta-analysis]
* Davis & Whalen Mol Psychiatry 2001
* Adolphs et al Nature 1998 [reviewed in lecture and worth skimming]
* Kennedy et al Nat Neurosci 2009 [reviewed in lecture and worth skimming]
* Choi & Kim PNAS 2010 [please do not worry about the technical details] [reviewed in lecture and worth skimming]
* Tovote et al Nature Rev Neurosci 2015 [for those interested in delving more deeply into amygdala circuitry; also, an excellent introduction to fear conditioning]

**Module 16: Splitting Negative Emotionality into its Key Constituents (Part 1)**

Required

* Grupe & Nitschke Nature Rev Neurosci 2013
* La Rosa Buzzfeed 2014
* Shackman et al. J Exp Psychopath in press

Optional

* MacLeod & Mathews Ann Rev Clin Psychol 2012 [ABM/CBM: reviewed in lecture and worth skimming]
* MacLeod Clin Psychol Sci 2015 [ABM/CBM: reviewed in lecture and worth skimming]
* Koster & Bernstein J Behav Ther & Exp Psychiatry in press [future directions for ABM/CBM]
* Linetzky et al. Dep and Anx 2015 [recent meta-analysis of ABM]
* Duits et al Dep and Anx 2015 [please do not worry about the technical details of the meta-analysis] [reviewed in lecture and worth skimming]
* Bar-Haim et al. Psychol Bull 2007 [please do not worry about the technical details of the meta-analysis] [reviewed in lecture and worth skimming]

**Module 17: Splitting Negative Emotionality into its Key Constituents (Part 2)**

Required

* Shackman et al Nature Rev Neurosci 2011
* Cavanagh & Shackman J Physiol Paris in press [please do not worry about the finer details of the analysis]

**SECTION IV: EXTRAVERSION / POSITIVE EMOTIONALITY & CONSTRAINT / SELF-CONTROL**

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

Required

* Kringelbach & Berridge Sci Amer 2012
* Pizzagalli Ann Rev Clin Psychol 2014
* Thomsen et al Frontiers in Behav Neurosci 2015
* Fleming Intell Life Mag 2015 [journalist hangs out with Kent Berridge for a week]

Optional

* Kotov et al. Psychol Bull 2010 [meta-analysis of associations between T&P and psychopathology; covered in lecture]
* Treadway & Zald Curr Directions Psychol Sci 2013
* Berridge & Robinson Brain Res Rev 1998 [seminal early review]
* Berridge & Robinson Neuron 2015
* Knutson & Greer Philo Trans Royal Soc B 2008
* The Neurocritic DBS RCT 2015 [popular science blog post on failed randomized clinical trials of deep brain stimulation for major depression]
* Scult Sci Amer 2016 [short blog post on the neural circuitry of reward and neurofeedback training]

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

Required

* Lopez et al. Psychol Sci 2014 [please do not worry about the more technical aspects of fMRI or EMA]
* Hare et al. Sci 2009 [please do not worry about any of the more technical aspects of this complex neuroeconomics study]
* Munro Nature 2015 [infographic on the psychoneurobiology of addiction]
* Yong The Atlantic 2016 [brief popular press piece on the neurobiology of impulsivity and risk aversion]
* Meurk International J of Drug Policy 2016 [how do addicts think about addiction]

Optional

* Kelley et al. Ann Rev Neurosci 2015
* Lehrer New Yorker 2009 [popular press piece on Walt Mischel]
* Mischel 2015 [Press release for the 2015 Congressional Golden Goose award to Mischel]
* Kotov et al. Psychol Bull 2010 [meta-analysis of associations between T&P and psychopathology; covered in lecture]
* Knutson & Greer Philo Trans Royal Soc B 2008 [review work linking the VS/NAcc to wanting and positive emotionality]
* Berridge & Robinson Brain Res Rev 1998 [seminal early review]
* Berridge & Robinson Neuron 2015 [recent review]
* Duckworth et al. Perspectives on Psychol Sci 2016 [*highly recommended* review focused on strategies for enhancing self-control in the real world; e.g. dieting, planning for retirement, quitting substances, etc.]

**Module 20: Semester Recap**

Required

* Shackman, Tromp, Kaplan, Stockbridge, Tillman & Fox, under review.

**tips for deciphering the assigned papers**

Here are some helpful tips to keep in mind as you read the assigned papers. Most of these apply equally well to review or empirical papers.

* First Steps
  + Begin by reviewing the title of the article. The title will indicate the central focus of the paper.
  + Next, read the abstract. The abstract will provide an overview of the study’s main research question, goals, and results. Don’t worry too much about the details or get hung up, just try to identify the big picture.
* Introduction
  + The introduction typically describes what the author hoped to achieve and states the problem being investigated. Normally, the introduction provides background and significance. It will summarize or at least foreshadow the experiment, the hypothesis(es) and the general experimental design or method.
  + Aims?
    - What were the aims of the paper? It can sometimes even be helpful to highlight the main study goals and hypotheses as you are reading the introduction. This will allow you to easily reference the aims as you dig deeper into the methods, results, and conclusions.
    - If a review paper, what was the scope of the review? In other words, what are the authors trying to accomplish?
  + Background & Significance?
    - What is at stake? Why is this line of research worthwhile or important? Are the goals important or trivial? Often, the larger significance of the work is highlighted at the beginning of the Introduction (and the end of the Discussion).
* Method and Participants?
  + **I do not expect students to fully understand every methodological detail or technique. But it is important that students do their best to understand the *gist* of what was done.**
  + What did the authors do? Are the methods a good fit for the aims or is there a gap of some sort?
  + Who participated and how were they enrolled in the study?
  + How representative is the sample? Is it a good fit for the aims or does it limit the conclusions that can be drawn from the study?
* Key results?
  + Did the results support the hypotheses?
* Discussion
  + The purpose of the Discussion is put the findings in the context of prior literature, acknowledge limitations of the current study, and suggest specific implications for future research and applications to prevention, intervention, or policy.
  + Often, the first paragraph of the Discussion summarizes the key results
  + Often, the final paragraph of the Discussion summarizes the broad implications
  + In between, the authors usually discuss the meaning and implications of the results as well as key limitations
  + Implications?
    - What are the implications for our understanding of T&P?
    - What are the main implications of the findings for theory and for practice?
    - Are there broader implications for our daily lives?
  + Limitations/Caveats, stated or otherwise?
    - Provide strong evidence for the stated conclusions?
    - Are the claims convincing? If not, what further evidence is needed? Are there other experiments or work that would strengthen the paper further?
    - Were important aspects of T&P neglected in the paper?
  + Future challenges
    - What are the most profitable, impactful future steps?

**LEARNING OBJECTIVES**

This section 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 or to the TA if you get stuck or have concerns that we can help you to address. Good luck and warm wishes,

**Module 2 – Impact (Mischel and Moffitt)**

* What did Walt Mischel suggest? Was he right? 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?
* In what 3 ways does contemporary culture magnify the impact of C/SC?
* Moffitt study
  + 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?

**Module 3 – Dimensions (Caspi)**

* How are temperament and personality similar vs. different? Are they categorically different or do they show continuity?
* What’s a hierarchy of traits?
* What are the Big 3? What are key characteristics of each of the 3 in terms of emotion, appraisal, motivation, and cognition/attention? Can we think of the Big 3 as purely cognitive or purely emotional?
* Where does BI fit in? What are the 2 major sub-divisions of N/NE?
* 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. job, culture)?
* Are dispositional traits very fixed/immutable, very plastic/malleable, or somewhere in the middle?
* Which test do we use to assess stability over time? Are long-term test-retest estimates biased upward or downward? Why?
* Why are traits stable at all? What are some mechanisms that explain continuity over time?

**Module 4 – Measurement Issues (Block and Tomarken)**

* What is the lexical hypothesis?
* In general, what are the Big 5 (OCEAN) and how did they come to be?
* In on-technical terms, what is factor analysis? Is it “lossy”? What is meant by “lossy”? Does it require subjective choices or is it an objective tool for discovery (like a telescope)? How does the choice of inputs determine the results?
* 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 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? What is construct validity? How is it related to sensitivity and specificity? To forward and reverse inferences?

**Module 5 – Traits and States Part 1 (Matthews chapter; Watson & Clark 1984)**

* What is Shackman’s definition of T&P (traits)?
* What are 3 models relating traits to states? Define each.
* What is the limitation of the traits = average of states model?
* What are 2 limitations of Fleeson’s probabilistic model? Does it address the role of the environment/situation?
* Describe the interaction or ‘reactive’ model.
* Eysenck, Allport and others 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 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 \_\_\_\_ ?
* McNulty’s Newly Weds Study & Damasio’s Lesion Study - Briefly describe 2 kinds of evidence suggesting that conscious and unconscious/preconscious processes guide behavior? What are 2 pieces of evidence suggesting that they are dissociable or separable or reflect distinct substrates?

**Module 6 – Traits and States Part 2 (Watson & Clark 1984; Canli; AS Fox)**

* What are 2 lines of self-report data that indicate that T&P is impactful in the absence of immediate reward or punishment? 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?
* Canli showed that N/NE is evident in the resting activity of the brain, as indexed using ASL fMRI imaging. Briefly describe what he found.
* In a nutshell how is ASL different than conventional fMRI techniques?
* 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 the key limitation of the Canli and Abercrombie studies? Were the subjects really at emotional ‘baseline?’
* Drew Fox extended this line of work by scanning monkeys in 4 conditions. 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 to this study?
* 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 (aka ‘anxious temperament’) evident in the spontaneous, on-going connectivity of the brain? Is this relevant to humans? Why or why not?
* 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)?

**Module 7 – What do traits do? (Davidson; Gray; Gable)**

* What are 4 key parameters of the emotional response to challenges? How might these be related to dispositional traits? How would you test differences in threshold? Does the amygdala only show differences in peak response? What are some other parameters that differ in individuals with higher levels of N/NE?
* 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 did they observe? Did High-BIS and High-BAS individuals difference in their average level of positive and negative emotion? How was BIS related to reactivity vs. exposure for negative events? How was BAS related to reactivity vs. exposure to positive events? What was the relationship between event exposure and momentary emotion for individuals high in BAS?
* Do traits only influence reactivity? What are some ways in which they influence motivated behavior which, in turn, can influence mood?

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

* What are the key strengths and weaknesses of each technique? Are they mechanistic/causal or correlational? Do they provide direct measures of neuronal firing?
* What is temporal resolution? What is spatial resolution? If an image appears blocky or pixelated, it reflects low \_\_\_\_\_\_\_? If an image appears blurry or smeared, it reflects low \_\_\_\_\_\_\_\_\_\_\_ ?
* Does fMRI measure blood oxygenation or neuronal firing?
* In very general terms, how do you compute an ERP?
* 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 the 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 complexity?
* 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?
* 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 \_\_\_\_\_\_\_\_\_\_\_ ?
* How are markers different than intermediate phenotypes? What are 2 kinds of markers? What are 2 kinds of intermediate phenotypes? What is special about endophenotypes?
* Has the endophenotype strategy ever identified genes, links from endophenotype 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 psychopathology?
* 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 (Visscher)**

* 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?
* 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 variation in a population? To social rules, taboos, or culture?
* What are 4 common misconceptions about heritability?
  + Are genes or phenotypes/traits passed down?
  + Are genes our destiny?
  + Are heritable traits amenable to intervention or are we stuck? Provide an example (e.g. height).
  + 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 are the major strength 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?
* Few genes with big effects or many genes with small effects?
* What are the consequences of small statistical effects for replication? For sample size?
* Is there any practical or biomedical value to weak genetic associations? Describe.
* Do weak genetic associations necessitate a lack of therapeutic value?
* Do genes always have ‘main effects’ or do they sometimes confer risk that is conditional on the environment (G\*E interaction)? Provide an example of a G\*E interaction.

**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? What can you potentially address?
* Is the serotonin transporter polymorphism related to amygdala activation? What does the most recent data teach us?
* Does the neurogenetics strategy rest on assumptions? Is it important to test these assumptions? Provide an example where the assumptions were disconfirmed.
* 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 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?

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

* What’s an emotional disorder
* How is N/NE related to the emotional disorders?
* What are key features of the anxiety disorders?
* Of major depressive disorder (MDD)?
* Common or rare disorders?
* 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. What are the 6 lines of evidence?
* 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)?
* Is there evidence that these disorders reflect shared brain or genetic substrates? Briefly describe the 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? Does temperament come on line earlier?

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

* Briefly describe BI and SAD
* How is BI assessed in children?
* In monkeys?
* In adults?
* How is BI related to Gray’s BIS?
* To the Big 3 and N/NE?
* 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? All individuals or only those who consistently show elevated BI?
* How might BI predict the normative acquisition of crucial social skills? How might this contribute to the development of psychopathology?
* Is BI a strong candidate endophenotype for SAD? Is it heritable?

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

* What is the relevance of the amygdala to fear conditioning? To more innate fears of predators?
* What is fear potentiated startle?
* What’s special about excitotoxic lesions? What do they spare?
* How is amygdala activity, specifically in the CeA, related to individual differences in BI in monkeys?
* To adults with a childhood history of extreme BI?
* To individual differences in N/NE?
* To anxiety 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?
* Does amygdala alter states of fear and anxiety in humans? What about trait-like (paper and pencil) measures of fear and anxiety?
* 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? To economic and behavioral choice?

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

* Briefly describe potential problems with trying to understand the origins of complex traits. What is one strategy for circumventing complexity and heterogeneity?
* How is uncertainty relevant to elevated levels of anxiety?
* What are the 5 key components of the anxious phenotype, according to Grupe & Nitschke?
* Briefly describe the dot probe task and its relevance for understanding hyper-vigilance to threat-related information
* Can hyper-vigilance be retrained?
  + What are the consequences for dispositional anxiety?
  + For clinical anxiety?
  + What does this imply in relation to causation or ‘active ingredient’?
* Briefly describe 2 ways in which the amygdala influences or prioritizes 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 🡪 NBM 🡪 sensory cortex circuit.
  + What is the relevant neurotransmitter?
  + What does the transmitter do to sensory processing in the cortex?
* Briefly describe the evidence for over-generalization and the maladaptive persistence of anxiety in objectively safe contexts.
  + Why is this a big deal?
  + What is the relevance for the development of future anxiety disorders? For childhood BI?
* Which brain regions support the maldaptive persistence of contextually inappropriate anxiety? What is the ‘central extended amygdala’?

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

* Briefly describe the 5 key constituents of individuals with an anxious, neurotic, behaviorally inhibited temperament
* Briefly describe the ERN. What evidence is there that errors are aversive?
* Briefly describe the relationship between this temperament and the magnitude of control-related ERPs generated in the MCC (enhanced or diminished?)
* Do individual differences in the amplitude of the ERN predict the future development of anxiety disorders?
* Imaging studies suggest that the MCC is recruited by what kinds of tasks?
* What is cognitive control?
* What is the adaptive control hypothesis (TACH)? How is it related to avoidance and behavioral inhibition?
* How are control-sensitive ERP signals generated in the MCC related to actual, overt behavior? Do individual differences in the size of the error and feedback-related negativities predict behavior on future trials? Do they predict increased or decreased caution, inhibition, and avoidance?
* Briefly describe some mechanistic/pharmacological evidence suggesting that these signals causally contribute to adjustments in behavior following errors or punishment. If you reduce anxiety, what happens to the control-sensitive ERP signals generated in the MCC? What happens to behavior?
* Are individuals with an anxious, neurotic disposition more or less sensitive to uncertainty?
* How is this altered by anxiety-reducing compounds, such as alcohol and benzodiazepines?
* Which brain region is especially sensitive to uncertainty?

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

* Briefly describe E/PE, BAS, and how they are conceptually related
* Describe key diagnostic criteria for depression
* Briefly describe the relationship between T&P and MDD (e.g. according to the Kotov/Watson meta-analysis)
* Briefly describe the key facets of ‘Reward’ according to Berridge and Robinson
* Are MDD and alcohol/substance use disorders a major or minor burden on global public health? Common and costly or rare and cheap?
* How do patients with depression respond to positive, pleasant, and rewarding stimuli in the lab?
* Briefly describe behavioral and EEG evidence for reduced wanting in MDD patients in the lab
* Briefly describe 2 major ways to behaviorally assess reducing wanting/appetitive motivation in the lab
* Are these 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 behavioral activation therapy. Does behavioral activation reduce depression? What does this suggest for causation?
* Is it easy or difficult to tease apart liking and wanting? Describe briefly please.
* Which regions does the MFB connect? Which transmitter system is it associated with?
* Briefly describe the behavior of rodents given the opportunity to SS the MFB. What about humans? Changes in self-reported liking, wanting, both?
* 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 hypothesis
* 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?
* Based on their work, which regions and transmitters mediate hedonic pleasure and liking?
* What about humans?
  + Does activation in the VS assayed using fMRI predict wanting?
  + E/PE?
  + Is VS activation trait-like?
* What about mechanistic evidence? Do neurofeedback and/or drug studies suggest a causal role?
* Is this system disrupted in MDD? Does it 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?

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

* What are key diagnostic features of SUD’s. What is the importance of impulsivity or loss-of-control?
* Is the VS/Nacc hyper or hypo-responsive to drug cues in addicts? What about everyday rewards and appetitive stimuli, such as food?
* How are trait-like individual differences in E/PE related to SUDs?
* What about C/SC?
* Why might addicts show reduced E/PE? Is there a blunting? An uncoupling of liking and wanting?
* 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?
* Who was Phineas Gage? Which brain region was damaged? What did this suggest about the OFC and self-control?
* 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?
* 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)?
* Does activation in this circuit predict impulse control in the real world?
* Which brain regions play a role in choosing between healthy and unhealthy options? Between tasty and untasty 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?
* What did we learn from Rangel/Hare studies that we did not already know based on Phineas Gage?
* 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?
* 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)?

**how to complete the extra credit assignment using sona**

Four points of extra credit will be available to students who complete the Department Mass Survey using the SONA system (see below for details).

1. Create an account at <https://umpsychology.sona-systems.com/default.aspx>. Please take care to enter your contact information correctly (i.e., errors = no extra credit). Choose PSYC 210 (SHACKMAN) as the course (incorrect course = no extra credit)
2. You should then be able to complete the pre-screen and continue on to the studies.
3. If you click on "View Available Studies", and scroll down, you should see an option that says “Mass Screening Questionnaire - 18 and older” or “Mass Screening Questionnaire - Under 18” - a collection of on-line surveys, many designed to assess facets of T&P. Separate versions of the Mass Survey are available for students who are above or below 18 years of age (owing to different procedures for obtaining consent).
4. ***The earlier you complete the Mass Screening Questionnaire, the better.*** If you encounter difficulties, please contact the TA. Be prepared to describe the problem in as much detail as possible.

**additional course policies**

***Students are responsible for making themselves aware of the relevant course and University policies. Some of these are described below.***

Late Policy

Students will lose 10% of total possible points for each day late without prior approval (barring compelling reasons). Prior approval requires at least 48 hours advance notice.

Grade Disputes

In the case of disputed grades, students are required to submit a written claim within 48 hours of receiving the disputed grade that describes the disputed item/grade, rationale for altering the grade, and suggested alteration.

Curving

Your grade will be determined by your individual performance on the exams and written response exercises. The course will *not* be graded on a curve. With the exception of calculation errors, no changes will be made to your final grade at the end of the semester. If earning a particular grade is important to you, please speak with Professor Shackman or the TA at the beginning of the semester so that we can offer some helpful suggestions for achieving your goal.

Final Grade for the Course

Final grades will be assigned in accord with the following rubric

>97 A+

94-96 A

90-93 A-

87-89 B+

84-86 B

80-83 B-

77-79 C+

74-76 C

70-73 C-

67-69 D+

64-66 D

60-63 D-

<60 F

XF-denotes failure due to academic dishonesty.

W indicates withdrawal from a course in which the student was enrolled at the end of the schedule adjustment period. This mark is not used in any computation of quality points or cumulative average totals at the end of the semester.

Course Evaluations

You will have a formal opportunity to evaluate the effectiveness of this course, although I first want to encourage you to schedule a meeting with me (Professor Shackman) or the TA if you have any questions, concerns, or suggestions for how we can help support your learning and engagement. Specifically, the University will ask you to evaluate all of your courses through the online system ([www.courseevalum.umd.edu](http://www.courseevalum.umd.edu)) at the end of the semester. As members of the campus learning community your feedback is crucial to the success of our program and therefore to the value of your degree. All I ask is that in evaluating of all your courses you approach it in the same way that you expect instructors to evaluate your performance: be open, honest, and objective.

Academic Integrity

Academic integrity is the foundation of science and the policies will be strictly enforced. **My goal is to protect the value and integrity of the grades that have been fairly earned by the vast majority of students.** Any indication of academic dishonesty (including but not limited to cheating, plagiarism and falsification) will be referred to the Office of Student Conduct ([www.osc.umd.edu](http://www.osc.umd.edu)) without hesitation. You are responsible for reviewing the Department of Psychology’s policy statement on academic integrity (<http://psychology.umd.edu/about-us/documents/documents/Syllabus_Supplement_on_Ethics_of_Scholarhip_in_Psychology.pdf>) for details.

The University of Maryland has a nationally recognized Code of Academic Integrity, administered by the Student Honor Council. This Code sets standards for academic integrity at Maryland for all undergraduate and graduate students. As a student, you are responsible for upholding these standards for this course. It is very important for you to be aware that the consequence for cheating, fabrication, facilitation, and plagiarism in this class is a grade of “F”. For more information on the Code of Academic Integrity or the Student Honor Council, please visit: <http://www.studenthonorcouncil.umd.edu/whatis.html>. The student-administered Honor Code and Honor Pledge prohibits students from cheating on exams, plagiarizing papers, submitting the same paper for credit in two courses without authorization, buying papers, submitting fraudulent documents and forging signatures.

**On every examination, paper or other academic exercise not specifically exempted by the instructor, students must write by hand and sign the following pledge: *I am not a cheater.* *I completed this with honor.***

Compliance with the code is administered by the Student Honor Council, which strives to promote a community of trust on the College Park campus. Allegations of academic dishonesty should be reported directly to the Honor Council (301-314-8450) by any member of the campus community. For additional information, consult the Office of Student Conduct. For a description of the University's definition of academic dishonesty, suggestions on how to prevent cheating, and practical answers to frequently asked questions about the Code of Academic Integrity, consult the Student Honor Council's webpage and click on the faculty tab.

Accommodations for Disabilities

The campus's Disability Support Service Office (DSS) works with students and faculty to address a variety of issues ranging from test anxiety to physical and psychological disabilities. If an instructor believes that a student may have a disability, DSS should be consulted (4-7682 or dissup@umd.edu). Note that to receive accommodations, students must first have their disabilities documented by DSS. The office then prepares an Accommodation Letter for course instructors regarding needed accommodations. Students are responsible for presenting this letter to their instructors by the end of the drop/add period ([www.counseling.umd.edu/DSS](http://www.counseling.umd.edu/DSS)).

Religious Observances

Students will not be penalized because of observances of religious beliefs. Please note that it is your responsibility to notify the instructor by email ASAP regarding any absences for religious observances.

Electronic Devices

I expect you to make the responsible and respectful decision to refrain from the temptation to use your cell phone or other mobile electronic devices, such as tablets and notebook computers in class. If you have critical communication to attend to, please excuse yourself from the room and return when you are finished. If I find myself or other students to be distracted by your behavior, I may ask you to leave the room.

Inclement Weather or Campus Emergency

If the University is closed due to inclement weather or a campus emergency (you can find this out by looking at the campus website <http://www.umd.edu> or the snow phone line (301-405-SNOW), classroom activities will be cancelled.

Learning Assistance Center

If you are experiencing difficulties in keeping up with the academic demands of this course, you are strongly encouraged to contact the Learning Assistance Service (www.counseling.umd.edu/LAS). Their educational counselors can help with time management, reading, math learning skills, note-taking and exam preparation skills. All their services are free to UM students.

Students in Distress

Services for students in various forms of distress are offered by the Counseling Center and the Mental Health Service in the Health Center. During evenings and weekends, the student peer-counseling hotline (4-HELP or 4-4357) is available. Faculty who wish to consult with professionals may call 4-7651 for immediate assistance. For non-emergency issues, faculty can call the Warmline (4-7653). A therapist will respond within a few hours.

***Continued…***

**ABOUT THE course**

Professor Alex Shackman

Dr. Shackman received his Ph.D. in Biological Psychology with a distributed minor in Neuroscience from the University of Wisconsin—Madison in 2008. His graduate research was supported by the National Science Foundation and National Institute of Mental Health. He subsequently conducted postdoctoral research in the laboratories of Richard Davidson, Brad Postle, and Ned Kalin in the Departments of Psychology and Psychiatry at Wisconsin. This work has appeared in a number of outlets, including the *Proceedings of the National Academy of Sciences USA*, *Nature Reviews Neuroscience*, *Journal of Neuroscience*, and *Psychological Science*. Professor Shackman serves as an Associate Journal Editor at *Cognitive, Affective & Behavioral Neuroscience* (CABN)*; Cognition and Emotion*; *Emotion*; and *Frontiers in Human Neuroscience*.

Dr. Shackman's major research interests include affective and cognitive neuroscience; neural bases of threat processing, anxiety, fear, and their application to anxiety, mood, and related psychiatric disorders; neural bases of personality; individual differences in anxiety and behavioral inhibition; cognition × emotion interactions; developmental psychopathology; extended amygdala; anterior cingulate cortex (ACC); prefrontal cortex (PFC).

Key methods used by the Shackman lab include multimodal neuroimaging (fMRI, PET, VBM); peripheral physiological techniques (cortisol, facial EMG, fear-potentiated startle), and behavioral assays (eyetracking and experience sampling). Populations of interest include children, adolescents, healthy adults, and psychiatric patients.

To learn more about the lab, please visit our website at <http://shackmanlab.org>

Rachael Tillman

Ms. Tillman is a graduate student in Clinical Psychology working with Dr. Shackman in the Affective and Translational Neuroscience Lab. She is interested in characterizing the neural mechanisms underlying the development of socioemotional processing and how it relates to psychopathology. Currently, her research focuses on dissociating the neural correlates of social fear and anxiety in adolescents with social phobia.

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