Research Statement

Broadly speaking, my research focuses on individual differences in stress risk and resilience, and potential mechanisms underlying these associations. As I have entered the senior stage of my career, my emphasis has shifted to a "team science" approach focused on interdisciplinary collaborations, as well as an increased emphasis on graduate mentoring and enabling lead authorship opportunities among my doctoral students whenever possible. I have approached key research questions using a variety of methodologies, including laboratory stress and emotion induction paradigms, ecological momentary assessment (EMA), and utilization of large nationally-representative databases. My principle areas of research are outlined below, with key findings and references (publications since tenure: 42; career total: 74).

Individual Differences in Stress Regulation. Despite the centrality of psychosocial stress in predicting adverse mental and physical health outcomes, the field of stress science has been limited by imprecise definitions and assessment. An advancement in stress research has been to consider stress as a set of processes—exposure, reactivity, recovery, and restoration—in an effort to better explicate mechanisms of association to health. Prior stress research has also been limited in not adequately considering individual differences across these stress processes. My research examines the inter-relations among personality, cognitive (especially executive) functioning, and neurophysiological factors (e.g., resting psychophysiology, resting-state fMRI functional connectivity) in the context of stress regulation. With graduate students and colleagues, I have published theoretical papers and chapters outlining a framework for considering individual differences in stress risk and resilience (e.g., Williams et al. 2017; Williams et al., 2011; Williams et al., 2009), with an invited chapter forthcoming in the *APA Handbook of Health Psychology* (Williams & Carlson, 2021).

Central empirical findings have included demonstrating associations between endophenotypic individual differences relevant to stress regulation, particularly executive function and resting high frequency heart rate variability (HF-HRV; an index of parasympathetic nervous system functioning) (Williams et al., 2019). Such research has implications for understanding self-regulation, broadly, as well as informing stress assessment and intervention. After identifying aesthetic engagement—connection to art, nature, and beauty—as a potential stress resilience factor in a laboratory stress study (Williams et al., 2009), my subsequent research has sought to understand these associations with a particular focus on the putative neurophysiological marker of aesthetic engagement—proneness to aesthetic chill. We established that individuals reporting high proneness to aesthetic chill have a distinct pattern of resting state functional connectivity relevant to stress resilience (Williams et al., 2018), have unique associations to the experience of awe (Williams et al., 2021), and are characterized by stress-related growth orientation assessed with behavioral coding during stress narratives (Johnson et al., in press). An NSF grant is in preparation with a colleague in Electrical and Computer Engineering to develop a reliable method to objectively measure aesthetic chill.

Individual differences in habitual sleep duration and subjective sleep-related dysfunction. Current recommendations are that adults sleep 7-9 hours per night to achieve optimal health (Watson et al., 2015). Yet, more than 30% of U.S. adults report routinely getting less than the recommended amount of sleep (Laukhaupt et al., 2010), costing the U.S. economy an estimated $411 billion per year in lost work productivity and mortality risk (Hafner et al., 2016). My research team has determined that a critical challenge to addressing this health risk is that approximately 1 in 3 habitual short sleepers (HSS)—an estimated 23 million U.S. adults—report having no daytime dysfunction (i.e., no subjective sleepiness or impairment) (Williams et al., in press; Curtis et al., 2016). However, we have also demonstrated that there may be a disparity between subjective versus objective risk in these short sleepers. With funding from the University of Utah Neuroscience Initiative, my doctoral students and I joined with colleagues in Neurology and Neuroradiology to identify these habitual short sleepers with no reported dysfunction (HSS-NRD) in the Human Connectome Project database. We showed that during resting fMRI assessment with instructions to remain awake, HSS-NRD had brain activation patterns consistent with sleep onset, suggesting these short sleepers have difficulty maintaining alertness under conditions of low environmental stimulation (Curtis et al., 2016). We also found that self-reported HSS-NRD had objectively poorer cognitive function and reward-related impulsivity (Curtis et al., 2018), as well as neural reward processing (Curtis et al., 2019). With a national colleague experienced in use of the National Health and Nutrition Examination Surveys (NHANES) database, we demonstrated that subjective sleep sufficiency among HSS did not confer protection against disease risk—HSS-NRD had higher cardiometabolic disease risk even has they reported better self-rated health (Williams et al., in press). Our central hypothesis is that HSS-NRD are characterized by high behavioral activation (reward drive and high activity levels). These HSS are hypothesized to seek environmental stimulation to override subjective sleep need, leading to a critical disparity between subjective versus objective daytime dysfunction. In turn, a lack of

perceived risk associated with short sleep is a major impediment to sleep behavior change (Khader et al., 2021). Thus, HSS-NRD are unlikely to seek out or respond to current sleep interventions which rely on self-assessments of impairment and motivation to improve sleep. My lab has also conducted a feasibility pilot study to obtain preliminary data in support of the conceptual model (Figure 1) forming the basis for a recent R01 grant submission to NIH-NHLBI (*Habitual short sleep duration: Neurobehavioral mechanisms and cardiometabolic risk*)[1]

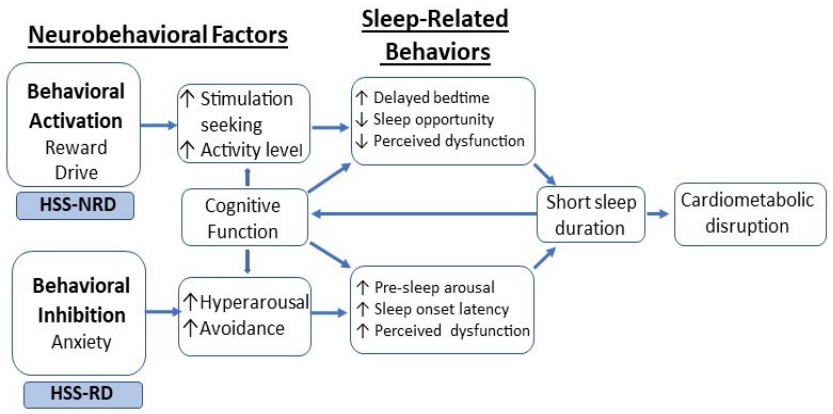

Figure 2. Conceptual model: Pathways to habitual short sleep duration. Habitual short sleepers not reporting dysfunction (HSS-NRD) are proposed to be characterized by high behavioral activation (reward drive) with concomitant high activity levels and stimulation seeking. These behavioral tendencies set the stage for bedtime delays and greater likelihood of diminished sleep opportunity leading to short sleep duration. The resultant cognitive function decrements are hypothesized to potentiate behavioral drive and further impair self-assessments of alertness and functioning in a feed-forward reciprocal cycle. This pathway is in contrast to that of habitual short sleepers reporting dysfunction (HSS-RD) via behavioral inhibition (anxiety) with concomitant hyperarousal driving delayed sleep onset.

Sleep, Stress, & Health. As part of my research on individual differences in stress regulation, a primary focus has been on stress restoration, particularly sleep. Research has focused both on individual differences that predict stress-related sleep disruption as well as the reciprocal effects of sleep on stress processes and health. Central findings have included demonstrating that recent sleep disturbance predicts laboratory stress responses (Williams et al., 2013) and that nonrestorative sleep (i.e., a lack of subjective restoration despite adequate sleep duration) is associated with EMA-assessed stress, cognitive function, and pre-sleep arousal (Tinajero et al., 2018). When the Covid-19 pandemic emerged as a significant global stressor, my graduate students and I obtained IRB approval to re-contact previous research participants to prospectively assess risk and resilience to sleep disturbance. We determined that positive reappraisal and higher positive affect were associated with resilience to stress-related sleep disturbance (Johnson et al., 2021). We also established that individuals with late chronotype (i.e., "night owls") experienced improved sleep duration during the pandemic, given alterations to morning obligations (i.e., less "social jet lag"; Carlson et al., 2021).

Self-Assessment vs. Objective Indicators of Health and Cognition. Assessment of health outcomes and psychosocial functioning relies on both self-assessed (i.e., subjective) measures as well as objective indicators. Research investigating disparities between subjective vs. objective assessment has been rare. Yet, such disparities have clear implications for self-regulation and health behavior. An overarching focus of my research has been to examine individual difference predictors of the correspondence between subjective and objective measures of sleep-related dysfunction, health (e.g., symptoms), as well as cognitive and psychosocial functioning. Recent research has focused on determining personality predictors of subjective vs. objective cognitive function (Williams et al., 2017), as well as understanding correspondence between self- and informant/spouse reports of personality (Smith & Williams, 2015). This line of research has been foundational in developing methods to examine discrepancies between self-assessments and objective indicators of functioning in the short sleeper research.

Personality, Cognition, and Aging. With clinical neuropsychology colleagues, my research on individual differences has extended to examine personality, cognitive function, and aging in older adults. This research has made contributions to our understanding of personality predictors of incipient cognitive decline (Suchy et al., 2018; Williams et al., 2013) and instrumental activities of daily living (Suchy et al., 2010), as well as explicating basic associations between personality factors and executive functioning (Williams et al., 2010).

[1] Following successful funding from the Department of Defense and two R21 submissions (positively reviewed, but not funded), a major surgery and cancer diagnosis & treatment, as well as a serious medical illness in my spouse, delayed plans to submit an R01 earlier. Given these delays, I sought to maximize the likelihood of successful funding by 1) applying for and participating in an R01 grant writing group sponsored by the Utah Center for Clinical and Translational Science; 2) assembling an outstanding team of interdisciplinary co-Investigators; and 3) working closely with NIH program officers to refine the focus to align with NHLBI funding priorities.

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*student co-author