



FIG. 2. Elution profile of cholinephosphotransferase from hamster liver microsomes by DEAE-Sephacrose chromatography. Solubilized hamster liver microsomes were applied to a DEAE-Sephacrose column (1.5×15 cm) equilibrated with 25 mM Tris-succinate (pH 6.0) -2 mM 2-mercaptoethanol. The column was washed with 100 ml of the same buffer and subsequently washed with 0.5 M KCl in the same buffer. Fractions (5 ml) were collected and the enzyme activity was expressed as nmol of product formed/min.

TABLE 2

Purification of Cholinephosphotransferase from Hamster Liver

	Total activity nmol/min	Protein mg	Specific activity nmol/ min/mg	fold
Microsomes	299.00	598.00	0.50	—
Solubilized microsomes	59.50	238.00	0.25	0.50
DEAE-Sephacrose ^a chromatography	47.88	25.20	1.90	3.80
Sephacrose 6B ^b chromatography	16.78	4.56	3.68	7.36

^aAfter DEAE-Sephacrose chromatography, only fractions 29 and 30 (containing 25.20 mg protein) were pooled and the total enzyme activity was calculated from this pooled sample.

^bAfter Sephacrose 6B chromatography, the total enzyme activity was calculated from fractions 21 and 22 (containing 4.56 mg protein).

cerol was substantially lowered when the enzyme was partially purified. The partially purified cholinephosphotransferase did not display any absolute requirement for neutral lipids or phospholipids (15). However, enzyme activity was activated (25%) by 0.5 mM phosphatidylcho-

line or phosphatidylethanolamine but was severely inhibited (>90%) by 0.5 mM lysophosphatidylcholine. The inhibition by lysophosphatidylcholine has been suggested as an important mechanism for the regulation of cholinephosphotransferase activity (17). The ability to solubilize and partially purify cholinephosphotransferase in hamster liver will enable us and other investigators to closely examine the control mechanism of this enzyme and its role in the regulation of phosphatidylcholine biosynthesis.

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ERRATUM

"Altered Arachidonic Acid Content in Polymorphonuclear and Mononuclear Cells from Patients with Allergic Rhinitis and/or Asthma" by Ross E. Rocklin, Lori Thistle, Leo Gallant, M. S. Manku and David Horrobin, *Lipids* **21**, 17-20, 1986. In Table 2, the difference in linoleic acid levels between control lymphocytes and those of atopic patients was significant at $p < 0.05$, and not at $p < 0.01$. The difference in arachidonic acid levels between the same two groups was not significant as opposed to being significant at $p < 0.05$ as stated in the table.