phosphatidylethanolamine and phosphatidylglycerol decreased. However, the more interesting observation was the considerable increase in the lyso- compounds derived from phosphatidylethanolamine and phosphatidylglycerol. The decrease in phosphatidylethanolamine was almost stoichiometric with the increase in the corresponding lyso- compound, but the decrease in phosphatidylglycerol was equivalent to the sum of the increase in lysophosphatidylglycerol and diphosphatidylglycerol. Similar increases in these lyso- compounds were also reported as a result of T_4 infection (8, 9, 27). This could be explained by possible damage caused by T_4 infection to the cell wall structure, resulting in the release of periplasmic enzymes located between the cell wall and the membrane (27). Phospholipase A_2, a membrane bound enzyme, could cause uncontrolled production of lyso- compounds. Alternatively, another enzyme, lysophospholipase, might be inhibited, causing an accumulation of lyso- compounds. In *E. coli* cells infected with M13, release of alkaline phosphatase, a periplasmic enzyme, has been reported (25). The present results may be explained by the damage to the cell wall of *E. coli* by the M13 infection. This may have made the cell membrane more susceptible to uncontrolled activities of the lipase, thus, producing increased amounts of lyso- compounds. The increase in diphosphatidylglycerol may have resulted in enhanced biosynthesis from phosphatidylglycerol caused by the M13 infection. The results in Table III showed that M13 infection had no significant effect on the composition of the component fatty acids of the phospholipids of *E. coli*.

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ERRATUM

An error was made in the December, 1974, issue of *Lipids*. In the article "New Series of Fatty Acids in Northern Pike," by R.L. Glass, Thomas P. Krick, and Allen E. Eckhardt (*Lipids* 9:1004 [1974]), the 2 subheadings, "Liver" and "Testes," of Table II are reversed. ■