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### **CENTRAL VASOPRESSIN V1A RECEPTORS ARE INVOLVED IN PRESSOR RESPONSE TO EVOKE FOOD SEEKING BEHAVIOR IN FASTING FREE-MOVING MICE**

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We previously reported that cerebral activation at the onset of voluntary locomotion suppressed baroreflex control of heart rate (HR) and increased arterial pressure (AP) via vasopressin V1a receptors in the brain. However, physiological role of these responses are unclear. Here, we examined whether these responses were associated with motivated behavior (e.g., food seeking) by using free-moving wild-type (WT, n=10), V1a receptor knockout (KO, n=9), and wild-type mice locally infused with a V1a receptor antagonist into the nucleus tractus solitarii (BLK, n=10).

For 3 consecutive days, mice were fed ad libitum (Fed) on the 1<sup>st</sup> day, food deprived (FD) on the 2<sup>nd</sup> day, and refed (RF) on the 3<sup>rd</sup> day under dark/light cycle (7pm/7am). Food was removed on day2 and restored on day3 at 6pm. Throughout the protocol, cerebral activity was determined from the power density ratio of  $\theta$  to  $\delta$  wave band ( $\theta/\delta$ ) on electroencephalogram every 4sec. Baroreflex control of HR was determined from cross-correlation function ( $R(t)$ ) between changes in HR and AP every 4sec. Their behavior was recorded with a CCD camera from 7pm-9pm.

We found that in WT, the linkage between cerebral activation and suppression of baroreflex control, determined from cross-correlation function between  $\theta/\delta$  and  $R(t)$ , at night was enhanced after FD ( $P=0.013$ ), accompanied by increased food-seeking behavior, twofold higher than those of Fed ( $P=0.00080$ ); thereafter, these variables returned to Fed level after RF ( $P>0.54$ ). However, none of these enhancements occurred in V1a KO or BLK ( $P>0.11$ ) despite maintained  $\theta/\delta$  level.

Thus, central V1a receptors are involved in motivated behavior through linkage between cerebral activation and suppression of baroreflex control.

#### **Keywords;**

Motivated behavior; Central pressor response; Vasopressin V1a receptor