Journal of Physiology and Biochemistry
Volume 73, Issue 2, 1 May 2017, Pages 245-258

IGF-1 modulates gene expression of proteins involved in inflammation, cytoskeleton, and liver architecture

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Abstract
Even though the liver synthesizes most of circulating IGF-1, it lacks its receptor under physiological conditions. However, according to previous studies, a damaged liver expresses the receptor. For this reason, herein, we examine hepatic histology and expression of genes encoding proteins of the cytoskeleton, extracellular matrix, and cell-cell molecules and inflammation-related proteins. A partial IGF-1 deficiency murine model was used to investigate IGF-1's effects on liver by comparing wild-type controls, heterozygous $igf1^{+/-}$, and heterozygous mice treated with IGF-1 for 10 days. Histology, microarray for mRNA gene expression, RT-qPCR, and lipid peroxidation were assessed. Microarray analyses revealed significant underexpression of igf1 in heterozygous mice compared to control mice, restoring normal liver expression after treatment, which then normalized its circulating levels. IGF-1 receptor mRNA was overexpressed in Hz mice liver, while treated mice displayed a similar expression to that of the controls. Heterozygous mice showed overexpression of several genes encoding proteins related to inflammatory and acute-phase proteins and underexpression or overexpression of genes which coded for extracellular matrix, cytoskeleton, and cell junction components. Histology revealed an altered hepatic architecture. In addition, liver oxidative damage was found increased in the heterozygous group. The mere IGF-1 partial deficiency is associated with relevant alterations of the hepatic architecture and expression of genes involved in cytoskeleton, hepatocyte polarity, cell junctions, and extracellular matrix proteins. Moreover, it induces hepatic expression of the IGF-1 receptor and elevated acute-phase and inflammation mediators, which all resulted in liver oxidative damage. © 2017, The Author(s).

SciVal Topic Prominence
Topic: Somatomedins | Insulin-Like Growth Factor I | growth factor-1
Prominence percentile: 73.629

Reaxys Database Information

View Compounds

Author keywords
Cytoskeleton; Extracellular matrix; Gene expression; Hepatocytes; IGF-1; Tight junctions

Indexed keywords

EMTREE drug terms:	acute phase protein; autacoid; lipid; messenger RNA; scleroprotein; somatomedin C; acute phase protein; autacoid; cadherin; cytoskeleton protein; insulin-like growth factor-1, mouse; scleroprotein; somatomedin C; somatomedin receptor; tight junction protein
EMTREE medical terms:	acute phase response; animal experiment; animal tissue; Article; cell junctioncell polarity; controlled study; cytoskeleton; extracellular matrix; gene expression regulation; gene over; expression; heterozygosity; histology; igf1 gene; inflammation; lipid peroxidation; liver structure; liver tissue; male; microarray analysis; mouse; murine model; nonhuman; oxidative stress; protein analysis; protein blood level; protein deficiency; protein function; reverse transcription polymerase chain reaction; wild type; animal; comparative study; cross breeding; desmosome; gene expression profiling; genetics; hepatitis; immunology; liver; metabolis; mpathology; subcutaneous drug administration; transgenic mouse
MeSH:	Acute-Phase Proteins; Animals; Cadherins; Crosses, Genetic; Cytoskeletal Proteins; Desmosomes; Extracellular Matrix Proteins; Gene Expression Profiling; Gene Expression Regulation; Hepatitis; Inflammation Mediators; Injections, Subcutaneous; Insulin-Like Growth Factor I; Lipid Peroxidation; Liver; Male; Mice; Mice, Transgenic; Oxidative Stress; Receptors, Somatomedin; Tight Junction Proteins

Chemicals and CAS Registry Numbers:
lipid, 66455-18-3; somatomedin C, 67763-96-6;

Acute-Phase Proteins; Cadherins; Cytoskeletal Proteins; Extracellular Matrix Proteins; Inflammation Mediators; Insulin-Like Growth Factor I; insulin-like growth factor-1, mouse; Receptors, Somatomedin; Tight Junction Proteins

- **ISSN:** 11387548
- **CODEN:** JPBIF
- **Source Type:** Journal
- **Original language:** English
- **DOI:** 10.1007/s13105-016-0545-x
- **PubMed ID:** 28124277
- **Document Type:** Article
- **Publisher:** Springer Netherlands