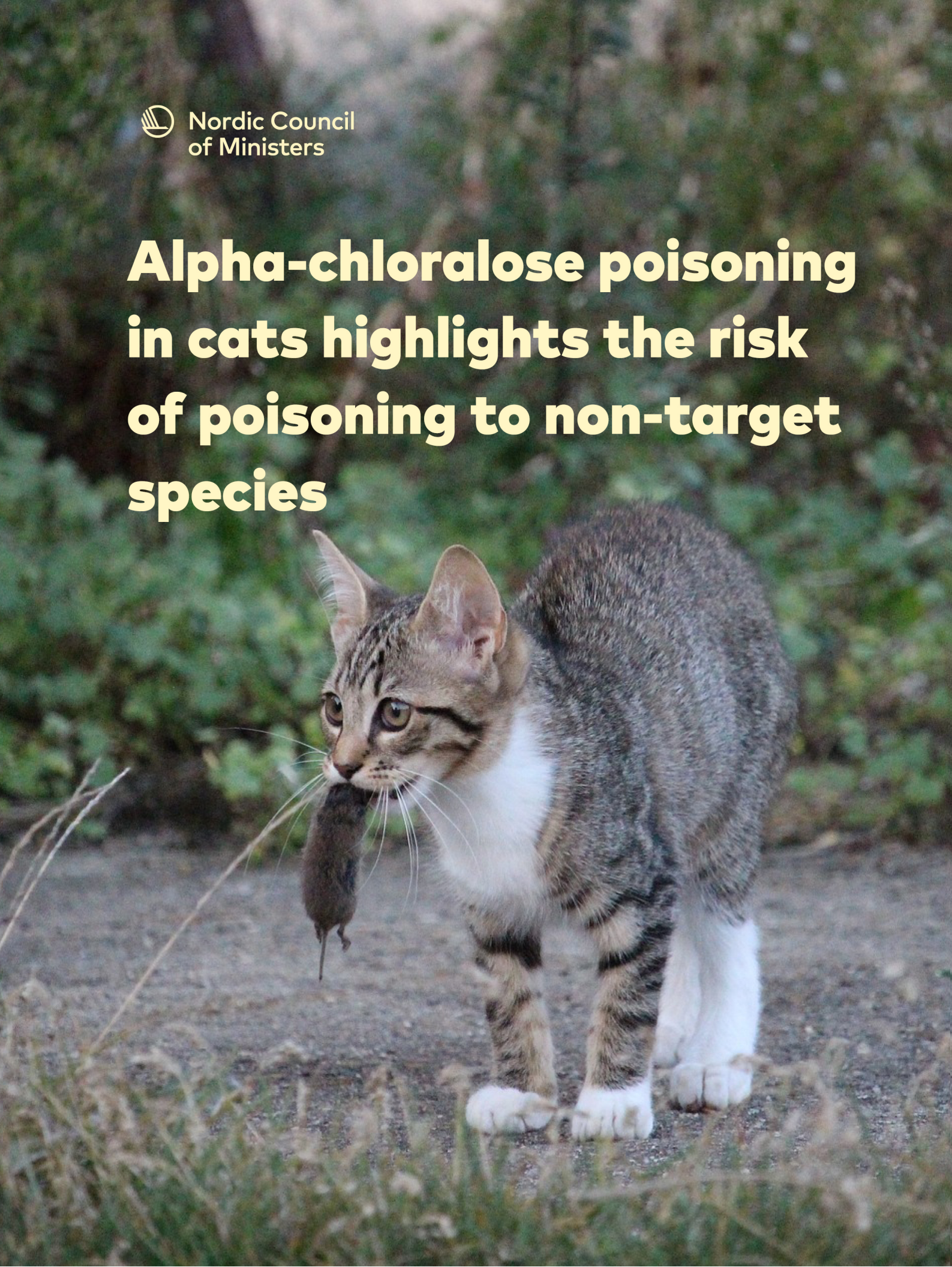
Nordic Council of Ministers

Alpha-chloralose poisoning in cats highlights the risk of poisoning to non-target species



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Preface

The Norwegian Environmental Agency took the initiative to and has been the project leader for this report. It was funded by the Nordic Council of Ministers, Nordic Working Group for Chemicals, Environment, and Health. At the time of initiating this project, other projects related to alpha-chloralose (AC) poisoning was ongoing both at the Swedish University Animal Hospital, the Swedish National Veterinary Institute and the Norwegian Veterinary Institute (2, 5,6). Knowledge from this work together with data from studies co-funded by the Nordic Council was compiled in a scientific publication in BMC Veterinary Research (1) with Ulrika Windahl as research manager. This publication is the main foundation to the present report. Terje Haraldsen and Hilde Andersen at the Norwegian Environment Agency has assembled this report in cooperation with the scientists who also contributed to the scientific article:

Sweden (Collecting and submitting blood serum and questionnaires from Swedish cats. Coordinating and analysing blood serum and veterinarian/owner questionnaires from all countries):

Swedish National Veterinary Institute

- Ulrika Windahl
- Annica Tevell Åberg

The University Animal Hospital at the Swedish University of Agricultural Sciences

- Sandra Lundgren
- Cecilia Tegner
- Kristoffer Dreimanis

Norway (Collecting and submitting blood serum and questionnaires from Norwegian cats. Performing the wild mice study and calculation of bait intake, coordinating urine samples and analysing these for metabolites):

The Toxinology Research Group at the Norwegian Veterinary Institute

- Fedor Kryuchov
- Morten Sandvik
- Aksel Bernhoft

Finland (Collecting and submitting blood serum and questionnaires from Finnish cats):

Finnish Food Authority

- Outi Simola

Finnish Safety and Chemicals Agency (Tukes)

- Sanna Koivisto

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Oslo, 04.11.2022

Terje Haraldsen
Project manager

Summary

A marked increase in suspected secondary cases of Alpha-chloralose (AC) poisoning in cats was reported to national veterinary and chemical authorities/institutes in Finland, Norway and Sweden in 2018 and 2019 by veterinarians working in clinical practices in the respective countries. This led to the initiation of several Nordic research projects, and a recently published study on AC-poisoning in cats in the three countries were co-funded by the Nordic council. This publication by Windahl et al. (1) is the main scientific foundation to the present report. Data on signalment, history and clinical findings were prospectively collected in Finland, Norway and Sweden using a questionnaire which the attending veterinarian completed and submitted together with a serum sample collected from suspected feline cases of AC-poisoning. The diagnosis was confirmed by quantification of AC in serum samples. Furthermore, feline urine samples were screened for AC metabolites, and bait intake and amount of AC consumed by wild mice was observed. The aim of the study was to investigate the possibility of secondary AC poisoning in cats from consuming poisoned mice, and to study metabolism and excretion of AC in cats. Findings in studies highlighted in this report (1,6) supports the theory that deficiency in enzymes responsible for conjugation reaction could make the cats more susceptible and prone to AC poisoning compared to e.g. dogs. Furthermore, the results from the studies showed that secondary poisoning of cats from ingestion of mice is possible and highlights the risk of AC poisoning to non-target species. Observations of wild mice revealed that they can consume significantly more AC-containing bait than earlier presumed.

1 Introduction

AC is a biocidal active substance that is used in rodenticide baits to control mice. AC has also been used as an avicide and an anaesthetic in clinical practice for human, feline and canine patients as well as laboratory animals, however it is currently not used clinically (1).

For many years the anticoagulants have dominated the Nordic market for control of mice. Based on their reproductive toxicity properties, all anticoagulants meet the exclusion criterion under the Biocidal Products Regulation (BPR – (EU) No 528/2012). Additionally, the 2nd generation anticoagulants meet the exclusion criterion because they are PBT (persistent, bioaccumulative and toxic) or vPvB (very persistent, very bioaccumulating) substances, i.e., they stay in the environment for a long time and accumulate in the food chain. This has led to a strict regulation of these products. AC was approved as a biocidal active substance in 2009 (2009/93/EC) under the Biocidal Product Directive (BPD (98/8/EC) in the European Union, and it has been considered a better alternative than the anticoagulants because it was less persistent and bioaccumulative.

AC containing biocidal products were first authorised for use by both the general public and professionals. As a response to an increased observation of suspected secondary poisoning in cats, the authorisation was restricted to trained professional use only, in Norway by the Norwegian Environment Agency from May 2020 and in Sweden by the Swedish Chemicals Agency (KEMI) from December 2019. In Finland restriction to trained professional use by the Finnish Safety and Chemicals Agency (Tukes) was appealed in December 2021 by the authorisation holders and thus the restrictions are not legally valid (the court case is in progress when finalising this report). In Denmark the products are authorised by the Danish EPA for both professional and public use, but they are restricted to use in bait boxes.

The marked increase in suspected cases of AC-poisoning in cats mentioned above, was reported to national veterinary and chemical authorities and institutes in Finland, Norway and Sweden in 2018 and 2019 by veterinarians working in clinical practices in the respective countries (1,2). This is exemplified by the graph showing annually suspected AC-poisoning cases at the University Animal Hospital, Small Animal Clinic in Uppsala in fig 1, reproduced by permission from the publication by Tegner et al. (2). The medical history led several veterinarians to suspect that the cats were poisoned secondarily from eating poisoned mice (1). The increase in cases led to concern among cat owners, and also received attention in the media (fig 2).

The same concern was not reported from Danish chemical authorities, but the Danish EPA have conducted an informal survey where they received a small number of suspected AC-poisoning cases of cats and one dog (personal communication, Jesper Johannesen, Danish EPA).

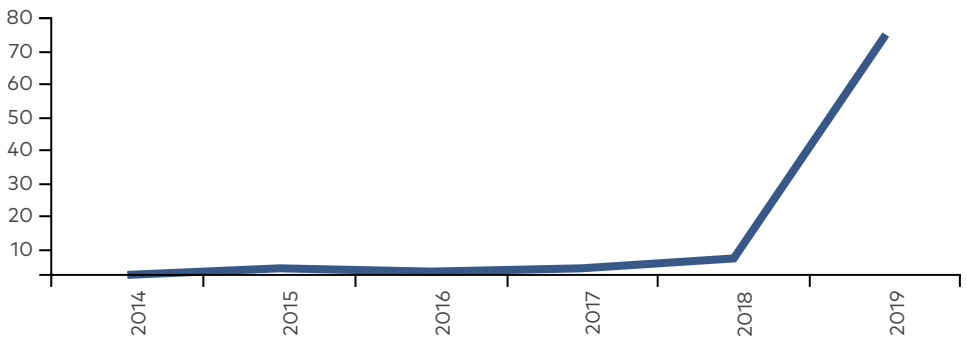


Figure 1: Number of cats presenting annually to University Animal Hospital, Small Animal Clinic, Uppsala, Sweden, with suspected AC poisoning in 2014–2019 (2). Reproduced from Tegner et al. (2022) by permission from the publisher

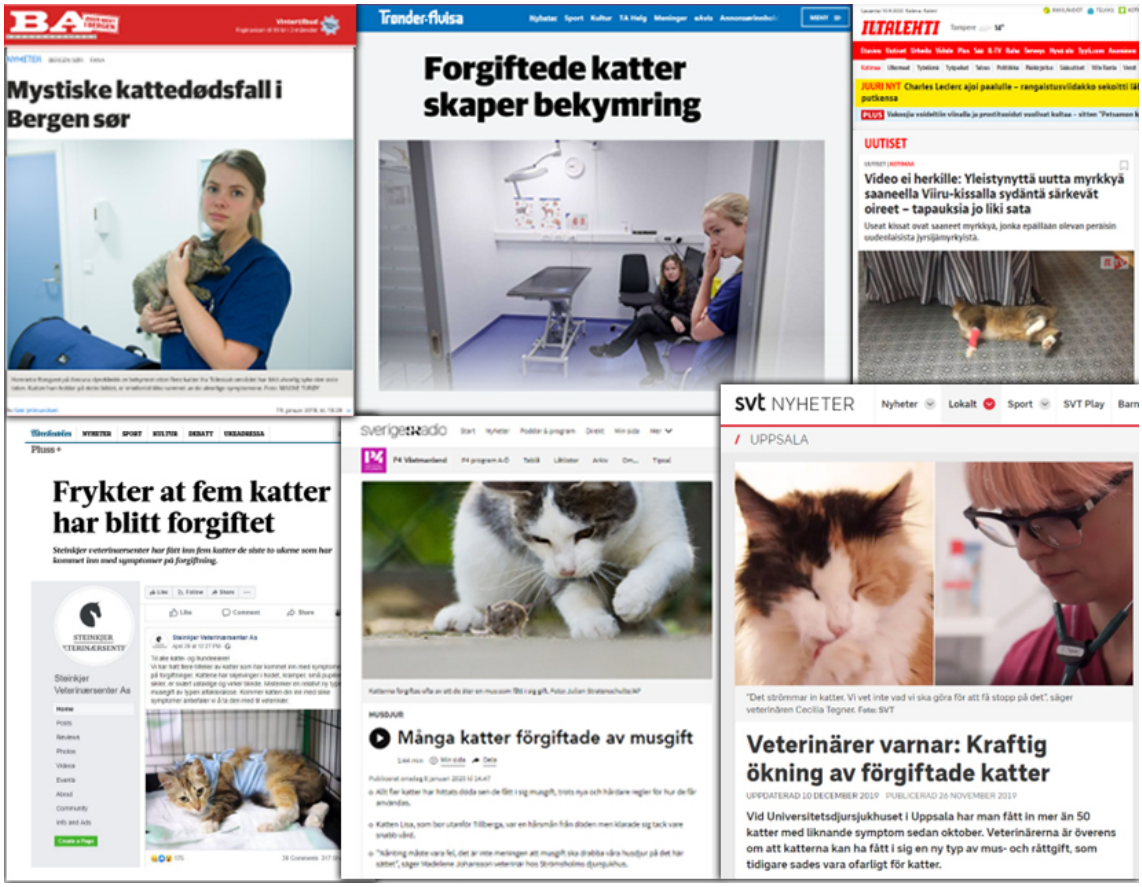


Figure 2: Examples of press releases on AC poisoning cases (3)

Only a few scientific publications on AC poisoning in companion animals were available before this surge in suspected cases occurred (4). The suspicion of secondary poisoning cases led to the initiation of several Nordic research projects (1,2,5,6). A recently published study on AC poisoning in cats in three Nordic countries (1) were co-funded by the Nordic council and provides the main scientific foundation to this report. Data on signalment, history and clinical findings were prospectively collected in Finland, Norway and Sweden using a questionnaire which the attending veterinarian completed and submitted together with a sample of blood serum collected from suspected feline cases of AC-poisoning. The diagnosis was confirmed by quantification of AC in the serum. Feline urine samples were screened for AC metabolites, and furthermore, bait intake and amount of AC consumed by wild mice was observed. The aim of this study was to investigate the possibility of secondary AC poisoning in cats from consuming poisoned mice, and to study metabolism and excretion of AC in cats.

More knowledge on the risk of secondary poisoning of non-target organisms, and thus any unacceptable effects on the welfare and health of animals, contributes to a more precise risk assessment of AC as an active substance in rodenticides in the reassessment for approval under the BPR. It should be emphasised that knowledge on the risk of AC poisoning in cats from the use in rodenticide baits to control mice is relevant regarding the risk for poisoning of other wild carnivores and birds of prey.

2 AC poisoning – mechanism of action, symptoms, toxicity, and metabolism

The exact mechanisms of action of AC are not well defined, but the compound has been shown to have both a dose-dependent depressant and stimulant effect on the central nervous system (CNS) in various animal species and in humans (1). Intoxication may lead to sudden onset of lethargy, ataxia, hypersensitivity, muscle tremors, seizures and comatose state. Increased drooling, and symptoms of cranial nerve disorders such as contracted or dilated pupils is frequently described in reports on intoxication in humans and other animal species. In individuals where lethargy is present or consciousness is lost, bradycardia, hypotension, bradypnea and hypothermia may be present, and if not treated or reversed in time hypothermia could contribute to a lethal outcome (6, 1). As there is no antidote or specific treatment for AC intoxication, treatment is supportive and symptomatic, including maintaining a normal body temperature, minimising external stimuli and, when indicated, anticonvulsants (2).

The toxicity of AC, including lethal dose (LD50), is reported to vary between different species (table 1). The therapeutic index in cats has never been fully determined, although reports on use of AC as a feline anaesthetic are available. For example, Kullman et al. used continuous intravenous infusion of AC at a dose of 5mg/kg/h after an initial bolus of 65–75mg/kg as anaesthesia in cats used for experimental research not related to AC toxicosis (7).

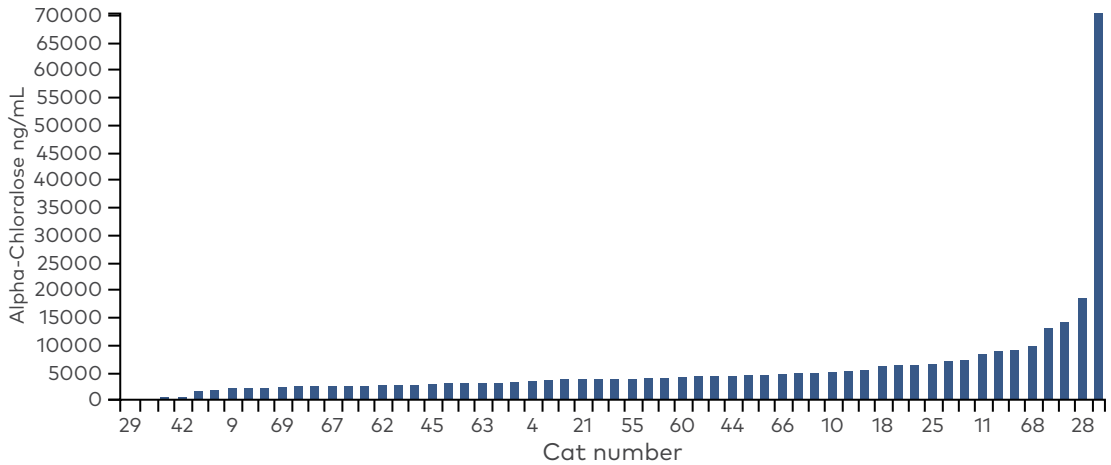
Table 1: Lethal dose (LD50) of alphachloralose for different animal species (8)

Species	Rats	Mice	Cats	Dogs	Starlings	Crows
Lethal dose (mg/kg)	400	300	100	600–1000	76	42

The cats were over-represented compared to e.g., dogs in the emerging AC-poisoning cases. The high susceptibility to AC in cats may among other reasons be related to their limited capacity to form glucuronide metabolites for drugs compared with other species (6). Glucuronic acid conjugation is a metabolic process that increases the polarity of xenobiotics and hence facilitates the excretion of several compounds including drugs and toxins via urine or bile. While the human UDP-glucuronosyltransferase gene (UGT1A) codes for nine different UGT enzymes, and the canine gene ten, the feline gene only encodes two different UGT enzymes that catalyses the conjugation with glucuronic acid, thereby leading to reduced excretion of substances secreted by this mechanism. Cats are also deficient in other enzymes responsible for conjugation reaction, *e.g.,* N-acetyltransferase and thiopurine methyltransferase (1).

3 Laboratory analyses for determining AC in feline blood

Detection of AC in blood or urine samples, or in body tissues collected post-mortem, is key for the diagnosis of clinical cases and a requirement for surveillance of secondary toxicosis, including potential cases in wild animals (5). At the time when the reports of suspected secondary AC-poisoning in cats were emerging, reports on poisoning of humans and non-laboratory animals confirmed by the detection of AC were available. Reports on clinical cases of AC-poisoning in cats were however rare. Furthermore, reports on clinical cases in domestic animals rarely report quantifications of AC in blood or body tissues. In a Swedish study performed by Windahl et al., the validation of a quantitative ultra high performance liquid chromatography--tandem mass spectrometry (UHPLC--MS-MS) method, fit for use in cases of suspected AC poisoning in cats, was described (5). The method was used in a study on AC poisoning in cats in Sweden, quantifying AC concentration in blood samples from 25 suspected cases (2). In the study, the intoxication severity was scored, and this correlated with the detected AC concentration. The validated method was also used later in the study by Windahl et al. (1) on AC-poisoning in cats in Nordic countries where the lowest measured serum concentration of AC was 127 ng/mL and the highest 70 100 ng/mL (fig. 3). The mean and median concentrations were 5 597 and 3 740 ng/mL, respectively.



4 Feline poisoning cases in Nordic countries

As there is no official register for reporting suspected AC-poisoning in the Nordic countries, the total number of poisoning cases is unknown. However, some studies and reports have been published indicating the extent of the problem. In a report from the Norwegian Veterinary Institute (NVI), AC-poisoning was verified in 30 cats and four dogs in samples collected from various Norwegian veterinary clinics from December 2019 to June 2020 (6). The Swedish Small Animal University Hospital had approximately 80 suspected and 25 confirmed feline cases admitted from January 2014–February 2020 (2). In Finland, 149 and 19 poisoning cases of cats and dogs, respectively, have been notified to Tukes in 2018–2021 (9). In the extensive study on AC poisoning in cats in three Nordic countries (1), AC-poisoning was confirmed in 59 Nordic cats (fig 4).

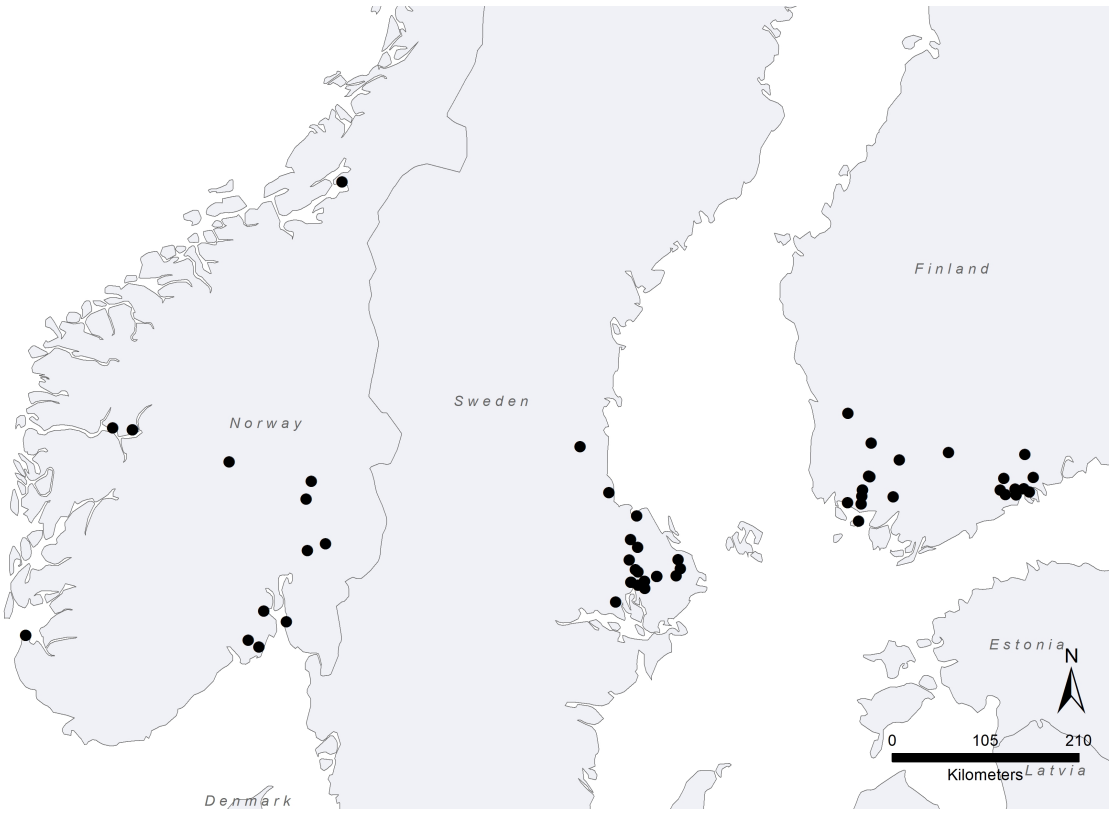


Figure 4: The geographical distribution of the cases in the Windahl et al. study (2022). Reproduced by permission from the publisher

AC poisoning has not only been a Nordic phenomenon. In comparison, data from the Belgian Poison Centre reported a similar emerging of AC poisonings in mostly dogs in the period 2016–2019, with a total of 1, 97, 128 suspected cases in 2017, 2018 and 2019 respectively (10). Furthermore, a survey among German veterinarians revealed that after authorization of AC-containing rodenticides in 2018, 39 out of 65 respondents noticed a significant increase in poisoning cases in cats and dogs attributed to AC (11). To our knowledge, this has not been confirmed by measurement of AC in samples from the animals, and the possibility of secondary poisoning has not been investigated in these countries.

5 Bait intake in mice

In the report published by the Norwegian Veterinary Institute (6), a calculation was made on possible intake of AC in mice to assess the possibility of secondary poisoning in cats and dogs. It was suggested that the mice (about 20 g body weight) ingest at least 1 g of bait before they die, possibly up to 2 g. One gram of bait contains 40 mg AC. A mouse that has ingested 40 mg AC, would give a 3 kg cat an oral dose of 13 mg/kg. If a cat ingests 4 mice, this will result in an oral dose of 50 mg/kg cat. These figures were considered conservative: If the mice ingested 2 g of the bait, the doses would double.

In the later study by Windahl et al. (1), bait intake and amount of AC of the formulation Trinol dry "No mouse korn" consumed by wild mice was studied. The mice were observed individually during an extermination of a rodent infestation. The calculated amount of AC ingested by each mouse was 33 to 106 mg with a mean of 61 mg (table 2). Thus, the real intake of bait in wild mice corresponded well with the doses calculated by Bernhoft et al. (6) The amount of ingested bait related to body weight varied from 3.9 to 13.7%, with a mean of 8.4%. A 3-kg cat that ingests a mouse containing the mean amount of bait with 61 mg AC, would result in a dose of 20 mg/kg cat, and four of such mice would give 80 mg/kg cat. If the 3-kg cat ingests a single mouse with the maximum amount of bait, 106 mg AC, the dose would be 35 mg/kg cat.

Table 2: Amount of consumed rodenticide bait Trinol No mouse korn® alpha-chloralose 40 mg/g in 14 wild mice (*Apodemus flavicollis*; Yellow-necked fieldmouse) caught in mousetraps, and calculated intake of AC for each mouse (1)

Mouse ID (number)	Body weight (g) after death	Bait placed in mousetrap (g)	Bait left in trap (g)	Bait consumed by mouse (g)	Bait consumed: Mouse body weight (%)	AC consumed (mg/mouse)
1	16.2	2.02	0	2.02	12.5	80.8
2	15.8	2.04	0	2.04	12.9	81.6
3	22.5	1.81	0	1.81	8.0	72.4
4	16.8	10.24	8.01	2.23	13.3	89.2
5	20.8	10.47	9.65	0.82	3.9	32.8
6	23.1	10.18	8.90	1.29	5.6	51.6
7	14.5	10.04	9.14	0.90	6.2	36.0
8	15.5	2.13	0	2.13	13.7	85.2
9	18.3	1.98	1.12	0.86	4.7	34.4
10	15.1	2.34	1.19	1.15	7.6	46.0
11	21.6	10.14	7.50	2.64	12.2	105.6
12	24.0	10.09	8.51	1.58	6.6	63.2
13	21.8	10.03	9.16	0.88	4.0	35.2
14	12.3	9.80	8.93	0.86	7.0	34.4
Mean	**17.7**	**-**	**-**	**1.51**	**8.4**	**60.6**

In an unpublished study by NVI, the intake of AC (Rodicum Express-green bait, 40 mg/g alpha-chloralose) was qualitatively studied by photo of the stomach in six mice. The stomach was filled with green coloured AC-bait (fig 5). All six mice in the study had similar findings.

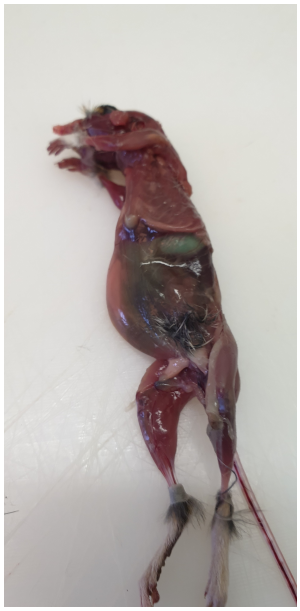


Figure 5: The picture shows a mouse with its stomach filled with the green bait formulation "Rodicum Express" containing alpha-chloralose 40 mg/g. Photo: Christin Plassen, NVI.

6 Risk of secondary poisoning

AC has been approved as a biocidal active substance in the European Union under BPD. In the competent authority assessment report from 2008 (12), it was concluded that "There is unlikely to be an issue of secondary poisoning since a limited exposure to the environment is expected. Chloralose is for indoor use only and immobilisation of mice occurs shortly after bait consumption. Reference should also be made to ESD (2003) (13), which states that the target animal, the mouse, will not eat large portions of the poison bait due to its rapid narcotic effect. Mammal predators may catch a poisoned mouse but with LD50 values no less than 100 mg/kg for cats and dogs, a secondary poisoning risk is considered negligible". However, data from the recent studies referred to in the present report contradicts several aspects of these assumptions.

In almost all (96%) of the 59 included cases in the Windahl et al. study (1), the cat was either reported to be a known rodent hunter or was known to have consumed mice prior to development of symptoms of intoxication (table 3). Consumption of mice was known in 10% of the cases through the owner's observation on the day of consumption or through the cats vomiting mice containing AC during the veterinary visit (in the Windahl et al. study (1), two of the cats included in the study vomited during the veterinary visit with the contents including one mouse each. AC was detected in these mice by analysis). As concluded in the study, these results do not directly prove that the majority of cases included in the study are cases of secondary poisoning through ingestion of mice. However, the collected data on exposure to AC together with the study of bait intake with calculation of the contents of AC in mice show the possibility of such secondary poisoning occurring. Notably, even if intoxication through direct ingestion of bait instead of mice may also occur, the availability of either bait or intoxicated mice outside the traps is a problem for susceptible animal non-target mammals and birds. The knowledge of either poisoning of other cats in the neighbourhood, or of presence of dead or somnolent mice in the cats´ outdoor living environment reported by approximately a fifth (22%) of the responders, indicate that non–domesticated animals were also at risk of AC-poisoning during the study period (table 3).

Table 3: An overview of reported anamnestic data for the 59 included cases in the Windahl et al. study (2022). Reproduced by permission from the publisher.

Access to rodents *Percentage and number of the included 59 reports*	
The cat was free roaming outdoors within 24 hours of onset of symptoms.	100% (n=59)
The cat was either a known rodent hunter or consumption of mice at the of development of symptoms was known.	96% (n=57)
The cat was a known rodent hunter, subcategories *Percentages presented are of the 57 cases where the cat was a known rodent hunter*	
The cat was spotted consuming a rodent or rodents on the day of onset of symptoms,	7% (n=4)
The cat regurgitated a mouse during the veterinary visit.	3.5% (n=2)
Suspicion or knowledge of use of rodenticides in the cats´ outdoor living environment[1] *Percentage and number of reports where data was included*	
The cat owner was unsure of presence of rodenticide products and had no knowledge of either other cats being poisoned, or of presence of dead or somnolent mice.	46% (n=27)
The cat owner reported knowledge of a rodenticide product or products being used in the cats´ outdoor living environment*[1]	32% (n=19)
Reports included either knowledge of poisoning of cats, or presence of dead or somnolent mice being noted in the cats´ outdoor living environment.	22% (n=13)
Suspicion or knowledge of use of rodenticides near the home of the cat, subcategories	
Mice regurgitated during the veterinary visit contained AC.	n=2
Knowledge of the cat consuming AC- based rodenticide mixed in cat food reported.	n=1
The veterinarian reported seeing the same cat due to similar symptoms on two separate occasions, with recovery in between.	n=2
Two owners had two of their cats developing similar symptoms during the course of one day.	n=2
The owner reported knowledge of other cats being poisoned with similar symptoms in the neighbourhood	n=1
Owner reported noting either dead or visibly somnolent rodents near the home of the cat on the day of onset of symptoms.	n=5

[1] Including use of rodenticides in buildings to which the cat had access, such as stables for large animals.

As pointed out by Tegner et al. (2), cats are known dietary neophobes, i.e., they tend to reject unknown or new foods. A pilot study on the use of AC as a poison for feral cat control showed that the AC-containing bait palatability was low, and the cats were very reluctant to eat it, even when it contained low concentrations of AC (14). Cornwell reported that cats did not eat AC bait, even when fasted for 36h (15). This may support that the poisoned cats in the studies presented were secondarily poisoned by eating mice, rather than having eaten bait directly. However, Bernhoft et al. have reported that there also are known cases of AC poisoning where cats have eaten the bait (6).

Additionally, the observations made on bait intake in mice (table 2) by Windahl et al. (1) do not support a theory that mice will not eat large portions of the poison bait due to its rapid narcotic effect. The amount of AC–containing bait consumed by the wild mice after being trapped in the study, is close to the expected daily amount feed intake at up to 12% of the body weight (1). In the same study, they also observed that the timespan from start to end of feeding was ten to fifteen minutes. Thus, even if AC is for indoor use only, there could be enough time after bait consumption for the mice to escape the building and be caught by a carnivore or bird of prey. Additionally, sedation will make the mice an easier prey.

We consider the statement from the EU assessment that "mammal predators may catch a poisoned mouse but with LD50 values no less than 100 mg/kg for cats and dogs, a secondary poisoning risk is considered negligible" to be incorrect. Poisoning symptoms can appear at considerably lower doses than LD50 (1). The calculated intake of bait by mice is below the LD50 for cats observed under animal test conditions, but such lower doses can as it has been shown in the Windahl et al. study (1) still cause significant poisoning symptoms in the cat and cause harm, due to for example heat loss, disorientation, and possible anaesthesia (6). The outcome of AC poisoning can be influenced by the ambient temperature, and there will also be individual variations in sensitivity. The prognosis is regarded as good if the cats receive supportive treatment (1,2). For animals that remain in a poisoned state outdoors in cold environments, the prognosis has been characterised as poor (6). In the report from the Norwegian Veterinary Institute, eight out of 37 cats with AC-poisoning had been euthanised or found dead (6). Some of these were euthanised even though it might have been medically possible to save them. Nevertheless, the report concludes that the calculated AC intake indicates that cats may be poisoned and die from eating mice exposed to AC. Also, even if the animals are treated successfully, there is no doubt that in these numerous poisoning cases the use of AC has led to an unacceptable suffering and undesirable effect on the health of animals. The poisoning cases have also caused major concern and financial costs for the cat owners. Finally, the risk of secondary poisoning of wildlife including birds of prey also has to be considered.

7 Significance of species-specific metabolism in cats

The increase in reports of suspected cases of AC-poisoning to national veterinary and chemical authorities/institutes in Finland, Norway, and Sweden during 2018 and 2019 were almost all reports involving cats, despite other carnivores including dogs may also consume rodents. There are several likely explanations, e.g. cats are free ranging and rodent hunters whereas the dogs do not roam freely in the same way as cats. Cats may also be more efficient mice hunters compared to dogs, and generally the cats have smaller body size compared with dogs. The higher susceptibility to AC in cats compared to other carnivores may also be related to differences in drug metabolism (6), however, exact metabolic pathways of AC are not clear. To investigate the significance of this deficiency of AC detoxification in cats, a study was conducted by the Norwegian Veterinary Institute (NVI) where urine samples were analysed both freely and indirectly for conjugated AC by determining UGT conjugates after deconjugation with β-glucuronidase (total AC) (6). The analyses showed that there was little conjugated AC in cat urine, indicating that most cats lacked or had little ability to metabolise AC in the form of glucuronic acid conjugates.

In the study by Windahl et al. (1), the urine samples were screened for AC-metabolites. Significant amounts of AC were excreted unchanged in the urine, and some metabolites existed as different isomers. In total, four different isomers of sulphate phase II metabolites and one glucuronidated phase II metabolite were detected as well as one dechlorinated and two oxidated phase I metabolites. The detection of the glucuronic acid conjugate of AC indicates that glucuronidation of AC might be possible in cats, even though cats are known to have a limited capacity to form glucuronide metabolites of drugs. Accurate quantification of the metabolites could not be performed without authentic standards, which were not commercially available. Furthermore, it was questioned if the results might be influenced by rodent metabolism, if the cats were subjected to secondary poisoning. The authors call for further investigations of the relevance of various metabolites of the compound in relation to development of clinical disease and death in various animal species including cats (1).

8 Conclusions

In several recent studies, the clinical suspicion of AC intoxication based on a healthy cat roaming free outdoors suddenly developing typical neurological symptoms correlated well with presence of AC in blood serum samples, indicating that there was a good correlation between clinical suspicion of AC poisoning and correct diagnosis.

Wild mice consume significantly more rodenticide bait than presumed in the EU assessment report (2009) (12) The calculated amount of AC–containing baits consumed by the wild mice after being trapped as described in the Windahl et al. study (1), is close to the expected daily amount feed intake in laboratory mice. Thus, the results in the study contradict the assumption that the mouse will not eat large portions of the poison bait due to its rapid narcotic effect presented in the EU assessment report.

AC metabolism was to our knowledge studied for the first time by Bernhoft et al. (6) and Windahl et al. (1) One glucuronic acid conjugate of AC was detected in feline urine from feline cases of AC– poisoning, however the studies support the theory that deficiency in enzymes responsible for conjugation reaction could make the cats more susceptible and prone to AC poisoning compared to e.g. dogs.

The calculated intake of bait by wild mice and calculated ingested amount of AC in the studied cats after ingesting poisoned mice may result in exposure below the reported LD50 for cats. However, the collected data on exposure of AC show that harmful symptoms may appear at considerably lower doses than LD50 (1). This shows that secondary poisoning of cats from ingestion of mice is possible and that the ingested doses of AC can be fatal due to heat loss, disorientation, and possible anaesthesia. For non-target wild animals that remain in a poisoned state in cold environments, the prognosis is characterised as poor. Results of the studies therefore also highlight the risk of AC poisoning to other non-target species, especially birds of prey that may be even more susceptible than cats.

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