The relative resistance of some experimental animal species (mice, rats, pigs, sheep, cattle) to pure T-2 toxin could be explained by natural resistance or alternatively these species may have developed, during thousands of years of evolution, specific enzymes capable of detoxifying trichothecenes because their natural foods have probably contained such toxins on occasion. Other species, such as humans, monkeys or cats are more selective or different in their food habits and therefore may not have had the chance to develop natural resistance. This hypothesis is further strengthened by a recent observation of the effects of T-2 toxin on invertebrates (a). Bertha Army worms which feed on green leaves are highly sensitive to T-2 toxin, whereas yellow mealworm larvae which feed naturally on cereal grains are less sensitive.

3.0 Metabolic and Toxicokinetic Fate of T-2 Toxin

Animal experiments have shown that T-2 toxin is relatively rapidly excreted. After four days of observation, 68 percent of radioactive-labelled T-2 toxin was excreted²). Most of the T-2 toxin is excreted as HT-2 toxin, suggesting that the liver may have been able to transform T-2 toxin into the deacytelated HT-2 toxin^{2,3}).

4.0 Stachybotryotoxicosis

Principally, Stachybotryotoxicosis is a mycotoxicosis of horses and other livestock caused by the toxins of the saprophytic fungus, <u>Stachybotrys alternans</u> or <u>S. atra</u>. In animals, which ingest infested hay or straw, the symptoms are rather similar to ATA in man. The toxic principles, Verrucarin, Roridin and Satratoxins, have been identified as belonging to the group of macrocyclic trichothecenes. Such macrocyclic trichothecenes are also produced by <u>Myrothecium</u> sp., and cause diseases in livestock known as Myrotheciotoxicosis or Dendrodochiotoxicosis.

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