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Exogenous nucleosome binding molecules: A potential new class of therapeutic drugs

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Chromatin is as an extraordinary example of molecular recognition that defines gene expression and genome integrity. Although the essential facts about the nucleosome were already revealed 17 years ago, new insights into its atomic structure and molecular mechanisms are still emerging. In this talk, I will feature the nucleosome surface as a drug target to control chromatin dynamics and phenotypic changes. I will cover the key aspects of chromatin architecture upon binding of protein and exogenous molecules (exogenous Nucleosome Binding Molecules-eNBMs) to the nucleosome. Moreover, I will discuss the impact and development of eNBMs, presenting some of our results in silico, *in vitro* and in cell-based assays. The particular scientific interest of my group is the basic mechanism of how the structure of chromatin regulates gene transcription. Hence, I will feature the nucleosome surface as a drug target to control chromatindynamics and consequently, gene expression and genome maintenance. Mainly, we try to understand the impact of Nucleosome Binding Molecules on chromatin architecture and in this conference, I will cover the key aspects of chromatin architecture upon binding of exogenous molecules (exogenous Nucleosome Binding Molecules-eNBMs) to the nucleosome. Moreover, I will discuss the impact and development of eNBMs, presenting some of our results in silico, *in vitro* and in cell-based assays. Certainly the Gordon Conference focused on Chromatin Structure and Function will be a unique opportunity to discuss our recent findings and establish future collaborations with the greatest scientists in the chromatin field. More importantly, we expect to learn a big deal about the future directions in the chromatin field.

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Fenofibrate protection against complications associated with obesity and type-2 diabetes mellitus

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The aim of the current study is to investigate the effect of fenofibrate alone and in combination with pioglitazone on serum sirtuin 1 and fetuin A of obese patients with Type 2 Diabetes Mellitus (T2DM). Intervention effect on inflammatory parameters was assessed before and after treatment. The study was conducted on 60 postmenopausal females of whom, only 44 patients completed the study. They were distributed as follows; obese patients without T2DM (n=15) who administered fenofibrate (160mg/day) once for 8 weeks, obese patients with T2DM (n=15) who administered fenofibrate (160mg/day) once for 8 weeks, obese patients with T2DM (n=14) who administered fenofibrate (160mg/day) and pioglitazone (15mg/day) combination once for 8 weeks. We measured fasting plasma glucose, glycated hemoglobin (HbA1c), serum lipids. Inflammatory markers (highly sensitive C-reactive protein "hs-CRP", interleukin-6 "IL-6", fetuin A, and sirtuin 1) of patients were measured in serum using enzyme-linked immunoassay (ELISA) kits. Sirtuin 1 levels in obese patients with T2DM were significantly lower than its levels in obese patients while fetuin A levels were significantly higher ($P<0.001$). Fenofibrate, alone and in combination with pioglitazone, significantly decreased triacylglycerol, hs-CRP, IL-6, fetuin A and increased sirtuin 1 levels ($P<0.001$) which suggests that it can be used to delay the complications of obesity and T2DM. There is a strong correlation between fetuin A, sirtuin 1, IL-6 and hs-CRP levels suggesting a shared common pathway. Fenofibrate was shown to increase serum sirtuin 1 and decrease serum fetuin A levels in obese patients.

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