Table 4. Effects of Lipoproteins and Insulin on PLTP mRNA in HepG2 Cells*

Treatment	PLTP mRNA (% control)
Control	100.00 ± 16.39
HDL (50 μg/mL)	102.58 ± 30.34
LDL (50 μg/mL)	88.72 ± 8.07
VLDL (50 μg/mL)	103.59 ± 36.57
Insulin (1 μg/mL)	94.63 ± 7.17

*Mean ± SD (n = 4).

kDa was found in the medium of HepG2 cells after purification by phenyl-Sepharose, heparin-Sepharose, and hydroxyapatite column chromatography followed by 8-fold concentration, and detection by mouse anti-human PLTP monoclonal antibody. It is postulated that PLTP could be detected in the conditioned medium due to the better binding of PLTP to its specific antibody after purification to remove interference. Tall[25] showed CETP mRNA levels in HepG2 and human colon carcinoma Caco-2 cells to be 1/10 those in vivo. It is also suspected that PLTP secretion by HepG2 cells was undetectable in our study possibly due to the fact that PLTP secretion by in vitro cells could be far lower than the physiological level.

After slot blotting and correction by β-actin mRNA, PLTP mRNA levels in HepG2 cells were not significantly different among the 5 groups (Fig. 3, Table 4). Jiang and Bruce[7] found significant increases in plasma HDL-cholesterol, HDL-phospholipid, VLDL+LDL-cholesterol, and VLDL+LDL-phospholipid concentrations, plasma PLTP activity, and lung PLTP mRNA levels in rats after a high fat and cholesterol diet. However, liver and adipose PLTP mRNA levels in rats were not affected by a high fat and cholesterol diet. It was suggested that a high fat and cholesterol diet could induce phospholipid fluxes into tissues by increasing plasma PLTP activity mainly due to increases in PLTP mRNA levels in the lung rather than in the liver. Our result revealed no changes in PLTP mRNA levels in hepatocytes in response to lipoproteins or insulin, which is consistent with no changes in PLTP protein expression in our finding. Since PLTP activity was not measured in this study, the possibility that PLTP activity was influenced by lipoproteins or insulin is not yet excluded.

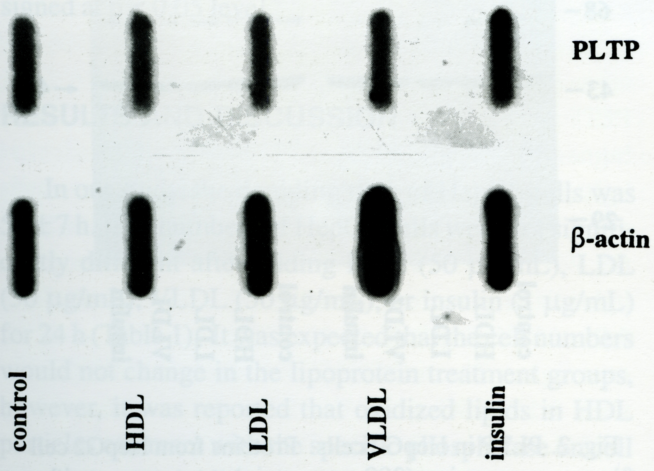

Fig. 3. PLTP mRNA expression in HepG2 cells. Total cellular RNA (30 μg) in HepG2 cells was transferred onto a nylon membrane by slot blotting. The blot was incubated with PLTP or β-actin cDNA probe, and exposed to x-ray film for 90 min.

In conclusion, our results show that exogenous HDL (50 μg/mL), LDL (50 μg/mL), VLDL (50 μg/mL), or insulin (1 μg/mL) did not significantly affect the cellular PLTP (44 kDa) or PLTP mRNA levels, indicating that lipoproteins or insulin do not directly regulate PLTP synthesis in hepatocytes. Therefore, plasma diet-induced PLTP may be derived from other tissues rather than from the liver stimulated by lipoproteins or insulin.

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