

Drug contamination of the equine racetrack environment: a preliminary examination

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Advances in analytical technology now make it feasible to detect and confirm exceptionally low concentrations (pg to fg/mL) of drugs and their metabolites in equine biological fluids. These new capabilities complicate the regulatory interpretation of drug positives and bring into question the fair application of medication rules. Such approaches and policies are further complicated by the possibility that drug positives may arise from contamination of the equine environment on the backstretch of the race track. This manuscript provides data demonstrating that the general environment of the backstretch in which horses live is contaminated with therapeutic drugs and drugs of human origin. The major contaminants are nonsteroidal anti-inflammatory drugs, such as flunixin, phenylbutazone and naproxen, present in the soil in stalls, on stall surfaces, in the dust that circulates and in the lagoon waters that accumulate on the backstretch. The presence of caffeine and cotinine suggest other possible vectors for contamination by humans. Concentrations of these compounds as well as their frequency of occurrence are provided.

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INTRODUCTION

The history of drug testing in the horse racing industry has closely mirrored the evolution of analytical technology (Stanley & Kollias-Baker, 1997). These advances in analytical technology have produced a 10^2 - to 10^6 -fold improvement in detection, depending on the instruments or immunoassays used. However, there is evidence that our instrumentation and methods have become so sophisticated and sensitive that we may now be detecting concentrations of drugs in equine urine and/or blood samples that arise from contaminants that may be common to both the human and equine environment, such as caffeine, cocaine and other compounds of both natural and human origins. In this regard, the present study is a preliminary examination of the contamination of the backstretch of a racetrack by drugs, with samples being collected from the floors of test barn and ship-in stalls, from wipes of stall walls, from the water waste created from washing out stalls and from the dust that collects after circulating in the track environment.

MATERIALS AND METHODS

Collection of samples from the backstretch

One of four racetracks in the State of Louisiana that had recently (within 6 months) been rebuilt following a hurricane, and for which racing was underway, was selected for examination.

Samples from the floor of test barn ($n = 5$) and ship-in ($n = 5$) stalls were collected in the following manner:

Stalls to be sampled were selected arbitrarily. All stalls had been recently 'cleaned', with raking and replacement of bedding, and were unoccupied at the time of sample collection. Wearing latex gloves and using a prewashed (water, methanol) stainless steel spatula, the technician selected an area inside each stall and gently brushed aside the wood shavings. **An effort was made to collect samples from the middle of the stalls in each case. All areas selected were dry, intentionally avoiding any areas considered to have been recently dampened or contaminated by urine and/or feces.** An approximately 40 g sample of the

underlying surface, collected over an approximately 10 cm × 10 cm area and less than 1 cm deep, was scraped into a 50 mL Nalgene, screwcap tube (Fisher Scientific, Pittsburgh, PA, USA), the cap closed and the tube labeled to identify the type of stall and its sequence in the collection. Gloves and spatulas were replaced prior to each new collection.

Water samples ($n = 4$) from the lagoons located near private barns on the backstretch of the track were collected in 50 mL Nalgene screwcap tubes. These lagoons capture the runoff from rain in the general area as well as the washings from the barns. Wearing latex gloves, the technician selected an area at the edge of each lagoon from which to collect the water sample. The cap was removed from the tube and the tube was submerged to allow collection of approximately 50 mL of water. The tubes were then capped and excess water was removed from the outside of the tube using a paper hand towel. The tubes were then labeled as to their location and collection sequence. Gloves were replaced prior to each new collection.

Dust samples ($n = 3$) were collected from the rafters of the ship-in stall barn. Wearing latex gloves and using a prewashed stainless steel spatula, the technician selected a rafter section inside the stall barn. Using a ladder to access the area, an approximately 20 g sample of the dust overlaying the rafter surface was scraped into a 50 mL Nalgene, screwcap tube, the cap closed and the tube labeled to identify the area of the stall and its sequence in the collection. Samples from the middle and ends of the barn were collected in sequence. Gloves and spatulas were replaced prior to each new collection.

Wipe samples were collected from the walls of the test barn and ship-in stalls also selected for collection of floor samples, as described above. Wearing latex gloves and using an ethanol-soaked Kimwipe (Fisher Scientific, Pittsburgh, PA, USA), the technician selected an area inside each stall and gently wiped the surface, covering an approximately 30 cm-by-30 cm total area. **An attempt was made to collect representative samples from all sides of the stalls in an arbitrary manner. Areas obviously contaminated with feces were intentionally avoided.** The wipe was placed into a 50 mL Nalgene, screwcap tube, the cap closed and the tube labeled to identify the type of stall and its sequence in the collection. Gloves were replaced prior to each new collection.

All samples were transported at room temperature to the Equine Medication Surveillance Laboratory at the School of Veterinary Medicine (SVM) of the Louisiana State University (LSU) in Baton Rouge, LA, for analyses. Lagoon water and wipe samples were stored refrigerated (4 °C) until analyzed. Floor soil samples were stored at room temperature.

Extraction and qualitative analysis of samples from the backstretch

Optima grade water, methylene chloride, ethyl acetate, isopropanol, methanol and 15 mL polypropylene and glass tubes were obtained from Fisher Scientific (Pittsburgh, PA, USA). Toxi-A[®] and Toxi-B[®] tubes were acquired from Varian Inc. (Lake Forest, CA, USA). Glucuronidase-sulfatase (*Patella vulgata*), mepivacaine hydrochloride, lidocaine hydrochloride, naproxen, flunixin

meglumine, phenylbutazone, caffeine, cotinine hydrochloride, furosemide, BSTFA with 1% TMCS, ascorbic acid, sodium carbonate, sodium acetate and ammonium hydroxide were obtained from Sigma-Aldrich (St Louis, MO, USA). Deuterated phenylbutazone (d_9) was obtained from Neogen Corporation (Lexington, KY, USA).

For the initial screening of stall floor and dust samples by GC/MS the following procedures were used:

Stall and dust samples (2.0 g) were placed in a polypropylene tube (15 mL) to which 12.0 mL of Optima grade water were added. The samples were placed on a roto-rack for 10 min and then centrifuged for 10 min. An aliquot of the supernatant (5 mL) was transferred to a second 15 mL conical polypropylene tube and 3 mL of glucuronidase were added (*Patella vulgata*; 1250 units/mL in pH 5 sodium acetate buffer). The tubes were capped and incubated at 65 °C for 1 h with gentle shaking. The samples were cooled and 5 mL of the sample were transferred to a Toxi-A tube (extraction of base/neutral drugs). The samples were capped and roto-racked for 10 min, centrifuged for 10 min (3000 *g*) and the upper organic layer transferred to a 5 mL conical glass tube. The solvent was removed by means of a dry nitrogen stream in a heated (30 °C) water bath. The dried residue was derivatized at 65 °C for 20 min using BSTFA containing 1% TMCS, placed into autosampler injection vials and submitted for total ion GC/MS analysis.

Stall floor and dust samples (2 g) were also extracted and derivatized in the same manner using Toxi-B tubes (acid/neutral drugs). Lagoon samples (5 mL each) were treated similarly, except glucuronidase was added directly and no dilution of the samples occurred otherwise prior to extraction with both Toxi-A and -B tubes.

Due to the relatively small surface area sampled, it was decided that all wipe samples (10) should be combined. Each wipe was transferred to a beaker and water (2 mL) was added. The sample was mixed on an orbital shaker for 10 min. The wipe and water extract were transferred to a 20 mL plastic syringe, with the plunger removed. The plunger was then reinserted and depressed to express the remaining water from the wipe. This was repeated for all of the wipes and the effluent from each was combined to afford a single sample of approximately 20 mL in volume. The sample was split into two tubes and treated as described for the lagoon samples above. Remaining aliquots were used for subsequent quantitative analyses.

Quantitative analysis of soil, lagoon and wipe samples

Following total ion GC/MS screening of the samples for various acid, base, neutral and amphoteric drugs, positive samples were re-extracted starting from fresh aliquots, along with surrogate matrix (negative soil collected from a residential site known to be drug free) fortified samples and internal standards, to provide quantification. Thus, blank soil (1 g) was fortified at 0, 10, 25, 50, 100, 250, 500 and 1000 ng/g or mL of the target analytes (flunixin, naproxen, phenylbutazone and caffeine) with and without the 250 ng/mL fortification of the internal standard, d_9 -phenylbutazone (d_9 -PBZ). For samples, 1 g of soil was placed

in a 10 mL conical glass tube and fortified with internal standard. Water was added (5.0 mL) and the sample was brought to pH 9.0 with dilute ammonium hydroxide. The samples were then roto-racked for 10 min and centrifuged for 10 min. The supernatant was transferred to a clean tube and adjusted to pH 4–5 with glacial acetic acid. Methylene chloride (5.0 mL) was added and the mixing and centrifuging steps repeated. The aqueous layer was aspirated to waste and the methylene chloride layer was evaporated to dryness.

Lagoon and wipe sample-extracts were similarly treated except the initial pH 9 extraction was not necessary. Thus, 5 mL of lagoon water or extract were placed in a 10 mL tube and treated and extracted as described above. Samples blanks and controls were prepared from Optima grade water.

Samples were analyzed by GC/MS both un-derivatized (extract residue dissolved in 150 μ L of dichloromethane) and derivatized. Thus, following GC/MS analysis of the un-derivatized extract, the aforementioned methylene chloride solutions were dried under nitrogen while remaining in the vial inserts, mixed with 50 μ L of BSTFA/1% TMCS and heated at 65 °C for 20 min. Dichloromethane (100 μ L) was then added, the sample mixed and submitted for a second GC/MS analysis.

Standards as well as quality control samples (method blank, soil minus internal standard) were extracted and handled as described above.

Immunoassay screens of samples

The initial aqueous extracts obtained from the stall floor and dust samples, the lagoon samples and the extract from the wipes were further analyzed by enzyme linked immunoassay tests. Following manufacturer's instructions, 20 μ L of each sample were assayed in the following kits: (TCC, Testing Components Corporation, Ithaca, NY, USA) amphetamines, atropine, azaperone, barbiturates, buprenorphine, butorphanol, dezocine, eltenac, etamiphylline, fentanyl, indomethacin, levallorphan, lidocaine, meperidine, methadone, generic opiates, zolpidem, (Neogen Corporation, Lexington, KY, USA) generic broncodilators, flunixin, phenylbutazone, generic promazines and furosemide. The 96-well format plates were read on a EL808 Biotek Instruments (Winooski, VT, USA) microplate reader. A positive response was considered to be any reading that was less than 50% of the appropriate blank control. A negative response was any reading that was within 50% of the negative control. Suspect samples were repeated in triplicate using the same criteria.

GC/MS analyses of samples

Gas chromatographic/mass spectrometric (GC/MS) analyses (qualitative and quantitative) for samples prepared from floor soil, dust, lagoon water and wipes were conducted using an Agilent 5973N GC/MSD. Samples were injected (1 μ L) using an injection port temperature of 250 °C and an injection port purge function activated at 0.5 min. The column was a 30 m, 0.2 mm i.d. DB5 (Agilent Technologies, Santa Clara, CA, USA) maintained at a flow rate of 1 mL/min of ultra-high purity helium

and heated with a temperature program; 50 °C, holding for 1 min and then increasing 30 °C to 300 °C/min and holding for 5 min. For the initial survey of samples the mass spectrometer was operated in the electron impact, total ion, positive ion mode at a sampling rate of 2.67 scans/s. The resultant scans were examined manually to identify the peak responses using both a reference mass spectral data base (National Institute of Standards and Technology, NIST; Agilent Technologies) and reference standards identified above.

Targeted drug analyses for quantitation were conducted in the positive, selected ion monitoring (SIM) mode using three diagnostic ions (m/z) for each compound (PBZ = 77, 183, 308 or (TMS) 246, 337, 380, d_9 -PBZ (TMS) = 248, 339, 390, naproxen (TMS) = 185, 243, 302, flunixin (TMS) = 263, 353, 368 and caffeine = 77, 109, 194) at a sampling rate of 3–9 cycles/s. Ion ratios were compared to reference standards and one ion was used for each for quantitation (PBZ = 183 or 380 (TMS), naproxen = 185, flunixin = 353, caffeine = 194).

To provide a measure of the relative amounts of drug being detected in the samples and to establish retention times, pure standards of the drugs were run before and after the samples. Initial estimates of drug concentrations were determined during the screening process and were based on the ratios of areas of total ion peaks for each to these standards. These estimates were used to establish a range for the standard curves subsequently created to quantitate the drugs following re-extraction and GC/MS analysis. Analyses for quantitation by GC/MS were conducted as described above. Peak areas of a selected diagnostic ion for each drug were compared to that of the appropriate internal standard and quantified against the appropriate standard curve for that drug, created by plotting the ratio of peak areas to that of the internal standard as a function of concentration.

Quantitation was accomplished by plotting the ratio of intensity of fortified standards to the intensity of the internal standard and comparing the results found in samples to the resulting line equation.

RESULTS

The results of extraction and analyses of stall floor samples from test barn and ship-in stalls, from 'lagoon' water, dust and wall wipes from the backstretch of a Louisiana racetrack are shown in Table 1. The drugs commonly observed in the samples were predominantly NSAIDs. However, caffeine was also observed as was the major metabolite of nicotine, cotinine (data not shown), which was observed in all samples tested. Table 1 also provides the quantitative results obtained as well as the frequency with which specific drugs were found. In some samples, detectable concentrations of some of the drugs were not initially observed in the total ion screen but were subsequently detected and quantified when analyzed in the selected ion monitoring mode.

Standard curves for the target analytes were linear (phenylbutazone = 0.993, flunixin = 0.994, naproxen = 0.994 and

Table 1. Concentrations (ng/g or mL) of drugs found in samples collected from the backstretch of a horse racetrack in Louisiana

Drug	Sample				
	Lagoon water (n = 4)	Test barn stalls (n = 5)	Ship-in stalls (n = 5)	Dust (n = 3)	Wipes (n = 10 = 1*)
Phenylbutazone	<1	ND	28.7	46.3	0.09
	1.0	55.2	34.3	31.5	
	<1	27.2	39.6	43.6	
	<1	46.0	85.8		
Flunixin		18.1	53.6		
	9.8	ND	78.8	5.8	1.04
	12.0	29.4	39.8	3.2	
	11.6	11.7	0.8	5.1	
Naproxen	3.0	15.5	251.1		
		ND	83.2		
	<1	1.6	1.8	ND	0.16
	<1	1.9	2.8	ND	
Caffeine	<1	1.9	1.9	ND	
	1.5	ND	97.5		
		1.7	2.6		
	1.9	13.8	9.6	ND	1.68
	2.1	ND	ND	ND	
	3.2	12.3	18.1	ND	
	3.7	ND	ND		
		7.1	15.5		

ND = not detected.

*The 10 wipe samples were combined into a single sample; values are expressed as ng/cm² with the total surface area of approximately 900 cm² having been sampled.

caffeine = 0.996) and bracketed the concentrations observed in the samples tested. Estimated limits of detection (3× baseline noise) were within an acceptable range (phenylbutazone = 0.2 ng/mL, flunixin = 0.5 ng/mL, naproxen = 0.8 ng/mL and caffeine = 0.5 ng/mL) for each compound. Absolute recoveries were not determined but were linear with concentration and linear relative to the internal standard.

The concentrations of each of the drugs found in the samples varied from undetected to low ng/g or mL to low µg/g or mL and were of sufficient quantity as to provide discernible full-scan spectra in many cases.

Based on relative peak area to internal standard, compared to extracted reference standards, flunixin in the lagoon water samples ranged from approximately 3 to 12 ng/mL and phenylbutazone was in the low ng/mL range, as was caffeine.

The composite sample of wall wipes, covering an approximately 30 cm-by-30 cm total area and eluted in a final volume of 20 mL of extraction solvent, contained approximately 76 ng/mL (1.68 ng/cm²) of caffeine and 47 ng/mL of flunixin (1.04 ng/cm²), with low ng/mL quantities of phenylbutazone and naproxen.

Dust samples were positive for phenylbutazone and flunixin and contained these compounds in the 3.2–46.2 ng/g range.

As may be expected, the highest concentrations of drugs were to be found in the samples collected from the floors of the stalls.

Overall, the concentrations of phenylbutazone, flunixin or naproxen ranged from undetected to 251.1 ng/g, with the highest concentrations being observed for flunixin (251.1 ng/g) and naproxen (97.5 ng/g) in the samples examined.

Although samples from the initial screening were treated with sulfatase–glucuronidase and efforts were made to detect them, no major metabolites of any of the parent compounds were observed. Thus, the analyses for quantitation were conducted without the use of enzyme and reflect the concentrations of free, parent drug found.

Immunoassay analyses for the following drugs or drug classes were negative (suppression of response <50% of control): amphetamines, atropine, azaperone, barbiturates, buprenorphine, butorphanol, dezocine, eltenac, fentanyl, indomethacin, levallorphan, lidocaine, meperidine, methadone, generic opiates, zolpidem, generic promazines and generic broncodilators. Positive responses (suppression of response by more than 50% of control) were observed for etamiphylline (which cross-reacts with caffeine), flunixin, phenylbutazone and furosemide immunoassays and corresponded to subsequent results obtained by GC/MS. Furosemide was present in three-of-four lagoon water samples, as indicated by immunoassay and by separate instrumental analysis (data not shown). However, the concentrations of furosemide were estimated to be 2 ng/mL or less.

DISCUSSION

Possible sources of drug contamination of horses are numerous and can be divided into three basic categories: (1) sample collection and handling (Sams, 1997; Kollias-Baker, 2002), (2) veterinary and pharmaceutical (Russell & Maynard, 2000; Ernst, 2005) and (3) environmental (Galey *et al.*, 1996; Harner & Herrman, 1996; Sams, 1997; Kollias-Baker, 2002; Camargó *et al.*, 2005). Given our ever increasing capabilities to detect ever smaller concentrations of drugs, all of these sources of contamination should be of concern to the industry and its regulators.

The data presented here document a further source of contamination, one that many would be aware of but for which no actual measurements have been made. It has been recognized for some time that medications administered to horses will be excreted and could continue, over time, to contaminate the environment and the horse (flunixin, for example; Norgren *et al.*, 2000). This has led to recommendations that such administrations should be conducted in separate 'medication stalls'. However, advances in our analytical capabilities, the detection of sub-nanogram quantities of drugs in biological fluids, make even this practice inadequate to avoid contamination of the horse. The data illustrate that the environment of the horse contains residues of drugs; in the soil beneath their hooves, in the water that washes from their barns, on the walls of their stalls and in the air they breathe, carried on the dust that circulates from all of these origins and sources. This contamination exists not only in the stalls in which horses are housed but also in areas that are under the control of the regulatory authorities, in the test barn stalls themselves.

In the present study, immunoassay screening for many drugs commonly used as therapeutics or as drugs of abuse in the equine (amphetamines, atropine, azaperone, barbiturates, buprenorphine, butorphanol, dezocine, eltenac, fentanyl, indomethacin, levallorphan, lidocaine, meperidine, methadone, generic opiates, zolpidem, generic broncodilators and generic promazines) indicated that, at least for the locations examined, they are not common contaminants of the equine environment. Given the rather sporadic and rare instances where drugs such as the amphetamines, fentanyl, barbiturates and other ARCI Category 1 and 2 drugs have been called as positives in the racing industry such a finding is not altogether surprising. However, the failure to detect clenbuterol, for example, a commonly used bronchodilator in Louisiana and elsewhere, or acepromazine, a commonly used sedative, is somewhat unexpected. This may be due to numerous factors that also influence the ongoing presence or absence of such compounds. Nonetheless, the failure to detect these and other compounds here does not preclude them from contributing to contamination.

The data indicate that a preponderance of the drug contamination is occurring from substances in the nonsteroidal anti-inflammatory class, with flunixin, phenylbutazone and naproxen being found in five-of-five ship-in stalls examined and most of the test barn stalls examined. Flunixin seems to be the most pervasive, also being commonly found in the lagoon water and in the dust from ship-in stable rafters as well as in the combined wipe sample. A close second is phenylbutazone, followed by naproxen.

Caffeine is also a common contaminant in the lagoon water and in 6-of-10 stalls tested. This is an interesting observation since it may be reasonably concluded that horses are not intentionally being administered caffeine and certainly not with the frequency or in the dose that flunixin and phenylbutazone are given. Further, caffeine is an ARCI Category 2 substance, the finding of which in a horse leads to severe financial penalties, disqualification and suspension in most racing jurisdictions. Nonetheless, these findings may be seen as evidence of general contamination of the equine environment by humans, the major consumers of caffeine, and the careless spreading of this compound in human sweat, saliva or urine and/or the disposal of coffee, colas, tea and the myriad other products that contain this drug. To underscore this fact, every sample tested was positive for the presence of the major metabolite of nicotine, cotinine, ranging from the low to high ng/g or mL concentration (data not shown). It is probable that this contamination occurs from the smoking of cigarettes and cigars, the chewing of tobacco and the careless disposal of cigarette butts, cigar stumps and tobacco laced spittle in and about the equine environment.

The only other drug that was detected as present was furosemide. This drug is a permitted 'bleeder' medication in Louisiana as well as other racing jurisdictions in the United States, being administered in doses up to 500 mg intravenously at 4 h prior to the scheduled race time of the horse. Thus, these administrations and much of the elimination occur on the backstretch of the track. This being the case, it seems somewhat incongruent that it is not found with greater frequency and at

higher concentrations than the NSAIDs, which, in Louisiana, are not permitted to be administered within 24 h of the scheduled race time of the horse. Indeed, a sensitive immunoassay test gave only limited positive results and three lagoon water samples were estimated to contain less than 2 ng furosemide/mL.

The degree to which the contamination documented here contributes to positive findings in race horses is not presently known. Indeed, most racing jurisdictions in the US have adopted thresholds in plasma for the nonsteroidal drugs reported here. Nonetheless, these drugs are emblematic of the problem and will not be the only drugs to ever be detected in the equine environment. Threshold considerations will be required for many other equine therapeutics as well as drugs commonly used by humans. However, it would not be desirable to establish such thresholds based on how long a laboratory can detect a drug, as the data presented here clearly show. The reason for this is that late-term withdrawal residues cannot be distinguished from contamination. Thresholds should more appropriately be based on the pharmacology of the drug and the concentrations found in blood and urine after it can be assured that the effects of the drug have ended. For many drugs this is at most 24 h after administration.

Other factors in the possible role of contamination to the creation of positives are, of course, the same as we see for any drug administration; the dose and frequency of the exposure, the route, the time after exposure and time prior to sample collection. More research into the concentrations of environmental contamination by veterinary drugs in the racetrack environment is needed, as are studies of mechanisms of exposure, concentrations of exposure required to lead to detection and the rates and mechanisms of degradation of drugs in the equine environment. This latter issue may explain why some drugs commonly used in equine therapeutics are present and others are absent. More effort also needs to be placed into training regulatory personnel, veterinarians, trainers and owners in how to avoid contamination and how to assure that they themselves are not the source of positives. It is hoped that these data and the accompanying discussion will assist in initiating such efforts.

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REFERENCES

- Camargo, F.C., Lehner, A.F., Karpiesiuk, W., Stirling, K., Kavanagh, P.V., Brennan, N., Dowling, M. & Tobin, T. (2005) Review of environmental morphine identifications: Worldwide occurrences and responses of authorities. *AAEP Proceedings*, **51**, 58–64.

- Ernst, E. (2005) Contamination of herbal medicines. *The Pharmaceutical Journal*, **275**, 167–168.
- Galey, F.D., Holstege, D.M., Francis, T., Hyde, W. & Jack, R. (1996) Residues of *Datura* species in horses. *Proceedings of the 11th International Conference of Racing Analysts and Veterinarians*, pp. 333–337, Queensland, Australia.
- Harner, J.P. III & Herrman, T. (1996) *Avoiding drug carryover during feed processing and delivery*. Publication MF-2055, Kansas State University Agricultural and Experiment Station and Cooperative Extension Service. <http://www.oznet.ksu.edu>.
- Kollias-Baker, C. (2002) A review of possible environmental sources of drug positives. *AAEP Proceedings*, **48**, 186–189.
- Norgren, A., Ingvast-Larsson, C. & Kallings, P. (2000) Contamination and urinary excretion of flunixin after repeated administration in the horses. *Proceedings of the 13th International Conference of Racing Analysts and Veterinarians*, pp. 377–380, Cambridge, UK.
- Russell, C.S. & Maynard, S. (2000) Environmental contamination with isoxsuprine. *Proceedings of the 13th International Conference of Racing Analysts and Veterinarians*, pp. 381–383, Cambridge, UK.
- Sams, R.A. (1997) Review of possible sources of exposure of horses to natural products and environmental contaminants resulting in regulatory action. *AAEP Proceedings*, **43**, 220–223.
- Stanley, S.D. & Kollias-Baker, C. (1997) Review of equine drug testing. *AAEP Proceedings*, **43**, 211–214.