Look at the labeling

LOPID® (gemfibrozil)—the only lipid medication specifically indicated to reduce the risk of CHD

240
TOTAL

<35
HDL

Low HDL with elevated LDL and triglycerides: A common denominator of many heart attack victims

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PARKE-DAVIS
LOPID is indicated for reducing the risk of coronary heart disease in type IIb patients with low HDL, in addition to elevated LDL and triglycerides, and who have had an inadequate response to weight loss, diet, exercise, and other pharmacologic agents such as bile acid sequestrants and nicotinic acid. LOPID is not indicated for the treatment of patients with low HDL cholesterol as their only lipid abnormality.

Reduced heart attack incidence up to 62%*
—in Helsinki Heart Study patients whose baseline HDL was < 35 mg/dL and median baseline LDL was 186 mg/dL. Incidence of serious coronary events was similar for LOPID and placebo subgroups with baseline HDL above the median (46.4 mg/dL).

Raised low HDL 25%
—in these Helsinki Heart Study patients.

RAISES HDL, LOWERS LDL AND TRIGLYCERIDES DRAMATICALLY REDUCES HEART ATTACK

Contraindicated in patients with hepatic or severe renal dysfunction, including primary biliary cirrhosis, preexisting gallbladder disease, or hypersensitivity to gemfibrozil. LOPID may increase cholesterol secretion into the bile, leading to cholelithiasis. Caution should be exercised when anticoagulants are given in conjunction with LOPID.

*Defined as a combination of definite coronary death and/or definite myocardial infarction. 

P = .013; 95% CI 13.3 to 111.5.


Please see last page of this advertisement for warnings, contraindications, and brief summary of prescribing information.
was a significant increase in the combined incidence of benign, and malignant liver nodules and liver carcinomas was significantly increased in high creatine kinase will prevent the occurrence of severe myopathy and kidney damage.

DEFINITELY DETERMINED THAT THE PROTHROMBIN LEVEL HAS STABILIZED

lovastatin and gemfibrozil does not outweigh the risks of severe myopathy, rhabdomyolysis and pancreatitis. The higher risk of rhabdomyolysis treated for gallbladder disease was confirmed.

During the Helsinki Heart Study and in the 1½ years follow-up period since the trial was halted, the mortality rate was decreased by 24% in the Lopid group and 55% (2.7%) in the placebo group. Mortality from any cause during the double-blind portion of the study was 44 deaths in the Lopid group and 43 in the placebo group. Because of the study design, not all deaths were statistically significant different from the 296 excess mortality seen in the clofibrate group in the separate WHO study Noncoronary heart disease related mortality showed a 58% greater trend in the Lopid group (43 vs 27 patients in the placebo group, p=0.09).

In the Helsinki Heart Study, the incidence of total malignancies discovered during the trial and in the 1½ years since the trial was completed was 39 in the Lopid group and 29 in the placebo group. Mortality from tumors other than noncardiovascular causes, malignancy, post-cholecystectomy, and pancreatic cancer was statistically significant different from the 296 excess mortality seen in the clofibrate group in the separate WHO study Noncoronary heart disease related mortality showed a 58% greater trend in the Lopid group (43 vs 27 patients in the placebo group, p=0.09).

A Brief Summary follows. Before prescribing, please see full prescribing Information.

Lopid® (Gemfibrozil Capsules and Tablets)

RAISES HDL, LOWERS LDL AND TRIGLYCERIDES DRAMATICALLY REDUCES HEART ATTACK

Additional adverse reactions that have been reported for gemfibrozil are listed below by system. These are categorized according to whether a causal relationship to treatment with Lopid is probable or not established.

SPECIAL HUMAN STUDIES

Gestational

References:

Fertility—Long-term studies conducted by the World Health Organization (WHO). 5000 subjects without known coronary heart disease were treated with clofibrate for five years and followed one year after discontinuation. None of the studies may also apply to gemfibrozil. In the first of those studies, the Coronary Drug Project in the United States investigated the effects of monosodium levulinate (MRL) therapy requiring surgery in 1951 and 1971.

Test for peroxisome proliferation has not been done in human individuals. Adharization of approximately three or ten times the human dose to rats for 10 weeks resulted in a dose-related decrease of fertility. Subsequent studies demonstrated that this effect was reversible after a drug-free period of about eight weeks, and it was not transmit-
Arteriosclerosis and Thrombosis

Scope and Purpose

Arteriosclerosis and Thrombosis is a forum for the publication of high-quality research from a variety of disciplines concerned with the biology, prevention, and impact of vascular diseases relating to arteriosclerosis and thrombosis. Major research areas and topics for relevant manuscripts and state-of-the-art reviews are cited below. This list is not meant to be all-inclusive but is intended as a guide for submitting and selecting articles for publication.

- The biology and pathology of vascular lesions related to arteriosclerosis, including the disciplines of biochemistry, biophysics, molecular cell biology, genetics, and metabolism
- The regulation of lipoprotein-cell interactions, adhesive molecules, and cytokines, including those involved in cell-cell interactions such as macrophage-endothelial cells, lymphocyte-endothelial cells, and platelet-endothelial cells
- Aspects of lipid and lipoprotein metabolism and transport related to vascular biology and disease
- Receptors on platelets, endothelial cells, and macrophages
- Extracellular matrix biochemistry and metabolism related to vascular biology, atherosclerosis, and thrombosis
- The molecular and cellular biology of clotting factors, platelets, and the fibrinolytic system
- Epidemiological, demographic, and genetic studies of atherosclerosis and thrombosis, including studies of the interplay of risk factors such as diabetes, nutrition, hyperlipidemia, and hypertension
- Development of atherosclerosis and thrombosis and its precursors in the young
- Animal models of atherosclerosis and thrombosis, including molecular and cellular engineering of the vascular system
- The detection and quantification of arterial lesions in vivo in humans and animals
- Evaluation of the effects of, for example, the prevention or treatment of intimal injury or the treatment of plasma lipoproteins on the established lesions of atherosclerosis
- The development of new thrombolytic agents
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