Although insomnia is generally considered as a disorder that leads to both nighttime and daytime sequela, acute insomnia can be an adaptive reaction to stress in life-threatening situations. After the stressor has been dissipated, the individual should then default back to normal sleep due to the plastic and automatic nature of normal sleep regulation. However, in patients with chronic insomnia, sleep does not normalize because of the activation of other processes that interfere with sleep regulation. Previous studies have shown that a significant proportion of cases of transient and/or milder insomnia might not develop into severe, chronic insomnia. It is of interest to explore the differential contributing factors for transient and chronic insomnia in order to understand why only some transient sleep disturbances develop into chronic problems. My talk will introduce three studies that aimed to address this issue by comparing the psychological and behavioral factors of insomnia as well as neurophysiological reactivity during sleep among good sleepers, chronic insomnia patients, and individuals vulnerable to transient insomnia. The results indicate a general hyperarousal in individuals vulnerable to stress-related transient insomnia, and a sleep-related cognitive abnormality in chronic insomnia patients. Clinical implications will also be discussed based on the findings of the studies.