BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors. Follow this format for each person. **DO NOT EXCEED FIVE PAGES.**

NAME: Travis Edward Baker

eRA COMMONS USER NAME (credential, e.g., agency login): travis.e.baker

POSITION TITLE: Assistant Professor

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Vancouver Island University, Nanaimo, Canada	BA	05/04 01/07	Psychology
University of Victoria, Victoria, Canada	MSc	04/12 08/15	Neuropsychology
University of Victoria, Victoria, Canada	PhD	08/16	Cognitive Science
University of Montreal, Montreal, Canada	Post-doc		Psychiatry
Montreal Neurological Institute, Montreal, Canada	Post-doc		Neuroimaging

A. Personal Statement

I am an Assistant Professor at the Center for Molecular and Behavioral Neuroscience, Rutgers University, and PI of the Laboratory for Cognitive Neuroimaging and Stimulation (www.neurostimlab.com). The main goal of my research program is to test and evaluate a new EEG-informed TMS intervention capable of precisely identifying and targeting the reward function of the anterior midcingulate cortex (MCC), a brain region strongly implicated in goal-directed behavior and substance use disorders (SUD). By developing a more effective TMS intervention aimed to modulate the MCC reward function, our approach may recovery the reward function of the MCC, reduce the frequency and severity of goal-directed deficits observed in SUD individuals, and may ultimately increase substance users' control over drug use and maintain treatment goals. I am a cognitive neuroscientist, with specific training and expertise in human electrophysiology, structural and functional neuroimaging, non-invasive brain stimulation (robot-assisted image-guided transcranial magnetic stimulation, Ri-TMS) and SUD. I have been at the forefront of investigations testing the theory that the anterior midcingulate cortex (MCC) utilizes dopaminergic reward signals to learn the value of goal-directed behaviors, and identified the *reward positivity* as a positive going deflection in the ERP highly sensitive to positive rather than negative feedback in reinforcement learning tasks. Further, my research has shown that drug-potentiated dopamine signals disrupt the reward function of the MCC and precipitate the abnormal goal-directed processes underlying SUDs. Building on this work, I designed and implemented a TMS intervention that restored the reward function of the MCC in abstained smokers, polysubstance and opioid users. As an Assistant Professor, I have made extensive use of combining Ri-TMS with neurocognitive endpoints (EEG, computational modeling, and behavior) to test the effects of various TMS protocols on reward processes in both healthy and substance using populations.

Ongoing and recently completed projects that I would like to highlight include:

2020-2022: National Institute on Drug Abuse of the National Institutes of Health [Award Number R21 DA049574] *Using combined EEG and non-invasive brain stimulation to examine and improve reward functioning in opioid use disorder.* Role, PI

2021-2023 National Cancer Institute | National Institutes of Health (NIH R21 CA262491). *"Candidate mechanisms for chemotherapy-induced neurocognitive deficits in pediatric solid non-CNS tumor patients".* Role: PI

Citations

1. Biernacki, K., Lin, M.H., **Baker, T.E.** (2020). Recovery of reward function in problematic substance users using a combination of robotics, electrophysiology, and TMS. *Int J Psychophysiol* 158, 288-298.
2. **Baker, T.E.,** Zeighami, Y., Dagher, A., Holroyd, C.B. (2020). Smoking decisions: Altered reinforcement learning signals induced by nicotine state. *Nicotine and Tobacco Research*, 22, 164-171.
3. **Baker, T.E.,** Lesperance, P., Potvin, S., Tucholka, A., Jutras-Aswad, D., Zhang, Y., Larcher, K., and Conrod, P. (2017) Reversing the atypical valuation of drug and nondrug rewards in smokers using multimodal neuroimaging. *Biological Psychiatry*, 82, 819-827.
4. **Baker, T. E.,** Stockwell, T., Barnes, G., Haesevoets, R., and Holroyd, C. B. (2016). Reward sensitivity of anterior cingulate cortex as an intermediate phenotype between DRD4-521T and substance misuse. Journal of Cognitive Neuroscience, 28, 460-471.

B. Positions, Scientific Appointments, and Honors

Positions and Scientific Appointments:

2016-present Assistant Professor, Center for Molecular and Behavioral Neuroscience, Rutgers University

Honors and Awards

2020	NIDA Cutting-Edge Basic Research Award
2019	Rutgers Research Council Award
2017	Rutgers Research Council Award
2015	Jeanne Timmins Costello Post-doctoral Fellowship, Montreal Neurological Institute
2013	Canadian Psychological Association Certificate of Academic Excellence in recognition of outstanding thesis
2012	Canadian Institute of Health Research Post-doctoral Fellowship
2011	Canadian Institute of Health Research Competition Silver Medal
2009	Frederick Banting and Charles Best Canada Graduate Scholarships Doctoral Award, Canadian Institute of Health Research
2007	Intersections of Mental Health Perspectives in Addictions Research Training Doctoral fellowship

C. Contributions to Science

1. **Anterior midcingulate cortex dysfunction in substance use disorder.** While much attention in the field has focused on the contributions of subcortical brain regions such as the ventral striatum to drug-induced changes to behavior, my research findings elucidate an important yet under-investigated role of the anterior midcingulate cortex (MCC) in substance use disorders (SUD). My findings suggest that drug-potentiated dopamine signals bias individuals to engage in goal-directed behaviors that lead to drug consumption, thereby associating these behaviors with enhanced reward value while de-potentiating the value of other, non-drug-related activities. Support for this proposal comes from three experiments indicating that people who abuse addictive substances produce a relatively small MCC electrophysiological response to nominal monetary incentives, but following an overnight period of abstinence, the aMCC reward response is normalized by feedback stimuli that signify a chance to puff on a cigarette. Moreover, the impairment appears to be mediated by the dopamine-MCC interface, as MCC electrophysiological activity (frontal midline theta power) statistically mediate the impact on substance dependence of a genetic polymorphism that codes for the expression of dopamine D4 receptors. Taken together, the results provided a theoretical framework that bridges the gap between genes, brain, and behavior in drug addiction.

   a. **Baker, T.E.,** Lesperance, P., Potvin, S., Tucholka, A., Jutras-Aswad, D., Zhang, Y., Larcher, K., and Conrod, P. (2017) Reversing the atypical valuation of drug and nondrug rewards in smokers using multimodal neuroimaging. *Biological Psychiatry*, 82, 819-827.
   b. **Baker, T. E**., Stockwell, T., Barnes, G., Haesevoets, R., and Holroyd, C. B. (2016). Reward sensitivity of anterior cingulate cortex as an intermediate phenotype between DRD4-521T and substance misuse. *Journal of Cognitive Neuroscience*, 28, 460-471.

c. **Baker, T.E**., Wood, J., and Holroyd, C. B. (2016). Atypical valuation of monetary and cigarette rewards in substance dependent smokers. *Clinical Neurophysiology*, 127, 1358-1365.
d. **Baker, T. E.,** Stockwell, T., Barnes, G., and Holroyd, C. B. (2011). Individual Differences in Substance Dependence: At the Intersection of Brain, Behaviour, and Cognition. *Addiction Biology*, 16, 458-466

2. **The application of transcranial magnetic stimulation in substance use disorders**. Extensive empirical work has detailed the potentiating effects of addictive substances on the reward function of the mesocorticolimbic dopamine system. Despite great efforts to identify methods to counteract such drug-induced neural alterations, brain-based treatments for this disorder remain underdeveloped. Building on my PhD research, my post-doctoral research focused on modulating reward signaling in the aMCC during the valuation of nondrug and drug rewards in abstinent smokers by applying robot-assisted image-guided repetitive transcranial magnetic stimulation (Ri-TMS). This study demonstrated that applying excitatory or inhibitory Ri-TMS repetitive to a specific frontal-cingulate pathway reversed the atypical valuation of monetary and cigarette rewards by aMCC in abstained smokers (Baker et al. 2017, Biological Psychiatry). As an assistant professor, my laboratory is making extensive use of Ri-TMS (1 of 3 systems in North America), in combination with EEG and neuroimaging, to develop new TMS interventions aimed to modulate reward-related decision-making functioning in SUD.

a. Lin, M.H., & **Baker, T.E.** (2022). A novel application of an adaptive filter to dissociate the effects of TMS on neural excitability and trial-to-trial latency jitter in event-related potentials, Brain Stimulation. 15, 388-390.
b. Biernacki, K., Lin, M.H., **Baker, T.E.** (2020). Recovery of reward function in problematic substance users using a combination of robotics, electrophysiology, and TMS. Int J Psychophysiology 158, 288-298.
c. **Baker, T.E.,** Lesperance, P., Potvin, S., Tucholka, A., Jutras-Aswad, D., Zhang, Y., Larcher, K., and Conrod, P. (2017) Reversing the atypical valuation of drug and nondrug rewards in smokers using multimodal neuroimaging. Biological Psychiatry, 82, 819-827.

3. **Deconstructing the role of the anterior midcingulate cortex in goal-directed behavior** Theories of anterior midcingulate cortex (MCC) function span a plethora of functions and continue to be hotly debated. I have been at the forefront of such investigations, namely using electrophysiological and behavioral methods to test the theory that the MCC utilizes dopaminergic reward signals to learn the value of goal-directed behaviors. My most notable contribution has been the identification of the event-related brain potential (ERP) called the *'reward positivity'*. A series of studies identified this component as a positive going deflection in the ERP highly sensitive to positive rather than negative reward prediction error (RPE) signals. Although studies of the reward positivity provides valuable insights into neural response patterns of MCC during reward processing, one cannot use correlative evidence from neuroimaging data to make causal inferences about the behavioral significance of the observed brain response. Because transcranial magnetic stimulation (TMS) offers a powerful tool for investigating causal brain-behavior relations, its experimental application may help reconcile or dispute theories of MCC function, thereby providing a deeper understanding of the functional significance of the reward positivity. Consistent with this idea, our study revealed that prefrontal theta-burst stimulation (TBS) suppressed the reward positivity and caused a decrease in post-feedback switch choices, supporting MCC's role in the valuation and selection of effortful behavior. We replicated and strengthened this suppressive action using individualized cortical thickness targets, a new TMS targeting method developed in my lab. By identifying a TMS method to up- and down-regulate reward positivity amplitude, my research has opened an exciting new era of investigative possibilities in the understanding of MCC function.

a. **Baker, T. E.,** Lin, M.-H., Gueth, M., Biernacki, K., Parikh, S. (2020). Beyond the Motor Cortex: Theta Burst Stimulation of the Anterior Midcingulate Cortex. Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 5, 1052-1060.
b. **Baker, T. E.,** & Holroyd, C. B. (2011). Dissociated roles of the anterior cingulate cortex in reward and conflict processing as revealed by the feedback error-related negativity and N200. *Biological Psychology*, 87, 25-34
c. **Baker, T. E.,** & Holroyd, C. B. (2009). Which way do I go? Neural activation in response to feedback and spatial processing in a virtual T-Maze. *Cerebral Cortex*, 19, 1708-1722

d. Li, P., **Baker, T.E.,** Warren, C., and Hong, L. (2016). Oscillatory profiles of positive, negative and neutral feedback stimuli during adaptive decision making. *International Journal of Psychophysiology*, 107, 37-43.

4. **Behavioral and computational explorations of decision-making functioning in health and disease**. I have also extensively studied the decision-making function of the frontal-striatal system using a combination of genetics, neuroimaging, behavior, and computational modeling methods, with a focus on SUDs. The premise of this work is based on an influential neurocomputational model of decision making, "the basal ganglia go/no-go model," which holds that dopaminergic reward prediction error signaling (RPEs) in the basal ganglia facilitates or suppresses action representations during reinforcement learning tasks: Phasic bursts of dopamine activity (positive RPEs) facilitate reward learning, whereas phasic dips in dopamine activity (negative RPEs) facilitate avoidance learning s (Frank et al., 2004). To date, my work has revealed that: 1) factors related to genetic, psychiatric, and personality can constrain the decision-making function of the basal ganglia, 2) susceptibility to early onset of alcohol misuse is related to genetic factors that modulate reward-related activations within the mesocorticolimbic system, and, 3) positive and negative RPE signaling are specifically altered during three states of smoking (smoking as usual, abstinence and consumption). Together, these studies hold out promise for integrating experimental, computational, and theoretical analyses of decision-making function together with research on addiction-related disorders.

   a. **Baker, T.E.,** Zeighami, Y., Dagher, A., Holroyd, C.B. (2020). Smoking decisions: Altered reinforcement learning signals induced by nicotine state. *Nicotine and Tobacco Research*, 22, 164-171.
   b. **Baker, T.E.,** Castellanos-Ryan, N., Schumann, G., Cattrell, A., Flor, H., Nees, F., . . . Conrod, P. and the IMAGEN Consortium. (2019). Modulation of orbitofrontal-striatal reward activity by dopaminergic functional polymorphisms contributes to a predisposition to alcohol misuse in early adolescence. *Psychological Medicine*. 18, 1-10.
   c. **Baker, T. E**., Stockwell, T., Barnes, G., and Holroyd, C. B. (2013). Constraints on Decision Making: Implications from Genetics, Personality, and Addiction. *Cognitive, Affective, and Behavioral Neuroscience*, 13, 417-436.
   d. **Baker, T.E.,** Lam, V., Ni, Lan., Uban, K., Weinberg, J. (2015). Chapter title: Of mice and wo/men: transdisciplinarity, stress and sex/gender differences in research on addictions/substance use disorders. Book title: The Alchemy of Addictions: Gender, Trauma, Transdiscplinarity. Edited by Greaves, L., Poole, N., and Boyle, E.

5. **Electrophysiological explorations of parahippocampus memory function**. Another major contribution to science concerns the existence of a generic, high-level spatial navigation system in humans. My work led to the discovery of the Topographical N170, an ERP component that was found to be highly focal over the right temporal cortex and sensitive to the spatial context of reward stimuli during navigation, occurring 5-10 ms earlier for reward-related stimuli encountered in maze locations following rightward turns compared to leftward turns. In a series of follow-up experiments, it was further demonstrated that this effect is sensitive to individual differences in spatial ability, localized to the right parahippocampus, and consistent with partial phase resetting of the ongoing EEG in the theta frequency range. For the first time, we show the phase of the theta rhythm in the human parahippocampus can be reset by salient events like reward delivery during navigation, reflecting a phase coded signal for encoding contextual information about reward location in the environment.

   a. Lin, M.H., Liran, O., Bauer, N**., Baker, T.E.** (2022). Scalp recorded theta activity is modulated by reward, direction, and speed during virtual navigation in freely moving humans. Nature: Scientific Reports, 12(1), 1-18.
   b. **Baker, T. E.,** Umemoto, A., Krawitz, A., and Holroyd, C. B. (2015). Rightward-biased hemodynamic response of the parahippocampal system during virtual navigation. Nature: Scientific Reports, 5, 1-8.
   c. **Baker, T. E.,** and Holroyd, C. B. (2013). The Topographical N170: Electrophysiological explorations of human parahippocampal cortex function. Biological Psychology, 94, 90-105.
   d. **Baker, T.E.,** Reid, A., Zhang, Y., Cole., M., Vainik, U., Lepage, C., Hunteburth, S.,Evans, A., Petrides, M., Dagher, A. (in prep) Charting the parahippocampal theta memory system across neuroimaging modalities.

Complete list of my publications is available in MyBibliography:
https://pubmed.ncbi.nlm.nih.gov/?term=Travis%20E%20Baker

Support

Active

1. Title: Using combined EEG and non-invasive brain stimulation to examine and improve reward functioning in opioid use disorder. 1R21DA049574-01A1. PD/PI: Travis E. Baker. Source of Support: NIDA. 3/1/2020 - 2/28/2022 (NCE - 2/28/2023). Total Award Amount (including Indirect Costs): $429, 834
2. Title: Candidate mechanisms for chemotherapy-induced neurocognitive deficits in pediatric solid non-CNS tumor patients. R21CA262491-02. PD/PI: Travis E. Baker. Source of Support: NIC. 07/05/2021 - 06/30/2023. Total Award Amount (including Indirect Costs): $403, 687
3. Title: Recovery of reward function in nicotine use disorder using a combination of robotics, electrophysiology, and TMS. UG3DA054787. PD/PI: Travis E. Baker. Source of Support: NIDA. 09/30/2022 - 8/31/2027. Total Award Amount (including Indirect Costs): $2,424,740.00

PENDING

1. Title: A novel therapeutic application of closed-loop neuromodulation of the brain reward system in nicotine use disorders. Project Number: 1R21DA057618-01. PD/PI: Baker, T.E. (co-PI, Headley, D.B.). Source of Support: NIDA. Project/Proposal Start and End Date: (MM/YYYY) (if available):09/2022-09/2024. Total Award Amount (including Indirect Costs): $ 423,213