Calcium serves many functions in the body; accordingly, low calcium intakes contribute to the development and severity of many chronic disorders, in addition to osteoporosis. In the skeleton, low calcium intake has two principal effects: 1) it causes the body to dip into its skeletal calcium reserves in order to offset daily calcium losses through skin and excreta, thereby increasing bony fragility because of reduction in bone mass; and 2) it causes increased secretion of parathyroid hormone, which greatly exaggerates the rate of bone remodeling, thereby decreasing bone strength because of the microarchitectural weakness that accompanies remodeling activity.

Like calcium, vitamin D also has many functions in the body, and the consequences of inadequate vitamin D status are still being explored. In regard to the calcium economy, vitamin D’s presence in adequate amounts in the body is necessary to allow the regulation of calcium absorption from ingested food and supplement sources. It serves this function by enabling the up-regulation of active, transcellular transport of calcium. Because a great deal of calcium enters the gut every day from the body’s calcium stores (in the form of digestive secretions and sloughed intestinal mucosa), some degree of active transport is almost always required if the gut is to be a source (rather than a sink) for calcium.

Daily obligatory losses of calcium from the body in typical adults (through skin and excreta) average about 200 mg. Hence, net absorption of calcium has to be at least that high to provide the body with sufficient calcium so that it does not tear down bone to make up for what it is not getting from diet. In the complete absence of vitamin D, calcium intake would have to be in the range of 2500–3500 mg/d to provide the needed, net absorption. By contrast, active absorption of about 16% lowers that calcium intake requirement to about 1000–1500 mg/d, i.e., precisely the currently recommended range of intakes. Thus current calcium intake recommendations presume vitamin D adequacy.

Calcium absorptive response to vitamin D is not an all-or-nothing affair. Available evidence indicates that absorption efficiency rises with vitamin D status, and that it plateaus at serum 25(OH)D concentrations of about 80 nmol/L, above which further increases in vitamin D status produce no further improvement in calcium absorption. The daily vitamin D input needed to sustain a serum 25(OH)D level of 80 nmol/L is about 4000 IU, most of which comes from cutaneous synthesis of vitamin D from solar UV-B radiation. Individuals deprived of this cutaneous input for long periods of time, for any reason, will require oral inputs much higher than the current AI for vitamin D.