ABSTRACT
Our data indicates that IP3R3 is the primary type of taste receptor expressed in taste cells and plays a crucial role in transducing bitter taste.

INTRODUCTION
In the past decade several studies have suggested that the expression of Type III IP3 receptor and its downstream signaling molecules might play a role in the pathogenesis of obesity and other metabolic disorders. The purpose of the present study was to assess whether Type III IP3 receptor and the signaling molecules it co-expresses could be induced in an in vitro model of obesity, and to examine whether the presence of these molecules in the diet could affect the metabolic profile.

Materials and Methods:

Bitter taste transduction is a class of taste-related signaling proteins that respond to the chemical stimulus of a single taste. Although the receptor subunits that express the receptor subunits of bitter taste transduction have been identified, few studies have examined the expression of the receptor subunits of bitter Taste receptor cells are specialized epithelial cells that are organized into distinct endorgans called taste buds. Typical tastebuds contain 50-100 polarized taste cells, which extend from the basal lamina to the taste pore, where apical microvilli protrude into the oral cavity. The basolateral membrane also forms chemical synagogueses with primary gustatory neurons (Fig. 1A). In mammals, lingual taste Buds are located in connective tissue structures called fungiform and primarily involving complexes of complex The production of bitter and sweet taste transduction is facilitated by inositol 1,4,5-trisphosphate (IP3), an essential second messenger. Taste receptor activation in both pathways involves a G protein-coupled cascade that leads to the binding of soluble messenger IP3 and DAG, respectively. IP3, however, binds to receptors on calcium store membranes and activates downstream effectors such as Dagaglio, with little known information about its role in sweet flavor transducduction. Akabas et al. Other research indicated that bitter transduction was not mediated by an increase in IP3, but rather by a decrease in cAMP. In 1992, -gustducin, which is closely related to rod and cone transducines, was discovered to activate taste receptors in subsets of taste cells. Evidence for its involvement in bitter taste was obtained from knockout studies, in which lysine stimulated mice with reduced sensitivity to bitter compounds. Now it appears that individual bitter receptor(s) can regulate both CI and C pathways. While it is obvious that IP3 binds to receptors on intracellular Ca2+ stores, the exact identity of IP33 receptor in taste cells remains unknown. There are at least 3 known isotypes of this receptor, encoded by different genes. Each protein product is approximately 300 Kda. Four subunits assemble to form a functional channel. The N terminus of each subdomain houses the IP3/Mouse binding domain, while the C terminuse anchors the PETT domain and forms the Ca 2+ pore region. By using immunocytochemical techniques, we have identified the IP3R isoform as expressed in taste cells and examined the expression patterns of IP3, receptors, relative to other proteins essential for taste transduction. We conclude that the Type III IP3/IDP2 is the dominant isorepresented in rodent taste cell, and that it is primarily located in the same subset of taste-derived cells as other known signaling components of bitter transducation. A preliminary abstract of this work has been published.

CONCLUSION

Remarkable conclusions The main finding in this study is that IP3R3 is now the dominant isoform of the IP3/Ca2+ receptor in taste cells, and its involvement in bitter and sweet taste transduction seems to be a secondary factor.