Australian Appetitive Motivation Symposium

Wednesday 6th of August, 2025

Registration Details at http://aams.psy.unsw.edu.au/

Organising Committee: Kelly Clemens, Karly Turner, Mike Kendig, Morgan James, Lizzie Manning

PROGRAM

Please note that morning tea and lunch are at your leisure. See the map below highlighting the symposium venues and some of our favorite coffee and food locations.

VENUE 1: ANITA B. LAWRENCE THEATRE (G001; H13) UNSW, LOWER CAMPUS

9.00 – 9.10 **Welcome:** *A/Prof Kelly Clemens and Dr Karly Turner (UNSW, AU)*

9.10 – 10.00 **Keynote:** Chair Dr Laura Bradfield, UTS, AU
***Prof Geoff Schoenbaum**, National Institute on Drug Abuse (NIDA), USA*
The curious case of dopaminergic error signals and learning beyond value.

~ Morning Tea ~

Session 1: Eating and Feeding: Chair Dr Poppy Watson, UTS, AU

10.30 – 10.50 **Dr Maya Opendak**, Kennedy Kreiger Institute, USA
Neural circuits supporting social transitions in infant rats in typical and perturbed development

10.50 – 11.10 **Dr Dominic Tran**, University of Sydney, AU
Consumption of a diet high in fat and sugar is associated with worse spatial navigation ability in a virtual environment

11.10 – 11.30 **Dr Mugdha Joglekar**, University of Western Sydney, AU
Analysis of gut metabolites during progression to diet-induced metabolic disease

Session 2: Addiction: Chair A/Prof Laura Corbit, U Tornoto, CA

11.30 – 11.50 **Dr Tristan Hurzeler**, University of Sydney, AU
Neurobehavioral effects of Cannabidiol (CBD) in individuals with Alcohol Use Disorder: A double-blind, randomised control trial

11.50 – 12.10 **Dr Liam Acheson**, UNSW, AU
Development of agonist like treatment approaches for the treatment of methamphetamine use disorder and acute withdrawal

12.10 – 12.30 **Dr Ivy Hoang**, Stanford University, USA
Changes to a hypothalamic-dopamine learning circuit with methamphetamine experience

~ Lunch ~

VENUE 2: CLANCY AUDITORIUM (C24) UNSW, UPPER CAMPUS

1.30 – 2.20 **Keynote:** Chair Prof Gavan McNally, UNSW, AU
A/Prof Susan Sangha, Indiana University, USA
Neurobiological correlates of behavioral resilience to chronic alcohol and acute stress

Session 3: Decision Neuroscience: Chair Dr Shauna Parkes, U Bordeaux, FR

2.20 – 2.40 **Dr Emily Sylwestrak,** University of Oregon, USA
Cell-type-specific encoding of reward variables in habenular neurons

2.40 – 3.00 **A/Prof Jay Bertran-Gonzalez**, UNSW, AU
At D2-Neuron's Discretion: Uncovering the Imperative Role of D2-to D1-neuron Transmodulation in Instrumental Learning

3.00 – 3.20 **Dr Claire O'Callaghan**, University of Sydney, AU
Windows into noradrenaline's role in decision making and learning through single dose drug studies in Parkinson's disease

3.20-5.00 Posters and Networking, Clancy Auditorium Foyer

IMPORTANT INFO

Transport: Arrive via Uber, Taxi, Tram or Bus. Parking is available at Barker Street and L6 Botany Street Parking Stations. If arriving after 9 am, Barker St will have more availability.

Morning Tea and Lunch: As AAMS is a free event, we encourage you to find your own morning tea and lunch as you continue your networking. UNSW has many food outlets https://www.unsw.edu.au/estate/food-retail/explore. We would recommend Coffee on Campus in the Ainsworth Building in the morning (lower campus) and Café Brioso or the Mathews Pavilion in the afternoon (upper campus).

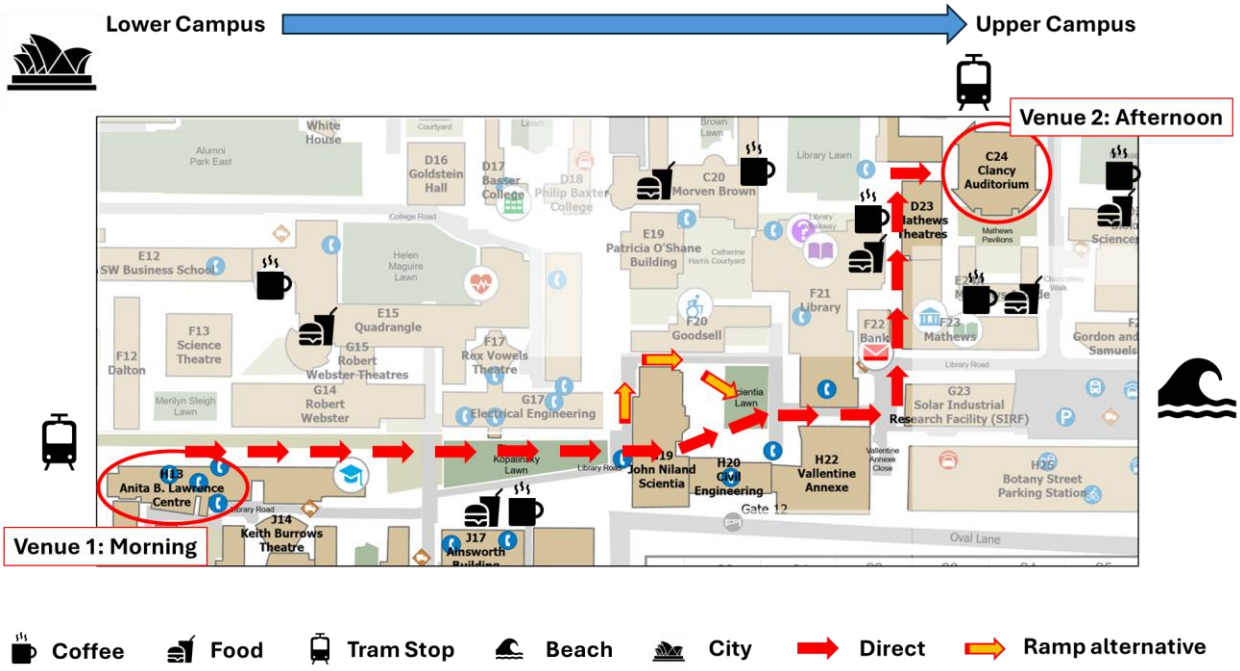
Talks: Keynotes are 40 min plus 10 min questions.
Session talks are 15 min plus 5 min questions.
Please bring your USB or laptop prior to the start of your session. We will be running strictly to time so technical issues might impact on your opportunity to speak.

Posters: Posters are AO Portrait format and can be put up from 1 pm. Poster boards are located on the East Wing of the Clancy Auditorium. Velcro spots will be provided.

Venues: Due to mid-term venue availability, AAMS will be held across two venues. In the morning AAMS is in the Anita B. Lawrence Theater, Ground Floor of H13 (The Red Centre). At lunch, take a stroll up through campus to the Clancy Auditorium.

Left: The Red Centre containing the Anita B. Lawrence Theatre

Right: The Clancy Auditorium



SPEAKER ABSTRACTS

Prof Geoffrey Schoenbaum

NIH Distinguished Investigator, NIDA, Baltimore, USA

The curious case of dopaminergic error signals and learning beyond value

Transient changes in the firing of midbrain dopamine neurons have been closely tied to the unidimensional value-based prediction error contained in temporal difference reinforcement learning) models. However, while there is now an abundance of work showing how well dopamine responses conform to the predictions of this hypothesis, there are far fewer studies that challenge its implicit assumption that dopamine is not involved in learning about value-neutral features of rewards and other events. Here we review studies in rats and humans that put this assumption to the test, and which suggest that dopamine transients provide a much richer signal that incorporates information that goes beyond integrated value.

A/Prof Susan Sangha

Indiana University School of Medicine, Stark Neuroscience Research Institute, Indianapolis, Indiana USA

Neurobiological correlates of behavioral resilience to chronic alcohol and acute stress

Alcohol use disorder (AUD) and stress-associated disorders show high comorbidity, with ~42% Post-Traumatic Stress Disorder (PTSD) individuals also exhibiting AUD. Increasing evidence suggests PTSD and chronic alcohol use may alter the ability to discriminate between different emotionally-relevant stimuli, leading to maladaptive behaviors such as overgeneralizing the impact of fear signals, and difficulties in using safety signals to reduce fear. Our lab has expertise in the neural circuits of fear regulation via safety/inhibitor cues and how stress and alcohol impacts this learning. Our recent work shows male and female Long Evans rats voluntarily consuming alcohol in the home cage (chronic intermittent 24h two-bottle choice access to water and 15% EtOH) and/or exposed to an acute footshock stressor have different behavioral outcomes in our conditioned inhibition task. This task includes trials of a fear cue paired with shock, a reward cue paired with sucrose, an inhibitor cue without an outcome, and compound trials of fear+inhibitor cues and reward+inhibitor cues without shock or sucrose. Depending on how fear and reward behaviors were downregulated by the inhibitor cue during the compound cues, rats were classified as one of 4 groups: those that still showed reduced fear and reward behavior during the inhibitor cue (resilient) and those that, despite the inhibitor cue, showed persistently high fear or persistently high reward behavior or both. Using immunohistochemistry, we assessed parvalbumin (PV), somatostatin (SOM), and protein kinase C delta (PKCδ) interneurons in subregions of the medial prefrontal cortex, amygdala, BNST and lateral septum. We found patterns of PV and PKCδ expression that correlated with behavioral resilience. Using bacterial sequencing, we profiled the gut microbiota of resilient and non-resilient subjects and found specific bacteria associated with behavioral resilience. Together, our conditioned inhibition task allows us to profile neurobiological changes unique to a subject's ability to regulate fear and reward seeking behaviors after alcohol and/or stress exposure.

Dr Maya Opendak,

Kennedy Kreiger Institute, USA

Neural circuits supporting social transitions in infant rats in typical and perturbed development

The development of social behavior reflects changing demands of age, environment, species and responses to early care quality. Indeed, the caregiver-infant social dyad provides a lasting template for lifelong social behavior patterns, with early caregiving adversity producing immediate and enduring impacts on the brain and behavior. However, the specific neural mechanisms translating early social experience to long-term reorganization of social behavior remain poorly understood. In this talk, I will present data from my lab adapting circuit dissection tools for infants to examine the typical changes that underlie social maturation from infancy to adolescence and how early adversity disrupts these circuits as they are being built to impair social behavior. I will present our functional work on the social brains of typically-developing infants and juveniles, as well as immediate and lasting effects of specific early stressors on central measures of brain structure and function (volume, gene expression, neuronal firing properties, circuit activity), peripheral measures and behavior.

Dr Dominic Tran

School of Psychology, University of Sydney, AU

Consumption of a diet high in fat and sugar is associated with worse spatial navigation ability in a virtual environment

Diets rich in saturated fats and refined sugars can lead to obesity, cardiometabolic disease, and certain cancers. These diets can also have adverse effects on cognitive function. Regular consumption of fats and sugars is associated with accelerated age-related cognitive decline in middle-aged and older adults. Rodent studies show that diets high in fats and sugars can impair the hippocampus, affecting spatial learning and memory. The present study examined the relationship between diet and spatial navigation ability in humans using a virtual reality maze. Successful performance in the task required participants to estimate distance and direction to track self-referential positioning and remember landmark locations. We found that young adults who frequently consumed foods high in fat and sugar were worse at remembering the location of a treasure chest in the virtual maze. This relationship remained after controlling for body mass index and performance on a non-spatial task. These results highlight the impact of diet beyond traditional physical health indicators and reveal a specific association between diet and spatial ability. The findings are consistent with animal studies and are the first to reveal the adverse effect of diet on spatial learning and memory in a task that requires navigation in three-dimensional space. The results confirm importance of healthy dietary choices for maintaining cognitive health.

Dr Mugdha Joglekar

Diabetes and Islet Biology Group, School of Medicine, Western Sydney University

Analysis of gut metabolites during progression to diet-induced metabolic disease

Short-chain fatty acids (SCFAs) are one of the gut metabolites that are demonstrated to play a critical role in the pathophysiology of obesity and diabetes. Our meta-analysis concluded that increase in SCFAs is significantly associated with lower fasting insulin levels and improved insulin sensitivity. However, longitudinal changes in the gut microbiome and SCFAs during the progression to obesity and diabetes are not well understood, which we aimed to profile using animal models of diet-induced obesity and prediabetes; specifically high fat-diet (HFD), cafeteria diet (CAF) and dietary weight cycling (OSC). Weight cycling comprised of 2-week dietary oscillation between HFD/CAF and normal (chow) diet for 24-weeks (study-endpoint). SCFAs and other gut metabolites were profiled (longitudinally and at study endpoint) using Liquid Chromatography–Mass Spectrometry. HFD and oscillating HFDled to overweight/obesity in mice with dysregulated glucose tolerance, while the animals on CAF and oscillating CAF diets had significant weight gain with higher fasting blood glucose levels. We observed a significant reduction in total faecal SCFAs (acetate, propionate, and butyrate) in mice on HFD and oscillating diet compared to chow diet at endpoint. Longitudinal SCFA analysis demonstrated a steady reduction in HFD group compared to control for each SCFAs. Infusion of butyric acid (with a flow rate of 0.11 µL/min for 4 days) in HFD mice improved glucose clearance. Further in vitro experiments showed upregulation of several transcripts including GLP-1, in colonic cells under high concentrations of SCFAs. Overall, our results suggest that gut SCFA concentrations can be early indicators of progression to obesity and prediabetes as well as potential therapeutic agents in metabolic disease

Dr Tristan Hurzeler

University of Sydney, AU

Neurobehavioral effects of Cannabidiol (CBD) in individuals with Alcohol Use Disorder: A double-blind, randomised control trial

Preclinical and clinical results suggest that cannabidiol (CBD) might be particularly well suited for the treatment of alcohol use disorder (AUD) and may reduce alcohol-cue and stress-cue-induced craving. This study aims to investigate this novel pharmacotherapy with a particular focus on neurobiological and psychophysiological mechanisms of CBD for AUD. Methods: In this double-blind, randomised, cross-over study, 22 non-treatment seekers were allocated to three days of CBD (800 mg/day) or placebo, with an 18-day washout period. Functional magnetic resonance imaging (fMRI) and psychophysiological measures were collected during cue reactivity tasks. Additionally, magnetic resonance spectroscopy (MRS) was conducted to collect neurometabolite levels in the dorsal anterior cingulate cortex (dACC). Outcomes included: i) regional activity during a functional magnetic resonance imaging (fMRI) cue reactivity task, ii) heart rate variability (HRV) and skin conductance levels (SCL) as a proxy for psychophysiological responses to alcohol stimuli, iii) neurometabolite levels (GABA+, NAA, Glx, Cho and GSH) within the dACC using MRS. Results: Region of interest analyses of the fMRI cue reactivity task demonstrated non-significant treatment effects in dorsolateral and ventromedial prefrontal cortex or caudate. However, whole-brain analysis indicated a significant treatment effect in the precuneus, independent of cue type. Throughout the

psychophysiological cue reactivity task CBD vs placebo was associated with elevated HRV and greater reductions in self-report anxiety and alcohol craving from exposure to cue recovery periods. While no main treatment effects were identified across neurometabolite, post-hoc analyses indicated that CBD vs placebo sessions were associated with significantly higher GSH, GLX, and GABA levels in participants who consumed alcohol the previous day compared to those who were abstinent. Conclusion: These findings suggest CBD administration modulates key dysregulated neural and autonomic pathways in AUD, and support further investigation in treatment-seeking populations to assess CBD's potential for AUD management.

Dr Liam Acheson

National Centre for Clinical Research on Emerging Drugs, National Drug and Alcohol Research Centre, NSW, AU

Development of agonist like treatment approaches for the treatment of methamphetamine use disorder and acute withdrawal

There are currently no pharmacological treatment options for methamphetamine use disorder or acute methamphetamine withdrawal. For other substances agonist treatment approaches are safe and effective. Lisdexamfetamine is an inactive prodrug of dexamphetamine, and is a likely candidate medication for both treating methamphetamine use disorder and withdrawal. This talk will give and overview of three clinical trials: LiMA, a randomised controlled trial of 250mg lisdexamfetamine once daily for 15 weeks for the treatment of methamphetamine use disorder; OLAM, a pilot clinical trial of a 5 day tapering dose of lisdexamfetamine starting at 250mg once daily for the treatment of acute methamphetamine withdrawal, and OLAM-II, a currently recruiting randomised controlled trial of a 5 day tapering dose of 250mg lisdexamfetamine and 2 day placebo washout for methamphetamine withdrawal. LiMA recruited 164 people, and throughout the 12-week treatment phase the lisdexamfetamine group had fewer days of methamphetamine use in total (difference = 8.8, 95% CI = 2.7–15.0; P = 0.005), however only weak evidence of a benefit at week 13 was apparent (difference = 2.2, 95% CI = –0.5 to 5.0; P = 0.49). The lisdexamphetamine group reported greater treatment effectiveness and satisfaction. The OLAM pilot study recruited 10 participants, 8 of whom completed treatment. No serious adverse events were reported, and the treatment was highly acceptable to participants. Withdrawal and craving severity reduced during treatment. The OLAM-II randomised controlled trial is currently recruiting at 5 sites in Australia, and will recruit 184 people. Lisdexamfetamine appears to reduce methamphetamine use over a 12-week treatment period in outpatient settings, and a short tapering treatment regimen is safe and feasible for managing acute withdrawal in inpatient withdrawal management units. Further exploration of the therapeutic potential of medications with a similar mechanism of action to the substance of disorder are warranted and ongoing, however further work is required to achieve consensus around codesigned therapeutic outcomes to ensure trial meet the needs of community

Dr Ivy Hoang

Stanford University, USA

Changes to a hypothalamic-dopamine learning circuit with methamphetamine experience

Environmental cues can shape our daily decision-making, guiding us towards or away from the outcomes they are associated with in a dynamic manner. However, cue-guided behavior can become maladaptive for individuals with a substance use disorder. Indeed, these individuals often exhibit high susceptibility to the profound influence of drug and other reward-related cues, directing them toward drug use behaviors and increasing the risk for relapse for those in recovery. Despite much research studying cue-evoked drug-seeking, it is unclear whether the heightened control these reward-paired cues exert over behavior is being driven by a cognitive representation of the reward itself. Here, we show that rats with a history of methamphetamine self-administration demonstrate enhancements in the ability of outcome-specific cues to guide behavior toward those specific outcomes. This could suggest that prior drug experiences may be strengthening key neural mechanisms underlying the acquisition and use of outcome expectations to influence behavior. In support of this hypothesis, we first characterized an understudied neural circuit comprised of the lateral hypothalamus and midbrain dopamine for outcome-specific cue learning using optogenetics. We then found that cue-evoked dopamine release patterns in the lateral hypothalamus across cue-reward learning was amplified in rats with prior methamphetamine experience. Altogether, these findings highlight a hypothalamic-dopamine circuit for acquiring outcome-specific expectations and expose how drug-induced changes to this circuit and its functions in learning may be underpinning maladaptive cue-driven behaviors seen with substance use disorders.

Dr Emily Sylwestrak

University of Oregon, USA

Cell-type-specific encoding of reward variables in habenular neurons

To maximize rewards in dynamic environments, animals must evaluate past outcomes, predict future events, and flexibly adjust their behavior. These reward-related computations are represented across many structures in the mammalian brain. Neurons in the habenula have been shown to encode both predictive and evaluative information, as well as signals related to stress, anxiety, and aversion. In addition to its functional heterogeneity, the habenula is molecularly diverse, with cells expressing a wide range of neuromodulatory receptors, neurotransmitters, and neuropeptides. However, it is unclear how these transcriptionally defined cell types map onto functional populations. Using Cre-mediated viral delivery in habenula subpopulations and fiber-based calcium imaging across a series of operant tasks, we identify cell types that encode reward history, predictive cues, and negative reward prediction errors. These data suggest that transcriptional markers could serve as a useful tool to target functional populations in the habenula to better understand how reward information is integrated with other behaviorally relevant information to support flexible behavior.

A/Prof Jay Bertran-Gonzalez

Neuromodulatory Systems and Behaviour group, Decision Neuroscience laboratory, School of Psychology, UNSW Sydney, Australia

At D2-Neuron's Discretion: Uncovering the Imperative Role of D2-to D1-neuron Transmodulation in Instrumental Learning

Striatal spiny projection neurons (SPNs) of the direct (D1) and indirect (D2) pathways possess remarkable molecular capacities and are intricately interspersed within a cellular mosaic. Yet, how these features contribute to the encoding of learning remains incompletely understood. My team capitalises on both the cytoarchitecture of the striatum and the intracellular signalling potential of SPNs to investigate how learning signals—such as dopamine—are captured and integrated within the basal ganglia to drive future behavioural change. We have identified a novel transmodulatory mechanism whereby D2-SPNs access and suppress plasticity in neighbouring D1-SPNs in a one-to-one fashion—a process critical for updating instrumental learning and likely constituting the standard modus operandi of the striatal network. To study this, we develop and deploy next-generation biosensors that enable the investigation of large-scale transmodulatory function in the striatum with high temporal resolution and molecular specificity. Our findings reveal a dominant cellular mechanism by which D2-SPNs can override substantial dopamine signals by rapidly and profoundly constraining D1-SPN plasticity—an imperative process with far-reaching implications for all forms of striatal-dependent learning.

Dr Claire O'Callaghan

University of Sydney, AU

Windows into noradrenaline's role in decision making and learning through single dose drug studies in Parkinson's disease

Single dose drug studies provide a powerful framework for understanding neuromodulatory systems, and they offer one of the rare opportunities for a causal manipulation in humans. In conditions like Parkinson's disease – which undergo profound changes in neuromodulatory function – these studies can provide important clinical insights, as well as being a test bed for current normative neuroscience theories. I will cover some of our recent findings using the noradrenergic reuptake inhibitor, atomoxetine in Parkinson's disease – focusing on areas that have typically been viewed through a dopaminergic lens.

POSTERS

1. Laura Corbit, U Toronto, CA**.** A link between inflammation and cognitive control, independent of diet-induced metabolic changes.

2. Christina Suhartono, U Melb, AU. Neurobehavioural Determinants of Diet-Induced Obesity

3. Alastair Hedge-Luzowoi, UNSW, AU. Examining Sex Differences in the Effects of Glucagon-like Peptide-1 on Alcohol Intake Behaviours

4. Lizzie Manning, University of Newcastle, AU. 5HT2C antagonists as a target for treating OCD and anxiety in Sapap3 knockout mice

5. Jessica Perry, UNSW, AU. Epigenetic Responses to Prenatal Methadone Exposure

6. Suzzan Luitel, UNSW, AU. Investigating Long-Term Deficits from Prenatal Opioid Exposure: Effects on Neurogenesis in Cerebral Organoids and Therapeutic Evaluation of Butyric Acid

7. Annabella Chen, UNSW, AU. Investigating Long-Term Deficits from Prenatal Opioid Exposure: Effects on Neurogenesis in Cerebral Organoids and Therapeutic Evaluation of Butyric Acid

8. Asena Bingul, UNSW, AU. Disconnection of the lateral hypothalamus-nigral projection result in motor deficits in rats: implications for Parkinson's disease.

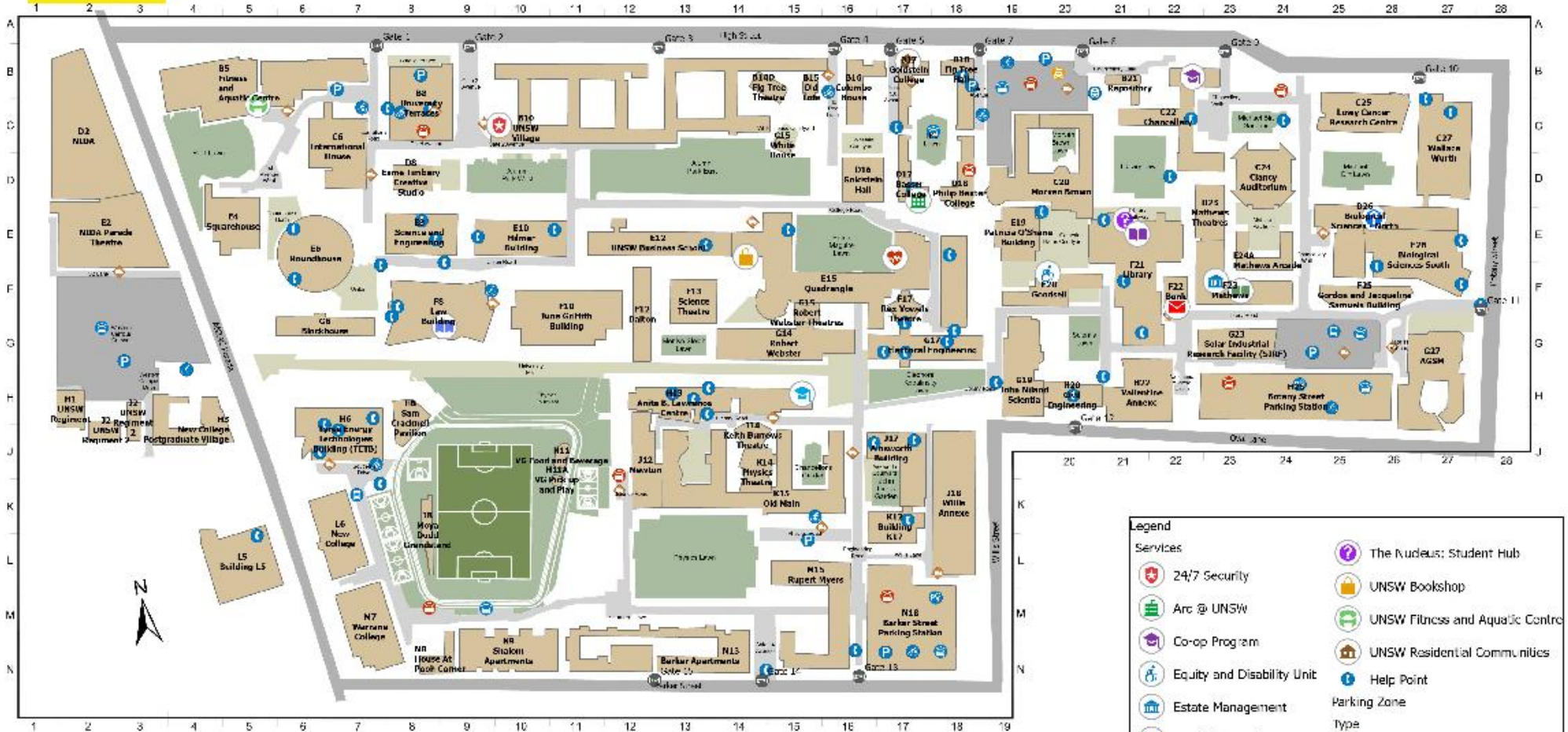
9. Isabel Chew, University of Newcastle, AU. Using Reversal Paradigms to Detect Changes in Flexible Learning in the SAPAP3-KO Model relevant to Obsessive Compulsive Disorder

10. Bixuan Lin, UNSW, AU. Striatal dopamine profiles during punishment learning

11. Lachlan Ferguson, UNSW, AU. Reference Point-Dependent Reinforcement Learning in Humans and Rats

12. Poppy Watson, UTS, AU. A lifetime of conditioning: Food Brand Logos, Dietary Habits, and Goal-directed Control

Kensington Campus Map



Date Exported: 20/02/2024