### Short Communication

# Chitinase-3-Like Protein 1 (YKL-40) As A New Biomarker in Inflammation, Oncology, and Neurology

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#### 1. Short Communication

The chitinase family includes 18 enzymes, and among them, the most studied are chitotriosidase and to a lesser extent acid AM-Case. Chitotriosidase activity has been shown to drastically increase in the serum of patients with Gaucher disease (a lysosomal storage disease); this biomarker is known to be useful for the diagnosis and theranostics of Gaucher disease.

At present, the biological function of chitinase-3–like protein 1 (CHI3L1, YKL-40) is unknown. YKL-40 is a secreted glycoprotein (without enzymatic activity), that is approximately 40 kDa in size and in humans is encoded by the CHI3L1 gene. The name YKL-40 is derived from three N-terminal amino acid residues present in the secreted form and from its molecular weight. YKL-40 is expressed and secreted by various cell types including macrophages, chondrocytes, fibroblast like synovial cells, vascular smooth muscle cells, and hepatic stellate cells. YKL-40 is a heparin-binding and chitin-binding glycoprotein. It belongs to a group of mammalian proteins with an amino acid sequence similar to that of 18 glucosyl hydrolases, a family of bacterial chitinases.

YKL-40, as a marker of inflammation and endothelial dysfunction, is associated with atherosclerosis and increased cardiovascular mortality in the general population [1, 2]. Serum YKL-40 is significantly associated with all-cause mortality in patients with heart failure and may be a new prognostic biomarker for these patients. Tumor cells secrete YKL-40 to induce endothelial-cell activation and stimulate tumor angiogenesis via the coupling of syndecan 1 with integrin receptors (Ismail et al., 2019). YKL-40 was shown to control VEGF activity in tumor cells, and the two cooperate to augment endothelial-cell-based angiogenesis [3].

Increased plasma YKL-40 concentration in Alzheimer's disease was reported recently [4, 5]. Determination of YKL-40 concentration in cerebrospinal fluid may help to distinguish between types of dementia and to identify patients with neurodegenerative diseases. According to recent findings, YKL-40 may be useful as a new biomarker for the diagnosis and prognosis of Alzheimer's disease (but not Parkinson's disease, where YKL-40 expression is unchanged).

Alzheimer's disease is a form of dementia that is reported to be closely linked with type 2 diabetes mellitus. Alzheimer's disease is characterized by confusion and loss of memory. Currently, Alzheimer's cannot be cured but can be treated to slow its progression. Therefore, prevention of risk factors of Alzheimer's disease can include early diagnosis and effective treatment of type 2 diabetes mellitus. The etiology of Alzheimer's itself is not well understood at present, and therefore the link between Alzheimer's disease and diabetes mellitus is not clear either.

One reason why Alzheimer's may be more prevalent among people with diabetes mellitus is that the latter can damage the small blood vessels that feed nerves and other relevant cells. Accordingly, damage to these blood vessels can lead to injury of the nerves and other cells that these vessels feed. If brain cells are damaged this way, then Alzheimer's disease can ensue.

## References

- Harutyunyan M., Christiansen M., Johansen J.S., Køber L., Torp-Petersen C., Jens Kastrup J. The inflammatory biomarker YKL-40 as a new prognostic marker for all-cause mortality in patients with heart failure. Immunobiology 2012; 217(6):652-656. Doi: 10.1016/j. imbio.2011.11.003
- Ismail H, Helby J, Hölmich LR, Chakera AH, Bastholt L, Klyver H, Sjøgren P, Schmidt H, Schöllhammer L, Johansen JS, Nordestgaard BG, Bojesen SE. Measured and genetically predicted plasma YKL-40 levels and melanoma mortality. Eur J Cancer. 2019; 121:74-84. doi: 10.1016/j.ejca.2019.08.025.
- Kognole AA, Payne CM. Inhibition of Mammalian Glycoprotein YKL-40: Identification of the physiological ligand. J Biol Chem. 2017;292(7):2624-2636. doi: 10.1074/jbc.M116.764985.
- Pouyafar A, Heydarabad MZ, Mahboob S, Mokhtarzadeh A, Rahbarghazi R. Angiogenic potential of YKL-40 in the dynamics of tumor niche. Biomed Pharmacother. 2018; 100:478-485. doi: 10.1016/j.biopha.2018.02.050.
- Villar-Piqué A, Schmitz M, Hermann P, Goebel S, Bunck T, Varges D, Ferrer I, Riggert J, Llorens F, Zerr I. Plasma YKL-40 in the spectrum of neurodegenerative dementia.
- 6. J Neuroinflammation. 2019; 16(1):145. doi: 10.1186