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354**ALTERATIONS IN PLASMA ENDOCANNABINOIDS (eCB) IN 4-WEEK ABSTINENT ALCOHOLICS: A BIOLOGICAL MARKER FOR HEDONIC TONE?**

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Chronic alcohol abuse significantly alters the CRF and noradrenergic responses to stress. Previous preclinical research has suggested that endocannabinoids (eCBs) like anandamide (AEA) play an important role in the regulation of stress-coping behaviors. Some basic research also indicates that eCBs may be involved in alcohol seeking behavior. However, there is no data on eCB levels and responses to psychological stress in humans. Thus, the aim of the present study was to assess endocannabinoid responses to individualized emotional stress and to alcohol cues in 4-week abstinent alcoholics compared to healthy social drinkers. Eight 4-week abstinent alcoholics engaged in inpatient treatment and 10 healthy social drinkers participated in three laboratory sessions in which they were exposed to individualized stress, alcohol cue and neutral relaxing situations using guided imagery procedures, one imagery per day with randomized order of presentation. Alcohol craving, subjective anxiety, cardiovascular and plasma anandamide levels were assessed at baseline and immediately following the imagery over the course of 75 minutes. Findings indicated that baseline anandamide levels were significantly lower in AD patients compared to social drinkers ($p < .0001$). Furthermore, anandamide response to neutral relaxing situations and alcohol cue exposure were higher than the stress condition (p 's $< .001$) in social drinkers but no such response differences were evident in AD patients. These findings indicate that alcoholics show suppressed endocannabinoid levels with a lack of responsivity to hedonic cues while healthy social drinkers showed increased peripheral endocannabinoid levels in response to positive emotional and hedonic stimuli. The findings suggest that chronic alcohol abuse is associated with a dysregulated hedonic state and that pharmacological manipulation of the endocannabinoid system may provide important therapeutic targets in the treatment of alcoholism (This research was supported by NIH grants R01-AA113892 and K02-DA17232).