

Subject: The public health risk of horse meat from American racehorses is unsubstantiated by the 2010 Dodman et al. paper published in the Journal, *Food and Chemical Toxicology*.

Esteemed Senators,

In their paper, "Association of phenylbutazone usage with horses bought for slaughter: A public health risk", the authors Nicolas Dodman, Nicolas Blondeau, and Ann Marini assert that human consumption of horsemeat following the administration of therapeutic amounts of the anti-inflammatory medication phenylbutazone (PBZ) could hold health risks similar to the direct consumption of the drug. Examples they included were case studies reported 25-62 years ago in which adults and children administered multiple human therapeutic PBZ doses experienced severe and sometimes fatal outcomes, most notably from aplastic anemia, a precursor to Leukemia.

Direct human administration of PBZ was discontinued in the US as a result of these findings. No studies on the risk of these serious side-effects developing from significantly lower (parts per million or less) exposures – such as those that could conceivably occur from consumption of horsemeat products produced from animals recently treated with PBZ – are present in the scientific literature. Experimental Pathologist and Toxicologist, K.N Woodward, author of "Surveillance of Veterinary Residues" in the book Veterinary Pharmacovigilance (1990) states that "it is difficult to associate human health problems with residues of veterinary drugs". In other words, there is no cogent frame of reference for the authors' arguments.

The Dodman et al. 2010 study did not actually measure PBZ levels in any of the horses that were used in this report. Therefore, no conclusions can be drawn – particularly conclusions linking the meat produced by these subject horses and the possibility of human risk. The authors **assume** that the consumption of horsemeat produced from animals treated with PBZ one week or longer prior to slaughter is unsafe for human consumption. As we consider the millions of pounds of horsemeat consumed each year and the length of time horses have been treated with PBZ we might ask: Why has this risk never been proven? Indeed, no cause-and-effect relation between consumption of meat products (from horse or other species) from PBZ-treated animals and a single case of aplastic anemia in any child worldwide has ever been reported.

The authors have taken one serious human health threat, in the form of treatment of *humans* with pharmacological doses (averaging 100 mg) of PBZ - a threat that has since been eliminated - and erroneously extrapolated it to the consumption of horsemeat. It is important to note that this study produced *not one thread of scientific evidence* supporting the supposed threats to human health resulting from horsemeat consumption. No reference was cited - in fact, no reference exists - to human disease or death associated with the consumption of horsemeat. In short, this study was specifically designed to be inflammatory, not scientific. The authors had political agendas that were promulgated by this publication. The level of scientific rigor both in

the design of the study as well as in the peer review process was lacking. It is indeed surprising that a study of this caliber should make it past the reviewers of this journal.

To better understand the level of possible PBZ residue in horses we suggest the following explanation (this applies to PBZ levels in blood, where it is primarily sequestered – levels in muscle would be far less than those calculated below) ...

The half-life for PBZ in horses is 5 to 6 hrs (MERCK Veterinary Manual). This is the time needed for a horse to naturally remove 50% of the drug from its system. So, by 6 hours post-administration, half of the PBZ has been eliminated from the horse; by 12 hours, half of this remaining half-concentration has been eliminated, and so forth.

Human PBZ dosages of 100mg were reported in the 1960's to increase the risk of bone marrow depression disorders. Considering the half-life of PBZ in the body of the horse, in order for a human to consume a 100mg dose of PBZ, assuming that PBZ is evenly distributed throughout the horse, that person would need to eat at least 100lbs of horse meat produced within one hour after that 1000 pound horse was administered a normal therapeutic dose of 1000mg PBZ.

If the same horse were slaughtered the following day, a person would have to consume 1000 lbs of its meat – in other words, an entire horse and about 2/3 of a second horse (assuming a dressing percentage yielding a 600 pound carcass). For horses processed after 2 days withdrawal, 10,000 lbs would need to be consumed. After 3 days, 100,000 pounds. After 4 days, 1 million pounds. After 5 days, 10 million pounds. After 6 days, 100 million pounds. So then, after just a single week of withdrawal, the shortest holding time reported in this study, a person would have to eat 1 billion pounds of horsemeat to consume 100mg of PBZ.

In effect, an adult would need to consume about 1.5 million horses to receive a single, potentially toxic dose of 100mg. That would be more than the total number of horses than have been exported since US horse processing ceased in 2007.

The authors skirt this issue by claiming that *any* amount of PBZ, even those that cannot be measured by current toxicological screening methods, has the potential to produce aplastic anemia in children. Where is the evidence for such a bold statement? The authors point to the EU regulations prohibiting PBZ administration at any time in the life of a horse intended to enter the human food chain as their “proof”. This doesn't prove a health risk, it merely highlights a regulation created because there have been no studies on a withdrawal period for this drug.

As reported by Dodman et al., PBZ and its metabolite, oxyphenbutazone, are not distributed evenly throughout the horse. Instead they accumulate in the kidneys and liver as they are naturally and continually removed or degraded from the animal. PBZ is not permanently retained by muscle or fatty tissues, thus its concentration in these edible tissues would be far less than in the blood that is drained away. Also noteworthy is that other potential causes of aplastic

anemia include toxic chemicals in gasoline and some pesticides, autoimmune disorders and some types of viral infections. <http://www.mayoclinic.com/health/aplastic-anemia/DS00322/DSECTION=causes>

As indicated in the 11<sup>th</sup> paragraph of the Dodman et al. 2010 discussion section, the FDA has set no safe levels of PBZ in livestock carcasses. A safe drug withdrawal period can be attained even in animals that have been administered PBZ at some time during their life, as there is a time following administration of PBZ where it has been completely eliminated from an animal's system with absolutely no detectible residues in any tissues. Perhaps the FDA should work to establish a timeline for withdrawal that results in zero PBZ levels in these carcasses.

If the FDA were to establish a withdrawal period, this would appropriately release an implied and unsubstantiated ban on this important veterinary NSAID for horses. Related public educational programs on drug residues in meat could help provide public assurance on food safety issues and make known how such issues affect food prices and animal agriculture.

Sincerely,

Dr William Day, PhD

Assistant Professor  
Morrisville State College Equine Institute  
Morrisville, NY

Dr Sheryl King, PhD, PAS

Professor  
Director of Equine Studies  
Southern Illinois State University  
Carbondale, IL

Dr Don Henneke, PhD

Professor  
Director of Equine Science  
Tarleton State University  
Tarleton, TX

Dr Pat Evans, EdD

Director of Equine Science  
Scottsdale Community College  
Scottsdale, AZ