Jenkins, J. N., Maxwell, F. G.; Lafener, H. M. *J. Econ. Entomol.* **1966**, *59*, 352.
Lukefahr, M. J.; L. W. Noble; Houghtaling, J. E. *J. Econ. Entomol.* **1966**, *59*, 817.
Minyard, J. P.; Tumlinson, J. H.; Hedin, P. A.; Thompson, A. C. *J. Agric. Food Chem.* **1965**, *13*, 599.
Minyard, J. P.; Tumlinson, J. H.; Thompson, A. C.; Hedin, P. A. *J. Agric. Food Chem.* **1966**, *14*, 332.
Minyard, J. P.; Thompson, A. C.; Hedin, P. A. *J. Org. Chem.* **1968**, *33*, 909.
Thompson, A. C.; Hanny, B. W.; Hedin, P. A.; Gueldner, R. C. *Am. J. Bot.* **1971**, *58*, 803.

Received for review April 5, 1985. Accepted July 9, 1985. Mention of a trade name, proprietary product, or specific equipment does not constitute a guarantee or warranty by the U.S. Department of Agriculture and does not imply its approval to the exclusion of other products that may be suitable. This article is approved as TA 20555 by the Director, Texas Agriculture Experiment Station.

Nitrosation of Phenol and 2,6-Dimethoxyphenol and Its Effect on Nitrosamine Formation

Mohammad S. Virk and Phillip Issenberg*

We measured modifying effects of the wood smoke constituents phenol and 2,6-dimethoxyphenol (syringol) on the rates of *N*-nitrosomorpholine (NMOR) and *N*-nitrosopyrrolidine (NPYR) formation in vitro. The second-order nitrosation rate constants (pH 3.0, 37 °C) were 127 M^{-1} min^{-1} for syringol and 1.16 M^{-1} min^{-1} for phenol. Under our standard conditions, syringol and ascorbic acid blocked NMOR formation by 91 and 77% and blocked NPYR formation by 71 and 79%, respectively. Phenol did not affect NMOR formation but enhanced NPYR formation by 358%. These results indicate that basicity of the amine and hence the rate of nitrosamine formation may alter the rate-modifying effect of phenol considerably. The results show that syringol can be a potent nitrosation inhibitor.

Formation of carcinogenic nitrosamines is influenced by various factors, including the presence and nature of modifying agents (Issenberg and Virk, 1974; Mirvish, 1975; Douglass et al., 1978; National Academy of Sciences, 1981). Phenols are important modifiers of amine nitrosation, because they are widely distributed in the environment. Furthermore, they are usually reactive with the nitrosating species, with which they react irreversibly to form *C*-nitroso products that may catalyze *N*-nitrosamine formation under acidic conditions (Davies and McWeeny, 1977; National Academy of Science, 1981; Walker et al., 1982). Reactive phenols compete with amines for the available nitrosating agents (Nakamura and Kawabata, 1981; Pignatelli et al., 1984; Kuenzig et al., 1984) and have been found either to enhance or to block *N*-nitrosoproline (NPRO) formation under different conditions (Pignatelli et al., 1982).

Nitrosation of phenol occurs readily in acidic solutions of sodium nitrite, yielding mainly *p*-nitrosophenol, with about 8–10% of the ortho compound (de la Mare and Ridd, 1959; Morrison and Turney, 1960). Substitution of electron-releasing groups such as alkoxyls, particularly at the 2- and/or 6-positions of the phenolic ring, appears to stabilize the positive charge on the carbon atom para to the hydroxyl group and may enhance the nitrosation rate of the phenolic compound.

Issenberg and Virk (1974) reported the blocking of *N*-nitrosomorpholine (NMOR) formation from morpholine (MOR) and nitrite by phenol and syringol, two phenolic components of wood smoke and smoked foods (Lustre and Issenberg, 1970). The nitrosation kinetics of phenol has been studied previously under different conditions (Suzawa et al., 1955; Morrison and Turney, 1960; Challis and Lawson, 1971; Challis, 1973), but the nitrosation kinetics of syringol has not been investigated.

The kinetics of nitrosation of phenol and syringol was examined in the present study to assess the possible role of the phenols in modifying nitrosamine formation rates. The nitrosation of MOR, a weakly basic and easily nitrosatable secondary amine, and of pyrrolidine (PYR), a strongly basic and slowly nitrosatable amine (Mirvish, 1975), was examined. Ascorbic acid was included as a reference inhibitor.

MATERIALS AND METHODS

Reagents. MOR, PYR, and syringol were purchased from Aldrich Chemical Co., Milwaukee, WI; and phenol was purchased from Mallinckrodt, St. Louis, MI; sodium nitrite, from Baker Chemical Co., Phillipsburg, NJ; and ascorbic acid from Fisher Scientific Co., Fair Lawn, NJ. Dichloromethane (DCM) was "distilled in glass" from Burdick and Jackson Inc., Muskegon, MI. NPYR solution (100 g/mL in 2,2,4-trimethylpentane) was purchased from Thermo Electron Co., Waltham, MA; and NMOR was provided by Dr. S. Mirvish (Eppley Institute). All other chemicals were reagent grade materials from commercial sources.

Nitrosation of Phenols. The reaction mixture (147 mL), containing phenol or syringol (4×10^{-4} M), and sodium nitrite (4×10^{-4} M), was adjusted to pH 3.0 with perchloric acid (approximately 6.8×10^{-4} M) and maintained at 37 °C in a water bath. Twenty-milliliter portions, drawn at intervals of 0, 20, 44, 68, 92, and 164 h for phenol and 0, 5, 10, 15, 20, 25, and 50 min for syringol, were transferred to a mixture of 0.5 mL of 1 M Na_2SO_3 (Mirvish et al., 1972) and 0.48 mL of 1 M $HClO_4$ to stop the reaction. A separate experiment, described below, evaluated the effectiveness of sulfite in removing nitrite. Unchanged

Eppley Institute for Research in Cancer and Allied Diseases, University of Nebraska Medical Center, Omaha, Nebraska 68105.

0021-8561/85/1433-1082$01.50/0 © 1985 American Chemical Society

phenols were extracted from the reaction mixture with 2 × 10 mL DCM, and the extracts were analyzed by gas chromatography with flame ionization detection (GC-FID, Model 3700 gas chromatograph, Varian Associates, Palo Alto, CA). Ni alloy columns, 2 m × 2 mm i.d., containing 10% Carbowax 20M-TPA on 100/120 mesh Chromosorb G-AW, were operated at 200 °C. Helium carrier gas flow rate was 29 cm^3/min, injector temperature 210 °C, and detector temperature 220 °C. Retention times were as follows: phenol, 8.4 min; syringol, 10 min. Amounts of phenols in the reaction mixture were computed by the digital integrator (System 1, Spectra Physics, Santa Clara, CA) after the system was calibrated by injecting standard solutions of phenol (1.0×10^{-4} to 4.0×10^{-4} M) and syringol (0.4×10^{-4} to 4.0×10^{-4} M), prepared and analyzed under identical conditions. Second-order rate constants were calculated from the integrated rate equation (Masterton and Slowinski, 1973)

$$x/[a(a - x)] = k_2t$$

where k_2 = second-order rate constant, a = initial concentration of phenol or nitrite, and x = concentration change of the phenol that took place in time t. In separate experiments, nitrite decomposition was measured in reaction mixtures that contained no phenolic compounds.

Effectiveness of Sodium Sulfite as a Nitrosation Quencher. Because of possible reaction of sulfamate with phenols to form sulfonates (Audrieth et al., 1940), we used sodium sulfite to quench the phenol nitrosation reaction mixtures. The effectiveness of sodium sulfite was compared with that of sodium sulfamate by monitoring the syringol absorbance at 268 nm of reaction mixtures containing 16 mL of deionized water, 1.7 mL of 5×10^{-3} M syringol, 2.4 mL of 3.5×10^{-3} M $NaNO_2$, 0.5 mL of 1 M $HClO_4$ (to pH 3.0), and 0.5 mL of 1 M sodium sulfite or sodium sulfamate.

Modification by Phenol and Syringol of the Rate of NMOR Formation. Twenty milliliters of aqueous solutions containing 10 mM MOR, 20 mM phenol, syringol, or ascorbic acid, and 12–20 mM $HClO_4$ (pH 3.0) were maintained at 37 °C in a thermostated water bath. The reaction was started by adding $NaNO_2$ (20 mM, pH 3). After 90 min, the reaction was stopped by adding 4 mL of an aqueous mixture of ammonium sulfamate (0.6 M) and $HClO_4$ (2.4 M). The mixture was made alkaline with 5 mL of 5 M NaOH to prevent interference by phenol or syringol in the subsequent GC analysis and extracted with 2 × 10 mL DCM.

The influence of pH on the blocking of NMOR formation by syringol and by ascorbic acid was determined in a reaction mixture containing 10 mM MOR, 20 mM nitrite, 20 mM syringol or ascorbic acid, and $HClO_4$ to adjust pH in the range of 1.0–5.0 in 0.5 pH increments. The temperature was 25 °C, and reaction time was 90 min.

The DCM extracts were analyzed by GC-FID. A Ni alloy column, 2 m × 2 mm i.d. containing 10% Carbowax 20M-TPA on 100/120 mesh Chromosorb G-AW, was operated at 190 °C. The helium carrier gas flow rate was 26 cm^3/min. The temperature of the injector was 200 °C, and that of the detector, 210 °C. NMOR concentrations were computed by the digital integrator, after the system was calibrated by injecting the NMOR standards (0.3×10^{-3} to 5.0×10^{-3} M) prepared and analyzed under identical conditions.

When the effect of pH on the NMOR yields was determined, the DCM extracts were analyzed by GC-FID. A Ni alloy column, 2 m × 2 mm i.d. containing 10% SP-1000 on Supelcoport, was operated at 175 °C with helium carrier gas flow rate of 30 cm^3/min. Peak heights were measured and amounts of NMOR were computed from response factors determined with standard solutions (8–800 μg/mL) analyzed under identical conditions.

Modification by Phenol and Syringol of the Rate of NPYR Formation. The reaction system contained 10 or 50 mM PYR, 20 or 100 mM phenol, syringol, or ascorbic acid, and perchloric acid (10 or 40 mM). The reaction was started by adding sodium nitrite (20 or 100 mM). The 20-mL reaction mixture was maintained at 25 or 37 °C in a constant-temperature water bath and kept at pH 3.0 during the reaction period with 280 mM citrate–phosphate buffer or adjusted to pH 3.0 with $HClO_4$. The reaction was stopped after 90 min by adding 4 mL of an aqueous solution of ammonium sulfamate (0.6 M) in $HClO_4$ (2.4 M). Five milliliters of 5 M NaOH was added, and the solution was extracted with 2 × 10 mL DCM. The extracts were analyzed by GC (Model 2200 gas chromatograph, Bendix Process Instruments Division, Ronceverte, WV) on a 2 M × 2 mm i.d. glass column containing 10% Carbowax 20M-TPA on 100/120 mesh Chromosorb G operated at 190 °C. The injector temperature was 210 °C, and the He flow rate was 20 cm^3/min. A thermal energy analyzer (Model 502, Thermo Electron Co., Waltham, MA) detector was used. Amounts of NPYR in the test samples were computed by the digital integrator after the system was calibrated by injecting a standard solution (1 μg/mL) of NPYR.

Table I. Rate Constants for Nitrosation of Phenol and Syringol at 37 °C, pH 3.0

compd	reactn time, min	extent of reactn, %	% nitrite dec	second-order rate const, M^{-1} min^{-1}
phenol	1200	30	43	0.89[a] (0.09)[b]
syringol	15	32	4	127 (48)

[a] Numbers in parentheses are standard deviations computed from three experiments with phenol and four experiments with syringol. [b] Correction for nitrite decomposition yields a value of 1.16 ± 0.12 M^{-1} min^{-1}.

RESULTS AND DISCUSSION

Rate Constants for Nitrosation of Phenol and Syringol. Rate constants for nitrosation of phenol and syringol are shown in Table I. The progress of the reaction was measured by the disappearance of phenol from the reaction mixture. The magnitude of the second-order rate constant for phenol nitrosation at 37 °C decreased from 0.89 to 0.27 M^{-1} min^{-1} during the 20- to 164-h time period. Nitrite decomposition was 43% at 20 h and 93% at 164 h. The rate constant at 20 h reflects the minimum effect of nitrite decomposition in this experiment. Correction for nitrite decomposition over the 20-h interval yielded a rate constant of 1.16 ± 0.12 M^{-1} min^{-1}, which did not differ significantly from the uncorrected value. Challis and Lawson (1971) reported a rate constant of 0.054 M^{-1} min^{-1} for nitrosation of phenol at 0.7 °C in 10^{-3} M $HClO_4$.

Nitrite decomposition during the 50-min syringol nitrosation experiment was 13%, and that during the first 15 min was 4%. Therefore, correction for nitrite decomposition was not necessary. The rate constant at 15 min was chosen to allow comparison (Table I) at a similar extent of reaction. The methoxyl groups at the 2- and 6-positions of the aromatic ring of syringol appear to increase the fractional negative charge on the –OH and thus enhance electrophilic attack by N_2O_3 compared with phenol. This observation supports Challis' (1973) suggestion that substituted phenols are more reactive toward nitrite than phenol.

Effectiveness of Sodium Sulfite as a Nitrosation Quencher. Absorbance at 268 nm of the mixture con-

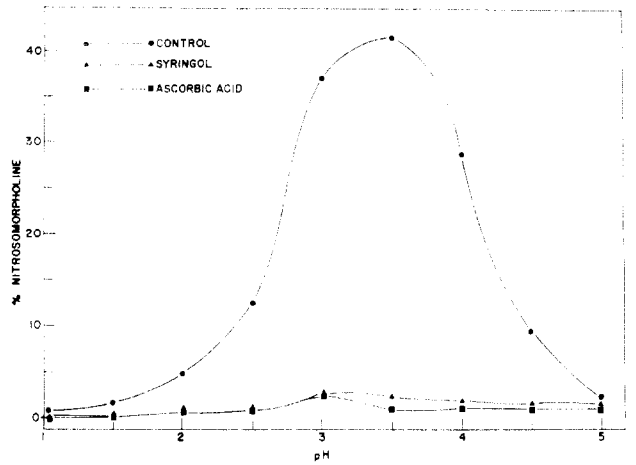

Figure 1. Effect of syringol and ascorbic acid on yield of NMOR. The aqueous reaction mixture contained the following: MOR, 10 mM; nitrite, 20 mM; phenol or ascorbic acid, 20 mM. $HClO_4$ was added to adjust pH. Temperature was 25 °C, and reaction time was 90 min.

Table II. Effect of Phenol and Syringol on NMOR Formation[a]

modifier	no. of samples	NMOR formed, % ±SD	blocking of NMOR formn, % ±SD
none	5	48 ± 3	
phenol	3	44 ± 1.5	8 ± 3
syringol	2	4 ± 0	91 ± 0
ascorbic acid	5	11 ± 4	77 ± 8

[a] Aqueous reaction mixture contained the following: MOR, 10 mM; modifier, 20 mM; sodium nitrite, 20 mM; perchloric acid, 12–20 mM, pH 3.0. Temperature was 37 °C, and reaction time was 90 min.

taining syringol, nitrite, and sodium sulfite decreased 2% during the 1-h observation period. The molar absorptivity of syringol under these conditions was 1195 M^{-1} cm^{-1}. Absorbance of the mixture containing ammonium sulfamate increased 8% under the same conditions. The sulfamate-containing mixture developed a pink color with Griess reagent, indicating the presence of residual nitrite. No color was observed when the sulfite-containing solution was treated with Griess reagent. Both sulfite and sulfamate reduced nitrite concentration in the reaction mixtures, but the results above show sulfite to be a more effective quenching agent in the phenol/nitrite reaction mixture.

Effect of Modifiers on NMOR Formation from MOR and Nitrite. Figure 1 shows that the blocking effects of syringol and ascorbic acid at pH 3.0 were true reductions in amounts of NMOR formed and not due to shifts of the optimum pH for the reaction. The results in Table II show that the blocking effect of syringol on NMOR formation was 9 times greater than that of phenol. These results are consistent with the rate constants for nitrosation of the phenols. The blocking by syringol is at least as effective as that by ascorbic acid.

Effect of Modifiers on Rate of NPYR Formation from PYR and Nitrite. The results in Tables III and IV indicate that the rate-modifying effects of syringol and of ascorbic acid on NPYR formation were similar, although concentration of the precursors and temperature in the two experiments differed. We do not know why the enhancing effect of phenol decreased substantially when the concentration of the precursors was increased 5-fold and the temperature was increased to 37 °C (Table IV). The blocking effect of syringol and ascorbic acid on NPYR

Table III. Effect of Phenol and Syringol on NPYR Formation at 25 °C[a]

modifier	NPYR formed, %	blocking of NPYR formn, %	enhancement of NPYR formn, %
none	0.11, 0.12		
phenol	0.54, 0.56		350, 367
syringol	0.03, 0.04	67,75	
ascorbic acid	0.02, 0.03	75,83	

[a] Aqueous reaction mixture contained the following: PYR, 10 mM; modifying agent, 20 mM; sodium nitrite, 20 mM; perchloric acid, 10 mM, added to adjust to pH 3.0. The reaction time was 90 min.

Table IV. Effect of Phenol and Syringol on NPYR Formation at 37 °C[a]

modifier	NPYR formed, % ±SD[b]	blocking of NPYR formn, % ±SD	enhancement of NPYR formn, % ±SD
none	10 ± 1		
phenol	14 ± 2		40 ± 29
syringol	4 ± 1	60 ± 8	
ascorbic acid	3 ± 0	70 ± 0	

[a] Aqueous reaction mixture contained the following: PYR, 50 mM; sodium nitrite, 100 mM; modifying agent, 100 mM; perchloric acid, 40 mM, added to adjust to pH 3.0; buffer (citric acid, 240 mM + phosphate, 100 m*M*). The reaction time was 90 min. [b] Standard deviations of three samples.

formation was consistent with that observed on NMOR formation (Table II), indicating that amine basicity or other structural differences did not influence the modifying effect of these compounds profoundly. Phenol caused a substantial increase in NPYR formation. Presumably, the relatively slow reaction of PYR with nitrite permits accumulation of larger concentrations of 4-nitrosophenol. Calculations based on the rate equation for nitrosation of phenol and measured rate constant indicate that 4-nitrosophenol concentration was 14 mM after 90 min under the conditions employed in this experiment (Table IV). The presence of 4-nitrosophenol at this concentration appears to be responsible for the observed enhancement of NPYR formation. Walker et al. (1979) and Pignatelli et al. (1982) reported an enhancing effect of 4-nitrosophenol on nitrosodiethylamine and NPRO formation. Davies and McWeeny (1977) attributed the catalytic activity of 4-nitrosophenol to formation of the tautomeric quinone monoxime. Syringol was at least as effective as ascorbic acid in blocking NPYR formation. Its effectiveness results from the rapid rate of nitrosation of syringol to form products such as 4-nitrososyringol, which are not reactive or which react rapidly to form inert products. Formation of a quinone oxime from nitrososyringol is unlikely.

This study demonstrates that the inhibitory effects of syringol and of ascorbic acid on the formation of NMOR and NPYR are comparable. The presence of syringol in smoked foods may contribute to reduced nitrosamine formation in these products.

ACKNOWLEDGMENT

We thank Douglas Klein for his technical help on the use of the TEA and Dr. Sidney Mirvish for providing NMOR and for his useful comments.

Registry No. NMOR, 59-89-2; NPYR, 930-55-2; PhOH, 108-95-2; syringol, 91-10-1; ascorbic acid, 50-81-7.

LITERATURE CITED

Audrieth, I. F.; Sveda, M.; Sisler, H. H.; Butler, M. *Chem. Rev.* **1940**, *26*, 49.

Challis, B. C. *Nature* **1973**, *244*, 466.

Challis, B. C.; Lawson, A. J. *J. Chem. Soc. B* **1971**, 770.
Davies, R.; McWeeny, D. *Nature* **1977**, *266*, 657.
de la Mare, P. B. D.; Ridd, J. H. "Aromatic Substitution-Nitration and Halogenation"; Academic Press: New York, 1959; p 94.
Douglass, M. L.; Kabacoff, B. L.; Anderson, G. A.; Cheny, M. C. *J. Soc. Cosmet. Chem.* **1978**, *29*, 581.
Issenberg, P.; Virk, M. S. "Abstracts of Papers", 168th National Meeting of the American Chemical Society, Atlantic City, NJ, Sept 1974; American Chemical Society: Washington, DC, 1974; Abstr. AGFD 53.
Kuenzig, W.; Chau, J.; Norkus, E.; Holowaschenko, H.; Newmark, H.; Mergens, W.; Conney, A. H. *Carcinogenesis* **1984**, *5*, 309.
Lustre, A. O.; Issenberg, P. *J. Agric. Food Chem.* **1970**, *18*, 1056.
Masterton, W. L.; Slowinski, E. J. "Chemical Principles"; W. B. Saunders Co.: Philadelphia, PA, 1973; p 379.
Mirvish, S. S.; Wallcave, L.; Eagen, M.; Shubik, P. *Science (Washington, D.C.)* **1972**, *177*, 65.
Mirvish, S. S. *Toxicol. Appl. Pharmacol.* **1975**, *31*, 325.
Morrison, D. A.; Turney, T. A. *J. Chem. Soc.* **1960**, 4827.
Nakamura, M.; Kawabata, T. *J. Food Sci.* **1981**, *46*, 306.
National Academy of Sciences "The Health Effects of Nitrate, Nitrite, and *N*-Nitroso Compounds", National Academy Press: Washington, DC, 1981; Chapters 1 and 4–8.
Pignatelli, B.; Bereziat, J. C.; Descotes, G.; Bartsch, H. *Carcinogenesis* **1982**, *3*, 1045.
Pignatelli, B.; Scriban, R.; Descotes, G.; Bartsch, H. *J. Am. Soc. Brew. Chem.* **1984**, *42*, 18.
Suzawa, T.; Yasouka, Z.; Manabe, O.; Hiyama, H. *Science Ind. (Jpn.)* **1955**, *29*, 7.
Walker, E. A.; Pignatelli, B.; Castegnaro, M. *J. Agric. Food Chem.* **1979**, *27*, 393.
Walker, E. A.; Pignatelli, B.; Friessen, M. *J. Sci. Food Agric.* **1982**, *33*, 81.

Received for review April 19, 1985. Accepted August 8, 1985. This research was supported by U.S. Public Health Service Grants P01-CA25100 and 1P30-CA36727 from the National Cancer Institute. The thermal energy analyzer was loaned to us by the National Cancer Institute under Contract N01-CP 33278.

Glucuronide Conjugates of T-2 Toxin and Metabolites in Swine Bile and Urine

Richard A. Corley,*[1] Steven P. Swanson, and William B. Buck

Metabolite profiles in the bile and urine of two swine were determined following the intravascular administration of tritium-labeled T-2 toxin. A total of 13.1 and 1.3% of the dose was found in the gallbladders in addition to 17.9 and 42.5% in the urine of the two swine 4 h after dosing. Free metabolites represented less than 20 and 30% of the total metabolite residues in bile and urine, respectively, with the parent compound, T-2 toxin, never exceeding 0.25%. The major free metabolites were 3′-OH HT-2 and T-2 triol. Glucuronide conjugates represented 63 and 77% of the metabolite residues in urine and bile, respectively. The major conjugated metabolites were glucuronides of HT-2, 3′-OH T-2, 3′-OH HT-2, and T-2 toxin. Neosolaniol, 4-deacetylneosolaniol, and T-2 tetraol were also identified in addition to three unknown metabolites.

INTRODUCTION

T-2 toxin, 4β,15-diacetoxy-8α-[(3-methylbutyryl)oxy]-3α-hydroxy-12,13-epoxytrichothec-9-ene, is a toxic fungal metabolite produced by several species of *Fusaria* (Bamburg and Strong, 1971; Pathre and Mirocha, 1977). T-2 toxin has been found in naturally contaminated corn, barley, and mixed feeds in the U.S. and Canada at concentrations of 0.076–25 ppm (Vesonder, 1983). When present in the diets of livestock and poultry, T-2 toxin has been associated with feed refusal, infertility, diarrhea, intestinal irritation, and possibly hemorrhage, perioral and pharyngeal irritation, and lowered immunity (Hsu et al., 1972; Palyusik and Koplik-Kovacs, 1975; Speers et al., 1977; Weaver et al., 1977; Weaver et al., 1978a; Weaver et al., 1978b; Rafai and Tuboly, 1982; Hoerr et al., 1982). Trichothecene mycotoxins, including T-2 toxin, and their effects on humans have attracted considerable international attention because of their possible use in chemical warfare as the agent "Yellow Rain" (Rosen and Rosen, 1982; Mirocha et al., 1983; Watson et al., 1984).

Many procedures have been reported for the analysis of T-2 toxin in grains and mixed feeds (Scott, 1982). Analytical procedures designed to detect T-2 toxin alone in body fluids, excrement, or tissues will probably fail to confirm exposure since several studies on the fate of T-2 toxin in laboratory animals, poultry, and livestock have demonstrated that the parent compound, T-2 toxin, is rapidly cleared from body fluids and tissues (Chi et al., 1978; Matsumoto et al., 1978; Robison et al., 1979; Yoshizawa et al., 1981). Toxicokinetic studies of T-2 toxin in growing gilts and heifers (Beasley, 1984) demonstrated that the disappearance of intravascularly administered T-2 toxin follows a two-compartment open model with mean plasma elimination phase half-lives of 13.8 min for swine and 17.4 min for cattle. In spite of administration of a lethal oral dose in swine (2.4 mg/kg) and a toxic oral dose in calves (3.6 mg/kg), no parent T-2 toxin was detected in plasma or urine at a detection limit of 25 ng/mL. These results indicate that the parent compound, T-2 toxin, is very rapidly eliminated in all species examined.

Studies of the in vivo metabolism of tritium-labeled T-2 toxin in laboratory animals, livestock, and poultry have demonstrated that hydroxylation at the 3′-carbon position and hydrolysis of ester linkages are important biochemical

Department of Veterinary Biosciences, College of Veterinary Medicine, University of Illinois, Urbana, Illinois 61801.

[1] Present address: Mammalian and Environmental Toxicology Research Laboratory, Dow Chemical, Midland, MI 48640.

0021-8561/85/1433-1085$01.50/0 © 1985 American Chemical Society