Vitenskapskomiteen for mattrygghet
Norwegian Scientific Committee for Food Safety

Opinion of the Head Committee of the Norwegian Scientific Committee for Food Safety

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Risk Assessment of White Phosphorus

SUMMARY

White phosphorus (WP, P_4) has been used by the Norwegian armed forces in military shooting ranges in Norway and particles of WP have been found in sediments, soil and water samples from the areas. WP is toxic in animals both after acute and chronic exposure and acute poisonings in humans have been reported. High mortality in populations of aquatic birds has been reported from a military shooting range in Alaska, and millions of fish were killed when WP was discharged into the marine environment from a WP producing facility in Newfoundland. Concern has been raised whether the occurrence of WP in the Norwegian shooting ranges may pose a health risk to humans and animals. The Norwegian Scientific Committee for Food Safety (VKM) has on request from the Norwegian Food Safety Authority, therefore assessed the risk for human and animal health from residues of WP in the environment.

WP is found in a range of tissues in exposed fish and birds from other contaminated sites and after experimental exposure. The highest concentrations are found in the gizzard and intestinal content and in the skin and fat of exposed animals. WP disappears rapidly from the tissues after cessation of the exposure with a half-life of 1-6 hours in fish. There is no indication of accumulation of WP in the food chain.

Acute and chronic intoxications of humans have been reported, but the available information does not allow any estimation of the dose-response relationship.

WP is highly acute toxic in rodents, birds and fish. Acute toxic effects have been observed in birds exposed to concentrations similar to those found in the Alaskan military training range. Symptoms of acute WP intoxication were lethargy, ataxia, convulsions and death and the more slowly developing liver and kidney damage. The LD50 values in birds were 3-4 mg WP/kg bw. The lethality of WP in fish is dependent on concentration and exposure time. No effect in fish has been found in water concentrations below 1 µg WP/l.

Effects of chronic exposure to low levels of WP, including effects on reproduction, have been studied in rodents and birds. A NOAEL of 0.015 mg WP/kg bw/day was found in a one-generation reproduction study in rats. Similarly, a NOAEL of 0.5 mg WP/kg bw/day has been found in reproduction studies with ducks. Based on these findings, the United States

Environmental Protection Agency (EPA) has set a Reference Dose (RfD, equivalent of TDI) for chronic oral exposure of WP to 0.02 µg/kg bw/day, but toxicological information is lacking for important endpoints, such as genotoxicity, carcinogenicity and immunotoxicity. The EPA has derived a lifetime health advisory value of 0.1 µg WP/l in drinking water from the RfD. VKM considers this limit to be acceptable as an upper limit for WP in drinking water for humans and animals.

Humans may be exposed to WP through consumption of water and food items derived from plants and animals in the contaminated areas in Norway. There is no information about the occurrence of WP in plants, berries or mushrooms. Due to the mechanisms of uptake and the oxidative metabolisms within these organisms, VKM considers that it is highly unlikely that WP is present in edible parts of plants and mushrooms. Wild birds and fish are not consumed continuously. Any WP present will therefore disappear from the body between each exposure. VKM therefore finds it most appropriate, in this risk assessment of exposure to food items from the areas, to evaluate the risk of acute intoxication associated with short-term intake. A theoretical worst-case estimation of the intake of WP from single meals of meat from birds using the highest level of WP in bird meat reported in the literature, gives a ratio of 40 between a worst-case scenario of intake of WP and doses leading to serious toxic effects in humans. The margin between a worst-case intake of WP from a meal of fish and toxic levels is 500.

The concentrations of WP in soil, sediments and water in the Norwegian shooting ranges exceed the values reported from other sites where lethal intoxications of wild birds and fish have occurred. Furthermore, ingestion of particles of unburnt WP may cause lethal effects in grazing animals. This implies that animals feeding in these areas may be at risk for acute WP intoxication. Previous reports concerning deaths of sheep (USA) and musk oxen (Norway) associated with ingestion of environmental WP support this conclusion. The probability for this to occur in the Norwegian shooting ranges cannot be estimated from the existing information.

BACKGROUND

White phosphorus (WP) is produced from phosphate rock and does not occur naturally in the environment. The commercial product is 99.9% pure and because it may have a slightly yellow colour it is often referred to as yellow phosphorus. WP has been used in the manufacture of rodenticides, pesticides, matches, firecrackers and ammunition for military purposes. WP enters the environment from industrial spills, during storage and transport as well as from military ammunition. Because of discharge of wastewater, WP is likely to be located in water and bottom deposits of rivers and lakes near industrial plants. Several incidents of WP pollution through wastewater release have been documented in the past [1-3]. The use of WP by the Norwegian armed forces was, however, discontinued in December 2005 due to the finding of elementary WP in water from ponds, small streams and soil in the artillery training ranges.

WP oxidises rapidly in contact with air with an estimated half-life of less than 5 minutes, and the prevailing views have been that WP has low negative ecological impact because of the vigorous reactivity with oxygen. As a consequence, early research on WP pollution was not concerned with its environmental fate. Focus was put on downwind deposition of aerosols from smoke clouds and small particles [4]. However, when WP is deposited in the anaerobic zones of water, sediment and soil, it may remain inert for decades [4;5]. It has been estimated that deployment of ammunition in the field may leave as much as 10 % of unburned WP [6]

and particles with diameters ranging from 0.015 to 3.0 mm have been isolated from sediment samples in the military training sites of Eagle River Flats in Alaska [7]. The WP pollution in Alaska has been identified as the cause of massive mortality in waterfowl populations in Eagle River Flats. Due to the findings in Alaska, concern has been raised whether the occurrence of WP in military training ranges in Norway may pose a risk for animal and human health. In response, the Norwegian Food Safety Authority asked the Norwegian Scientific Committee for Food Safety (VKM) to prepare a scientific risk assessment on white phosphorus pollution in Norway. The previous lack of understanding of the environmental fate and transport of WP residues has precluded quantitative ecological risk assessment and decision making regarding its production and use.

An *ad hoc* Working Group of experts was appointed by VKM with the mandate to draft a risk assessment. Members of this *ad hoc* working group have been Gunnar Sundstøl Eriksen (chair), Ragna Bogen Hetland, Jan Ludvig Lyche and Rose Vikse. The draft assessment has been commented by the Panel on Food Additives, Flavourings, Processing Aids, Materials in Contact with Food and Cosmetics, the Panel on Contaminants in the Food Chain and the Panel on Animal Health and welfare, and the risk assessment was adopted by the Head Committee of the Norwegian Scientific Committee for Food Safety.

TERMS OF REFERENCE

White phosphorus is used by the Norwegian armed forces in military training ranges. WP is toxic both after acute and chronic exposure. It has been demonstrated that residues of WP can be found in military training ranges for some time, especially in streams, ponds, swamps and wetlands.

In 2003, the Norwegian Pollution Control Authority (SFT) asked the Norwegian Armed Forces to make a survey of the use of white phosphorus in Norwegian military training ranges. In the light of this report, the Norwegian Defence Estates Agency (Forsvarsbygg) and the County Governor of Troms established a project to investigate environmental effects of WP used in three of the military training ranges in the northern part of Norway (Mauken, Blåtind and Setermoen). A report from Forsvarsbygg was published in January 2006 with contributions both from the Norwegian Defence Research Establishment (FFI) and the Norwegian Institute for Water Research (NIVA). The Norwegian Food Safety Authority (Mattilsynet) has asked the Norwegian Scientific Committee for Food Safety to assess the risk for human and animal health from residues of WP, and has specified that the following aspects should be considered in the assessment:

- Food safety:
    - Can white phosphorus be incorporated in muscle tissue of grazing animals, birds and fish, and if so, will white phosphorus be available in a chemical form that implies that eating this meat will pose a risk to human health?
    - Can white phosphorus be incorporated in plants, berries and mushrooms, and what risk will be associated with eating these plants/crops?
- Drinking water:
    - What levels of white phosphorus in drinking water can be considered as acceptable for human and animal health?
- Animal health:
    - If domestic and wild grazing animals eat white phosphorus, what consequences can be expected and what levels of white phosphorus can be considered as acceptable for animal health?

- Should other consequences regarding animal health for fish and birds be taken into consideration?

ASSESSMENT

HAZARD IDENTIFICATION AND CHARACTERIZATION

PHYSICAL AND CHEMICAL PROPERTIES

White phosphorus (WP, CAS No. 12185-10-3), also known as yellow or elemental phosphorus, is a wax-like solid that ignites spontaneously in air between 30 and 40°C [8]. White smoke with a garlic odour is characteristic when WP burns. At room temperature, WP is a tetrahedral crystal with the molecular formula P_4. WP is virtually insoluble in water and highly fat-soluble. Therefore, it remains inert in aquatic environments. Storage of WP in water prevents it from burning spontaneously. In nature, WP can exist in water in the colloidal state, as large particles of WP, in a particle-absorbed state or it can undergo oxidation to hypophosphorus acid, phosphorus acid and phosphoric acid. In water with low oxygen pressure, WP may react with water to form phosphine (PH_3). Phosphine is a highly toxic gas that quickly moves from water to air by volatilisation. In air it is converted to less harmful chemicals in less than a day. The rate of the reactions depends on the dissolved oxygen concentration, temperature, state of phosphorus in water (dissolved, absorbed, colloidal or particle form), and probably on the pH of the solution. At concentrations well below the solubility limit of 3 mg/ml, WP will have a half-life of 2 hours at 10°C and 0.85 hours at 30°C, and the rate of phosphorus disappearance increases with the oxygen concentration and pH of the water [8]. The reported half-life of colloidal phosphorus is 80 hours at 30°C and 240 hours at 0°C at concentrations between 10 and 50 mg/l. In anoxic water, the half-life of a solid chunk of WP (1.8 g) was 2.4 years [8]. In a previous Norwegian study, approximately 90 % of the elemental phosphorus that remained non-oxidised in water was deposited in the sediment after gravitational settling, while around 10 % remained in the colloidal form [9].

EXTRACTION AND ANALYTICAL METHODS

White phosphorus is extracted by carbondisulfide or another suitable non-polar solvent under anaerobic conditions to prevent WP from being oxidised. The analytical method used is a gas chromatographic method with a nitrogen-phosphor detector (NPD) where the quantity of WP (P_4) is determined [8;10].

TOXICOKINETICS

The toxicokinetic properties of WP are poorly understood. Very little is known about absorption, distribution, metabolism and excretion of WP itself and possible metabolites. The fate of WP following exposure by any route is an open question. However, absorption of WP by oral, dermal and inhalation routes is likely since it is highly lipid soluble. The observation of toxic effects in humans following exposure to WP suggests that WP and/or metabolites are absorbed. In studies on fish [11;12] and birds [13], elemental WP has been detected in organs, indicating uptake via the oral route in those species. Furthermore, toxicokinetic studies on fish and American kestrels have detected WP in tissues of living organisms only for a short time, indicating that degradation and/or elimination processes are active [11-14]. No studies that quantify absorption or tissue concentrations in animals and humans following dermal exposure are available. Qualitative evidence for dermal absorption is the observation that dermal burns by WP produce additional systemic effects. Experimental studies on rats and rabbits exposed via the dermal route reported severe systemic effects and mortality rates up to

100 % depending on the dose [15;16]. There are no studies available that report deaths in humans or wild animals due to WP absorption via the dermal and inhalation routes [8].

In a military training range in Alaska, levels of 1600 µg/kg muscle have been found in birds killed by WP poisoning. The gizzard and intestine of these birds contained 14 978 000 and 820 000 µg WP/kg and the fat contained 45 000 µg WP/kg [17]. WP accumulated in fat and skin of American kestrels given 6.4 µg WP/g feed for up to 7 days, and 0.03 µg WP/g was found in thigh muscle in one bird, while no WP was detected in the remaining 14 exposed birds. The levels in skin and fat decreased rapidly after cessation of the exposure [13]. WP is shown to be transferred to eggs in exposed herring gulls, and concentrations of 3-15 µg/kg were found in the yolk of herring eggs [8].

In a toxicokinetic study on cod and salmon, it was observed that WP accumulated in all tissues, including liver and muscle when WP was added continuously to the water. After the end of the treatment WP disappeared rapidly from the tissues, and the half-lives were estimated to 6 h for cod and 1 h for salmon [12]. Furthermore, the concentrations of WP detected in fish tissues did not correlate directly with the level of damage. Based on this observation the author hypothesised that the tissue damage and/or death may be related to the amount of WP "metabolised" or oxidized in the animal, suggesting that low O_2 pressure in a contaminated individual may reduce the extent of damage [11;12].

FOOD CHAIN BIOACCUMULATION

The biomagnification potential for WP in predators consuming contaminated prey organisms has not been studied systematically. However, in one study, WP was found in the tissue remains of ducks that had been preyed upon by bald eagles, herring gulls and common ravens, thus providing positive evidence that predators were exposed to WP. Subsequently, WP was detected in the fat tissue of one eagle and in one herring gull egg, indicating the ability of WP to relocate from prey to predator [7;18;19]. The potential for chemicals to bioaccumulate in an organism and biomagnify in food chains depend on their lipophilicity and/or specific affinity to other tissues and the ability to resist degradation. WP is highly fat soluble, but it seems to be effectively degraded in living organisms, suggesting a low bioaccumulation and biomagnification potential. In nonliving biological tissues, however, such as fish tissues preserved by freezing, salting and drying [12;20], and frozen duck tissues [17], WP levels were relatively stable for weeks.

TOXICITY

Acute toxicity

WP is extremely toxic both to humans and animals following inhalation, ingestion or absorption through burned areas of the skin [21]. Many case reports from intentional and accidental ingestion of rat- or cockroach poison and firecrackers have been published [8]. Symptoms usually include vomiting and extreme abdominal pain, fall in blood pressure, intense thirst, restlessness and delirium followed by death resulting from multi-organ failure, usually of the liver, kidney, heart and brain. Acute oral exposure to high levels of WP may develop through three stages: The first stage is dominated by gastrointestinal effects, the second stage (2-7 days) is apparently symptom free, and the third stage is characterised by severe gastrointestinal, kidney, liver, cardiovascular and CNS effects. In such cases, the exposure dose is usually unknown because vomiting expels part of the ingested dose. However, in one case, a woman ingested a single dose of 2 mg WP/kg bw without vomiting before dying and this identifies an acute toxic dose [8]. It has also been reported that oral

intake of 1 mg/kg bw of WP may be lethal to humans [22]. Furthermore, serious toxic effects have been observed after human intake as low as 0.2 mg WP/kg bw [8].

Animal data support the acute oral toxicity data of WP observed in humans described above. Early experimental work in waterfowl found that WP produced liver and kidney damage and could be lethal in concentrations as low as 1.7 mg/kg bw [23]. High mortality rates have been reported in rats and mice experimentally exposed to single doses ranging from 3 to 6 mg WP/kg bw [24;25]. The oral LD50 value for Charles-River rats was found to be 3.03 mg WP/kg bw for females and 3.76 mg WP/kg bw for males. For female and male mice, the LD50 values were reported to be 4.82 and 4.85 mg WP/kg bw, respectively [8].

Farm-reared mallards experimentally dosed with WP in concentrations similar to the high levels found in Eagle River Flats, showed behavioural symptoms nearly identical to those of wild ducks observed in the field [17]. One study has described two sets of acute toxicological effects in WP exposed mallards. Rapid effects resulted in symptoms consistent with anoxia, including lethargy, ataxia, convulsions and death. Slower acting effects lead to liver and kidney damage with demonstrable hepatomegaly, lipid accumulation in liver and cellular necrosis. These slowly developing effects may or may not be linked to mortality. The study also determined the LD50 dose in mallards to be 3.9 mg WP/kg bw [14]. In mute swans the LD50 dose was found to be 3.65 mg/kg bw when WP was administered as pellet with water [26].

WP is toxic to both fish and marine invertebrates [12]. The lethality of WP to fish depends on the concentration in the water, and the LC50 was found to be 14.4 µg/l. No observable adverse effect levels (NOAELs) were found at concentrations below 1 µg/l [27]. The exact cause of the toxicity is unknown. However, WP may act on both external and internal structures and tissues, such as the gills, liver and brain. Damage to both gill epithelia and liver tissue has been reported [11;12]. Furthermore, haemolysis has been observed in several of the WP polluted fish species suggesting a possible mechanism for toxicity. However, this effect was not universal among fish because two of the species studied did not exhibit haemolysis. The ubiquitous distribution of WP observed in fish indicates that it has potential to affect any tissue in the body [11;12].

Chronic toxicity

Effects of chronic exposure to low levels of WP, have been observed among workers in occupational settings such as production of fireworks and matches. In these cases, the total exposure was a result of both inhalation and ingestion of WP-containing airborne particles, as well as dermal contact when handling WP. Some of these workers developed phossy jaw, a degenerative condition affecting the soft tissue, bones and teeth of the oral cavity. Furthermore, increased mortality, chronic cough and alterations in haematological parameters have been observed in workers chronically exposed to WP [8].

Up to 1940, WP was used for therapeutic purposes in treatment of rickets in small children. The therapeutic doses ranged from 0.03 to 0.08 mg WP/kg bw/day for 3-6 months. During treatment, the children developed increased density bands in the ends of tibia, fibula and femur, representing decreased absorption of the calcified cartilage matrix [8]. It should be noted that the patients were rachitic children and the relevance of the observed effects to potential effects of WP in healthy children cannot be ascertained. Treatment effects of WP exposure on growing bones have also been found in rabbits and rats [8]. Young, growing rabbits exposed to 0.3 mg/kg bw/day for an intermediate duration (9 days) had bands of increased density in metaphyseal regions of fibula and tibia. Likewise, weanling rats exposed

to 1.25 mg/kg bw/day WP for an intermediate duration (16 days) had changes in bone composition that were related to alterations in morphology and function of osteocytes resulting in widening trabeculae and denser metaphysis [28].

In a one-generation reproduction study, groups of rats were given different oral doses of WP (0, 0.005, 0.015 or 0.075 mg/kg bw/day) prior to mating and continuing through weaning of two complete reproductive cycles. Effects that were attributed to the intake of WP were loss of hair on the forelimbs, death while giving birth (53% at the highest dose), decreased number of viable pups at birth and increased number of stillborn pups. At dosage levels of 0.015 mg WP/kg bw/day or less, no adverse effects were observed [29]. The findings were confirmed in another one-generation reproduction study. In this study, pregnant rats were dosed orally with 0.075 mg WP/kg bw/day for 145 days. Late in gestation or during parturition 30-47% of the rats died, suggesting that reproductive mechanisms may be disturbed [8].

In order to characterise possible chronic effects of WP in wild waterfowl, feeding studies were conducted on captive birds using lower levels of WP relevant for less contaminated parts of the Eagle River Flats. Sublethal effects observed in mallards experimentally exposed to 0.5 mg WP/kg bw included reduced blood haematocrit and haemoglobin, elevated blood liver enzymes and altered leukocyte profiles [30]. Similar changes in blood constituents have also been found in American kestrels [31]. In mute swans exposed to 2.98 and 3.64 mg WP/kg bw, histological changes were found in liver and other organs one week after exposure. Among surviving birds, changes in liver enzymes were also observed in all dose groups [26]. Reproductive effects, such as significant reduction of laying frequency and translocation of WP from hen to eggs, have been reported in hens receiving 1 mg WP/kg bw for 5 days [32]. These data raised the possibility that wild waterfowl may also suffer adverse reproductive effects when repeatedly exposed to low levels of environmental WP. In mallards exposed via the oral route to doses as low as 0.5 mg WP/kg bw/day for 7 days, fertility and hatchability were reduced and the occurrence of teratogenic effects was increased in their embryos [33]. In male mallards exposed to 1 mg WP/kg bw/day, the plasma testosterone levels were significantly reduced. Furthermore, in the same experiment WP was detected in eggs for at least 48 hours at all dose levels. These data suggest that reproductive capacity of free-ranging waterfowls may be impaired when exposed to WP at typical field levels. Decreased testosterone in males indicates a possibility for an endocrine effect [33].

There is no information available for other important endpoints of toxicity, such as genotoxicity, carcinogenicity or immunotoxicity.

Table 1. Values of toxicity after oral intakes of white phosphorus reported in the literature.

Species	Effects	Level	Reference
Humans	Acute lethal dose	1-1.4 mg/kg bw, single dose	M.E. Welsh* US EPA
	Serious toxic effects	0.2 mg/kg bw, single dose	Reference in Søbye et al., 2003
Mammals	Variable dead	1-10 mg/kg bw, single dose	Johnsen et al., 2002
Rats	Acute lethal dose LD50	3-4 mg/kg bw, single dose	M.E. Welsh*
	Sublethal effects	0.05 mg/kg bw/day	Johnsen et al., 2002
Dog	Sublethal effects	0.05 mg/kg bw/day	Johnsen et al., 2002
Ducks	LD50	4.05 mg/kg bw, single	Sparling et al., 1997

		dose	
	LD50 cubicle particle	1.3 mm	Roebuck et al., 1998
	Teratogenic	0.5-2 mg/kg bw/day	Vann et al., 2000
	Hatching success	0.5 mg/kg bw/day	Vann et al., 2000
	Fertility	1.0 mg/kg bw/day	Vann et al., 2000
Swans	LD_{50}	3.65 mg/kg bw, single dose	Sparling et al., 1999
Fish	Lethal concentration LC_{50}	14,4 µg/l	Reference in Løvik and Rognerud, 2005
	No observed adverse effect level (NOAEL)	1.0 µg/l	Reference in Løvik and Rognerud, 2005

* M.E. Welsh, US Army Corps of Engineers: Personal communication to S. Rognerud, NIVA.

Field observations

The most significant evidence for a cause-effect relationship between WP exposure and toxic effects in wildlife populations is the massive mortality among waterfowl caused by ingestion of residual WP particles from smoke-producing grenades in Eagle River Flats, Alaska [34]. In wet anoxic sites in Eagle River Flats considerable amounts of solid WP particles were detected (see Table 3). The concentrations ranged from 0.0025 to 198 mg/kg in the sediment samples collected [7;17;35]. The analysis of samples collected before and after explosion tests indicated that the level of WP increased as a result of artillery training. The particles of WP in this area appear to be the major risk to waterfowl feeding in the sediments and their predators. The acute symptoms of poisoning observed in wild ducks included repeated head shaking and rolling. Death was preceded by severe trembling and convulsions, and autopsy findings resemble those found in rodents and humans, including liver degeneration, necrosis, haemorrhages and fatty infiltration. WP was found in the tissues and the gizzard of intoxicated waterfowls. Mortality was mainly found in waterfowl species feeding in the bottom sediments, and it was hypothesised that the poison was located in the bottom sediments. However, incidents of secondary poisoning of predators were also discovered. The concentrations detected in dead birds collected in Eagle River Flats are given in Table 2. As a consequence of these findings, experiments were conducted to determine the lethal dose of WP to waterfowl and to study possible sublethal effects, the distribution of WP in ducks, the risk of secondary poisoning to predators of polluted ducks, the identification of possible biomarkers of WP intoxication and possible reproductive toxicity [34].

Table 2. The concentration of WP detected in birds collected in Eagle River Flats.

Species, tissue	**Mean (max) WP level in mg/kg**	**References**
Swans, gizzard	52 (207)	Racine et al., 1992
Swans, fat	0.67 (7)	
Swans, skin	0.060 (0.140)	
Duck, gizzard	304 (3140)	Racine et al., 1992
Duck, fat	0.21 (0.43)	

Duck, skin	0.07 (0.13)	
Duck, liver	0.05 (0.14)	
Dead bald eagle, fat	0.06	Bird, 1991
Herring gull, egg yolk	0.003-0.015	Bird, 1991

Discharge of WP into a marine environment from a WP producing facility at Newfoundland, Canada, led to millions of fish being killed and closure of the entire fishery for several months [1]. Moreover, during the same incident, huge mortality rates were observed in bottom living organisms. In dead lobster that had been exposed to WP via the wastewater prior to their death, damaged cells were found in several organs [36].

WP polluted pasture may pose a threat to grazing domestic and wild ruminants. Acute toxic effects leading to death of sheep ingesting soil containing WP have been reported. Prior to death the animals exhibited symptoms such as abnormal thirst and extreme muscle weakness. Following autopsy, WP was identified in the gastrointestinal tract of all cases [37]. Furthermore, the death of five musk oxen in a former military training site at Hjerkinn, Norway, was associated with intake of WP residues from the pasture [38]. Pathological changes were found in the kidneys and the gastrointestinal tract, and it was concluded that the animals died due to WP intoxication [38]. At Hjerkinn, WP was detected 1.5 years after the military activity was ended [10] supporting the finding from Alaska that unburnt WP may remain inert in nature for years and pose a potential threat to wild and domestic ruminants in the polluted areas. Few data exist on the effect of chronic exposure for either water- or land-based animals. The effects of long-term exposure to low to medium doses of WP on important target organs, such as the immune system and the central nervous system, are not characterised. Toxic effects on the CNS could potentially affect feed intake, lead to adverse emotional reactions and aberrant behaviour.

EXPOSURE CHARACTERISATION

HUMAN EXPOSURE

EXPOSURE FROM FUNGI AND PLANTS, INCLUDING BERRIES

To our knowledge, there are no available data on WP in plants, berries or fungi in Norway or elsewhere.

Plants and fungi are normally only capable of absorbing ions and simple organic molecules such as some amino acids, but not fat soluble, non-polar compounds like WP. Furthermore, both plants and fungi normally grow in aerobic soil, and root systems into the anaerobic layers are uncommon. The metabolic processes within these organisms are largely aerobic. The relatively few plant species growing in anaerobic soil often transport oxygen from the upper parts to the lower parts of the plant to supply the roots with oxygen. As WP is very unstable under aerobic conditions, it is not likely that any phosphorus would remain in the reduced form and be transported to upper parts of the plants. VKM therefore considers it unlikely that consumption of fungi or berries from the contaminated areas would give any

significant intake of WP, unless particles of white phosphorus adhere to the surface. This could be verified by analysis of plants or fungi from contaminated sites.

EXPOSURE FROM GAME BIRDS AND EGGS

There is no information available about levels of WP in wild birds from the contaminated sites in Norway.

WP has been found in muscle and other tissues in birds intoxicated by WP in the field, as well as in birds and eggs under experimental conditions (see chapter on toxicokinetics and Table 2). The levels in all tissues decreased rapidly after cessation of the exposure. Experimentally, a small fraction of WP is transferred to the egg, but the public consumption of eggs from wild birds is low. The consumption of eggs from wild birds is therefore not considered to pose any risk to the consumer, even for the consumers of eggs from game birds.

The existing data on concentrations of WP in bird meat are derived either from poisoned and dead birds or from experimental conditions with birds exposed to high doses of WP. These data are considered to be an overestimate of the concentrations found in the shooting ranges since WP is rapidly excreted (see chapter on toxicokinetics), and no traces are found in muscle a short time after exposure.

Since there may be a certain time delay from consumption until any visible sign of toxicity occurs, there is a low probability of hunting a bird shortly after it has ingested a particle of WP, before the bird is either ill or WP has disappeared from the muscle. It is not possible to estimate the probability of catching such a bird. Furthermore, the absorption rate is unknown, and it is not possible to predict the tissue concentration during the period shortly after ingestion.

The highest concentration reported in bird muscle is 1.6 mg WP/kg found in a poisoned dead, swan. This may be considered as a worst-case scenario, and it is unlikely that muscle concentrations in apparently healthy birds could reach this level. Consumption of one meal (200 g) of this meat would give an intake of 0.32 mg WP, or 0.005 mg/kg bw for a person weighing 70 kg.

The average and high intake (95 percentile) of game bird in Norway is 2 or 3 g/day respectively [39]. The estimated worst-case average intake based on the maximum concentrations measured in intoxicated birds would be 3.2 µg (2 g/day x 1.6 µg WP/g muscle). The corresponding high intake would be 4.8 µg/day. Assuming a body weight of 70 kg, this would correspond to 0.05 or 0.07 µg WP/kg bw/day after average and high intakes, respectively. However, it is considered to be highly unlikely that all the game bird consumed meat would have levels close to the highest levels measured in wild birds killed by WP intoxication.

EXPOSURE FROM FISH

There is no information available about levels of WP in fish from the contaminated sites in Norway. Very few data from edible parts of fish are available from other contaminated sites, but 3.13 – 138.8 µg WP/kg was found in muscle of channel catfish and 30 µg WP/kg in mackerel muscle collected at heavily contaminated sites with a mass death of fish (8). The reported concentrations were a result of very heavy pollution and are not regarded as a realistic measure of concentrations in the military training ranges where the fish may periodically be exposed by oral intake of WP particles. There is a low probability of catching a fish that has recently ingested a particle of WP from the sediments, but the existing

information does not allow an estimation of this probability. The highest reported levels from intoxicated fish may be used in an estimation of a worst-case intake of WP from fish. The intake of WP from one meal (200 g) of a fish with a concentration of 139 µg WP/kg would lead to intake of 0.03 mg WP, or 0.0004 mg/kg bw (bw = 70 kg).

The average and high intake (95 percentile) of freshwater fish amongst consumers in Norway is 4.4 and 21 g/day respectively [40]. Assuming that all the ingested muscle contained the highest reported levels of WP from fish muscle, this would lead to a daily intake of 0.611 µg WP/day (4.4 g muscle/day x 139 µg WP/kg muscle) or 0.0087 µg WP/kg bw/day. The corresponding high intake would be 2.919 µg/day or 0.042 µg WP/kg bw/day. However, it is considered to be highly unlikely that all the consumed fish would contain the maximum measured level of WP since this value is measured in fish dead from WP intoxication.

EXPOSURE FROM GRAZING ANIMALS

There is no information regarding amounts of white phosphorus that may occur in mammals, but the possibility of finding white phosphorus in animal-derived food products can not be ruled out. Findings from fish and birds indicate a rapid elimination of white phosphorus from muscles (se above). If these findings are representative for mammals, only consumption of meat from animals slaughtered during or shortly after exposure is likely to give an intake of WP.

STABILITY OF WP IN FOOD ITEMS

WP is stable in eggs at room temperature for at least 48 h (unpublished data referred by Nam et al., [32]). Similarly, WP has been found to be stable for weeks in non-living biological materials, such as frozen fish muscle and frozen duck tissues [20]. The findings suggest that, any edible part may contain WP if an animal is slaughtered shortly after ingesting WP.

EXPOSURE FROM DRINKING WATER

The registrations of high values of WP in the streams running out of Setermoen, Mauken and Blåtind shooting ranges (Table 3) led to an immediate analysis of WP in drinking water originating from the drainage basin of these shooting areas. All houses with drinking water supplies connected to streams and rivers flooding out of the shooting ranges were registered. In January 2006, a total of 22 samples of drinking water (14 from ground water, 8 from streams), were collected and analysed for WP [41]. None of the collected samples of drinking water demonstrated WP content above the detection limit (< 0.015 µg/l). This result suggested that no WP was transported from the military training areas in Troms to the surrounding drinking water wells [41]. It is, however, important to take into account that WP in the drinking water of concern has been analysed only once.

ANIMAL EXPOSURE

From three military training ranges in Troms County (Norway), Setermoen, Mauken and Blåtind, a total of 26 and 50 samples from water and sediment, respectively, were collected and analysed for WP. The levels are summarised and compared with the levels found in Eagle River Flats in Table 3. The samples were collected August 22nd to 26th 2005 during a period of nice and dry weather except for one day with a heavy rainfall [42]. This rainfall resulted in increased flow of water in the streams and rivers and may have led to episodic higher levels of

WP in the water samples due to whirling up of particulate WP from sediments. The water samples were from ponds, small streams within the military training ranges and from streams leaving the area. The samples were not filtered before analysis. Eighteen samples contained WP above the detection limit (0.015 μg/l) in concentrations from 1.3 to 5.1 μg/l. The concentrations of WP in streams running out of the area ranged between < 0.015 to 3.7 μg/l [42]. It must be emphasised, however, that these samples were collected during one specific month in 2005. Concentrations of WP in water may differ considerably throughout a year, depending on e.g. rainfall and snow melting. In addition, it has been reported that these data are hampered by additional analytical uncertainties due to contamination of samples due to to carry-over between samples and consequently an overestimation of the WP concentrations [43]. The existing data are used in the evaluation despite this uncertainty, since no other data are available.

In the three shooting ranges included, soil and sediment samples were collected, based on previous knowledge, at spots suspected to contain high levels of WP. As reported from Eagle River Flats, Alaska, remaining WP in soil and sediments will not be evenly distributed throughout the shooting ranges, but may be found as "hot spots". Since WP is easily oxidised in air, such "hot spots" will mainly be found in water filled shell craters and other wet anoxic areas. As a consequence, the concentrations of WP in soil and sediments show huge variations within limited areas. Analysis of the collected samples demonstrated concentrations from below the detection limit, 0.005 mg/kg, and up to 5700 mg/kg, exceeding the levels reported from Eagle River Flats (Table 3). Hot-spots were targeted as far as possible during the sampling, but there is no information available about how widespread these hot spots are. There are also uncertainties related to the estimates of the total amount of WP spread in the areas.

Table 3. Concentrations of WP measured in soil, sediments and water from samples collected from areas where WP has been produced or used. Norwegian samples are collected between 22nd and 26th of August, 2005 (n = number of samples).

Country/Area	Soil/sediment mg/kg (n)	Water µg/l * (n)	References
Norway/Kobbryggdalen	<0.005-2000 (17)	1.1-3.3 (6)	Strømseng et al., 2005
Norway/Liveltskardet	<0.005-2300 (8)	<0.015-4.9 (4)	Strømseng et al., 2005
Norway/Mauken	<0.005-5700 (11)	<0.015-5.1 (6)	Strømseng et al., 2005
Norway/Blåtind	<0.005-0.11 (14)	<0.015-3.7 (10)	Strømseng et al., 2005
Norway/Hjerkinn	<0.4	0.023	
USA/Eagle River Flats, Alaska	<0.0001-198		Racine et al., 1992
USA/Pine Bluff Arsenal, Arkansas	5.2		Spanggord et al., 1985
USA/Yellow Lake, Arkansas		<0.02-40.4	Pearson et al., 1996
Canada/Long Harbour, Newfoundland	5.5-2910		Idler, 1972

* In a newly published FFI note, technical problems with the analysis of WP in the water samples collected in the Norwegian shooting ranges were reported. These problems may have led to an overestimation of the concentrations of WP in water [43].

The concentrations of WP detected in soil and sediments in military training areas in Norway are higher than those measured in Eagle River Flats and other polluted areas (Table 2), indicating that the occurrence of WP in Norwegian military training ranges may pose a threat to wild animals and birds living in the areas. Likewise, WP polluted soil may also cause harm to domestic animals on pasture. The highest concentrations of WP measured in water samples from Setermoen, Mauken and Blåtind shooting fields were higher (3.3-5.1 µg/l) than the NOAEL (1.0 µg/l) for fish, suggesting a potential for adverse health effects to fish in the areas. However, such high concentrations are measured in water from small ponds and streams, and the levels will undergo extensive dilution in the biggest rivers and lakes far away from the sources of pollution. Animals, however, may drink still water from craters that theoretically may contain much higher concentrations than measured in streams and rivers.

RISK CHARACTERISATION

PREVIOUS EVALUATIONS

HEALTH RISK FOR HUMANS EVALUATED BY US EPA

The United States Environmental Protection Agency (EPA) has set a Reference Dose (RfD, equivalent to tolerable daily intake, TDI) equal to 0.02 µg/kg bw/day for chronic oral exposure to WP. No studies have been located regarding cancer in humans after oral exposure to WP. WP is therefore not classifiable with regard to human carcinogenicity (EPA Class D). The reference dose is based on the reproductive study by Condary et al. [29] described earlier. The NOAEL of 0.015 mg/kg bw/day from this study was used for derivation of the oral RfD. Uncertainty factors used are a factor of 10 for interspecies variation, 10 for intraspecies variation, and 10 for incomplete reproductive/developmental data and a less than adequate lifetime study. The oral RfD has been given low confidence. The study does not provide unequivocal evidence of adverse effects of WP at the doses tested because the exact nature of the deaths was not conclusively related to WP. Supporting studies indicate significant WP-related body weight and/or bone changes, but these studies have design deficiencies that lower the confidence of the reported observations [44-46]. Screening of more recent toxicological literature pertinent to the RfD of WP has not identified any important new studies. The RfD of 0.02 µg/kg bw/day for chronic oral exposure is therefore used in the risk assessment of WP. An acute RfD value was not established due to lack of data [8].

HEALTH RISK FOR HUMANS EVALUATED BY FFI

In August 2005, the Norwegian Defence Research Establishment (FFI) performed a risk assessment on the military use of WP in Troms County, Norway [42]. Such evaluations generally use a guideline for risk assessment provided by The Norwegian Pollution Control Authority [47]. This guideline is based on the assumption that pollution in a restricted area is relatively homogenously distributed. However, the levels of WP in soil and sediments are not evenly distributed throughout the shooting ranges in Troms (Table 3). Therefore, FFI has developed a tool for risk evaluation based on Bayesian network and Bayesian statistics. This is a mathematical method that allows calculations of probabilities for exposure to harmful concentrations of WP and other compounds with uneven distribution. The FFI report concludes that today's use of the areas pose no risk to human health. If water and rivers within the shooting ranges are used permanently as drinking water, health effects cannot be excluded. However, the concentrations measured in the water samples are encumbered with

uncertainties due to technical analytical problems and the risk may be overestimated according to this report [43].

HEALTH RISK FOR ANIMALS EVALUATED BY US EPA AND FFI

Wild and domestic animals, birds and fish have been exposed to WP from spills from WP producing plants, from leakage during storage and transport and military use in ammunition. As discussed above incidents of deaths among waterfowl, fish and grazing ruminants exposed to WP pollution in the environment have been reported. Based on research conducted in military training ranges in Eagle River Flats, Alaska, levels for various acute and toxic effects in waterfowl have been established (Table 1). Furthermore, LC50 and NOAEL for fish have been estimated to be 14.4 µg/l and 1 µg/l, respectively [27]. However, no reliable values of toxic levels are available for wild and domestic ruminants. Consequently, risk assessment of WP exposure in wild and domestic mammals has to be based on values derived from studies on humans, laboratory animals and birds. By using the mathematical model discussed above and toxic values derived for humans, FFI estimated a probability of 6% for adverse effects due to repeated exposures for grazing ruminants when residing in the areas for longer periods of time [42]. The increased probability for animals compared to humans was based on the assumption that animals may ingest more soil and drink still-water from grenade craters. Furthermore, the report concluded that wild animals residing in the areas their entire life may develop chronic effects.

HEALTH RISK FOR HUMANS

FOOD

Food items originating from the shooting ranges, e.g. berries, mushrooms, fish and game meat are not likely to be consumed on a regular basis. Since WP is eliminated after a few hours in tested species of birds and fish, it is unlikely that the long-term intake of WP from these food items will represent any health risk. Food items derived from plants and fungi are not likely to contain any WP. However, there are no analytical data available to confirm this assumption.

There is a low probability of catching a fish or a bird shortly after they have ingested a particle of WP. Muscle tissue and other edible parts may contain WP for a certain period of time after ingestion. The estimated intake of WP from consumption of meat from birds exposed to lethal doses of WP may lead to an intake of 0.005 mg WP/kg bw from a single meal. Similarly, a worst-case estimation of intake of WP from fish from one single meal would reach 0.0004 mg/kg bw. Acute effects in animals, including humans, are observed after ingestion of 0.2 mg/kg bw. These intake estimates may be compared to the acute toxic effects observed after intake of doses of WP exceeding 0.2 mg/kg bw (Table 1) leading to a ratio of 40 between a theoretical worst-case scenario of intake of WP from bird meat and doses leading to serious toxic effects. The same margin between a worst-case intake of WP from a meal of fish and toxic levels is 500. When considering the margin between a potential worst-case intake and any acute toxic effect level, one should bear in mind that the effect levels are close to lethal doses.

An average intake of fish and game birds from the contaminated areas may lead to an intake of WP exceeding the RfD if all the meat is containing concentrations similar to the maximum concentration of WP measured in dead fish or bird. VKM considers this to be highly unlikely, since a high concentration in muscle only would be found shortly after ingestion of a particle of WP due to the short disappearance time from muscle. VKM therefore considers that

chronic exposure to WP from food items derived from these areas does not pose any risk to consumers.

DRINKING WATER

The concentrations of WP in the River Kobbrygg, running out of the Setermoen military training range where the highest concentrations of WP were found, were measured to be 1.1 - 3.3 μg/l [42]. Given that a person weighing 70 kg drinks 2 l /day, regular consumption of this water would represent an intake of up to 0.1 μg/kg bw/day. The reference dose (RfD or TDI) of WP set by EPA is 0.02 μg/kg bw/day. If drinking water is the only source of WP, the daily intake of this person will exceed the RfD five times, while the lifetime US EPA health advisory (HA, see below for definition) for WP in drinking water will be exceeded by a factor of 33. Thus, if water from the River Kobbrygg was to be used regularly as drinking water, health effects could not be excluded. Daily consumption of water from within the shooting ranges will exceed the RfD and therefore pose a risk to human health.

The water from streams and rivers leaving the discussed Norwegian shooting ranges will be diluted several times before reaching populated areas where it may be used for household purposes. Drinking water collected in all the concerned houses was analysed, and no WP was detected. However, soil and sediments that are polluted by WP may function as reservoirs. The WP particles may be mobilised in the streams and rivers, e.g. during periods with heavy rainfall or as a result of melting snow and ice. Care should therefore be taken when the water is used as drinking water during such periods and factors such as distance from the contaminated sites, water source, filtration etc should be considered. The probability of mobilising WP from sediments and soils can not be estimated since there is no information available about how widespread the “hot spots” are.

The drinking water in the area is most likely not to be contaminated with WP, but the tap water should be reanalysed during flood periods when there is a probability of remobilisation of particles, to ensure that such remobilisation has not occurred.

US EPA provides lifetime health advisories (HA) for several contaminants found in drinking water, including WP [48]. HA describe non-regulatory concentrations of drinking water contaminants at which adverse health effects would not be anticipated to occur. In derivation of a lifetime HA for WP, US EPA has converted the RfD value to a concentration of WP in drinking water. The lifetime HA for WP in drinking water estimated by EPA is 0.1 μg/l [48]. In derivation of lifetime HA, the chronic RfD is multiplied by typical adult body weight (70 kg) and divided by daily water consumption (2 l) to provide a Drinking Water Equivalent Level (DWEL). To determine the lifetime HA, the DWEL is multiplied by a percentage (often 20 %) believed to be the level of exposure resulting from drinking water compared to other sources. The contribution of WP from drinking water is set to 20 %. VKM has not found any documentation that gives reason to anticipate a different contribution from drinking water, even if the probability for intake of WP from other sources is regarded to be very low.

VKM finds that the concentration of 0.1 μg/l, estimated as HA by the US EPA, for the protection of human health is acceptable as an upper limit for content of WP in drinking water.

HEALTH RISK FOR ANIMALS

Theoretically, wild and domestic ruminants may be exposed to WP via the oral route by consuming soil and/or water. The concentrations detected in the training ranges under

investigation (Mauken, Blåtind, Setermoen) range from 0.015 µg/l to 5.1 µg/l for water, and from 0.005 mg/kg to 5700 mg/kg for sediments. In worst-case scenarios, ingestion of soil containing the highest levels may represent a lethal dose to grazing ruminants. For example, a sheep (50 kg) or reindeer (150 kg) ingesting 50 g soil containing 5700 mg/kg WP will receive doses of 5.7 mg/kg bw and 1.9 mg/kg bw, respectively. These doses are within the range of the lethal dose for mammals (1-10 mg/kg; Table 1). Grazing animals may also ingest single doses of WP from particles exceeding 0.2 mg/kg bw, which leads to chronic damage to organs in humans. Considering that some of the samples collected in Troms generated smoke, an indication of an ongoing oxidation process, it is not unlikely that large particles of unburnt WP are present in the fields. In Eagle River Flats particles of WP with a diameter as big as 3 mm were found [4]. WP particles were also isolated from the gizzard of a mallard carcass in which over 3 mg of WP was measured [4]. White particles such as these may be mistaken as small salt particles and ingested by ruminants. Typically, in the shooting ranges in Troms, very high concentrations of WP were found in relatively new craters filled with water. Grazing ruminants may drink water from such craters and at the same time ingest soil from the bottom, thereby taking in high doses of WP. At present, there are no reports of deaths or any other adverse effects in grazing animals or other species in the training ranges in Troms. The probability that grazing animals may ingest highly contaminated soil or water can not be estimated since there is no information about how widespread the “hot spots” are. However, at Hjerkinn the death of five musk oxen were associated with WP intoxication.

To date no systematic research has been conducted on possible health effects in animals, birds or fish in the Norwegian training ranges. Effects of long-term exposure of low to medium doses of WP on the different target organs, including the immune system and the central nervous system, have not been possible to assess. Toxic effects on the CNS could affect feed intake, and lead to adverse emotional reactions and aberrant behaviour. These effects clearly affect animal health and welfare over time.

A clear association was found between huge mortality rates among waterfowls and oral intake of WP polluted sediments in military training sites in Alaska. In those fields, the poison was mainly located in anaerobic zones in the bottom sediments in wet areas, and in accordance with this, acute intoxication was generally observed in waterfowl species feeding in bottom sediments, such as ducks and swans. In the same studies, the risk of secondary poisoning to predators eating contaminated ducks was also reported. Considering that the levels of WP detected in Norwegian military training ranges exceed the levels in Alaska, birds and their predators feeding in these ranges may be at risk for WP intoxication. To date, however, no mortality in bird or fish populations has been observed in the military training ranges in Norway, but no systematic observations have been conducted.

The highest concentrations of WP measured in water samples from Setermoen, Mauken and Blåtind shooting ranges are higher (3.3-5.1 µg/l) than the NOAEL (1.0 µg/l) for fish, indicating a potential for adverse health effects in fish in those areas. Furthermore, the concentration in water may increase during snow melting and flooding after heavy rainfalls. The risk for fish will also increase immediately after artillery training because it has been shown that the level of WP in water and sediments is increasing following explosion tests. Fish populations in larger rivers and lakes may be at less risk of intoxication due to dilution of the WP concentrations.

GAPS IN THE DATABASE AND NEED FOR FURTHER STUDIES

The residues of WP in the military ranges are a potential source of contamination of drinking water. More data about the flux, degradation, solubilisation and oxidation of WP in the contaminated areas is needed. In order to quantify an eventual leakage of WP, more water samples should be analysed. Samples should be collected under different climatic conditions, e.g. dry periods, before and after rainfalls and during periods of snow melting.

Due to the huge gap in database regarding toxicokinetic and toxicodynamic properties of WP, controlled exposure studies on larger animals, such as wild and domestic grazing ruminants, are needed. Experimental studies may provide data on absorption and tissue levels that enable more appropriate risk assessment.

There is no available data on analysis of WP in grass, plants, fungi or berries growing in contaminated sites. Such information may be desirable, even if it is considered to be unlikely that these items will contain WP in any significant amounts.

Furthermore, if WP is to be allowed in shooting ranges in the future, research should include investigations of possible effects on the welfare of relevant bird and mammalian species. Such studies should focus on effects of long-term exposure to low to medium doses of WP.

CONCLUSIONS

FOOD SAFETY

Can white phosphorous be incorporated in muscle tissue of grazing animals, birds and fish, and if so, will white phosphorus be available in a chemical form that implies that eating this meal will pose a risk to human health?

White phosphorous may be incorporated as reactive WP into muscle tissues of fish and birds and probably also grazing mammals, however, there is no data that verify this. Muscle from poisoned (dead) animals, birds and fish may contain high concentrations of WP. However, muscle tissue of animals that have recently ingested WP particles may contain WP for a short time period after intake of WP. Theoretical worst-case estimations of intake of fish or bird muscle from such food indicate a ratio between intake and acute toxic levels of 40 and 500, respectively. The probability of catching such a bird or fish is low, and cannot be estimated from the available information. When considering the safety margin between a potential worst-case intake and any effect level, one should bear in mind that the toxic effect levels are close to lethal doses.

Can white phosphorus be incorporated in plants, berries and mushrooms, and what risk will be associated with eating these plants/crops?

There are no analytical data available in the literature to answer this question. Due to the mechanisms of uptake of nutrients from soil and the oxidative metabolism within plants and mushrooms, VKM considers that it is unlikely that WP will be present in edible parts of plants and mushrooms. However, particles may adhere to the surface of plants, fungi, or berries.

DRINKING WATER

What levels of white phosphorus in drinking water can be considered as acceptable for human and animal health?

The oral reference dose (RfD or TDI) of WP is set to be 0.02 μg/kg bw/day. The RfD is derived from a reproductive study in rats using the obtained NOAEL of 0.015 mg/kg bw/day. Uncertainty factors included in the estimation are 10 for interspecies variation, 10 for intraspecies variation and 10 for incomplete reproductive/developmental data representing less than a lifetime study.

Humans may be exposed to white phosphorus by consumption of drinking water containing WP and/or ingestion of food items originating from the contaminated areas. In derivation of a lifetime health advisory (HA) for WP, US EPA has converted the RfD value to a concentration of WP in drinking water. In this process, a relative intake of WP of 20% from drinking water compared to intake from other sources and the consumption of 2 l/day by a 70-kg adult is assumed. The lifetime HA for WP in drinking water estimated by EPA is 0.1 μg/l.

VKM has screened more recent toxicological literature of WP, but has not identified any relevant new studies. Hence, VKM therefore finds that the estimated HA by the US EPA of 0.1 μg/l is acceptable as an upper limit for content of WP in drinking water for humans and animals.

ANIMAL HEALTH

If domestic and wild grazing animals eat white phosphorus, what consequences can be expected and what levels of white phosphorus can be considered as acceptable for animal health?

Ingestion of WP may produce acute and chronic effects in domestic and wild animals. In the Norwegian military training ranges relatively high concentrations of WP have been detected, and it is likely that animals on pasture in those areas will ingest doses that may cause both acute and chronic adverse effects affecting animal health and animal welfare. Ingestion of particles of unburnt WP may cause lethal effects. The previous reports about deaths of sheep (USA) and musk oxen (Norway) associated with ingestion of environmental WP support this conclusion.

No studies have been located regarding toxic effects of WP in domestic and grazing animals. As a consequence, VKM has assessed the risk based on values of toxic effects as reported in humans and other wild animal species (Table 1). Based on these data, toxic effects on domestic and grazing animals cannot be ruled out.

Should other consequences regarding animal health for fish and birds be taken into consideration?

In Alaska, high mortality rates among waterfowl have been associated with WP pollution in military training ranges. The fact that the levels of WP detected in Norwegian training ranges exceed the levels in Alaska implies that birds feeding in these areas may be at risk for WP intoxication.

The concentrations measured in some of the water samples from the training ranges in Troms were higher than the NOAEL derived for fish, thus representing a possible health risk for fish.

WP levels in water may increase during snow melting, flooding after heavy rainfalls and immediately after artillery training, thereby increasing the risk for adverse effects.

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REFERENCES

1. D. R. IDLER, COEXISTENCE OF A FISHERY AND A MAJOR INDUSTRY IN PLACENTIA BAY, *CHEMISTRY IN CANADA* PP. 16-21 (1969).

2. P. M. JANGAARD, EFFECTS OF ELEMENTAL PHOSPHORUS ON MARINE LIFE, *CIRCULAR NO. 2, L-6.* (1972).

3. J. W. SIMMERS, R. A. PRICE, K. F. MEYERS, R. A. KARN, R. D. KRESS, H. E. TATEM, K. C. JENSEN, G. S. WILHELM AND D. S. CHERRY, IMPACT AREA CONTAMINANT INVENTORY: FORT MCCOY, *US ARMY ENGINEER WATERWAYS EXPERIMENTAL STATION REPORT EL-97-4* P. 100 PP (1995).

4. M. E. WALSH, C. M. COLLINS AND C. H. RACINE, PERSISTENCE OF WHITE PHOSPHORUS (P-4) PARTICLES IN SALT MARSH SEDIMENTS, *ENVIRONMENTAL TOXICOLOGY AND CHEMISTRY* **15**, PP. 846-855 (1996).

5. M. R. WALSH, M. E. WALSH AND C. M. COLLINS, REMEDIATION METHODS FOR WHITE PHOSPHORUS CONTAMINATION IN A COASTAL SALT MARSH, *ENVIRONMENTAL CONSERVATION* **26**, PP. 112-124 (1999).

6. R. J. SPANGGORD, R. REWICK, T. W. CHOU, R. WILSON, R. T. PODOLL, T. MILL, R. PARNAS, R. PLATZ AND D. ROBERTS, "ENVIRONMENTAL FATE OF WHITE PHOSPHORUS/FEH AND RED PHOSPHONDBUTYL RUBBER MILITARY SCREENING SMOKES, ", *FINAL REPORT, CONTRACT NO. DAMD 17-82-C-2320, AD-A176 922, SRI INTERNATIONAL, MENLO PARK, CA* (1985).

7. S. T. BIRD, C. H. RACINE, M. E. WALSH, C. M. COLLINS, D. CALKINS, B. D. ROEBUCK, L. W. METKER AND R. NEWSOME, WATERFOWL MORTALITY IN EAGLE RIVER FLATS, ALASKA: THE ROLE OF MUNITIONS COMPOUNDS AND HUMAN HEALTH RISK ASSESSMENT, *PROCEEDINGS, CARRIBEAN HAZTECH INTERNATIONAL CONFERENCE AND EXHIBITION,* NOVEMBER 13-15, (1991).

8. ATSDR, TOXICOLOGICAL PROFILE FOR WHITE PHOSPHORUS, *NO. 205-93-0606 U. S. DEPARTMENT OF HEALTH AND HUMAN SERVICES PUBLIC HEALTH SERVICE AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY SEPTEMBER 1997* (1997).

9. A. JOHNSEN, K. S. LONGVA, H. RINGNES AND A. E. STRØMSENG, HELSE- OG MILJØSMESSIGE KONSEKVENSER VED FORSVARETS BRUK AV RØYKAMMUNISJON MED HVITT FOSFOR, *FFI/RAPPORT-2002/04042* (2002).

10. E. SØBYE, A. JOHNSEN AND A. E. STRØMSENG, KARTLEGGING AV HVITT FOSFOR FORURENSING I HJERKINN SKYTEFELT, *FFI-RAPPORT-2003/01224. 42S* (2003).

11. G. L. FLETCHER, THE ACUTE TOXICITY OF A YELLOW PHOSPHORUS CONTAMINATED DIET TO BROOK TROUT (SALVELINUS FONTINALIS), *BULL. ENVIRON. CONTAM TOXICOL.* **10**, PP. 123-128 (1973).

12. G. L. FLETCHER, THE DYNAMICS OF YELLOW PHOSPHORUS IN ATLANTIC COD AND ATLANTIC SALMON: BIOLOGICAL HALF-TIMES, UPTAKE RATES AND DISTRIBUTION IN TISSUES; *ENVIRON. PHYSIOL BIOCHEM.* **4**, PP. 121-138 (1974).

13. S. I. NAM, B. D. ROEBUCK AND M. E. WALSH, UPTAKE AND LOSS OF WHITE PHOSPHORUS IN AMERICAN KESTRELS, *ENVIRONMENTAL TOXICOLOGY AND CHEMISTRY* **13**, PP. 637-641 (1994).

14. D. W. SPARLING, M. GUSTAFSON, P. KLEIN AND N. KAROUNARENIER, TOXICITY OF WHITE PHOSPHORUS TO WATERFOWL: ACUTE EXPOSURE IN MALLARDS, *JOURNAL OF WILDLIFE DISEASES* **33**, PP. 187-197 (1997).

15. N. BEN HUR, A. GILADI, Z. NEUMAN, B. SHUGERMAN AND J. APPLEBAUM, PHOSPHORUS BURNS--A PATHOPHYSIOLOGICAL STUDY, *BR. J. PLAST. SURG.* **25**, PP. 238-244 (1972).

16. N. BEN HUR AND J. APPELBAUM, BIOCHEMISTRY, HISTOPATHOLOGY AND TREATMENT OF PHOSPHORUS BURNS. AN EXPERIMENTAL STUDY, *ISR. J. MED. SCI.* **9**, PP. 40-48 (1973).

17. C. H. RACINE, M. E. WALSH, B. D. ROEBUCK, C. M. COLLINS, D. CALKINS, L. REITSMA, P. BUCHLI AND G. GOLDFARB, WHITE PHOSPHORUS POISONING OF WATERFOWL IN AN ALASKAN SALT MARSH, *J. WILDL. DIS.* **28**, PP. 669-673 (1992).

18. B. D. ROEBUCK, M. E. WALSH, C. H. RACINE, L. REITSMA, B. STEELE AND S. I. NAM, PREDATION OF DUCKS POISONED BY WHITE PHOSPHORUS - EXPOSURE AND RISK TO PREDATORS, *ENVIRONMENTAL TOXICOLOGY AND CHEMISTRY* **13**, PP. 1613-1618 (1994).

19. B. D. ROEBUCK, S. I. NAM, D. L. MACMILLAN, K. J. BAUMGARTNER AND M. E. WALSH, TOXICOLOGY OF WHITE PHOSPHORUS (P-4) TO DUCKS AND RISK FOR THEIR PREDATORS: EFFECTS OF PARTICLE SIZE, *ENVIRONMENTAL TOXICOLOGY AND CHEMISTRY* **17**, PP. 511-518 (1998).

20. W. J. DYER, D. F. HILTZ AND R. G. E. AL. AGMAN, IN VIVO ASSIMILATION BY COD MUSCLE AND LIVER TISSUE OF ELEMENTAL PHOSPHORUS FROM POLLUTED SEA WATER. *JOURNAL OF THE FISHERIES RESEARCH BOARD OF CANADA* PP. 1131-1139 (1970).

21. A. ELDAD AND G. A. SIMON, THE PHOSPHORUS BURN - A PRELIMINARY COMPARATIVE EXPERIMENTAL-STUDY OF VARIOUS FORMS OF TREATMENT, *BURNS* **17**, PP. 198-200 (1991).

22. NATIONAL RESEARCH COUNCIL, *TOXICITY OF MILITARY SMOKE AND OBSCURANTS*, PP. 18-44. THE NATIONAL ACADEMY PRESS, WASHINGTON DC (2000).

23. D. R. COBURN, J. D. DEWITT, J. V. DERBY AND E. EDIGER, PHOSPHORUS POISONING IN WATERFOWL, *J. AM. PHARM. ASSOC. AM. PHARM. ASSOC.* **39**, PP. 151-158 (1950).

24. A. HURWITZ, EFFECTS OF MICROSOMAL ENZYME INDUCERS ON ANIMALS POISONED WITH HEPATOTOXINS, *TOXICOL. APPL. PHARMACOL.* **22**, PP. 339-346 (1972).

25. M. V. TORRIELLI, P. PANI AND E. AL. GABRIEL L, THE PATHOPHYSIOLOGICAL SIGNIFICANCE OF THE HEPATIC DRUGMETABOLIZING ENZYME SYSTEM IN THE LIVER

DAMAGE INDUCED BY CCL, AND WHITE PHOSPHORUS. *DUNCAN WAM, ED. EXPERIMENTAL MODEL SYSTEMS IN TOXICOLOGY AND SIGNIFICANCE IN MAN, 15, AMSTERDAM, EXCERPTA MEDICA INTERNATIONAL CONGRESS SERIES NO. 3 11* (1974).

26. D. W. SPARLING, D. DAY AND P. KLEIN, ACUTE TOXICITY AND SUBLETHAL EFFECTS OF WHITE PHOSPHORUS IN MUTE SWANS, CYGNUS OLOR, *ARCHIVES OF ENVIRONMENTAL CONTAMINATION AND TOXICOLOGY* **36**, PP. 316-322 (1999).

27. S. ROGNERUD AND J. E. LØVIK, VURDERING AV MILJØRISIKO VED FORSVARETS BRUK AV HVITT FOSFOR I SKYTEFELT I TROMS, *NIVA RAPPORT LNR 5085-2005* (2005).

28. J. P. WHALEN, N. O'DONOHUE, L. KROOK AND E. A. NUNEZ, PATHOGENESIS OF ABNORMAL REMODELING OF BONES: EFFECTS OF YELLOW PHOSPHORUS IN THE GROWING RAT, *ANAT. REC.* **177**, PP. 15-22 (1973).

29. J. R. CONDRAY, ELEMENTAL YELLOW PHOSPHORUS ONE-GENERATION REPRODUCTION STUDY IN RATS, *IR-82-215; IRD NO. 401-189.* (1985).

30. D. W. SPARLING, S. VANN AND R. A. GROVE, BLOOD CHANGES IN MALLARDS EXPOSED TO WHITE PHOSPHORUS, *ENVIRONMENTAL TOXICOLOGY AND CHEMISTRY* **17**, PP. 2521-2529 (1998).

31. D. W. SPARLING AND N. E. FEDEROFF, SECONDARY POISONING OF KESTRELS BY WHITE PHOSPHORUS, *ECOTOXICOLOGY* **6**, PP. 239-247 (1997).

32. S. I. NAM, D. L. MACMILLAN AND B. D. ROEBUCK, THE TRANSLOCATION OF WHITE PHOSPHORUS FROM HEN (GALLUS DOMESTICUS) TO EGG, *ENVIRONMENTAL TOXICOLOGY AND CHEMISTRY* **15**, PP. 1564-1569 (1996).

33. S. L. VANN, D. W. SPARLING AND M. A. OTTINGER, EFFECTS OF WHITE PHOSPHORUS ON MALLARD REPRODUCTION, *ENVIRONMENTAL TOXICOLOGY AND CHEMISTRY* **19**, PP. 2525-2531 (2000).

34. EAGLE RIVER FLATS: BIBLIOGRAPHY, *HTTP://WWW. CRREL. USACE. ARMY. MIL/ERF/BIBLIOGRAPHY/JOURNALS/).* (2006).

35. S. M. RICHARDSON AND WESTONR.F., WHITE PHOSPHORUS CONTAMINATION OF WETLANDS: EFFECTS AND OPTIONS FOR RESTORATION. *PROCEEDINGS OF THE FEDERAL ENVIRONMENTAL RESTORATION '92, VIENNA, VA* PP. 186-189 (1992).

36. D. E. AIKEN AND E. H. BYARD, HISTOLOGICAL CHANGES IN LOBSTERS (HOMARUS AMERICANUS) EXPOSED TO YELLOW PHOSPHORUS, *SCIENCE* PP. 1434-1435 (1972).

37. W. L. STEWART, A CURIOUS CASE OF PHOSPHORUS POISONING IN SHEEP, *AGRIC JRL MIN AGRIC (GREAT BRITAIN)* PP. 56-59 (1030).

38. J. A. TØRNES, BESTEMMELSE AV HVITT FOSFOR I PRØVER FRA FORSVARETS SKYTEFELT PÅ DOVRE, *FFI/RAPPORT-6009* (1998).

39. C. BERGSTEN, THE CONSUMPTION OF FOODS THAT MAY BE IMPORTANT WHEN ASSESSING THE DIETARY INTAKE OF MERCURY, CADMIUM AND PCB/DIOXINS, WITH A FOCUS ON

POPULATION GROUPS LIVING ON THE COAST AND IN THE INLAND OF NORWAY., *FISH- AND GAME STUDY, PART B, NORWEGIAN FOOD SAFETY AUTHORITY* (2005).

40. H. M. MELTZER, C. BERGSTEN AND H. STIGUM, FISK- OG VILTUNDERSØKELSEN: KONSUM AV MATVARER SOM KAN HA BETYDNING FOR INNTAKET AV KVIKKSØLV, KADMIUM OG PCB/DIOKSIN I NORSK KOSTHOLD, *SNT-RAPPORT 6 • 2002* (2002).

41. A. E. STRØMSENG, A. JOHNSEN, LONGVA K.S. AND VOIE Ø.A., ANALYSE AV HVITT FOSFOR I DRIKKEVANN I TROMS, *FFI/NOTAT-2006/00412* (2006).

42. A. E. STRØMSENG, Ø. A. VOIE, A. JOHNSEN AND K. S. LONGVA, RISIKOVURDERING AV FORSVARETS BRUK AV HVITT FOSFOR I TROMS, *FFI-RAPPORT-2005. 73S* (2005).

43. A. JOHNSEN, Ø. A. VOIE, K. S. LONGVA AND A. E. STRØMSENG, ANALYSETEKNISKE PROBLEMER VED BESTEMMELSE AV KONSENTRASJONEN TIL HVITT FOSFOR I VANN. TILLEGGSNOTAT TIL FFI RAPPORT – 2005/03531, RISIKOVURDERING AV FORSVARETS BRUK AV HVITT FOSFOR I TROMS., *FFI-NOTAT - 2006/00512* (2006).

44. C. O. ADAMS AND B. G. SAMAT, ADAMS CO, SAMAT BG. 1940. EFFECTS OF YELLOW PHOSPHORUS AND ARSENIC TRIOXIDE ON GROWING BONES AND GROWING TEETH. ARCH PATHOL 30:1192-1202., *ARCH PATHOL* PP. 1192-1202 (1940).
45. R. B. L. FLEMING, J. W. MILLER AND V. R. JR. SWAYNE, SOME RECENT OBSERVATIONS ON PHOSPHORUS TOXICOLOGY, *J IND. HYG. TOX.* PP. 154-158 (1942).

46. T. SOLLMAN, STUDIES OF CHRONIC INTOXICATIONS ON ALBINO RATS. VIII, *J PHARMACOLEXP THER* PP. 119-122 (1925).

47. SFT, EKSEMPELSAMLING FOR VEILEDNING OM RISIKOVURDERING AV FORURENSET GRUNN, *VEILEDNING 99:01B HTTP://WWW. SFT. NO/PUBLIKASJONER/KJEMIKALIER/1648/TA1648. PDF* (1999).

48. U.S.ENVIRONMENTAL PROTECTION AGENCY (EPA), HEALTH ADVISORIES FOR DRINKING WATER CONTAMINANTS, *OFFICE OF WATER HEALTH ADVISORIES. LEWIS PUBLISHERS, ANN ARBOR.* (1993).