Chemical residues in poultry and eggs produced in free-range or organic systems

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Summary

Residues in poultry arise from accidental or intended exposure to chemicals. The exposure routes are by oral ingestion (feed, water, soil and preening), by inhalation, by dermal absorption or by injection. Chemical contaminants are not a major issue in the principles of organic or free-range systems. Risk management of chemicals has not been addressed widely and if so the attention was focused on intentionally used chemicals. This paper aimed to review and identify possible contamination sources in organic or free-range production and substantiate some possibilities with available data.

High levels of dioxins in eggs from organic systems have been reported from several European countries. Eggs from other outdoor systems seemed to contain lower levels. No explanation for this difference is at hand. The higher contamination in organic systems seems to be related to soil contamination and soil ingestion. Other materials like Pentachlorophenol (PCP) treated wood also constitute a risk for dioxin contamination of farm animals.

Drugs residues are generally not higher in systems using litter, but due to recirculation via the litter, persistence of residues is often considerably longer.

Introduction

Consumers concern about the environment and animal welfare transformed intensive animal production facilities into more sustainable agriculture. This transformation, that started about two decades ago, has had its impact on both quality and safety of the agricultural products. Nardone and Valfré (1999) reviewed the effects of changing of production methods on quality of products. They identified nutrition, management and animal health as important factors in relation to residues in poultry meat and eggs. However, they do not support this statement with literature data. They only make a claim about the concern around ochratoxin residues in eggs, not supported by experimental evidence. Woese *et al.* (1997) compared literature data on composition of organic and conventional foods. Data on meat and eggs proved to be scarce at that time. The reviews on organic livestock production by Berg (2001) and Sundrum (2001) also pay very little attention to product characteristics. Hermansen *et al.* (2004) discuss at length the integration of organic poultry production in that of plants and their possible role in reducing pests. Residues in poultry or eggs are not mentioned in this paper. Miao *et al.* (2005) reviewed very recently free-range poultry production (especially in Australia). They only cite a quite old paper (Holmes *et al.*, 1969) on observed differences in contents of organochlorine pesticides in eggs from different production system. This study concerned DDT and lindane residues in eggs, due to the use of thermal vaporizers, which bears no relevance to the situation of 2005.

This paper aims to review the possibilities for chemical contamination in organic or free-range poultry production. It intends further to substantiate some possibilities with available data.

Chemical contaminants in food animals products have been reviewed e.g. by Biehl & Buck (1987) with special emphasis to the US situation. Kan (2004) reviewed recently the information available on chemical residues in broilers with emphasis on the European situation.

No specific or detailed overviews on chemical residues in meat or eggs from free-range or organic production are presently available. A general assumption is that residues might me lower in more extensive production system, as less or no protective or curative substances are used. However more contact with materials from the environment and more fungi producing toxins, may also lead to more residues from these sources in products from extensive animal farming systems.

Risk assessment and communication

The general assumption - for non-carcinogenic substances – is, that a threshold limit exists below which exposure of humans to chemical residues does not pose a threat to human health. This limit expressed as an acceptable or daily intake (ADI), however, is set often for individual compounds and does not take into account possible interactions between different chemicals neither aggravating nor alleviating. The concept however does still allow certain conclusions on the risks of human exposure to chemicals via the food. Nasreddine & Parrent-Massin (2002) e.g. reviewed the risks of intake of certain pesticides via plants and heavy metals via food in general. Pesticide intake was quite low compared to the tolerances but the intake of certain consumer groups in Europe of heavy metals proved to be near or above the assumed safe levels. Thus more attention should be paid to high risk food items and to a more solid support of the tolerances, as a number of assumptions are still part of the calculations. Recently Finamore *et al.* (2004) have used a new approach to substantiate the supposition that organic food might be safer than conventionally produced one. They fed wheat grown under organic and conventional conditions to rats and measured the immuno-competence of the rats after being on those diets for 30 days. No major differences were observed between organic and conventional wheat. Unfortunately the authors only determined the levels of deoxynivalenol (a mycotoxin) in the wheat samples and those were higher in the organic wheat. Other potentially immuno-toxic compounds might have been present in either organic or conventional wheat and a wider screening for these compounds might have revealed a clearer answer.

Next to risk evaluation is risk communication leading to perception by both authorities and the general public. Trautman (2001) gives a nice overview on the importance of risk communication in the perspective of risk analysis as a whole. Risk communication has proven to be a key element in public perception of food safety in recent years and deserves continuous attention both from authorities and from scientists.

Control of contamination

Kan (2002) stated in his review that prevention and control of contaminants and pesticides in the poultry production chain requires:

1. Knowledge of the sources of contamination and their consequences
2. Knowledge of the possibilities to exclude these sources from the system or to reduce their effect.

Preventive measures are preferred over "end-of-pipe" approaches due to (cost) effectiveness (Kan, 1994).

Hazard Analysis by Critical Control Points (HACCP) is a systematic approach to the identification, assessment and control of hazards in the food chain. Ropkins *et al.* (2003) were one of the first to extensively describe the templates to be used for the control of organic chemical hazards in food production. They discriminate between applied chemicals, accidental chemicals and background chemicals and this distinction will be followed in this paper.

Applied chemicals are generally absent for the organic poultry but feed additives and veterinary drugs are used in free-range animals. Accidental chemicals e.g. cleaning materials, disinfectants and machine lubricants can be used in both organic and free-range production and background chemicals are also a threat to all production systems. Ropkins *et al.* (2003) subdivide the background chemicals for beef production into airborne, waterborne, vegetation and soil contaminants. For poultry, housing related contaminants seem to be also of importance and vegetation should be expanded to include feed. Both Ropkins *et al.* (2003) and Kan (2002) point out that applied chemicals are the ones likely to be controlled by a HACCP approach. Accidental and background chemicals are rarely considered by the system and hard to be controlled by HACCP.

Dioxin residues in eggs

Data from several countries are now available that indicate differences in levels of dioxins and dibenzofurans in eggs produced by hens from different management systems. The number of samples is often restricted, but a certain overall trend does seem to exist.

Belgium: Pussemier *et al.* (2004) reported similar dioxin levels in egg from conventional and organic farms but a much increased dioxin levels in eggs from some private owners. Also in 2004 free-range eggs with a much too high PCB level were recalled from a supermarket chain (Anonymous, 2004c). Contaminated soil was indicated as the possible source of the pollution.

Germany: Fürst *et al.* (1993;1998) reported that eggs sampled around 1992 and 1996 from free-range hens did contain the highest proportion of samples with increased dioxin levels as compared to hens kept on deep-litter or on batteries. Also the average value in the free-range eggs was the highest. Data from 1999-2003 showed a similar trend (Bioland, 2005) for dioxin levels in eggs, with a certain percentage exceeding the value of 3 pg/g TEQ. Organic eggs did not contain amounts over this value. These results aroused considerable media interest in Germany and political statements. There have been (newspaper) reports (Anonymous, 2005) that samples taken in early 2005 showed on average lower dioxin levels.

France: Eggs and hens were found contaminated in the North of France in 2003 (Anonymous, 2004a). Soil pollution was also assumed to be the origin of the problem.

Ireland: The Food Safety Authority of Ireland analyzed a number of battery, barn, free-range and organic eggs for dioxins (Ireland, 2004). Organic eggs (although only four samples) showed a much higher average and especially maximum level than the other three types. The mean value approached the critical 3 pg/g level.

Sweden: Eggs were sampled in 2003: some organic eggs had a level around 3 pg/g (Anonymous, 2004b)

Switzerland: Schmid *et al.* (2002) investigated a number of food items and found levels up to 21 pg/g in eggs. In some instances the feed could be identified as the source of contamination.

The Netherlands: The National Plan for controlling products of animal origin for residues (an obligation brought about by EU legislation) revealed in 2001 one that one egg sample exceeded the 5 pg/g level. Tracking and tracing revealed that the sample originated from an organic farm. This prompted a survey in eggs by the Dutch Food Inspection Service in the fall of 2001. Out of 68 samples 6 exceeded the 3 pg/g and 3 samples exceeded the 5 pg/g level (de Vries, 2002). Follow up studies suggested strongly that the origin of the contamination was not the feed but the environment. During 2003 a large inventory was made amongst Dutch organic egg producers to try to relate external factors to the dioxin levels in the eggs. Not only samples from soil, worms etc were taken and analyzed but also possibly relevant management factors were recorded. Also in this survey a certain percentage of eggs displayed dioxin levels above 3 pg/g (Brandsma *et al.* , 2004).

In different countries thus, the organic production system seems to add an extra risk for dioxin contamination of eggs. A clear explanation for this phenomenon is not yet available.

Possible sources for dioxins

Three major sources causing dioxin residues in poultry have been described so far:

1. Pentachlorophenol (PCP) related dioxin residues in wood and derived products.
2. Residues in soil mainly caused by deposition of airborne residues caused mainly by (incomplete) industrial combustion processes. However man-made fires are a possible source too.
3. Certain clay minerals e.g. kaolin or ball clay

Landfills were PolyChlorinated Biphenyl (PCB) containing materials have been dumped, are however also recognized as a major source (Hansen and O'Keefe, 1996) Till now they have not been connected to residue problems in poultry. River sediments have also been found to be a potential dioxin source in both the US (Vorhees *et al.* , 2003) and Europe (Schulz *et al.* , 2003). Also for this source a connection with residue problems in poultry has not yet been reported. “Kieselrot” (red slag); a residue from a copper production process was found to contain considerable amounts of PCDD/F. The bioavailability of PCDD/F *in vitro* in this material, which is generally used as coverage for pavements and playground was found to amount to 60 % (Wittsiepe *et al.* , 2001). Poultry being in contact with this material, could thus well display higher dioxin levels. Attention is generally focused to oral absorption but absorption of lipophilic compounds through the skin or the airways should not be excluded (Holmes *et al.*, 1969; Fishwick *et al.*, 1980; Qiao *et al.*, 1997; Rohrer *et al.*, 2003; Imsilp and Hansen, 2005).

Ad 1: Ryan *et al.* (1985) analyzed some 26 chicken samples from all over Canada with the methodology available in the early 1980’s. They already found a remarkable similarity in the dioxin and dibenzofurans pattern between chicken samples and PCP treated wood indicating that PCP was the source of contamination. Quite recently Huwe and coworkers (2004) analyzed over 150 cattle samples originating from research facilities all over the USA. The PCDD/F pattern in the cattle samples was also quite similar to that of PCP treated wood. At three facilities with high levels, they could find PCP treated wood being used and accessible to the animals. Butler and Frank (1991) and Fries *et al.*

(1999) showed similar transfer of PCP and PCDD/F from treated wood to pork and milk respectively. The relationship in pattern between source and animal material has been also nicely illustrated by Huwe (2002).

There is thus no reason to believe that things would not be similar in some poultry facilities. Harnly *et al.* (2000) analyzing soil and home-produced chicken eggs near a former (burnt) facility using PCP, conclude, that the pattern of PCDD/F found in the samples, clearly points to PCP as the origin of the contamination.

Ad 2: The fire in a PCP applying facility in California USA caused a local contamination with a specific PCDD/F pattern in the soil. In a series of studies, carry-over of the contaminants from soil to poultry and eggs was demonstrated (Chang *et al.*, 1989; Stephens *et al.*, 1990; Petreas *et al.*, 1991; Stephens *et al.*, 1995). Similarly Lovett *et al.* (1998a;1998b) showed that the dioxin pattern close to a waste incinerator in Wales UK, was similar to that found in poultry and eggs of animals living in the close neighbourhood. Intake of contaminated soil was thought to be the major cause of the problem. Schuler *et al.* (1997) investigated five small free-range laying hens farms in Switzerland. Two of them were quite close to known sources and the other three in rural areas. PCDD/F concentrations were highest in the soil samples of the two located near the point sources. Dioxin contents were quite high in the egg samples of one of those, but quite similar for all other four, although higher than reported for battery eggs. Thus soil contamination certainly plays a role but not always a decisive one. Pussemier *et al.* (2004) report a good similarity in PCDD/F pattern between soil and eggs, which suggests soil to be a major source of dioxin contamination for laying hens

Ad 3: Certain clay minerals e.g. ball clay or kaolin have been proven to be a dioxin source. The US authorities issued a statement on it in 2001 after investigation which already started in 1997 (Anonymous, 2001). Schmid *et al.* (2002) reported further that some of the contaminated Swiss poultry and eggs samples displayed the typical kaolin pattern of PCDD/F congeners. The recent Dutch problems with dioxins in milk, also originated from the use of kaolin which ended up in the diet of dairy cattle.

Likely causes for dioxin contamination (of eggs)

Feed, soil, wood treated PCP etc are possible causes for dioxin contamination in eggs from free-range or organic hens, but are they also the likely causes? The second question then is; if you remove those sources from the system, do the dioxin levels then drop?

Air *et al.* (2003) state there are many questions and only some answers. They have found that some poultry holders show consistently high or moderate levels of contamination, while others show great variation in their levels. They find soil contamination to be a relevant contributor to PCDD/F levels in eggs, but also reported similar dioxin levels in eggs from two pens with a considerable difference in soil contamination (40 vs. 148 pg/g TEQ). Similarly the large Dutch inventory (Brandsma *et al.*, 2004) could unfortunately give no clear clue on the cause of the problem. Some statistical relationships between the many variables measured were found; e.g. small farms had a higher chance than large farms to produce eggs with high contents. Also flocks receiving vitamin treatments showed lower dioxin levels. Flocks with high dioxin levels also had higher ash content in the manure suggesting increased soil intake. A clear causal relationship did not emerge form the study. Some of the possible solutions like (reduction of) soil intake and reduction of time outdoors, were tested in an intervention study in late 2004, but no clear reduction in dioxin levels in eggs was obtained (A. Kijlstra, personal communication). Air *et al.* (2003) state that time spent outdoor and picking might be an important item in determining dioxin contamination. The amount soil or sand taken up by hens was still unknown. For that reason, we included up to 30 % sand in a layers ration (in pellet form) and fed it to young layers for 5 weeks. The hens were, much to our surprise, able to increase their feed intake to (almost) fully compensate the dilution brought about by the sand inclusion. Hens consumed up to 45 grams of sand per day (van der Meulen, Kwakernaak, Kan, 2005, unpublished observations). Thus uptake of considerable amounts of soil while maintaining good productivity seems possible.

Experimental studies on carry-over of dioxins

Older studies have been reviewed previously (Kan, 1994). Iben *et al.* (2003) fed broilers rations with different concentrations of dioxins for 2, 4 or 6 weeks and analyzed the fat after slaughter at 6 weeks. The residues in fat were dose related and the absolute amount in the broiler did not decrease during the period on uncontaminated feed. Similarly Hoogenboom *et al.* (Hoogenboom *et al.*, 2004) found only dilution of residues in broilers, during a three week withdrawal period after one week on a dioxin contaminated diet. Ueberschär and Matthes (2004) fed chlorinated paraffin's to broilers at different levels in the diet for 31 days and established dose-response related residues in abdominal fat, leg muscle and some other tissues. However about 95 % of the ingested dose was not recovered from the carcass and apparently metabolized and excreted by the animals. These substances are thus much less resistant towards metabolism.

Traag *et al.* (2004) carried out three out three carry-over studies with dioxins in laying hens. The first study with five different dioxin levels in the feed, demonstrated that a level of 0. 750 ng TEQ/kg in feed (the current tolerance) led within two weeks to levels in egg fat above 3 pg/g (the current tolerance). This study consisted of 56 days exposure to contaminated feed and 56 days withdrawal on uncontaminated feed. The dioxin residues in egg and body fat showed a dose response relationship to those in feed. The data were used to construct a PBPK model describing the pharmacokinetics of dioxin residues in laying hens. Steady state in egg residues was not yet obtained after 56 days. The second study tested carry-over of dioxins from two contaminated soils included at 10 % in the feed, to eggs. Residues increased much faster in this trial and the dioxins were almost fully bio-available. Three different binders added to the feed were tested for their effect on dioxin residues in eggs. Unfortunately but not unexpectedly (Kan, 1994) no effect of the binders on dioxin levels in eggs was observed.

Other contamination sources for (lipophilic) compounds

Coating material of (concrete) farm silos has shown to be a possible source of PCB's (Willett *et al.*, 1985). Leaching from the coating to the material contained in the silo especially during silage formation was shown to occur. Another unexpected PCB source was a vapor seal being part of the insulation material on the ceiling of a turkey house, which decomposed over time, fell down and was taken up by the animals (Hansen *et al.*, 1989). The spraying of propoxur containing formula in poultry houses (not on animals) to combat the poultry red mite (*Dermanyssus gallinae)* did result in violative residues in eggs (> 50 µg/kg) for some days after administration especially in battery housing (Hamscher *et al.*, 2003).

Drugs and coccidiostat residues

The incidence of gastrointestinal helminths in free-range and backyard poultry was higher than in deep litter systems (Permin *et al.*, 1999), but increased use of anti-parasitics or higher incidence of residues has not been reported.

The persistence of nicarbazin in both broilers and layers, in eggs and manure depending also on housing conditions has been studies by quite a number of scientists. Friedrich *et al.* (1984) fed laying hens during 7 days feed with 129 mg/kg nicarbazin being the normal dose to give to young birds to prevent coccidiosis. The hens in cages excreted the nicarbazin via both eggs and faeces during and rather short after exposure. Very small amounts remained detectable for quite some time in the yolks. The hens on deep litter were divided at five days after cessation of the administration in two groups; one group remaining on the old litter and one being transferred to clean unshed litter. The excretion in faeces and eggs on the clean litter showed a decline similar to the eggs from the hens in cages. Residues in faeces and eggs from hens on the old litter remained high for weeks. These results clearly prove the stability of nicarbazin (or one active component DNC) in litter and the recirculation of DNC from litter- probably through oral ingestion of litter – to the hens. Administration of 2 mg/kg nicarbazin in feed during 29 days gave similar results (Friedrich *et al.*, 1985). Macri *et al.* (1990) gave nicarbazin to broilers housed both on litter and in cages. The broilers in cages had no detectable nicarbazin levels in liver after 9 days of withdrawal but on litter the amount was unchanged. They ascribe these results to uptake of litter with nicarbazin by the broilers; however the residues should have gone down due to rapid growth of the broilers and the dilution resulting from that growth. Kan *et al.* (1995) found indeed that recirculation of nicarbazin in broilers kept on litter occurred, but that residues went down over time. Penz *et al.* (1999) used nicarbazin in a three consecutive broiler trials using the build-up litter system with no new addition of litter between rounds. They found DNC residues in muscle, liver and litter samples. The average nicarbazin (DNC) values found in litter at the end of each round reflected the concentrations used in the feed. Residues of DNC were still found in the litter after the third round, although nicarbazin had only been used during the first one. This proves the stability of the compound in litter also reported in a laboratory study (van Dijk and Keukens, 2000). Penz *et al.* (1999) also found DNC residues in liver samples of the two consecutive flocks not receiving nicarbazin in the feed but grown on the litter of the first flock receiving nicarbazin in the starter diet. This proves once again the presence of oral uptake of litter as a possible contamination source. The longer persistence of nicarbazin residues in broilers housed on litter than in cages has also been demonstrated by Cannavan and Kennedy (2000).

The importance of litter as a (re)contamination source has also been demonstrated for meticlorpindol (another coccidiostat) in laying hens trials (Hafez *et al.*, 1988). They demonstrated like Schwarzer and Dorn (1987) did for chloramphenicol, that administration of drugs to replacement hens till about two weeks before the start of lay can lead to detectable residues in the first eggs. Thus, caution is required in exposing replacement hens intentionally or unintentionally to any unwanted substances in the period shortly before the start o egg laying. The explanation is most likely, that yolks are already developing at that time point and the chemicals can be deposited and trapped in those developing yolks. This leads afterwards to detectable residues.

Cross-contamination in a slaughterhouse was found the cause of chloramphenicol residues in broiler meat by Dorn *et al.* (1986). This phenomenon has not been reported for other pharmaca, but its presence can not be ruled out. In cattle it has been proven that licking rather than dermal absorption was the cause of systemic ivermectin residues after use of a pour-on formulation (Laffont *et al.*, 2001). Pour-on formulations are seldom used in poultry but dusting with certain anti-parasitica might happen. The preening behaviour of birds might then cause a similar phenomenon to happen.

Other chemicals

Heavy metals like cadmium and lead, mycotoxins and polycyclic aromatic hydrocarbons (PAH) do occur in animal and the animal surroundings. Specific risks of residues in poultry hip in free-range or outdoors or on the contrary lower levels than in intensive barn housed poultry have not been reported in the literature.

Conclusions

Poultry in outdoor farming systems is more exposed to infectious agents and chemical contaminants than those in barn housed.

Higher dioxins levels in eggs from some organic systems than from conventional ones substantiate this. Both general and local environmental pollution with dioxins are probably responsible for it.

Increased incidence of diseases due to more exposure to infectious agents from the environment has been predicted and sometimes found for outdoor systems. An increased disease incidence could lead to more use of veterinary drugs and more or higher drug residues in products of animal origin. Reports substantiating this assumption have not (yet) been published.

Increased attention for risk management in the design and operation of outdoor systems seems appropriate.

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