UNIVERSITY of WASHINGTON
PSYCHIATRY & BEHAVIORAL SCIENCES
School of Medicine

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Faculty Profile

Summary of Research Program:

My lab uses rat models to investigate mechanisms contributing to excessive alcohol drinking, mechanisms mediating resilience or vulnerability to developing PTSD after traumatic stress, and interactions between stress and alcohol drinking. Our main methods include a range of behavioral models, pharmacologic and hormonal interventions, gene expression analyses within microdissected brain areas, analyses of hormonal responses, and use of selectively-bred rat strains. Most of our current studies have immediate translational utility – informing and in some cases derived from clinical studies by our collaborators.

The organization of our ongoing studies reflects the current grants that support them. The studies in the first (NIH R01, Noradrenergic Agents As Potential New Pharmacotherapies for Alcohol Drinking) all use the selectively-bred alcohol-preferring (P) rats to address: [1] optimal prazosin dosing parameters, specificity; [2] use of prazosin alone or in combination with naltrexone; [3] phases of the alcohol addiction/relapse process sensitive to prazosin; [4] gender effects; [5] responses to other adrenergic agents; [6] effects on alcohol seeking vs alcohol consumption; and [7] initial studies of mechanisms. A recent high impact finding from these studies is that combinations of drugs addressing different mechanisms mediating excessive alcohol drinking can be much more effective than either of the individual drugs (e.g., prazosin + naltrexone, prazosin + propranolol). Studies in the second (DoD, Stress and PTSD Mechanisms as Targets for Pharmacotherapy of Alcohol Abuse, Addiction and Relapse) use an outbred rat model to address: [1] whether anxiety behavior and/or acoustic startle response can predict which subjects will develop high levels of alcohol drinking when provided intermittent repeated opportunities to drink, which subjects will progress to compulsive alcohol drinking, and if prazosin will be effective in reducing this alcohol drinking once it is established; [2] whether anxiety behavior and/or startle response can predict the effectiveness of prazosin in blocking initial development of high levels of alcohol drinking; [3] whether prazosin will block stress-induced increases in alcohol drinking; [4] whether alcohol dependence increases vulnerability to developing a PTSD-like condition after experiencing a traumatic stress; [5] whether hyperexcitability and/or anxiety associated with development of a PTSD-like condition can predict how effective prazosin will be in reducing associated alcohol drinking; and [6] whether administration of prazosin at the time of traumatic stress will prevent subsequent development of a PTSD-like condition and increased alcohol drinking.