PEDIATRICS—T(oxin) OF AUTISM *continued from page 11*

adding urea or an available ammonia source to a fermentation broth. The fermentation process for producing CA is particularly sensitive to concentrations of ammonia in the system. While ammonia is needed to catalyze the fermentation system, it needs to be kept at an acceptably low level in order to avoid the repression of one or more enzymes.

Under general fermentation conditions, where the ammonia levels exceed 100ug/ml, it has been found that ammonia added to the system will act to repress the number of enzymes involved in the metabolism of nitrogen, including urease, which catalyzes the conversion of urea to ammonia and carbon dioxide.[17]

Since urea can accumulate in the CA fermentation broth, it can cause significant problems both in the production and in the potential purity of the CA and its salt which is ultimately used in the pharmaceutical preparation. Therefore, the urea buildup can act as an explosive to the fermentation broth, or can potentially pollute it. Numerous patents have been written to date covering novel ways to produce CA in ways which are more stable and commercially viable for the manufacturer. The use of ammonia in the fermentation broth is therefore exceedingly unstable, and the general purity hovers around the 85% mark which is considered pharmaceutically acceptable.[18,19,20]

Urea and nitrogen poisoning is well-known in the agricultural literature. While much has been written about nitrate and nitrite poisoning in livestock, little has been written about it in humans. There is generally a two-fold effect of urea poisoning: 1) The neurotoxic effect on brain tissue, and 2) the highly alkaline corrosive effects of NH3 levels in the digestive tract. Moderate to acute signs of urea poisoning include abdominal colic, bloating, diarrhea, muscle tremors, incoordination, weakness, poor appetite and other signs generally associated with neurological toxicity. Additionally, high levels of NH_3 in the digestive tract may cause damage to the secretory cells of the small intestine.[21,22,23]

A study by Frick et.al. examined the role of tricarcillin (T) and clavulanate (C) in human neonates. Three groups, one comprised of full-term babies, one comprised of pre-term babies with normal birth weight, and a third of pre-term babies with low birth weight. The ratio of T:C was 25:1. It was found that in groups 1 and 2, the full term and pre-term normal weight, the peak serum concentrations of the drugs was achieved in 15-30 minutes after the infusion. In group 3, the pre-term with low birth weight, the peak serum concentration was found at 120-240 minutes.[24] Since the amoxycillin/CA preparation has a known hepatotoxicity, the fact that the prolongation in group 3 occurred is significant.[21, 24]

In light of these findings, it is important that further studies be undertaken. Since Augmentin™ is one of the most widely prescribed drugs for children and that its introduction into the marketplace for use in the treatment of childhood illnesses corresponds with a significant increase in the incidence of autism, it is imperative that further research be undertaken to determine if a subset of children are at risk for neurotoxicity due to the use of clavanic acid in pharmaceutical preparations.

Autism is a disability of monumental proportions in the pediatric and adult populations. It is therefore crucial that we determine its causative factor. Researchers are looking for what we chiropractors call the T of toxicity. We [chiropractors] have always been aware that toxins can affect mental and physical health and cause severe and debilitating illness. Whether the causative factor of autism be clavanic acid or some other toxin, I believe that examining the T(oxin) of antibiotics is very important not only for the current population of children but to future generations as well.

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Speaker: Richard Christie, D.C.,J.D.

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