INFECTIONS IN ANIMALS

If vitamin C affects the immune system in a nonspecific manner as an antioxidant, it is probable that the effects are not strictly limited to the respiratory viruses, which in fact consist of half a dozen unrelated viruses with over 100 serotypes. Consequently, it is possible that vitamin C intake affects susceptibility to and severity of infections by some of the nonrespiratory viruses and possibly by some bacteria as well.

Most mammals synthesize vitamin C in the liver. The guinea pig is one of the rare species that have lost the capability to synthesize vitamin C (196,197), and therefore it provides a good experimental model for studies dealing with the effects of low vitamin C levels on susceptibility to infections. Low vitamin C intake has been found to decrease the resistance of guinea pigs to *Mycobacterium tuberculosis* (198-205), other bacteria (2,95,206-211), Rickettsiae (212), *Endamoeba histolytica* (213), and *Candida albicans* (214). Supplementation of guinea pigs with vitamin C has been reported to increase resistance to the rabies virus (215-217). In some studies vitamin C supplementation had no
effects on bacterial infections (218-220), but there is such a large number of experimental variables of potential importance that discrepancies in results are not surprising.

In guinea pigs infected with *M. tuberculosis* vitamin C supplementation slightly increased the hemoglobin level (221). In histological studies fewer caseonecrotic lesions, more collagenous tissue within and around the tuberculous centers, and less dispersion of tubercle bacilli were observed in vitamin C-supplemented animals (202,203,222). Furthermore, in guinea pigs infected with *M. tuberculosis* there was a decrease in vitamin C level in the adrenals, the liver, and urine (223,224).

Primates lack the ability to synthesize vitamin C (196,197). In some studies with rhesus monkeys vitamin C was reported to decrease the incidence of poliomyelitis (225,226), while in one study no effect was observed (227). Nonetheless, in the latter study it was noted that many rhesus monkeys on a scorbutic diet died of spontaneous infections, chiefly pneumonia and enterocolitis, while those receiving adequate amounts of vitamin C remained well (227). In rhesus monkeys vitamin C intake affects the bacterial flora in the oral cavity (228,229). In marmosets vitamin C supplementation decreased the rates of morbidity and mortality due to parainfluenza infection (230). In macaque monkeys malarial infection decreased the vitamin C level in plasma (231).

Fishes require exogenous vitamin C (196,232). In catfish (232) and rainbow trout (233,234) vitamin C supplementation decreased the mortality rate of bacterial and parasitic infections.

Rats and mice synthesize vitamin C in the liver and consequently cannot be used to study the effects of low vitamin C intakes. However, the effect of vitamin C supplementation and the effect of infections on vitamin C metabolism can be studied in these species and in others that synthesize vitamin C. In mice infected with *Pseudomonas aeruginosa* (235) and *Candida albicans* (135) vitamin C supplementation increased the proportion of surviving animals. In mice infected with rodent malaria parasites, vitamin C depressed parasitemia and extended the mean survival time of the infected mice (236). Vitamin C inhibited the multiplication of *Mycobacterium lepra* in mouse foot pads (237). In mice infected with *Streptococcus pneumoniae* vitamin C supplementation enhanced the clearance of bacteria from the lungs, apparently through an increased influx of neutrophils to the lungs; however, the survival rate was not significantly changed in the vitamin C group (238). In rats infected with *Trypanosoma hippicum* there was a decrease in the vitamin C concentration in the liver, spleen, and adrenals, but the level of vitamin C in plasma was doubled (239). In cats vitamin C supplementation decreased the duration of rhinotracheitis (240). In chickens vitamin C supplementation increased the resistance to *Salmonella gallinarum* (241), *E. coli* (242), and viral bronchitis (243).

It is possible that the amount of the infecting agent affects the role of vitamin C intake. If vitamin C has only moderate effects on the immune system it is possible that it shows effects when the infectious dose is rather small, whereas there may be no effect when the infectious dose is very large. In rhesus monkeys vitamin C provided moderate protection when quite a small dose of polio virus was used, while it was without effect when a large dose was used (226), but the number of animals was so small that the conclusion is not strong. In a study with guinea pigs infected with bacteria, it was also pointed out that the infectious dose seemed to affect the role of vitamin C intake (206).

From the studies examining the effects of vitamin C on the immune system and on various animal infections it seems possible that vitamin C intake may have effects on the susceptibility of humans to infections other than the common cold.