SELENIUM AND KESHAN DISEASE

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Keshan disease is an endemic cardiomyopathy prevailing throughout a wide belt from the northeast to the southwest of China.[1,2] The most susceptible populations are children and women of child-bearing age. The disease has a high rate of fatality. Clinically, it can be divided into four types, i.e., acute, subacute, chronic, and latent.[3] The primary pathological change is multifocal necrosis of the myocardium.

Two facts indicate that selenium deficiency plays an important role in the occurrence of this disease. First, populations in all the endemic areas are in very poor selenium status as compared to those in the nonendemic areas. The Se levels in staple cereals, blood, and hair of populations in the endemic areas are much lower than those in the nonendemic areas. The average blood glutathione peroxidase activities of people in endemic areas are slightly but significantly ($p < 0.05$) lower than those in nonendemic areas. Second, selenium supplementation protects people against this disease. A prophylactic trial was conducted in 12,000 children 1-9 years old in 1974-75. Half of the children were given sodium selenite tablets once a week (ages 1-4 years, 0.5 mg; ages 6-9, 1.0 mg) and the other half were given a placebo. The incidence rate and number of fatal cases were far less in the Se-supplemented group than in the control group. Recently, similar trials with 500,000 subjects were carried out in other provinces, and the results were consistent with previous results.

The etiology of Keshan disease is not clear yet. There are still some epidemiological characteristics that cannot be explained solely by the Se-deficiency hypothesis. Results from epidemiological surveys suggest that viral infection might be another factor. Therefore, the combined effect of Se deficiency and viral infection was studied in mice and their offspring.[4] Weanling mice were divided into a control group (stock diet), two low-Se groups (including grains produced in a Keshan disease area and a semisynthetic diet), and a group supplemented with sodium selenite. Animals in similar groups were mated after six weeks of feeding. The offspring on their seventh day of life were injected intraperitoneally with 0.07 ml of either a virus culture or a virus-free tissue culture. The suckling mice and the adults were sacrificed on the seventh day after injection. Blood selenium content was determined, and pathological examination of viscera was performed. The virus used in these experiments was isolated from the blood of a child who suffered from subacute Keshan disease in 1974. This strain was identified serologically as Coxsackie virus B_4. The results indicated that the offspring of all Se-deficient groups, no matter whether they consumed a natural or a semisynthetic low-Se diet, were much more sensitive to the virus injury. Both the frequency and severity of the heart lesions in the Se-deficient animals were significantly higher than those of the Se-adequate group. Se supplementation depressed virus-induced myocardial necrosis in Se-deficient suckling mice.

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