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Abstract

Since Hahn's observation of the postalimentary lipemia clearing actIvity following the injection of heparin, physiological, biochemical and clinical significances of the postheparin lipoprotein lipase have been well clarified. The presence of the endogenous lipoprotein lipase in human blood, which was at first doubted, has been repeatedly confirmed2∼8. Recent papers9,10 described elevated endogenous lipoprotein lipase activity in patients with essential hyperlipemia after ample fat uptake. In this preliminary report, changes of the lipoprotein lipase activity during oral glucose tolerance test is illustrated.

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BRIEF NOTES

CHANGES OF THE ENDOGENOUS LIPOPROTEIN LIPASE ACTIVITY DURING ORAL GLUCOSE TOLERANCE TEST

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Since Hahn's observation of the postalimentary lipemia clearing activity following the injection of heparin, physiological, biochemical and clinical significances of the postheparin lipoprotein lipase have been well clarified. The presence of the endogenous lipoprotein lipase in human blood, which was at first doubted, has been repeatedly confirmed[2~8]. Recent papers[9,10] described elevated endogenous lipoprotein lipase activity in patients with essential hyperlipemia after ample fat uptake.

In this preliminary report, changes of the lipoprotein lipase activity during oral glucose tolerance test is illustrated.

Measurement of the lipoprotein lipase (LPL) activity was performed by determination of the release of free fatty acid (FFA) *in vitro*[11~13].

In normal persons (10 cases) (Fig. 1), no LPL activity was demonstrated in the plasma before and 30, 120 and 180 minutes after glucose intake. And, elevated LPL activity was observed at 60 and 90, and 150 minutes after glucose intake, showing two peaks of the LPL activity. After incubation (at 37°C for half an hour) of the plasma mixed with sesame oil (Fatgen), the identical changes of the LPL activity curve were observed. But the peaks of the enzymatic activity of the postincubation plasma were about three times as high as that of the preincubation plasma.

In diabetics (9 cases) (Fig. 2), the LPL activity in plasma before incubation was quite different from changes in normal persons. No activity was observed before glucose intake, but the activity in the plasma rose at 30, 60 and 90 minutes, when the activity reached a peak, and then the activity decreased 120, 150 and 180 minutes after glucose intake, finally showing almost no activity. The elevation of the enzymatic activity in diabetics was higher than that in normal persons. After incubation of the plasma mixed with sesame oil (Fatgen), identical changes of the LPL activity curve were also observed, demonstrating

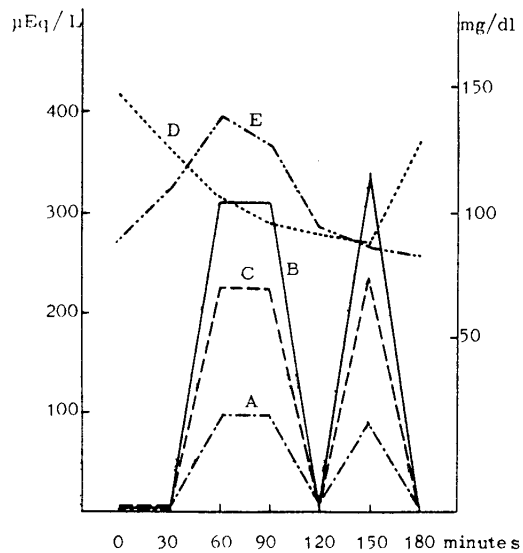

Fig. 1 Plasma LPL and FFA and blood sugar levels in normal persons
A: Preincubation LPL activity (μEq/L) B: Postincubation LPL activity (μEq/L)
C: (Postincubation LPL activity)-(Preincubation LPL activity) (μEq/L) D: FFA (μEq/L), E: Blood sugar (mg/dl)

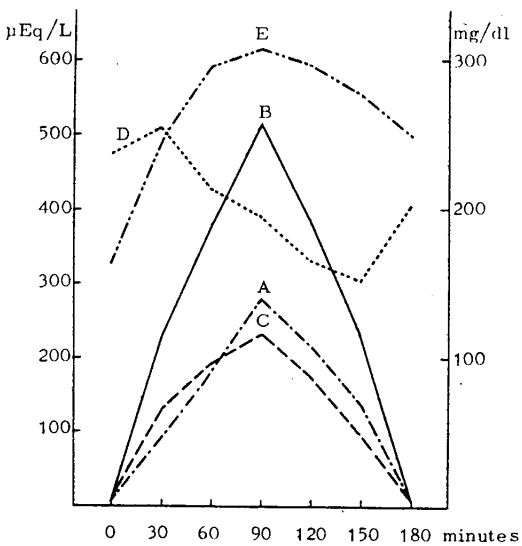

Fig. 2 Plasma LPL and FFA and blood sugar levels in diabetics.
A: Preincubation LPL activity (μEq/L) B: Postincubation LPL activity (μEq/L),
C: (Postincubation LPL activity)-(Preincubation LPL activity) (μEq/L) D: FFA (μEq/L), E: Blood sugar (mg/dl)

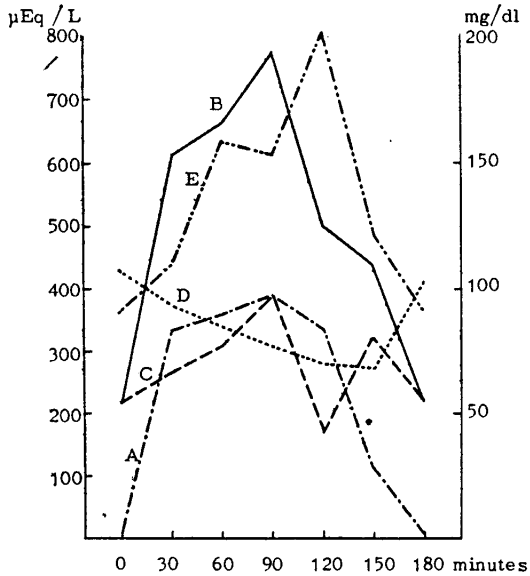

Fig. 3 Plasma LPL and FFA and blood sugar levels in patients with cirrhosis of liver having diabetic GTT curve.
A: Preincubation LPL activity (μEq/L) B: Postincubation LPL activity (μEq/L), C: (Postincubation LPL activity)-(Preincubation LPL activity) (μEq/L) D: FFA (μEq/L), E: Blood sugar (mg/dl)

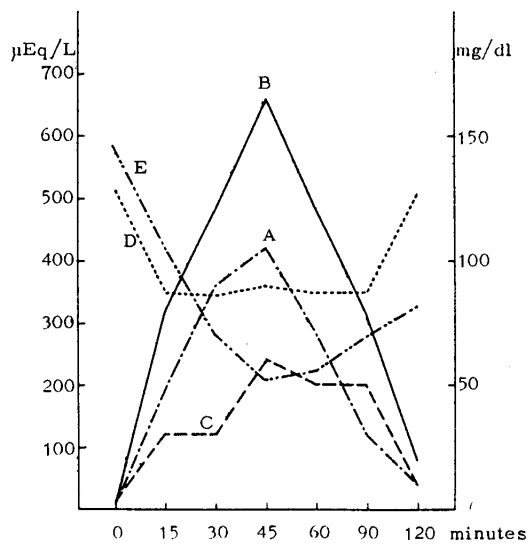

Fig. 4 Plasma LPL and FFA and blood sugar levels in diabetic during insulin sensitivity test.
A: Preincubation LPL activity (μEq/L), B: Postincubation LPL activity (μEq/L), C: (Postincubation LPL activity)-(Preincubation LPL activity) (μEq/L) D. FFA (μEq/L), E: Blood sugar (mg/dl)

a single peak at 90 minutes and no activity before and after glucose tolerance test. The LPL activity of the postincubation plasma was elevated about twice as high as that of the preincubation plasma. But differences of the LPL activity between the pre- and postincubation plasma showed almost same value in normal persons and diabetics.

FFA levels were decreased until 150 minutes and elevated at 180 minutes during oral GTT in normals. In diabetics, FFA levels remained elevated for 30 minutes and then decreased until 150 minutes and again elevated at 180 minutes, keeping higher levels than normals throughout oral GTT.

In patients (3 cases) with cirrhosis of the liver having diabetic GTT curve (Fig. 3), the LPL activity of the preincubation plasma showed a single peak curve as in diabetics, but the LPL activity of the postincubation plasma demonstrated considerably elevated activity before GTT and 180 minutes after glucose intake. At these moments no LPL activity was usually revealed both of the normal and diabetic. The enzymatic activity curve showed a single peak curve having the highest level at 90 minutes as in diabetics.

In insulin sensitive test (Fraser's method[14]) in the diabetic patient (Fig. 4), an identical LPL activity curve as in oral GTT of diabetics was observed in spite of lowered blood sugar level.

These enzymatic activities were completely inhibited by the administration of protamine sulfate and 10 per cent NaCl solution *in vitro*.

These observations are clearly demonstrated that this LPL is the endogenous lipoprotein lipase activated by glucose metabolism and strongly suggested that this enzyme has some sorts of close relationship with insulin activity in the human subject.

The endogenous LPL activity was also observed during fat tolerance test and food uptake.

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