The influence of Coenzyme compositum and Ubichinon compositum preparations on the function of left and right heart ventricles in patients suffering from “Metabolic X Syndrome”

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Reprint from: Biologicheskaya Terapia, Ukraine, 2001;2:10-14

Limited efficiency of traditional therapy of heart disease makes the search for alternative approaches valuable. This paper presents the results of an investigation of the influence of the products Coenzyme compositum and Ubichinon compositum on the function of left and right heart ventricles in patients suffering from metabolic syndrome with early heart disease. 49 patients suffering from ischemic heart disease together with the metabolic syndrome, with age ranging from 50 to 70 years, took part in the study. The first therapeutic group was composed of 20 patients prescribed with traditional therapy along with angiotensin-converting enzyme inhibitor, Capoten, in a 50 mg daily dose. 15 patients of the second group were given Ubichinon compositum in addition to the standard therapy. The third group was composed of 10 patients prescribed standard medicines together with Coenzyme compositum. Both biological preparations were given at a rate of 1 ampoule on alternative days, 10 injections in total. Additionally, 148 patients suffering from ischemic heart disease without metabolic syndrome symptoms were investigated. The control group was composed of 30 practically healthy persons of similar age. The inclusion of Ubichinon compositum in the traditional therapeutic scheme permitted achievement of the normalization of several parameters of both ventricles’ diastolic function within 3 weeks. It was also shown that Coenzyme compositum was effective in increasing velocity and volume features of early and active ventricular filling as well as the contractile activity of the myocardium in heart disease patients. There is a need to take into account the possibility of the development of sympathicotonia when prescribing this preparation. It was concluded that the use of composite biological preparations, such as Coenzyme compositum and Ubichinon compositum, in the traditional treatment scheme for early heart deficiency patients, is expedient.

Keywords: Ubichinon compositum - Coenzyme compositum - metabolic syndrome - ischemic heart disease

Dual inhibition of 5-lipoxygenase/cyclooxygenase by a reconstituted homeopathic remedy; possible explanation for clinical efficacy and favorable gastrointestinal tolerability

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Reprint from: Inflammation Research 2004;53:150-157

Objective: In order to elucidate potential anti-inflammatory activities of Zeel comp. N and its constituents, the inhibition of the synthesis of Leukotriene B4 (LTB4) and Prostaglandin (PGE2) by 5-lipoxygenase (5-LOX) and cyclooxygenase 1 and 2 (COX 1 and 2) respectively were examined in vitro.

Materials: Human HL-60 cells, differentiated for 6–8 days with DMSO (1.2% v/v) were used for the 5-LOX assay. The COX activity assays were carried out with purified enzymes, COX 1 (ram seminal vesicles), COX 2 (sheep placenta) and with human THP-1 cells, differentiated for 24 h with PMA (50 nM).

Methods: LTB4 and PGE2 production in the 5-LOX and COX assays respectively were determined by enzyme-linked immunoassays.

Results: A reconstituted Zeel comp. N combination as well as its constituent mother tinctures of Arnica montana, Sanguinaria canadensis and Rhus toxicodendron (Toxicodendron quercifolium) showed distinct inhibitory effects on the production of LTB4 by 5-LOX (IC50 values of 10, 20, 2 and 5 mg/ml respectively) and on the synthesis of PGE2 by COX 1 (IC50 values of 50, 80, 40 and 20 mg/ml respectively) and COX 2 enzymes (IC50 values of 60, 110, 50 and 20 mg/ml respectively). The mother tincture of Solanum dulcamara inhibited the production of PGE2 by COX 1 (IC50 40 mg/ml) and COX 2 (IC50 150 mg/ml), but not the production of leukotriene LTB4 by 5-LOX.

Conclusions: The observed dual inhibition (modulation) of both LOX- and COX-metabolic pathways may offer an explanation for the reported clinical efficacy and the favorable gastrointestinal tolerability of the original remedy Zeel comp. N.

Keywords: Zeel comp. N - mother tinctures - in vitro assays - dual 5-LOX/COX inhibition