PPAR Research

Hindawi

Special Issue on

PPARs and Their Coactivators in Obesity and Metabolic Disease

CALL FOR PAPERS

Peroxisome proliferator-activated receptors (PPARs) are a subfamily of ligand-inducible nuclear hormone receptors including PPARα (NR1C1), PPARβ/δ (NR1C2), and PPARγ (NR1C3), which have distinct tissue distribution and specific functions in response to their agonists. PPARs have been recognized as master regulators of a number of physiological processes in multiple organs and cell types, participating in cellular differentiation, development, carbohydrate and lipid metabolism, energy homeostasis, tumorigenesis, and inflammation. Given that PPARs are known to play central functions in metabolic homeostasis, highly effective PPAR agonists have been developed and are widely used for the treatment of chronic metabolic diseases.

Upon specific ligand-binding, PPARs heterodimerize with the retinoid X receptor (RXR) and bind to the peroxisome proliferator responsive element (PPRE) on target genes. Importantly, the transcriptional activation of specific target genes set in motion by liganded PPARs is also determined by a number of coactivators that partner with PPARs in a tissue- and cell type-specific manner. Three major categories of PPAR coactivators have been identified: (1) those with chromatin remodeling effects, such as those carrying histone acetyltransferase (HAT) or methyltransferase activity (i.e., members of the p160/SRC family); (2) anchoring coactivators, which serve as linkers between cofactor complexes and the basal transcriptional machinery (i.e., members of the mediator complex); (3) coactivators without intrinsic enzymatic activity which function by providing a scaffold to aggregate large transcriptional complexes on tissue specific target genes: these cofactors include PGC1 family members among others. These coactivators have also been shown to be recruited in response to ligand/agonist activation to a number of nuclear receptors and transcriptional factors in addition to PPARs.

As one of the most serious public health problems of the 21st century, obesity is mainly caused by the combination of excessive caloric intake, sedentary lifestyle, and genetic susceptibility. Obesity is the leading risk factor for many physical disorders commonly known as the metabolic disease/syndrome which includes but is not limited to type 2 diabetes, hypertension, hyperlipidemia, cardiovascular diseases, hepatic steatosis, and certain types of cancer ultimately reducing life expectancy. Thus, understanding the molecular basis of PPARs and coactivator functions in controlling the expression of selective gene programs may be helpful for the development of possible strategies to prevent/treat obesity and metabolic comorbidities.

We invite investigators to submit original research articles and review articles that aim to increase our knowledge on the molecular basis of the effects of PPARs and of cofactors on metabolism and on energy homeostasis, in a PPAR dependent or independent manner. Studies describing strategies targeting PPARs and cofactors to prevent/treat obesity and metabolic diseases are also highly welcomed.

Potential topics include but are not limited to the following:

- Role of PPARs and cofactors (a) in energy homeostasis and metabolic disease, including obesity, type 2 diabetes, hypertension, hyperlipidemia, cardiovascular diseases, hepatic steatosis, and chronic inflammation; (b) in certain types of cancer and aging-related diseases; (c) in adipocyte biology
- Role of noncoding RNAs in the regulation of PPARs and cofactors in homeostasis and metabolic disease
- Epigenetic modifications of PPARs and cofactors affecting homeostasis and metabolic disease
- Mechanisms of preferential recruitment of cofactors to PPARs to specify lipid storage and thermogenic gene programs, under certain physiological conditions
- New PPARs ligands and/or cofactors agonists as therapeutic agents for metabolic diseases
- Novel strategies targeting PPARs and cofactors for preventing/treating metabolic disease

Authors can submit their manuscripts through the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/ppar/comd/.

Papers are published upon acceptance, regardless of the Special Issue publication date.

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