Diet-induced obesity in animal models: points to consider and influence on metabolic markers

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Abstract (600 Word Limit):

Overweight and obesity are a worldwide public health problem. Obesity prevalence has increased considerably, which indicates the need for more studies to better understand these diseases and related complications. Diet induced-obesity (DIO) animal models can reproduce human overweight and obesity, and there are many protocols used to lead to excess fat deposition. So, the purpose of this review was to identify the key points for the induction of obesity through diet, as well as identifying which are the necessary endpoints to be achieved when inducing fat gain. For this, we reviewed the literature in the last 6 years, looking for original articles that aimed to induce obesity through the diet. All articles evaluated should have a control group, in order to verify the results found, and had worked with Sprague–Dawley and Wistar rats, or with C57BL-/-6 mice strain. Articles that induced obesity by other methods, such as genetic manipulation, surgery, or drugs were excluded, since our main objective was to identify key points for the induction of obesity through diet. Articles in humans, in cell culture, in non-rodent animals, as well as review articles, articles that did not have obesity induction and book chapters were also excluded. Body weight and fat gain, as well as determinants related to inflammation, hormonal concentration, blood glycemia, lipid profile, and liver health, must be evaluated together to better determination of the development of obesity. In addition, to select the best model in each circumstance, it should be considered that each breed and sex respond differently to diet-induced obesity. The composition of the diet and calorie overconsumption are also relevant to the development of obesity. Finally, it is important that a non-obese control group is included in the experimental design.

Importance of Research (200 Word Limit):

Studies that used non-commercial diets, such as cafeteria diets, were excluded, since the nutritional composition varied widely compared to diets produced from standardized ingredients and commercial diets. Processed foods can contain food additives and be low in vitamins and minerals, which can influence the composition of the intestinal microbiota and, consequently, the occurrence of obesity and other metabolic changes. So, in these cases, it is difficult to determine whether a metabolic outcome is only due to the high content of lipids or whether the high amount of food additives or low content of micronutrientes may influence it. In addition, diets produced from food may contain food additives, which make it difficult to assess the real effect of nutrients on the development of obesity High-fat diets are commonly used to induce obesity in animals since they generate adverse metabolic effects, meaning that diet is one of the major contributors to the obesity epidemic. All 35 studies evaluated used a high-fat diet to induce

obesity; however, the amount of calories from lipids ranged from 41 to 60% (Table 2). Despite looking like a wide margin, according to Research Diets, diet induced-obesity (DIO) animal models usually provides between 45 to 60% of calories from fats; therefore, all selected studies follow this recommendation. Nine studies did not provide the composition of the macronutrients directly, which made it difficult to calculate the amount of calories from fat. Consumption of diets rich in fat can result in the development of human-like obesity, since it increases body adiposity and leptin, and stimulates the development of hypertension and glucose intolerance. Matias et al. observed that offering a diet rich in sugar did not lead to the development of metabolic changes that characterize obesity. On the other hand, offering a diet with an excessive amount of fat leads to an increase in the adiposity index and visceral and body fat gain in comparison with sugar or control diets .In addition, some studies highlighted that in their high-fat diets.

Biography (150-200 Word Limit):

Mariana de Moura e Dias is a second-year doctoral student in Environmental Health Sciences at Yale University where he has gained experience from his teaching fellowship roles in both the Introductory Biostatistics and Introductory Toxicology courses. His research interests include understanding how cells of the central nervous system respond to both endogenous and exogenous stressors. His interest in climate change grew from a belief that climate change is the most consequential problem facing the world in the 21st century. Prior to his doctoral studies, Brian obtained a BS in Biochemistry from the University of Massachusetts Amherst. Ocular development is composed of a carefully orchestrated set of events that are easily perturbed, which results in a syndrome of diseases termed MAC (microphthalmia, exophthalmia and coloboma). For decades, previous research has largely been focused on elucidating the role of transcription factors in directing eye development. However, it is increasingly realized that oxidative stress also plays an important role in the eye development process. Despite these realizations, much remains to be known about the mechanisms by which oxidative stress influences eye development.

Information of Institute/ University/ Laboratory :(200 Word Limit)

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Other clients have included UPMC, University of Washington, Yale University, Columbia University, Duke University, Oklahoma University, University of Nebraska, Henry M. Jackson Foundation, Robert Wood Johnson Foundation, and RAND Corporation.

1. Benfey PN, Ren L, Chua NH. Tissue-specific expression from CaMV 35S enhancer subdomains in early stages of plant development. The EMBO journal. 1990 Jun;9(6):1677-84.

2. Horvath AJ, Forsyth SL, Coughlin PB. Expression patterns of murine antichymotrypsin-like genes reflect evolutionary divergence at the Serpina3 locus. Journal of molecular evolution. 2004 Oct 1;59(4):488-97.

3. Ludwig Y, Zhang Y, Hochholdinger F. The maize (Zea mays L.) AUXIN/INDOLE-3-ACETIC ACID gene family: phylogeny, synteny, and unique root-type and tissue-specific expression patterns during development PloS one. 2013 Nov 1;8(11):e78859.

4. Segerman B, Jansson S, Karlsson J. Characterization of genes with tissue-specific differential expression patterns in Populus. Tree Genetics & Genomes. 2007 Oct 1;3(4):351-62.

5. Campbell PD, Marlow FL. Temporal and tissue specific gene expression patterns of the zebrafish kinesin-1 heavy chain family, kif5s, during development. Gene expression patterns. 2013 Oct 1;13(7):271-9.

6. Li IM, Liu K, Neal A, Clegg PD, De Val S, Bou-Gharios G. Differential tissue specific, temporal and spatial expression patterns of the Aggrecan gene is modulated by independent enhancer elements. Scientific reports. 2018 Jan 17;8(1):1-2.

7. Ong CT, Corces VG. Enhancer function: new insights into the regulation of tissue-specific gene expression. Nature Reviews Genetics. 2011 Apr;12(4):283-93.

8. Reed NA, Castellini MA, Ma H, Shearer TR, Duncan MK. Protein expression patterns for ubiquitous and tissue specific calpains in the developing mouse lens. Experimental eye research. 2003 Apr 1;76(4):433-43.

9. Qian J, Jiang Z, Li M, Heaphy P, Liu YH, Shackleford GM. Mouse Wnt9b transforming activity, tissue-specific expression, and evolution. Genomics. 2003 Jan 1;81(1):34-46.

10. Buggs RJ, Elliott NM, Zhang L, Koh J, Viccini LF, Soltis DE, Soltis PS. Tissue-specific silencing of homoeologs in natural populations of the recent allopolyploid Tragopogon mirus. New Phytologist. 2010 Apr;186(1):175-83.

11. Wong QW, Li J, Ng SR, Lim SG, Yang H, Vardy LA. RPL39L is an example of a recently evolved ribosomal protein paralog that shows highly specific tissue expression patterns and is upregulated in ESCs and HCC tumors. RNA biology. 2014 Jan 1;11(1):33-41.

12. Guo Y, Liu J, Zhang J, Liu S, Du J. Selective modes determine evolutionary rates, gene compactness and expression patterns in Brassica. The Plant Journal. 2017 Jul;91(1):34-44.

13. Yang R, Wang X. Organ evolution in angiosperms driven by correlated divergences of gene sequences and expression patterns. The Plant Cell. 2013 Jan;25(1):71-82.

14.Bayer E, Thomas C, Maule A. Symplastic domains in the Arabidopsis shoot apical meristem correlate with PDLP1 expression patterns. Plant signaling & behavior.