ABSTRACT

Many researchers have concluded that secondary or delayed ettringite is responsible for serious premature deterioration of concrete highways. In some poorly performing Iowa concretes, ettringite is the most common secondary mineral but its role in premature deterioration is uncertain since some researchers still maintain that secondary ettringite does not itself cause deterioration. The current research project was designed to determine experimentally if it is possible to reduce secondary ettringite formation in concrete by treating the concrete with commercial crystallization inhibitor chemicals. The hypothesis is such that if the amount of ettringite is reduced, there will also be a concomitant reduction of concrete expansion and cracking. If both ettringite formation and concrete deterioration are simultaneously reduced, then the case for ettringite induced expansion/cracking is strengthened.

Our experiments used four commercial inhibitors – two phosphonates, a polyacrylic acid, and a phosphate ester. Concrete blocks were subjected to continuous immersion, wet/dry and freeze/thaw cycling in sodium sulfate solutions and in sulfate solutions containing an inhibitor. The two phosphonate were effective in reducing ettringite nucleation and growth in the concrete. Rapid experiments with solution growth inhibition of ettringite without the presence of concrete phases were used to explore the mechanisms of inhibition of this mineral.

Reduction of new ettringite formation in concrete blocks also reduced expansion and cracking of the blocks. This relationship clearly links concrete expansion with this mineral – a conclusion that some research workers have disputed despite theoretical arguments for such a relationship and despite the numerous observations of ettringite mineralization in prematurely deteriorated concrete highways. Secondary ettringite nucleation and growth must cause concrete expansion because the only known effect of the inhibitor chemicals is to reduce crystal nucleation and growth, and the inhibitors cannot in any other way be responsible for the reduction in expansion. The mechanism of operation of the inhibitors on ettringite reduction is not entirely clear but our solution growth experiments show that they prevent crystallization of a soluble ettringite precursor gel.

The present study shows that ettringite growth alone is not responsible for expansion cracking because our experiments showed that most expansion occurs under wet/dry cycling, less under freeze/thaw cycling, and least under continuous soaking conditions. It was concluded from the different amounts of damage that water absorption by newly-formed, minute ettringite crystals is responsible for part of the observed expansion under wet/dry conditions, and that reduction of freeze resistance by ettringite filling of air-entrainment voids is also important in freeze/thaw environments.

On the basis of these experiments, we conclude that the reduction of ettringite formation and expansion cracking in highway concretes is chemically feasible in laboratory experiments. The chemicals used in the project are relatively inexpensive and are of low toxicity. We strongly recommend additional research to determine whether treatment of highways with a commercial phosphonate inhibitor will reduce secondary ettringite formation and premature deterioration under field conditions.