

## **Macrophages as a therapeutic target for Type 2 Diabetes.**

Easy access to rich food and an increased sedentary lifestyle has tilted the energy balance of westernized countries towards a high-energy intake but a low-calorie consumption. This has dramatically increased the prevalence of obesity worldwide, even in developing countries. The stigma of obesity not fitting the criteria of beauty has unfortunately depreciated the seriousness of this condition. Indeed, obesity is a high-risk factor for many diseases including insulin resistance and Type 2 Diabetes. In healthy people insulin is produced by the pancreas to lower the amount of glucose in the blood after a meal. In patients with Type 2 Diabetes, the insulin production is impaired as well as its action (the body is resistant to insulin) leading to high blood glucose levels. Our objective is to understand the mechanisms whereby obese people develop Type 2 Diabetes to facilitate the development of therapeutic strategies.

Macrophages are cells of the immune system that are usually known to defend the body against infection. However, macrophages have been recently proposed to contribute to the development of Type 2 Diabetes in obesity. Although their main role was described as inducing inflammation leading to resistance to insulin, we have discovered that macrophages in metabolic tissues could regulate insulin sensitivity independently of their inflammatory status. Using cutting edge technologies, we found that multiple populations of macrophages are present in metabolic tissues such as liver and adipose tissues. These distinct populations of macrophages have both beneficial and detrimental effect on insulin sensitivity. We are now deciphering the particular factors that drive the ability of these macrophage populations to induce insulin resistance. We aim to better our understanding of the role of immune cells in the development of Type 2 Diabetes to facilitate the generation of efficient therapies.

## **References**

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