

Acute oral toxicity of *p*-aminopropiophenone to stoats (*Mustela erminea*)

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Abstract No toxic baits are currently registered in New Zealand for stoat (*Mustela erminea*) control. PAPP (*p*-aminopropiophenone) has potential application as a vertebrate pesticide for stoat control in New Zealand, and in the hydrochloride form was highly toxic (LD₅₀ 9.3 mg/kg) to stoats in pen trials. The first visible signs of toxicosis, cyanosis and salivation, generally appeared within 20 min of gavage administration of PAPP. These signs were generally followed by rapid respiration, lack of co-ordination and lethargy. Death generally followed within an hour of receiving a lethal dose. Stoats remained at least partly conscious until just prior to death, or may have had periods of intermittent consciousness in the later stages. PAPP toxicosis in stoats appeared relatively rapid (in comparison with other vertebrate pesticides in mustelids) and on preliminary assessment appears relatively humane. Further investigation and development of targeted bait delivery of PAPP for stoat control in New Zealand is recommended. This should be conducted alongside formal assessment of humaneness and the potential for unwanted effects such as non-target mortality and environmental contamination.

Keywords control; lethal dose; poisoning; *p*-aminopropiophenone; *p*-aminopropiophenone hydrochloride; stoat; time to death; vertebrate pesticide

INTRODUCTION

Mustelid control is a critical component of many New Zealand strategies for local conservation of native birds. Potential control techniques for stoats (*Mustela erminea*) include trapping, tracking with dogs and baiting, although no toxic bait formulations are currently registered for stoat control. Sodium fluoroacetate (1080) in whole egg baits has been investigated experimentally for stoat control (Dilks & Lawrence 2000; Spurr 2000); and while they appeared to be effective, this bait type was not easily applied in the field, and 1080 is not target specific. The availability of an effective and humane toxicant, combined with a targeted delivery system for stoats, would improve future protection of native wildlife from stoat predation. Given the increasing public awareness of the risks of pesticide use, and increasing regulatory scrutiny of hazardous substances applied to the environment, any new toxic bait formulations will need to demonstrate field efficacy, target specificity, acceptable environmental risk, and relative humaneness.

Oral administration of *p*-aminopropiophenone (PAPP) can significantly elevate methaemoglobin (MetHb), because a *p*-hydroxy metabolite of PAPP is thought to be responsible for the direct formation of MetHb (Marrs et al. 1991). The metabolite *p*-hydroxyaminopropiophenone, produced by *N*-hydroxylation of PAPP *in vivo*, is a potent MetHb-forming compound (Graffe et al. 1964). Methaemoglobin reduces the capacity of blood to carry oxygen to tissues (Friedell 1954; Salerno & Friedell 1954). Elevation of MetHb can cause anaemia (Smith 1969). Sufficiently high concentrations of MetHb in blood produce anoxic effects (Marrs & Bright 1987). The toxicity of PAPP is assumed to be largely due to this mode of action (Goldstein & Doull 1973). Controlled MetHb formation is one potential strategy to counter cyanide toxicity, and PAPP has been previously studied in this context (e.g., Marrs & Bright 1987; Rockwood 1999).

The potential for PAPP to be used as a vertebrate pesticide has also been investigated (Savarie et al.

1983; Marks et al. 2004). Published LD₅₀ values for PAPP in mammals and birds indicate a wide range in oral toxicity, although mammalian carnivores appear to be generally more susceptible than either rodents or birds (see Discussion), suggesting that stoats may be quite susceptible to PAPP. Females of some species may be intrinsically more susceptible to PAPP toxicosis. Female beagles produced more methaemoglobin for a given dose of PAPP than males (Bright et al. 1987), and female rats had approximately twice the susceptibility of males to oral doses of PAPP (Scawin et al. 1984). Given the lack of information regarding the susceptibility of stoats to PAPP, documentation of acute toxicity was considered a necessary first step in evaluating whether this compound might be suitable for further development as a stoat control tool in New Zealand.

MATERIALS AND METHODS

Wild-caught stoats were housed in outdoor cages (60 × 150 × 90 cm) at the Landcare Research Animal Facility, Lincoln. Each stoat was provided with a nest box (40 × 33 × 15 cm) containing shredded paper as nesting material. Stoats were fed a daily rotation of dead day-old chicks, chicken pet mince and mutton/beef pet mince and had free access to water. They were acclimatised to captivity for at least 8 weeks before being used in trials.

Pilot trial

A pilot trial was conducted to determine whether the susceptibility of stoats to PAPP was within the range of high acute toxicity that would be required for an effective vertebrate pesticide. A standard index of toxicity following ingestion of a pesticide is the LD₅₀, which is the amount of compound required to

kill 50% of a sample of animals. Oral LD₅₀ values in birds and mammals are often expressed as milligram of pesticide per kilogram of animal bodyweight (mg/kg), equivalent to parts per million (ppm). An LD₅₀ of PAPP in stoats of less than 25 mg/kg was considered a sufficient level of toxicity to proceed with trials to refine an estimate of acute oral toxicity.

Although PAPP is nominally water soluble (Merck 1989), stable PAPP solutions of suitable concentration for gavage delivery to stoats were most easily achieved by the formulation of the hydrochloride salt. Dosing solutions were prepared to final PAPP concentrations of 10–50 mg/ml, depending on the treatment group. An appropriate quantity of crystalline PAPP (Merck Ltd, New Zealand) was ground in a mortar and pestle, and equal parts water and monopropylene glycol (MPG) added. This mix was homogenised in an ultrasonic bath, then 35% hydrochloric acid was added (at a 1:0.7 ratio w/w) and further homogenised. This mixture was warmed until solution was complete, then made up to appropriate volume with MPG and water. The maximum dose volume of PAPP hydrochloride solution administered to stoats was 2.5 ml/kg, and control stoats received 1 ml MPG only.

For the pilot trial, light anaesthesia with fluothane was necessary to minimise handling time and stress to the wild-caught stoats. From previous experience, stoats were expected to recover from fluothane anaesthesia within 5 min, so that the onset of signs of PAPP toxicosis were not expected to be masked by the effects of anaesthesia beyond this time. On separate days, a group of stoats ($n = 6$) was dosed with 50 and 25 mg/kg PAPP respectively, alongside corresponding groups of control stoats (Table 1). All stoats were monitored by direct observation for 1 h following dosage, with the control animals providing a reference for normal behaviour during recovery

Table 1 Treatment groups of stoats used to estimate toxicity of PAPP. BW = body weight (mean ± SE).

Date dosed	No. and sex dosed	Mean weight dose group (g)	Dose (mg/kg)	No. and sex of controls	Mean weight control group (g)
Pilot trial					
22 Feb 2000	4 M, 2 F	308.2 ± 24.8	50	2 M, 1 F	323.7 ± 30.5
12 Apr 2001	6 M	296.0 ± 12.5	25	6 M	309.7 ± 23.2
Acute toxicity trial					
17 Dec 2001	6 M	311.7 ± 24.0	12.5	2	362.0 ± 11.0
4 Feb 2002	6 M	357.0 ± 15.2	18.75	3	363.0 ± 8.5
22 Mar 2002	6 M	344.3 ± 15.7	15.63	3	330.3 ± 21.6
1 Jul 2002	6 M	329.7 ± 21.1	9.38	*	—

*Control animals were unavailable for the final dose group. However, substantial experience with gavage dosing and observations of normal recovery from anaesthesia was available by this time.

from anaesthesia and gavage. All procedures were carried out in a room with a temperature between 18° and 23°C.

Acute oral toxicity trial

Adult male stoats only were used in this trial because they were likely to be less susceptible than females to PAPP. Having a larger average body weight than females, males also represented the upper limit for calculating effective toxic loadings for PAPP delivery in baits. On the mornings of four different days, four groups ($n = 6$) of stoats were administered different doses of PAPP using the procedures already described, alongside corresponding control groups (Table 1). Following dosing, stoats were placed back into clean nestboxes and monitored constantly by direct observation for the following 2 h. Their behaviour, any signs of toxicosis, and time to death were recorded. Death was confirmed by lack of heartbeat and corneal reflex. Dosed stoats that appeared to recover fully, i.e., were displaying normal alert behaviour 6 h after dosing, and the control stoats, were returned to outdoor housing. The behaviour of these stoats was recorded for a further 2 h, i.e., until 8 h after dosing, and they were then monitored at least daily for 2 weeks thereafter, including observations of food intake, general health, and behaviour.

One stoat in the first group dosed in the pilot trial (50 mg/kg) and another dosed in the acute toxicity trial (18.75 mg/kg group) died within 8 min of gavage dosing. Necropsy indicated that some of the dose had entered the lungs, and these stoats were discounted from statistical analysis. Mortality data of all remaining stoats dosed with PAPP were used in both logistic and probit regression models (Genstat) to approximate lethal dose (LD_{50} , LD_{95} , and

LD_{99}) values and corresponding 95% confidence intervals.

RESULTS

All control stoats survived, and always recovered from anaesthesia within 3 min. Within this time, a raised head was generally observed within the first minute, rapid but uncoordinated movement about the box within the following 2 min, and apparently normal behaviour thereafter, e.g., lying in a curled position but alert and responsive to stimuli.

Stoats dosed with PAPP showed recovery from anaesthesia consistent with that of control stoats. However, after this there was a generally consistent progression of signs of PAPP poisoning, in most instances leading to death. Within approximately 20 min of recovery from anaesthesia, movements of PAPP-dosed stoats appeared slower and less coordinated, and cyanosis (a bluish discoloration of the skin resulting from inadequate oxygenation of the blood) began to affect the lips and nose (Table 2). By 30 min after recovery from anaesthesia, stoats were less responsive to disturbance, e.g., noise or touch, often lying curled with head down and displaying rapid, shallow respiration. After this, in comparison with control stoats, the behaviour of PAPP-dosed stoats was visibly affected. They showed little activity, often lying in a curled position, with cyanosis evident in exposed skin. Some stoats first displayed excessive salivation at this stage, with respiration remaining rapid and increasingly laboured. Any movement attempted appeared very uncoordinated, often with legs splayed sideways and an inability to raise their heads. Excessive salivation was ob-

Table 2 Mortality and time to death and to first observation of cyanosis, as a distinctive symptom of poisoning, in groups of stoats gavage-dosed with PAPP (mean \pm SE).

PAPP dose (mg/kg)	Mortality (no. dead/no. tested)	Time to death (min)	Time to first obs. cyanosis (min)
Pilot trial			
50	5/5	40.6 \pm 2.6	Not recorded
25	6/6	36.7 \pm 3.6	Not recorded
Acute toxicity trial			
18.75	5/5	35.6 \pm 1.9	18.2 \pm 3.5
15.63	6/6	42.0 \pm 3.1	24.0 \pm 4.4
12.5	3/6	39.3 \pm 1.4	20.8 \pm 2.5
9.38	2/6	68.5 \pm 20.5	13.8 \pm 4.0

served in 10 of 24 PAPP-dosed stoats, and vomiting or retching observed in two of 24 stoats. Retching was observed once in one stoat, which survived, in the lowest dose group (9.38 mg/kg), and retching with resultant vomitus in one stoat, which died, in the 15.63 mg/kg group. Mean times to death were consistently around 40 min after dosing, except for the lowest-dose group (9.38 mg/kg) where the mean time to death ($n = 4$) was just over 1 h after dosing (Table 2). Stoats that died appeared to lose

consciousness just prior to death (unresponsive to noise or touch), with breathing slow, irregular, and "gasping". Just prior to death, some stoats showed writhing or paddling movements, which appeared involuntary.

Those stoats that survived dosing all reached the stage where they were visibly affected, as described above. They remained in curled or prostrate positions for at least 1 h and up to 3 h after dosing, during which time it was difficult to ascertain whether they were conscious. Recovery was usually preceded by a raised head and shaky movements, followed gradually by increasing coordination of head and leg movement. Stoats that survived dosing in the morning all ate food presented to them that night and, from daily checks, appeared to be eating and behaving normally over the following 2 weeks.

Using either probit or logit analysis, the LD_{50} for PAPP in stoats was calculated at 9.3 mg/kg (upper 95% CL 11.8 mg/kg). Other LD values were also calculated with 95% confidence intervals (Table 3).

Table 3 Lethal dose values calculated for PAPP in stoats.

Lethal dose value (mg/kg)	95% CI	Genstat analysis
$LD_{50} = 9.3$	0.4–11.8	Probit
$LD_{50} = 9.3$	0.01–11.8	Logit
$LD_{90} = 15.1$	11.9–167	Probit
$LD_{95} = 17.3$	13.5–902	Probit
$LD_{99} = 22.4$	16.0–20 500	Probit

Table 4 Reported oral lethal dose (LD_{50}) values for PAPP in various species. Where available the sex, species, and strain of the animals tested are given. In the absence of these in some references, "mouse" was assumed to be laboratory (*Mus musculus*) and "rat", laboratory (*Rattus norvegicus*).

Species	LD_{50} (mg/kg)	Reference
Dog (<i>Canis familiaris</i>) ¹	7.5	Coleman et al. (1960)
Coyote (<i>Canis latrans</i>)	5.6	Savarie et al. (1983)
Kit fox (<i>Vulpes velox</i>)	14.1	Savarie et al. (1983)
Cat (<i>Felis libyca domestica</i>)	5.6	Savarie et al. (1983)
Bobcat (<i>Lynx rufus</i>)	10	Savarie et al. (1983)
North American badger (<i>Taxidea taxus</i>)	c. 100	Savarie et al. (1983)
Raccoon (<i>Procyon lotor</i>)	142	Savarie et al. (1983)
Striped skunk (<i>Mephitis mephitis</i>)	>400	Savarie et al. (1983)
Guinea pig (female) (<i>Cavellio porcinus</i>)	1020	Scawin et al. (1984)
Mouse (albino)	223	Savarie et al. (1983)
Mouse (female)	>5000	Scawin et al. (1984)
Mouse (male) (Swiss Webster strain)	168	Pan et al. (1983)
Rat (female) (Porton Wistar strain)	223.7	Scawin et al. (1984)
Rat (male)	475	Scawin et al. (1984)
Rat	177	Savarie et al. (1983)
Rat (male) (Sprague-Dawley)	221	Pan et al. (1983)
Golden eagle (<i>Aquila chrysaetos</i>)	>50	Savarie et al. (1983)
Coturnix quail (<i>Coturnix coturnix</i>)	>316	Savarie et al. (1983)
Starling (<i>Sturnus vulgaris</i>)	>316	Savarie et al. (1983)
Red-winged blackbird (<i>Agelaius phoeniceus</i>)	133	Savarie et al. (1983)
Black-billed magpie (<i>Pica pica</i>)	178	Savarie et al. (1983)
Common crow (<i>Corvus brachyrhynchos</i>)	≥178	Savarie et al. (1983)

¹Dosing route not specified, assumed to be oral.

DISCUSSION

On the basis of an oral LD_{50} of 9.3 mg/kg, PAPP appears to be sufficiently toxic to stoats for further development as a pesticide for this species. Upper confidence limits calculated for the PAPP toxicity values in stoats reflected the shape of the probit function, so that as the mortality rate approached 1, a large change in dose was required to gain a small increase in mortality. The lower confidence limits for the toxicity values estimated for PAPP in stoats were less robust, and depended on the analysis used (Table 3), because the data only just extended into groups showing 50% mortality. 1080 is more toxic to stoats than PAPP, with oral LD_{50} for 1080 in stoats estimated as 0.49 mg/kg (0.29–0.70 95% CI), and LD_{90} at 0.70 mg/kg (0.47–0.87 95% CI) (Spurr 2000). In comparison, relatively broad confidence intervals for toxicity estimates of PAPP in stoats are reported here, suggesting that future bait formulations will need to contain sufficient PAPP to account for potentially wide variation in susceptibility within field populations of stoats. Gavage dosing with PAPP hydrochloride in solution probably resulted in higher bioavailability than would be expected after the ingestion of PAPP in food. Further trials with PAPP delivered in baits, rather than by oral gavage, are required to confirm an effective toxic concentration and bait formulation that can achieve high kill efficacy in stoats. Encapsulated formulations of PAPP may overcome this practical limitation, and are currently being developed and pen-tested for efficacy when delivered in food to stoats (Murphy et al. 2005).

Oral toxicity of PAPP to stoats was within the range reported for some other mammalian carnivores (Table 4). Durie & Doull (1968) and Savarie et al. (1983) noted that the toxicity of PAPP can vary significantly between species and even between strains, and attributed this to variations in the metabolic pathways responsible for the detoxification and excretion of the compound. Distinct intraspecific differences in the urinary excretion of aromatic amines, including PAPP, were noted by von Jagow et al. (1966), who measured the percentage of PAPP excreted as *N*-hydroxy metabolite by dogs at 1%, guinea pigs approximately 15%, and rabbits 30%. The oral toxicity of PAPP in rodents and birds is generally lower than in mammalian carnivores. Median LD_{50} values of 178 and 223 mg/kg for birds and rodents, respectively (Table 4) indicate a relatively reduced susceptibility. In the context of using PAPP for stoat control, this is a promising indication of selective toxicity. However, it should be noted that

these evaluations, in birds especially, cover a limited range of species and confidence of estimates. Currently available toxicity figures for PAPP do not include New Zealand native species, or marsupials, so it is difficult to make assessments of potential non-target hazards of PAPP. Further development should focus on stoat-specific delivery of PAPP, and quantification and minimisation of non-target poisoning risks.

The clinical consequences of methaemoglobinaemia are related to the blood concentration of MetHb. In humans, dyspnoea, nausea and tachycardia occur at levels $\geq 30\%$, while lethargy, stupor, and deteriorating consciousness are induced by MetHb levels approaching 55%. Higher levels may cause cardiac arrhythmia, circulatory failure and neurological depression, while levels of 70% are usually fatal (Coleman & Coleman 1996). Although MetHb levels in PAPP-dosed stoats were not measured in this trial, it was assumed that an oxygen supply to cells necessary for sustaining life would fail at MetHb levels approximating 85% (Bodansky 1951). Observation of cyanosis and progressive lethargy until death in PAPP-dosed stoats was consistent with this mode of action. Observations indicated that poisoned stoats remained at least partly conscious until just prior to death, or may have had periods of intermittent consciousness in the later stages.

Mean times to death were consistently around 40 min after dosing with PAPP, except for the lowest-dose group, 9.38 mg/kg, where the mean time to death ($n = 4$) was just over 1 h after dosing (Table 2). While this difference suggests dose-dependence there were insufficient data to test this statistically. Spurr (2000) reported that stoats poisoned with 1080 died within 12 h. Stoats poisoned by cholecalciferol in pen trials had an average time to death of 6.3 days, and some had lost significant amounts of body weight (Spurr et al. 2001). In comparison, the mean times to death following a lethal dose of PAPP in stoats were much shorter, and the maximum time to death in an individual stoat was 89 min after receiving a dose of 9.38 mg/kg PAPP. Even assuming that stoats were conscious right up to death, this gives a preliminary indication that PAPP is relatively humane, at least compared with other vertebrate pesticides that have been considered for stoat control. Further published studies of other pesticide toxicoses in stoats would allow a more thorough comparison with PAPP, and further evaluations against a recognised humanness standard would be required to make a more formal assessment of the humaneness of PAPP for stoat control. Bait delivery of PAPP to stoats may

be improved by encapsulated formulations, which could facilitate delivery of an acute toxic dose, and also overcome possible taste aversion to PAPP.

Various effective treatment regimes have been described for methaemoglobinaemia (Coleman & Coleman 1996), so that if PAPP was used as a vertebrate pesticide, there is the desirable possibility of prescribing an effective treatment for accidental poisoning. If PAPP is further developed as a toxic bait formulation for stoats, issues of non-target susceptibility (including ecotoxicity) and the potential for bioaccumulation and environmental contamination in the New Zealand context, will need to be considered.

CONCLUSIONS

The high oral toxicity of PAPP to stoats suggests that this compound is very suitable for continued development in a new toxic bait formulation for stoat control. Advantages of PAPP as a stoat control tool could include the potential for effective treatment of accidental poisoning, and a relatively humane action compared with existing vertebrate pesticides. Information gaps in the potential non-target and environmental risks of using PAPP as a control tool for stoats in New Zealand remain as future priorities.

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REFERENCES

Bodansky O 1951. Methemoglobinemia and methemoglobin-producing compounds. *Pharmacological Reviews* 3: 144–196.

Bright JE, Woodman AC, Marrs TC, Wood SG 1987. Sex differences in the production of methaemoglobinaemia by 4-aminopropiophenone. *Xenobiotica* 17: 79–83.

Coleman IW, Little PE, Grant GA 1960. The induction of methemoglobinemia as an adjunct to therapy for tabun poisoning. *Canadian Journal of Biochemistry and Physiology* 38: 667–672.

Coleman MD, Coleman NA 1996. Drug-induced methaemoglobinaemia: treatment issues. *Drug Safety* 14: 394–405.

Dilks P, Lawrence B 2000. The use of poison eggs for the control of stoats. *New Zealand Journal of Zoology* 27: 173–182.

Durie RH, Doull J 1968. Factors influencing the toxicity of para-aminopropiophenone in rats. *The Pharmacologist* 10: 172.

Environmental Risk Management Authority (ERMA) 2001. User guide to the HSNO thresholds and classifications. Wellington, ERMA New Zealand.

Friedell L 1954. Further studies on the relationship between oxygen tensions and the protective actions of cysteine, mercaptoethylamine and p-aminopropiophenone. *Radiation Research* 1: 559–560.

Goldstein GM, Doull J 1973. The use of hyperbaric oxygen in the treatment of p-aminopropiophenone-induced methemoglobinemia. *Toxicology and Applied Pharmacology* 26: 247–252.

Graffe W, Kiese M, Rauscher E 1964. The formation in vivo of p-hydroxylaminopropiophenone and p-aminopropiophenone and its action in vivo and in vitro. *Naunyn-Schmiedeberg's Archiv für Experimentelle Pathologie und Pharmakologie* 249: 168–175.

Marks CA, Gigliotti F, Busana F, Johnston M, Lindeman M 2004. Fox control using a para-aminopropiophenone formulation with the M-44 ejector. *Animal Welfare* 13: 401–407.

Marrs TC, Bright JE 1987. Effect on blood and plasma cyanide levels and on methaemoglobin levels of cyanide administered with and without previous protection using PAPP. *Human Toxicology* 6: 139–145.

Marrs TC, Inns RH, Bright JE, Wood SG 1991. The formation of methaemoglobin by 4-aminopropiophenone (PAPP) and 4-(N-hydroxy) aminopropiophenone. *Human & Experimental Toxicology* 10: 183–188.

Merck 1989. The Merck index. 11th ed. In: Budavari S ed. Rahway, New Jersey, USA, Merck and Co. Inc. P. 488.

Murphy EC, Lavrent A, MacMorran D, Robbins L, Ross P 2005. Development of a humane toxin for the control of introduced mammalian predators in New Zealand. Proceedings of the 13th Australasian Vertebrate Pest Conference, 2–5 May 2005, Wellington, New Zealand. Pp. 137–142.

- Pan HP, Savarie PJ, Elias DJ, Felton RR 1983. Alkyl chain length and acute oral toxicity of *p*-aminophenones. *General Pharmacology* 14: 465–467.
- Rockwood GA 1999. Comparison of hematologic consequences and efficacy of *p*-aminophenones in mice. *Environmental Toxicology and Pharmacology* 7: 237–252.
- Salerna PR, Friedell HL 1954. Further studies on the relationship between oxygen tensions and the protective actions of cysteine, mercaptoethylamine and *p*-aminopropiophenone. *Radiation Research* 1: 559–560.
- Savarie PJ, Ping Pan H, Hayes DJ, Roberts JD, Dasch GJ, Felton R, Schafer EW Jr 1983. Comparative acute oral toxicity of *para*-aminopropiophenone (PAPP) in mammals and birds. *Bulletin of Environmental Contamination and Toxicology* 30: 122–126.
- Scawin JW, Swanston DW, Marrs TC 1984. The acute oral and intravenous toxicity of *p*-aminopropiophenone (PAPP) to laboratory rodents. *Toxicology Letters* 23: 359–365.
- Smith RP 1969. The significance of methemoglobinemia in toxicology. In: Blood FR ed. *Essays in toxicology*. New York, Academic Press. Pp. 83–113.
- Spurr EB 2000. Hen eggs poisoned with sodium monofluoroacetate (1080) for control of stoats (*Mustela erminea*) in New Zealand. *New Zealand Journal of Zoology* 27: 165–172.
- Spurr E, O'Connor CE, Airey A, Kerr J 2001. Feracol for control of stoats (*Mustela erminea*). Unpublished report LC0102/049, Landcare Research, Lincoln, New Zealand. 9 p.
- von Jagow R, Kiese M, Renner G 1966. Urinary excretion of *N*-hydroxy derivatives of some aromatic amines by rabbits, guinea pigs and dogs. *Biochemical Pharmacology* 15: 1899–1910.