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

Most hyperlipidemas—elevated cholesterol and triglycerides—are common among heart attack victims, and nearly two thirds of people who developed myocardial infarction in the PROCAM trial had a level less than 35 mg/dL baseline level of HDL cholesterol. HDL lowering is not indicated for the treatment of patients with low HDL cholesterol as their only lipid abnormality.

HEART ATTACK PATIENTS
(PROCAM TRIAL)

HDL under 35 mg/dL
64%
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.

Raised low HDL 25%
—In patients whose baseline HDL was < 35 mg/dL and median baseline LDL was 186 mg/dL in the landmark Helsinki Heart Study (HHS).  

Reduced heart attack incidence up to 62%*
—in these HHS patients. Incidence of serious coronary events was similar for LOPID and placebo subgroups with baseline HDL above the median (464 mg/dL).

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. 

References
3. Data on file, Medical Affairs Dept, Parke-Davis.

Please see last page of this advertisement for warnings, contraindications, and brief summary of prescribing information.
GEMFIBROZIL (Gemfibrozil Capsules and Tablets)

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

CONTRAINDICATIONS. 1. Hepatic or severe renal dysfunction, including primary biliary cirrhosis.

2. Preexisting gallbladder disease (See WARNINGS).

WARNINGS. 1. Because of chemical, pharmacological, and clinical similarities between gemfibrozil and clofibrate, the adverse findings with clofibrate in two large clinical studies have been applied to gemfibrozil. In the first of those studies, the Coronary Drug Project, 1000 subjects treated with gemfibrozil for over 5 years, died 12 patients died, 6 with clofibrate. There was no difference in mortality between the clofibrate-treated subjects and 3000 placebo-treated subjects, but twice as many clofibrate-treated subjects died from congestive heart failure (cheloiditis requiring surgery). In the other study, 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 later for mortality. The 29% higher total mortality in the clofibrate-treated than in a comparable placebo-treated control group. The excess mortality was due to a 33% increase in noncardiovascular causes, including malignancy, post-cholesteroidal syndrome, and pancreatitis. This higher risk of clofibrate-treated subjects for gallbladder disease was confirmed.

During the Helsinki Heart Study and in the 1 1/2 year follow-up period since the trial was completed in June 1975, no deaths occurred, and in the rabbit at 2 and 6.7 times the human

BID

LOWERS LDL, Lowers LDL AND TRIGLYCERIDES DRAMATICALLY REDUCES HEART ATTACK

LDL AND TRIGLYCERIDES

2. A gallstone prevalence study of 450 Helsinki Heart Study patients showed a trend toward a greater prevalence of gallstones during the study within the Lopid treated group (7.5% vs 4.5% in the placebo group, a 55% excess for the gemfibrozil group). A trend toward a greater incidence of gallstones was observed for the Lopid group (17 vs 11 subjects, a 54% ex-

ces). This result did not differ statistically from the increased incidence of cholecystectomy observed in the WHO study in the group treated with clofibrate. The reported cholecystectomy may increase cholesterol excretion into the bile leading to cholelithiasis. If cholecystitis is suspected, gallbladder studies are indicated. Lopid therapy should be discontinued if gallstones are found.

3. Concomitant Anticoagulants—Caution should be exercised when anticoagulants are given concomitantly with Lopid. The dose of anticoagulant should be reduced to maintain the prothrombin time at the desired level to prevent bleeding complications. Frequent prothrombin determinations are advisable until it has been definitely determined that the anticoagulant dosage is adequate.

5. Concomitant Therapy with Lopid and Mevacor® (lovastatin) has been associated with rhabdomyolysis, markedly elevated creatinine kinase (CK) levels and myoglobinuria, leucopenia, anemia, and elevated liver enzymes to acute renal failure. In most subjects, this occurred with doses greater than 400 mg of Lovastatin or almost 15 times the human dose. Patients receiving Lopid and complaining of muscle pain, tenderness, or weakness should have prompt medical evaluation for myositis, including serum creatinine kinase level determination. If myositis is suspected, Lopid should be discontinued.

6. Cataracts—Subcapsular bilateral cataracts occurred in 10%, and unilateral in 6.3% of male rats treated with gemfibrozil at 10 times the human dose.

PREGNANCY Category B. Lopid therapy should be determined to ascertain that the lipid levels are consistently abnormal. Before instituting Lopid therapy, every at
temt should be made to control serum lipids with appropriate diet, exercise, weight loss or in other ways. If diet alone is not successful in lowering serum lipids, or if diabetes mellitus and hyperlipidemia are found to be contributing to the lipid abnormalities.

2. Continued Therapy—Periodic determination of serum lipids should be obtained, and drug withdrawn if lipid response is inadequate after 3 months of therapy.

3. Drug Interactions—Lopid has no effect on platelet aggregation and has occurred when used in combination with gliotoxin and lovastatin. It may be seen as early as 3 weeks after initiation of combined therapy or after several months. In most subjects with an unallevia-
tory lipemia, no increase in postprandial lipemia was seen. In one study, a beneficial interaction in patients with severe hypertriglyceridemia and moderate hyper-
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