



## Blood and Tissue Biomarkers for Prevention of Fatty Liver Disease / Non-Alcoholic Steatohepatitis & Associated Clinical Trial Patient Segmentation

### *Application*

- ◆ *Development of blood indicators of Non-Alcoholic Steatohepatitis (NASH)*

### *Advantages*

- ◆ *Development of a panel test could detect NASH*
- ◆ *NASH is a reversible condition achieved by aggressive weight loss*

### *Introduction*

An estimated 20% to 30% of adults in the United States have excess fat accumulation in the liver. Of these, about 10% (or 2% to 3% of all adults) have fatty liver related inflammation (hepatitis) and/or fibrosis and meet the diagnostic criteria for non-alcoholic steatohepatitis (NASH).

NASH begins with fatty liver (steatosis) and progresses to inflammation (steatohepatitis), then to fibrosis and bridging fibrosis. This eventually can lead to end-stage cirrhosis, which is treatable only by transplantation. Prior to fibrosis, NASH is a reversible condition, but only if treated with aggressive weight loss such as that achieved by bariatric surgery.

NASH is diagnosed by liver biopsy; however, few physicians use this test on obese patients because of their size, geometry and layers of adipose tissue. NASH is a particularly difficult condition to treat due to the lack of accurate, non-invasive and safe diagnostics to analyze the progression of the disease. Because of problems associated with existing tests (liver function tests and liver biopsies), there is a pressing and widely-recognized need for new and accurate diagnostics in obese patients. Approximately 16 novel circulating blood biomarkers have been discovered as indicators of NASH. With continued development of these indicators, a panel test may be able to detect NASH.

Geisinger has licensed NASH technology to Hepgen ([www.hepgen.com](http://www.hepgen.com)).

### CONTACT



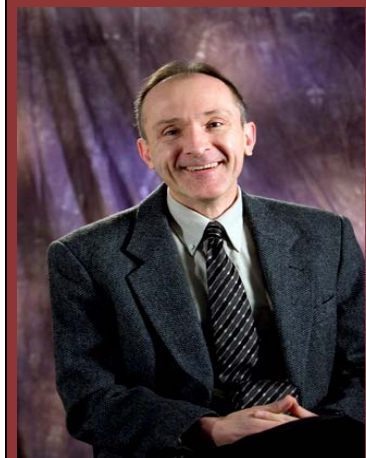
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### *About the Inventor*

Glenn Gerhard, MD is a board-certified Clinical Pathologist with research training in Human Genetics at the University of Pennsylvania and residency training in Pathology at Dartmouth. He is the Director of the Geisinger Clinic Genomics Core, Staff Scientist at the Weis Center for Research, and Associate Medical Director of the Geisinger Molecular Diagnostics Laboratory in the Department of Pathology and Laboratory Medicine at Geisinger. Dr. Gerhard is a co-inventor on two patent applications filed in May 2006 and May 2007. In 1997, he co-Founded Targeted GastroIntestinal Therapeutics (TGIT), a firm based upon developing diagnostics and therapeutics for hereditary hemochromatosis, a disorder of iron.

### **Selected publications includes:**

1. Grundy MA, Gorman N, Sinclair PR, Chorney MJ, **Gerhard GS**. High-throughput non-heme iron assay for animal tissues. *J Biochem Biophys Methods*. 2004 May 31;59(2):195-200.
2. **Gerhard, GS**, and Kasales, CJ. Aging and kyphosis. *Journal of Gerontology: Biological Sciences*, Nov; 58(11): 968, 2003.
3. **Gerhard, GS**. Comparative aspects of zebrafish as a model for aging research. *Experimental Gerontology*, Nov-Dec; 38(11-12): 1333-41, 2003. Zacharski, L. and Gerhard, GS. Atherosclerosis: a Manifestation of Chronic Iron Toxicity? *Vascular Medicine*, 8:153-5, 2003.
4. Chorney, MJ, Yoshida, Y, Meyer, PN, Yoshida, M and **Gerhard, GS**. The Enigmatic Role of the Hemochromatosis Protein in Iron Absorption. *Trends in Molecular Medicine*, Mar;9(3):118-25, 2003.
5. Sinclair, PR, Gorman,N, Trask,H, Bement, J, Szakacs, JG, Elder, GE, Balestra,D, Sinclair, JF, and **Gerhard, GS**. Uroporphyrin caused by ethanol in HFE (-/-) mice as a model for porphyria cutanea tarda. *Hepatology*, Feb;37(2):351-8, 2003.
6. **Gerhard, GS** and Cheng, KC. A call to fins! Zebrafish as a gerontological model. *Aging Cell* 1(2), 104-111, 2002.
7. **Gerhard, GS**, Kauffman, EJ, Wang, X, Stewart, R, Moore, J, Kasales, CJ, Demidenko, E, and Cheng, KC. Life spans and senescent phenotypes in two strains of Zebrafish (*Danio rerio*). *Experimental Gerontology*, Aug(8-9): 1055, 2002.



### **CONTACT**