We thank the reviewers and editors for their consideration of this work. Specific points addressed are noted below, with our comments **in bold** and verbatim additions to the text in **bold red text**.

1. The article acknowledges an incomplete elucidation of the precise mechanistic underpinnings that establish a linkage between gestational early time-restricted feeding (eTRF) and the onset of glucose intolerance.

**We thank the reviewer for this acknowledgement**

1. Multifaceted Nature of Variables: The study encompasses a multitude of variables, encompassing maternal diet, progeny diet, gender, and the temporal deployment of interventions. While this intricate framework contributes to a more comprehensive comprehension of the ramifications, it concurrently introduces the potential for unaccounted confounding elements and interactions within the study's design.

**We have added a note on lines 408-410 of the revised manuscript noting this potential limitation:**  
  
**Second, we assessed the effect of a dietary insult in young adulthood by switching all animals to HFHS. As such, disentangling the effect of HFHS diet from that of aging and gestational eTRF is not possible in this model.**

**We also added this statement in the discussion on lines 419-421 describing potential unaccounted for confounding elements:**

**Furthermore, while dams were manipulated simultaneously, we cannot rule out that our eTRF treatment induced other confounding differences that we have not accounted for, including potential maternal stress or chronodisruption.**

1. Restricted Horizon of Longitudinal Effects: The study predominantly concentrates on near-term and young adult outcomes, thereby potentially omitting protracted consequences of gestational eTRF. Manifestations of certain health conditions, particularly metabolic disorders, could emerge in later stages of life. It is prudent for the article to acknowledge the constraint imposed by short-term observation and advocate for protracted investigations to ascertain enduring implications.

**We agree with this statement and the need for broader studies across the lifespan, and have added this caveat and suggestion to lines 423-426 of the discussion:**

**It is also worth noting that several metabolic diseases are highly linked to age, and while our study ended at approximately six months of age, mice can live much longer under laboratory conditions typically 26-30 months. As metabolic, physical, cognitive, and other phenotypes that do not appear until towards the end of the mouse’s lifespan were not detectable, and we look forward to future studies on geriatric mice treated *in utero* with eTRF.**

1. Conceivable Researcher Bias in Data Analysis: Given the intricate nature of the statistical analysis carried out in the study, encompassing recurrent measures and interaction variables, there exists the potential for inherent researcher bias in the selection of specific statistical methodologies. Such bias could conceivably influence the interpretation of results and conclusions derived from the analysis.

**We agree that this is a potential limitation, and so we put in several safeguards in our data collection and data analyses that we now describe in the revised methods section on lines 204-206:**

**To minimize potential bias, animals were identified by ear tags and as such the researchers generally did not know which group mice were in during the experiments. Furthermore, the analysis plan was chosen prior to the start of experiments, and unchanged upon data analysis.**

**We also added this note to the discussion section on lines 435-437**

**To minimize researcher bias, we remained blind to offspring maternal exposure until the time of data analysis.**