The Annual Meeting of the AACR was a very comprehensive meeting that outlined findings from a broad range of topics within the cancer research field. Abstracts presented at the meeting are now freely available at www.aacr.org. Those that garnered media attention, such as the finding that there is no link between aspartame intake and cancer incidence, are also highlighted on the AACR website. The 98th Annual Meeting will be held April 14th to 18th, 2007, in Los Angeles, CA and promises to be another excellent venue for the dissemination of ideas and findings of cancer researchers from around the world.

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The function of lipin in obesity and lipodystrophy

Experimental Biology meeting notes, San Francisco, April 2006

Experimental Biology is the lead scientific conference for the American Association of Anatomists, the American Physiological Society, the American Society for Biochemistry and Molecular Biology, the American Society for Investigative Pathology, the American Society for Nutrition, and the American Society for Pharmacology and Experimental Therapeutics. The conference was attended by ~15,000 scientists, physicians, and trainees. Thematic symposia and poster presentations were presented over four days. With such a large meeting it is impossible to attend every event, especially as many overlap. Of the symposia I attended, one of the most important presentations was work out of Rutgers University where the function of the lipin gene was determined. Lipin is one of only a handful of genes that can adjust body fat up or down, depending on its expression level. Increased levels of lipin result in heightened insulin sensitivity as well as obesity. Decreasing lipin levels results in insulin resistance and lipodystrophy. Lipin is thus considered to be a very good target for the development of drugs for the treatment of both obesity and lipodystrophy[1].

At the Experimental Biology conference, lipin was recognized as the missing gene in the biochemical pathway that converts fatty acids into triacylglycerol. New research presented at the conference, identified the function of the lipin protein as an enzyme called phosphatidic acid phosphatase (PAP), which converts a lipid (fat) metabolite called phosphatidic acid into diacylglycerol [2]. Subsequent to this enzymatic conversion by lipin, a fatty acid is then added to diacylglycerol to produce an intact triacylglycerol fat molecule. Excessive PAP leads to an increase in triacylglycerols and consequently fat deposition. Identifying the mechanistic basis of lipin function now enables direct screening of small molecules for inhibition or activation of lipin encoded PAP activity. With this discovery, the hope of new therapies for obesity and lipodystrophy are closer at hand.

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