selenoproteins than was plasma glutathione peroxidase. Therefore, we concluded that selenoprotein P was a better biomarker for whole-body selenoprotein expression than was plasma glutathione peroxidase.

Two approaches are being used to determine the selenium nutritional requirement. One approach is disease-related and will require intervention trials to determine the selenium intake needed to prevent pathologic conditions and ensure optimal health. At present, this approach supports an intake of ≈20 μg selenium/d to protect against Keshan disease. Intervention trials are being carried out to determine the effect of higher selenium supplements, and they may lead to an increase in this recommendation.

The second approach is the use of biomarkers for selenoproteins to assess the full expression of selenoproteins in the body. This approach was used in 2000 by the Institute of Medicine to support a dietary reference intake of 55 μg selenium/d, which is based on plasma glutathione peroxidase activity (7). The same glutathione peroxidase results were used by a Chinese panel to set a dietary reference intake of 50 μg selenium/d (8). When adequate data on optimization of selenoprotein P by selenium intake are available, the current recommendations will likely require an upward revision.

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Do unsaturated fatty acids function as endogenous antibacterial and antiviral molecules?

Dear Sir:

The recent study in the Journal by Merchant et al (1) that showed that higher intakes of α-linolenic and cis-linoleic acids (ALA and LA, respectively) and fish may reduce the risk pneumonia is interesting but not surprising. Previously, I proposed that long-chain polyunsaturated fatty acids (LCPUFAs), such as γ-linolenic acid (GLA), dihomo-GLA (DGLA), arachidonic acid (AA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA), may behave as endogenous antibacterial, antifungal, antiviral, and immunostimulating agents (2).

Kodiczek (3) showed that both LA and ALA have a bacteriostatic effect on gram-positive and gram-negative bacteria. Lacey and Lord (4) observed that cultures of Staphylococcus aureus seeded onto human skin were rapidly killed after the skin was covered with ALA and suggested that ALA had all the attributes of an ideal antibacterial agent. A variety of bacteria were found to be sensitive to the growth-inhibitory actions of LA and ALA in vitro (5). Hydrolyzed linseed oil, which contains 52% ALA and pure LA, killed methicillin-resistant Staphylococcus aureus (6). Both LA and AA inactivated animal herpes, influenza, Sendai, and Sindbis viruses within minutes of contact (7). The oral administration of LA as safflower oil (which contains 76% LA) produced the remission of mycosis fungoides, a rare skin disease of viral etiology, in dogs; the remission correlated with an increase in plasma LA and AA concentrations (8). AA, EPA, and DHA induced the death of Plasmodium falciparum both in vitro and in vivo (reviewed in 9). Furthermore, both prostaglandin E2 and prostaglandin A, which are derived from DGLA, AA, and EPA, inhibit viral replication and behave as antiviral compounds (10, 11). These observations suggest that both LCPUFAs and their products have antibacterial, antifungal, and antiviral actions. Both lymphocytes and macrophages contain significant amounts of LCPUFAs and are capable of releasing them with the appropriate stimulation. In addition, LCPUFAs stimulate NADPH-dependent superoxide production by macrophages, neutrophils, and lymphocytes, which is capable of killing the invading microorganisms (12). In view of these evidences, it is reasonable to believe that an increased intake of LA, ALA, EPA, and DHA protects against or reduces the risk of pneumonia.
Recent studies showed that AA, EPA, and DHA could give rise to antiinflammatory compounds such as lipoxins and resolvins, which are essential for the limitation and resolution of inflammation (13). These studies imply that a deficiency of lipoxins and resolvins could lead to the perpetuation of inflammation and tissue damage.

In light of these facts, it will be interesting to study whether a subclinical deficiency of LCPUFAs and a decreased formation of lipoxins and resolvins occurs in subjects who develop various types of pneumonia and its complications. Because LCPUFAs can inactivate enveloped viruses, including influenza (7), it is probably worthwhile to study the effect of various fatty acids on the bird flu virus and, if the fatty acids do inactivate the bird flu virus, to study whether increased intake of these fatty acids could reduce the risk of flu.

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