TARGETING PEROXISOME PROLIFERATOR-ACTIVATED RECEPTOR-B/D FOR CANCER PREVENTION AND THERAPY

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Abstract:
The peroxisome proliferator-activated-β/δ (PPARβ/δ) was identified in 1994, but not until 1999 was PPARβ/δ suggested to be involved in carcinogenesis. Initially, it was hypothesized that expression of PPARβ/δ was increased during colon cancer progression, which led to increased transcription of yet-to-be confirmed target genes that promote cell proliferation and tumorigenesis. It was also hypothesized at this time that lipid metabolizing enzymes generated lipid metabolites that served as ligands for PPARβ/δ. These hypothetical mechanisms were attractive because they potentially explained how non-steroidal anti-inflammatory drugs (NSAIDs) inhibited tumorigenesis by potentially limiting the concentration of endogenous PPARβ/δ ligands that could activate this receptor that was increased in cancer cells. However, during the last twenty years, considerable research was undertaken describing expression of PPARβ/δ in normal and cancer cells that has led to a significant impact on the mechanisms by which PPARβ/δ functions in carcinogenesis. Whereas results from earlier studies led to much uncertainty about the role of PPARβ/δ in cancer, more recent analyses have revealed a more consistent understanding. The focus of this presentation is on the fundamental roles of PPARβ/δ in carcinogenesis and how this nuclear receptor might be targeted for cancer prevention and therapy.

Host: Dr. Jonathan Shannahan