Over the past few years there have been major changes in the way scientists view many diseases. There has been a shift to a focus of the role of chronic inflammation and its effects on those significant worldwide diseases. Inflammation is generally beneficial for the body because it brings the immune system’s attention to the area of the body at risk of infection from any pathogens. In some cases however, the inflammation does not stop, and this may be caused by changes in the normal bacteria that are usually in the body.

The 100 trillion bacteria thriving in a normal human gut are not just a stagnant force; they outnumber the body’s own cells and aid in the digestion of foods, the protection of the body, the secretion of vitamins and hormones, and the basic function of the intestinal tract. Therefore, when these bacteria change due to a modified diet high in fats, they can become harmful to the body and cause inflammation. For example, the bacteria eventually escape the intestine and they can elicit chronic systemic inflammation in different areas of the body, with the help of lipo-poly-saccharide (LPS), and cause a multitude of inflammatory diseases, such as Type 2 Diabetes, Alzheimer’s Disease, Crohn’s Disease, and Rheumatoid Arthritis.

In our last three weeks of research, we aimed to better understand how the bacteria changes in the normal bacteria that are usually in the body. Therefore, when these bacteria change due to a modified diet high in fats, they can become harmful to the body and cause inflammation. For example, the bacteria eventually escape the intestine and they can elicit chronic systemic inflammation in different areas of the body, with the help of lipo-poly-saccharide (LPS), and cause a multitude of inflammatory diseases, such as Type 2 Diabetes, Alzheimer’s Disease, Crohn’s Disease, and Rheumatoid Arthritis.

The pictures above are sections of the small intestine and colon from different mice fed with either a high-fat diet (HFD) or normal chow (NC). Each section is cut seven microns thick using a cryostat and stained with GFP and DAPI. The GFP highlights CX3CR1GFP+ cells in a green color. CX3CR1GFP+ cells are also known as macrophages. Macrophages are white blood cells involved in the innate immune response. The DAPI highlights the nucleus in all cells in a blue color.

These graphs depict the number of macrophages that we counted in each villus from the distal small intestine and colon of high fat diet (HFD) and normal chow (NC) mice. Each point on the graph represents the number of macrophages that we identified in one villus of one type of mouse. A general trend throughout all of the graphs is that normal chow mice tend to have more macrophages per villus than high-fat diet mice do, in the small intestine as well as the colon. This data could mean that mice fed with normal food are more inflamed than mice fed with fatty foods, or that there is a defect with the immune system in high-fat diet mice.

The data that we generated regarding macrophage and eosinophil counts in both the distal small intestine and colon show a reduction in immune activity in mice fed with a high-fat diet compared to the ones fed with normal chow. Unlike our initial perception that the gut would be more inflamed in high-fat diet mice due to the inflammation in the rest of the body, there were fewer macrophages in high-fat diet mice than in normal chow mice. These results show that there is a diminished immune response in mice fed with fatty foods, which could also explain how bacteria from the gut are able to escape the intestine and infect other parts of the body without being detected, causing chronic systemic inflammation. The gut microbiota that is corrupted by the fat and cholesterol could escape detection from the macrophages and eosinophils because the immune system’s cells could be engulfing the extra fat causing them to be unable to function properly.

The experiments have been consistent with information from several studies that suggest that high fat diets can influence the number of macrophages in the gut. The research of Stein, Conova, and Ackerman, among others, support our findings that high fat diets can lead to a decrease in immune response, which could be due to changes in the bacteria present in the gut. However, further research is needed to fully understand the mechanisms behind this phenomenon.

This data could mean that mice fed with normal food are more inflamed than mice fed with fatty foods, or that there is a defect with the immune system in high-fat diet mice.

**References**