LEGENDS FOR SUPPLEMENTAL TABLES AND FIGURES

Supplemental Table 1. This table provides a list of the: (A) Primer sequences for real-time quantitative PCR; (B) Blocking serum solutions and antibodies used for single- and double-labeling immunofluorescence reactions ([a]Sigma, St. Louis, MO; [b]Jackson Immunolaboratory, West Grove, PA; [c]Santa Cruz, Santa Cruz, CA; [d]Novus Biol, Littleton, CO; [e]Chemicon, Temecula, CA; [f]Abcam, Cambridge, MA); and (C) Catalog and lot numbers for the antibodies.

Supplemental Table 2. Prenatal HFD produced behavioral, physiological and neurochemical changes (mean ± SEM) in adult female offspring (70 days of age) that are similar to those observed in adult male offspring (see Fig. 1). This is indicated by a significant increase (*, $p<0.05$), in both the HFD and HFD-BD compared to BD groups (n=6-8/diet group), in measures of body weight, daily caloric intake and preference (% of total diet) for fat vs carbohydrate; of triglycerides, non-esterified fat acids (NEFA), and galanin mRNA and peptide levels in the PVN; and of body fat pad weights, leptin and insulin levels, with no change in glucose, CORT or NPY levels in the ARC. Data are mean ± SEM.

Supplemental Table 3. Prenatal HFD produced significant effects (*, $p<0.05$) in both the HFD and HFD-BD offspring at P8 (n=4-6/diet group) that were similar to those described in the P15 offspring (see Fig. 2). Compared to the BD group, these effects were an increase in GAL peptide levels in the PVN, ORX peptide immunoreactivity in the PFLH, and circulating levels of triglycerides. In contrast, the HFD and HFD-BD groups at P8 showed no change in measures of body weight, leptin, glucose, insulin and corticosterone, with an increase in NEFA seen only in the HFD offspring. Data are mean ± SEM.

Supplemental Table 4. Prenatal HFD as compared to the BD had little or no effect on serum levels of glucose, leptin, insulin and corticosterone in dams during pregnancy (E14-E18) and their offspring at birth (P0) and also on the body weight and daily food intake of the dams (n=4-8/age/diet group). Data are mean ± SEM.

Supplementa1 Figure 1. Prenatal HFD had no effect on cell proliferation in the ARC or hippocampus compared to BD (n=4-6/diet group). This was demonstrated by a lack of change in $BrdU^+$ cell density in P0 offspring of dams injected with BrdU from E13-E14 and E14-E15. BrdU injections from E11-E13 yielded few labeled cells in these areas. Data are mean ± SEM.

Supplemental Figure 2. Prenatal HFD stimulated cell proliferation in the PVN and PFLH compared to BD (n=4-6/diet group). This was demonstrated by a significant increase in $BrdU^+$ cell density (*, $p<0.05$) in the HFD or HFD-BD vs BD offspring at P8 or P15 of dams given i.p. injections of BrdU from E11-E13 and exposed to the diet on embryonic day 6 (E6) or day 9 (E9). Data are mean ± SEM.

Supplementa1 Figure 3. Prenatal HFD had no effect on gliogenesis in the PVN and PFLH of HFD and HFD-BD postnatal offspring compared to BD offspring (n=4-6/diet group). This was demonstrated by no change or a slight reduction of single-labeling immunofluorescence for GFAP at P15 and P25, a marker for astrocytes, and for GalC at P15, a marker for oligodendrocites. Data are mean ± SEM.

Supplementa1 Figure 4. Embryos (E14) of HFD vs BD dams exhibited no change in gliogenesis in the ventral neuroepithelial lobe (VL) of the hypothalamic third ventricle (3v) and surrounding hypothalamic area (HYP) (n=4-6/diet group). (A) This is demonstrated by no difference between the groups (mean ± SEM) in single-labeling immunofluorescence in the VL and HYP for glial-restricted precursors (nestin) or radial glia and tanycites (vimentin); and (B) This is illustrated by immunofluorescence photomicrographs of these glial markers (scale bar: 100μm).