Introduction

The prevalence of overweight in general population is considerably higher in women (36%) than in men (15%) (1). Overweight exacerbates many aspects of women's health by enhancing risk for heart disease, diabetes, breast cancer, and infertility. One reason for the gender difference in obesity may be that fluctuations in reproductive hormone concentrations throughout women's lives uniquely predispose them to excess weight gain (2). Phase related changes in food intake were evident in women, with lowest energy intake during follicular relative to the luteal phase (3). Changes in body composition at menopause may be caused by the decrease in circulating estrogen, and for fat distribution shifts, the relative increase in the androgen-estrogen ratio (4). Androgen-estrogen ratio is a critical physiological signal for feeding related brain areas in female mammals (5).

Feeding behavior and body weight regulation are sexually dimorphic in the rat in that male and female rats differ in terms of their growth rate (6), response to different diets (7), ability to show metabolic adjustments in feeding behavior (8), diurnal feeding patterns (6) and taste preference (7). Similarly, female rats also show phase related changes in food intake (FI), water intake (WI) and body weight (BW) (9-12). Interestingly, female rats show a greater inhibition (48%) of food intake in response to emotional stress than male rats (22%) and this gender difference in the inhibition of food intake in emotional states has been attributed to estrogen. The inhibition of food intake by emotional stress in female rats was more prominent during proestrus than the other phases of estrous cycle. (13). It has been well established that there is decreased plasma estrogen to a negligible level in ovariectomized female rats (14), which results in increase in FI, WI and BW,
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while estrogen substitution reverses either centrally (15) or peripherally (16), suggesting that lack of estrogen causes abnormal increase in ingestive behaviors and body weight in female rats similar to postmenopausal women. Therefore, ovariectomy in rats provides a useful model (17) to study the mechanism by which estrogen deficiency causes obesity in women.

The neurotransmitters or neuromodulators that mediate estrogenic effects on FI, WI and BW were largely obscure and contradictory. It has been reported that many peptides like leptin (18), neuropeptide Y (NPY) (17), cholecystokinin (CCK) (19) and glucagon (20) mediate FI and BW induced by estradiol in ovariectomized (OVX) rats and angiotensin-II (21) is the proposed peptide hormone in the estrogenic modulation of WI in ovariectomized rats.

On the other hand, studies have shown that the direct actions of estradiol on central dopaminergic system may be critical to control the FI and BW (3). Previous studies have demonstrated that injection of dopamine into hypothalamus decreases feeding (22,23), which is mainly mediated via dopamine D2 receptors (24). The evidence for possible interaction between estradiol and dopamine comes from animal models in which sulpiride (dopamine D2 receptor antagonist) induced body weight gain and obesity in female rats, was totally prevented by estradiol (25) and bromocriptine (26).

Based on the literature presented above, we have investigated the role for dopamine in mediating ovariectomy and estradiol induced effects on feeding behaviors and body weight. However, to the best of our knowledge, there are no reports available to suggest the possible interactions between estrogen and dopamine in the control of ingestive behaviors and body weight in ventromedial hypothalamus (VMH), nucleus septal lateralis (NSL) and
basolateral amygdala (BLA) in ovariectomized rats, also there is no literature on the effect of dopamine in lateral septum on FI and BW. Therefore, in the present study we have attempted to understand whether estradiol and dopamine interacting each other in these nuclei in the control of FI, WI and BW in ovariectomized rats.