Abstract

During high salinity stress, β-alanine accumulates to high levels in the sea anemone,*Bunodosoma cavernata*. Following a salinity increase from 26‰ to 40‰ β-alanine increased 28-fold from 1.5 to 41.9 μmoles/g dry weight. Both whole animal studies and experiments with cell free homogenates indicate that under high salinity conditions an increase in the rate of β-alanine synthesis from aspartic acid as well as a decrease in the rate of β-alanine oxidation are responsible for the observed accumulation of β-alanine. The rate of aspartic acid decarboxylation to β-alanine is about 3 times greater in anemones acclimated to 40‰ than for those in normal salinity water (26‰). β-alanine oxidation to CO_2 and acetyl-CoA proceeds 2.5 to 3 times slower in high salinity adapted*B. cavernata* than in those acclimated to normal salinity. There is always a rapid degradation of uracil to β-alanine, but this does not change with salinity.