The region specific decrease in the magnitude of FI, WI and BW following central injections of EB may be due to variance in the receptor expression in respective nuclei. The changes in FI and WI, following ovariectomy and EB treatment were transient and this may be an attempt to overcome the persistent changes in the BW. Subsequently, intake returns to a level at which the change in the BW stabilizes. These findings suggest that there should be additional mechanisms, which regulate the BW other than FI and WI. Although, subcutaneous injections of EB caused significant inhibition of FI, WI and BW, it was not as great as when these nuclei were intact. It is likely that in the absence of estrogen receptors in these nuclei, other areas of the brain can monitor estrogen effects and suppress these measures or some other suppressant mechanisms remained functional.

The findings that ovariectomized rats treated with bromocriptine displayed inhibitory effects on FI and BW similar to EB, are novel and interesting given the paucity of reports available with respect to actions of BC on ovariectomy induced changes in feeding behaviors and body weight. We suggest that ovariectomy induced increase in FI and BW may be due to alterations in the dopamine receptor transmission in the brain, particularly, the D2 receptors. In addition, prevention of sulpiride induced increase in FI and BW by EB was marked in VMH and NSL compared to BLA and this finding raises the possibility of D2 receptors involvement in the control of these measures. Our present study supports the view that down regulation of D2 receptors may represent a common pathogenic mechanism contributing to hyperphagia and obesity.

If low dopamine D2 receptor expression is crucial for the development of hyperphagia and obesity, then by analogy, it is possible that ovariectomy induced
changes in hyperphagia and body weight gain in the present study could also be due to down regulation of D2 receptors in VMH, NSL and BLA. D1 receptor down regulation would expect to bring alterations in fluid balance. Further, we assume that both EB and bromocriptine might have stabilized ovariectomy induced increase in meal size and corresponding decrease in meal number by improving dopaminergic neurotransmission in these nuclei. In addition, both EB and bromocriptine may also have enhanced physical activity and increased D2 receptors density in these brain regions and consequently resulted in the inhibition of FI and BW in ovariectomized rats.

Though, our study outlines the possibility for dopamine in mediating effects of both ovariectomy and estradiol replacement on FI, WI and BW, obviously many other neuromodulators in addition to dopamine, such as norepinephrine, serotonin, leptin, neuropeptide-Y (NPY), cholecystokinin (CCK) etc and also hormones that regulate ingestive behaviors and body weight, may be involved. The interactive and integrative activities of these neurotransmitters and neuromodulators that participate in the control of these behaviors merit research to have a more comprehensive view of the complex and intricate mechanisms associated with control of ingestive behaviors and body weight. However, given the paucity of information known about the functional interaction between estradiol and dopamine in the regulation of appetitive behaviors and body weight in ovariectomized rats, current study is of great interest and may hold promise for understanding the mechanisms involved in postmenopausal obesity.
Perspectives

Our study outlines some mechanisms that contribute to hyperphagia and obesity in ovariectomized rats. Direct actions of estradiol on central dopaminergic system seem to be critical to control the FI and BW in female rats. We speculate that altered dopamine receptor expression particularly, D2 receptors in VMH, NSL and BLA may account for pathophysiology of hyperphagia and obesity in ovariectomized rats. Further studies to evaluate the interaction between estradiol and dopamine receptors with particular attention to D2 receptors might be beneficial to understand the mechanisms involved in the pathophysiology of ovariectomy induced hyperphagia and body weight gain. Recording of physical activity, MZ and MN may give better perspective as to what mechanisms contributed for the development of hyperphagia and obesity in ovariectomized rats. A perspective direction of research may include direct assessment of neurotransmitters, neuromodulators and diurnal rhythm of hormones to understand functional interaction between estradiol and dopamine in the control of ingestive behaviors in ovariectomized rats. It merits further investigation in animal models whether strategies to enhance dopaminergic function will ameliorate ovariectomy induced hyperphagia and obesity. We are aware of the fact that long-term treatment of estrogen and drugs that improve dopaminergic function will have certain side effects. Our speculation is that combined treatment of estradiol and bromocriptine in low doses might have therapeutic implications for prevention and early intervention of postmenopausal obesity, and host of related disorders.