Effects of Ingesting Soy or Egg Lecithins on Serum Choline, Brain Choline and Brain Acetylcholine

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ABSTRACT Rats were fed lecithins, derived from eggs or soybeans, to determine whether the fatty acid composition of the phosphatidylcholine altered choline availability. Rats were fed either a single meal containing 5 g phosphatidylcholine or a lecithin-containing diet for 3 weeks, including approximately 5 g phosphatidylcholine per day. Each form of dietary lecithin elevated blood choline, brain choline and brain acetylcholine significantly (P < 0.05). There was no difference in response to egg- or soy-derived lecithin. J. Nutr. 111: 166–170, 1981.

KEY WORDS lecithin · choline · acetylcholine · phosphatidylcholine

Consumption of choline, an important nutrient for mammals, elevates blood choline concentration, with subsequent increases in brain choline and acetylcholine levels (1). Based on this finding, pharmacologic doses of choline have been used to treat tardive dyskinesia (a movement disorder) and are being evaluated as potential therapy for other neurologic diseases believed to stem from inadequate cholinergic activity (2, 3). Such choline doses cause only transient (2- to 3-hour) elevations in blood choline levels, thus necessitating frequent administration to maintain adequate therapeutic levels (4). Also, significant amounts of orally ingested choline are degraded by gut bacteria (forming trimethylamine), which both wastes the compound and gives the patient a “fishy” body odor (5–7).

For these reasons, lecithin, a choline-containing phospholipid that provides most of the choline in the diet (8), has, in most centers, replaced choline chloride as the preferred method of administering choline molecules in therapy of diseases involving inadequate cholinergic activity (9–11). Lecithin does not release significant amounts of free choline within the intestinal lumen; therefore, little trimethylamine is formed by gut bacteria and “fishy” body odor is not a problem. Also, lecithin raises blood choline concentrations far more effectively than equimolar doses of choline chloride (4), causing elevations that persist for more than 8 hours (12).

Most commercial lecithin is prepared from soybeans (soy lecithin) or eggs (egg lecithin) which differ significantly in their fatty acid compositions (table 1). Egg lecithin contains relatively more saturated fat (41.8%); soy lecithin includes primarily unsaturated fats such as linoleic acid [83.4%] (12). It is not known whether the fatty acid moieties of a lecithin molecule influence its efficacy in raising serum choline or brain choline or acetylcholine levels. This study was designed to examine the relationship be-

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TABLE 1

<table>
<thead>
<tr>
<th>Fatty acid</th>
<th>Soy lecithin % wt</th>
<th>Egg lecithin % wt</th>
</tr>
</thead>
<tbody>
<tr>
<td>16:0</td>
<td>12.8</td>
<td>29.7</td>
</tr>
<tr>
<td>16:1ω7</td>
<td>0.2</td>
<td>1.7</td>
</tr>
<tr>
<td>18:0</td>
<td>2.9</td>
<td>12.1</td>
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<tr>
<td>18:1ω6</td>
<td>—</td>
<td>30.0</td>
</tr>
<tr>
<td>18:1ω9</td>
<td>10.6</td>
<td>—</td>
</tr>
<tr>
<td>18:2ω6</td>
<td>65.9</td>
<td>14.9</td>
</tr>
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<td>18:3ω3</td>
<td>6.5</td>
<td>0.4</td>
</tr>
<tr>
<td>20:1ω9</td>
<td>0.2</td>
<td>0.3</td>
</tr>
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<td>20:4ω6</td>
<td>—</td>
<td>4.2</td>
</tr>
<tr>
<td>20:5ω3</td>
<td>—</td>
<td>0.1</td>
</tr>
</tbody>
</table>

1 Fatty acid analyses were performed by Unilever Laboratories by gas chromatography (13).

BETWEEN THE SOURCE OF A LECHITHIN (Egg versus Soy) AND THE IN VIVO UTILITY OF ITS CHOLINE CONTENT.

MATERIALS AND METHODS

Male Sprague-Dawley rats (150 g; Charles River Breeding Laboratories, Wilmington, MA), housed singly in a controlled environment (23–24°C), were given ad libitum access to water and food. The

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Fig. 1 Effect of ingesting a single lecithin-rich meal on choline and acetylcholine levels. After a 16-hour fast, rats (groups of 12) each ate 25 g of a diet containing 20% pure lecithin (5 g total ingestion) or its control (footnote 2). All animals consumed all of the diet offered within 2 hours. Ten hours after initiation of the meal, animals were killed by focused microwave irradiation after cardiac puncture. Data are expressed as the mean ± the standard error of the mean.

* P < 0.05, different from control.
animals were exposed to light from 0700 to 1900 hours daily (Vita-Lite, 50–75 footcandles, Duro-Test Corp., North Bergen, NJ). Animals that were given single, acute doses of lecithin were fasted for 16 hours before feeding; animals in the chronic study consumed the lecithin-containing diet ad libitum for 3 weeks before use. Control animals ingested diets in which shortening (Crisco, Proctor & Gamble, Cincinnati, OH) replaced lecithin. All animals were offered 25 g of diet (containing 5 g pure lecithin [phosphatidylcholine]) 10 hours before death; all animals consumed all the diet offered within 2 hours.

At the time of death (1800 hours), each animal was anesthetized lightly with ether, and a blood sample was taken by cardiac puncture; the animal was then killed by a microwave beam focused on its head. At the time of death, rats were still unconscious as a result of ether administered during cardiac puncture. We did not note differences in brain acetylcholine levels between these anesthetized animals and unanesthetized rats studied in the past. The brain was cooled on ice and frozen until assayed. Choline and acetylcholine were assayed with a radioenzymatic technique (14) after extraction with tetraphenylboron dissolved in 3-heptanone. Data were analyzed by Student’s t-test.

RESULTS
In confirmation of previous findings (4), consumption of a single lecithin-con-
taining meal significantly elevated serum choline, brain choline and brain acetylcholine levels after 10 hours (fig. 1). The effects of egg and soy lecithin did not differ. Consumption of the lecithin-rich diet for 3 weeks also significantly elevated these choline and acetylcholine levels (fig. 2). Although these levels tended to be slightly lower when the lecithin source was egg, differences between effects of the egg and soy materials were not statistically significant.

DISCUSSION

These data show that the biologic source of a lecithin preparation (soybeans or eggs) does not influence significantly its effect on serum choline, brain choline or brain acetylcholine levels. This finding is compatible with the recent report (12) that humans ingesting either soy or egg lecithin exhibited similar increases in serum choline concentrations.

The increases in brain acetylcholine seen 10 hours after a single lecithin meal (fig. 1) or after 3 weeks of chronic consumption of such meals (fig. 2) were also similar. This finding suggests that lecithin has no cumulative effect on blood choline during long-term administration, possibly because lecithin’s metabolism during each day’s fasting period causes serum and brain choline levels to revert to baseline, or because each high lecithin meal saturates choline transport or its enzymatic conversion to acetylcholine. The data also indicate no diminution of lecithin’s ability to affect choline levels, suggesting that there is no induction of choline degradation.

Apparently, the source of lecithin ingested does not affect utilization of its choline in the brain: both soy and egg lecithins raised serum and brain choline and brain acetylcholine to the same level, whether they are ingested only once or frequently over a 3-week period. However, the fact that lecithin source does not affect brain acetylcholine levels need not imply that it does not affect acetylcholine release. Chronic consumption of large amounts of soy lecithin, by providing the animal with large quantities of linoleic and other polyunsaturated fatty acids, may change the amounts of these substances stored in membranes [partly as endogenous lecithins] (15); this change could influence the amount of arachidonic acid available for conversion to prostaglandins (16). If such changes occur in the vicinity of synapses, then soy lecithin could affect neurotransmitter release and neurotransmission by mechanisms other than the enhancement of acetylcholine synthesis described previously (1).

LITERATURE CITED


