Birds and pesticides: is the threat of a silent spring really behind us?

What would Rachel Carson say if she came back today? Would she still be sounding the alarm about a world without the sound of birds in our fields and forests? In this year's ***Rachel Carson Memorial Lecture****, Senior Research Scientist for Environment Canada* ***Pierre Mineau*** *describes why Rachel Carson warned of a 'silent spring.' Dr. Mineau discusses what happened when we switched from organochlorines like DDT to newer pesticides that were less persistent but dangerous to birds in different ways. Forty-seven years after the publication of Carson's ground-breaking book, uncounted millions of birds around the world continue to die from pesticides. The industry still resists regulation and governments are slow to deal with the problem. Nevertheless, Dr. Mineau sees some reasons for hope.*

When Rachel Carson wrote Silent Spring in 1962, the pesticides being used in massive quantities on farms and forests were organochlorine insecticides, one of the earliest generations of synthetic pesticides.

Application rates were shockingly high by today's standards: in those innocent days, it was mistakenly believed that if a little pesticide was good, more was better. Few people before Rachel Carson understood or even speculated about the possible impact of such profligate use[1].

This early group of compounds had characteristics that made them particularly damaging to the environment and to birds. They were persistent (remained unchanged for a long time in the environment), they were soluble in lipids (fats), and they were not easily eliminated (metabolised and excreted) by the organisms that ingested them. Because of these characteristics, these compounds accumulated in fatty tissue and their concentration increased with each step up the food chain.

The long environmental persistence of organochlorines was initially considered a benefit, as claimed by Paul Müller, the father of DDT. In his Nobel Prize acceptance speech in 1948, Müller had declared that stability and environmental persistence were ideal attributes for a pesticide. According to him, it did not matter if DDT took a little longer to kill insects; its environmental persistence would make it prevail over competing products.

Of course, it is precisely because of their lengthy persistence in living organisms and the environment that these early organochlorine insecticides came to be restricted and sometimes banned in the decades after *Silent Spring* was published. Because of their potential to achieve ever-increasing concentrations with each step up the food chain, the deleterious effects of these persistent organochlorine pesticides were most clearly seen in top carnivores – birds of prey or fish-eating birds.

DDT became the most infamous of the organochlorines because it caused the thinning of birds' eggshells, although this specific effect was not known when *Silent Spring* was published[2]. Not all birds are affected equally; chickens and other species of galliformes, like quail and pheasants, for example, are among the least affected. This fact is important because galliforme species have been traditionally used to test for pesticide toxicity.

The most severe effects from eggshell thinning were found in the peregrine falcon (*Falco peregrinus*), the brown pelican (*Pelecanus occidentalis*), the double-crested cormorant (*Phalacrocorax auritus*), the osprey (*Pandion haliaetus*) and the bald eagle (*Haliaetus leucocephalus*).

Also very damaging to bird life were the cyclodiene insecticides such as aldrin, dieldrin and endrin. Among the pesticides available to Rachel Carson's generation, these particular organochlorines were among the most toxic to birds. However, even the less toxic organochlorines – as long as they persisted in bird tissue rather than being readily cleared from the body – could reach lethal levels in the brain during periods of food shortage or when high energy demands forced the organism to call upon fat reserves.

Rachel Carson would be pleased to see that the use of DDT and most of the persistent organochlorine pesticides has now been banned in most of the world although it is clear that the ban had a lot more to do with concerns about human health than worry about birds. The continued use of DDT against the insect vectors of human diseases like malaria in some parts of the world remains controversial, and there are arguments for allowing continued use on a small scale where malaria control alternatives are impractical or ineffective. Realistically, birds are unlikely to get serious exposure to pesticides from small-scale indoor surface spraying in tropical climates.

Of greater concern is that we are still seeing extremely high levels of DDT and other organochlorines in areas where deep-burrowing earthworm species and other insects are bringing them up to the surface in their bodies[3]. An Australian study showed that virtually all birds living near cotton fields had residues of DDT and its metabolites in their stomach contents 15 years after the product was discontinued[4].

Nevertheless, the good news is that populations of falcons, ospreys, pelicans and cormorants have now recovered in many areas once affected by the agricultural use of organochlorines. Rachel Carson would be pleased.

Numerous organophosphates replaced organochlorines

In *Silent Spring*, Rachel Carson mentioned a few organophosphorous insecticides, notably parathion, as being exceptionally toxic to birds. She writes about parathion being used to control red-winged blackbirds (*Agelaius phoeniceus*), considered by many to be crop pests. She probably could not have imagined the dominance of this group of insecticides in the decades that followed the publication of the book.

Because government regulators saw that organophosphorous pesticides and carbamate insecticides stayed active in the environment for only days or weeks at the most, they quickly authorized their use to replace the organochlorines. The extreme toxicity of the newer compounds was glossed over or ignored. The main concern became how to limit exposure for the people applying these products or working in fields where the products had been applied. This problem has not been fully solved yet and many pesticide-exposure poisonings among agricultural workers continue to be recorded every year.

Both organophosphorous and carbamate insecticides have a similar mode of action: they bind to, and put out of commission a vital enzyme called cholinesterase. They are therefore lumped as 'cholinesterase inhibitors'. The cholinesterase enzyme, which is present across the animal kingdom, is active both in the gap between nerve cells (the synaptic cleft) and in the junctions between nerve and muscle cells (neuromuscular junctions). The role of the cholinesterase enzyme is to neutralize the neural messenger acetylcholine after it has allowed the nerve impulse to pass from one cell to the next. The sudden disabling of the critical cholinesterase in the bird's brain and peripheral nervous system leads to the equivalent of a 'short-circuiting' of neural connections with a multitude of consequences, none of them good. No other enzyme in the body has such extensive and complex functions; in fact, there is hardly a physiological mechanism or response that

Table 1. Toxicity of representative insecticides to birds and rats. The Aspirin index is the number of sparrow-sized birds killed by one tablet-sized quantity.

Pesticide	Average bird toxicity (median of available values) (mg/kg)	Average rat toxicity (mg/kg)*	Aspirin™ Index for birds of average sensitivity (No. median lethal doses)
Organochlorines			
aldrin	19.8	38 - 67	899
dieldrin	35.1	37- 87	507
lindane	90.8	88 - 270	196
DDT	1334	113 - 250	13
OPs and carbamates			
fenamiphos	1.1	2.3 - 19.4	16198
carbofuran	1.65	8.8	10799
monocrotophos	2.51	18 – 20	7099
aldicarb	2.82	0.62 - 1.23	6319
diazinon	5.25	300 - 1250	3394
methamidophos	15.8	13.0 - 15.6	1126
pirimicarb	20.5	142	868
chlorpyrifos	27.4	135 - 155	651
malathion	466	1000 - 1375	38
New insecticides			
imidacloprid	35.4	450	504
fipronil	39.2	96	455
chlorfenapyr	8.3	441 - 1152	2147

**Sources: International Program on Chemical Safety (World Health Organisation) and British Crop Protection Council Manual.*

does not involve cholinesterase.

Hence, the cholinesterase-inhibiting insecticides have a fundamental mode of action that affects many more species than just insects. If non-target species such as birds or mammals could break down and eliminate the insecticide before it could poison them, then it would not be a problem. Organophosphorous insecticides were relatively easy to produce, and chemists succeeded in creating a wide variety with slightly different molecular structures and properties. Some of these indeed had a lower toxicity profile to mammals. As it turns out, however, birds are singularly ill-equipped to deal with cholinesterase inhibitors because their detoxification machinery simply is not up to the task. Even insecticides developed to be far less toxic to humans and therefore allowed for applications with high human exposure (such as diazinon which was a mainstay of home and garden use) still prove to be exquisitely toxic to the average bird. We do not know why. The answer is no doubt buried in their evolutionary past.

It is hard to convey just how toxic to birds these organophosphorous and carbamate insecticides really are. We can start by listing LD_{50} values in milligrammes of active ingredient per kilogramme of bodyweight, the usual measure in toxicology circles (Table 1). This is the quantity of a substance estimated to be able to kill half of the exposed individuals. The toxicity of the pesticides to the laboratory rat (*Rattus norvegicus*), the usual human surrogate, is also shown for comparison.

In addition, to provide a more graphic illustration, I am borrowing from Rachel Carson's 'Aspirin™ index.' Recognising that the average person could not relate well to milligramme/kilogramme values, she calculated how many birds a tablet-size amount of pesticide could kill on average. For example, an aspirin-sized tablet of aldrin could kill an average of almost 900 sparrow-sized birds[5]. Table 1 provides the Aspirin™ index for several common insecticides and birds of average sensitivity.

We have already noted that an aspirin tablet-sized amount of the most acutely toxic organochlorine insecticide, aldrin, represents approximately 900 lethal doses for the average sparrow-sized bird. By contrast, the most toxic organophosphorous insecticide, fenamiphos, has an Aspirin™ index of more than 16,000 lethal doses, a significant jump in toxicity. Sensitivity of different bird species usually ranges from 10 times higher to 10 times lower than the average. This means that our Aspirin™ index is likely in the vicinity of 100,000 lethal doses for some species exposed to the most toxic organophosphorous insecticides.

The extremely popular insecticide carbofuran, applied to one hectare of maize (*Zea mays*)[6], represents more than 41 million lethal doses for the average bird. It is no wonder that with this and other insecticides of similar toxicity, high bird mortality has been frequent and unavoidable.

As the world adopted a wide variety of cholinesterase-inhibiting insecticides to replace persistent organochlorines, we saw a shift of effects from top-of-food-chain species (birds of prey and fish-eating birds) to insect- and grain-eating songbirds that breed in and around our farm fields. Because birds can move rapidly into pesticide-treated areas, they risk being exposed to pesticides simply by being in the wrong place at the wrong time.

Affected bird species are typically small and, when breeding, are present in relatively low densities. Thus, mass bird kills from pesticides tend to be spread over large areas.

The pesticide industry in the early- to mid-1980s conducted a number of definitive studies that showed how seldom bird mortality comes to light without considerable resources to detect it. This problem is not unique to pesticide kills but extends to bird mortality from other human factors such as communication towers, office buildings and windmills. The fact that small bird carcasses are hard to see and are quickly scavenged ensures that the loss of a few songbirds per hectare is hardly noticeable (Figure 1); yet, this loss may represent a large proportion of the breeding population of a given species once the deaths over the entire treated surface are accounted for.

I have analysed quite a few of the available avian pesticide impact field studies and constructed predictive models[7]. This work has led me to conclude that a lot of birds are dying in fields and pastures sprayed for insect control. Bird species that inhabit farmland and open areas or those that use farmland during migration are at risk. Waterfowl and game birds are at risk because they eat large quantities of foliage. Songbirds are attracted to pesticide-treated seeds. Birds that feed on agricultural pests, such as grasshoppers, grubs and cutworms, gorge on the freshly poisoned insects. (The loss of the latter bird species is doubly tragic because of the important role they can play in helping to control pest populations naturally[8].) Scavengers and predators in turn are poisoned when they consume the gut contents of their prey. All bird species are also exposed through their skin and eyes; they ingest residues when they preen their feathers, and inhale small droplets and pesticide vapours when they enter treated fields or spend time in field borders.

Some of the methods of pesticide delivery unknown in Rachel Carson's day have made the situation even worse for birds. An example is the coating of the active ingredients of pesticides onto small inert granules (sand, clay, or granulated dried maize cobs).

Figure 1. The observer is pointing to a typical bird kill in freshly-seeded oilseed rape – not the easiest to see *Photo: Pierre Mineau*

Figure 2. Pinkish carbofuran granules (circled) can be seen alongside weed seeds and other plant material retrieved from a poisoned duck
Photo: Canadian Wildlife Service

Agricultural engineers thought that preparing pesticides this way would ensure a slow release of the active ingredients around the roots of a developing plant[9]. Unfortunately, they probably did not foresee that the tiny granules would prove attractive to birds looking for grit (needed to grind food in the gizzard), weed seeds or broken crop seeds[10]. Depending on the product, the type of agricultural machinery and where in the field you look, anywhere from less than one per cent to more than 50% of the granules actually stay on the surface in full view of foraging birds[11]. Some species like waterfowl pick up the granules as they sift through puddles that develop in fields (Figure 2).

I would bet that Rachel Carson, with her keen understanding of the natural world, would have foreseen the problem of granular insecticides and grit-seeking birds. She might also have understood that we could not solve bird-pesticide conflicts by moving to cholinesterase-inhibiting insecticides.

Why continue to use such highly toxic pesticides?

Reports of mass bird mortality made Rachel Carson speak of a 'silent spring.' It was a powerful metaphor for everything that was wrong with the way pesticides were used (and abused) in the early days of the synthetic pesticide era. Undoubtedly, she would be pleased that governments now demand far more rigorous testing to anticipate the impact of pesticides on the environment in general and on birds in particular. However, she would surely be discouraged to learn that, despite the requirements for testing, governments have allowed farmers and forest managers to continue using products with an appalling record for killing birds even though their full impact has been known for decades.

A notorious example of such a product is the carbamate insecticide carbofuran (principal trade name Furadan™). Despite its extreme toxicity to birds, it appears to be one of the most widely used insecticides in the world, either in the form of a liquid spray or as a granular insecticide. The granular formulation is especially dangerous to birds because the carrier medium is sand (silica) and therefore very attractive to birds as the ideal grit material.

Two detailed US studies by the Food Machinery and Chemical Corporation (FMC), the maker of carbofuran in the 1980s[12], revealed that the typical loss of songbirds following the use of the granular formulation in mechanically seeded maize fields ranged from 3 to 16 birds per hectare after accounting for search efficiency and scavenging. This was despite concerted efforts to bury granules as much as possible to make them less visible to birds. At the time, given the popularity of this insecticide, this represented an estimated yearly kill of 17 to 91 million songbirds in US maize fields alone[13]. The same product was also registered for use with soybean, sorghum, peanuts, tobacco, cotton, sunflowers and rice as well as several less popular crops, undoubtedly adding to the total loss of birdlife. In a third company-led field study, a total of 799 birds of the same species horned larks – (*Eremophila alpestris*) – as well as a number of other species were found dead in a few seeded fields[14]. The kill rate uncorrected for finding and scavenging in that study was over eight birds per hectare, exceeding the typical breeding density of birds in farmland.

In a rather perverse way, carbofuran has been useful in helping us understand which birds search for food in crop fields and may therefore be at risk of poisoning. Based on the data I collected from studies and bird mortality incidents, I have documented more than 80 bird species known to have been killed by this insecticide just in Canadian and US cropland.

Today, the granular formulation of carbofuran is no longer legal in North America but is still sold all over the world for a variety of crops (although the fate of the closely-related product – furathiocarb – is still being debated in Europe). Often, the carbofuran granules are applied by hand to the fields and only crudely worked into the soil, if at all (Figure 3).

The principal manufacturer of carbofuran, FMC Corporation, continues to claim that the product is safe when used as directed.

'Furadan remains a useful product, vital to the sustainability of agriculture. FMC believes the proper use of Furadan does not create a risk to human health, wildlife, or the environment, and we will continue to promote its responsible use.' [15]

Another mass killer of birds is the organophosphorous insecticide monocrotophos. Within a few weeks in 1995/6, an estimated 20,000 Swainson's hawks (*Buteo swainsoni*) were poisoned to death in farm fields in the Argentine pampas after feeding on grasshoppers sprayed with the chemical (figure 4)[16].

Although the kills came as a shock to the conservation community, the carnage was surely no surprise to the manufacturers. As early as 1970, the two leading manufacturers of monocrotophos[17] had conducted small-scale tests in Europe in response to reported bird mortality. These unpublished tests had uncovered 74 dead or paralysed birds in only two hectares of crop, an amazing bird density by today's standard[18]. The following year, two Colorado researchers carried out a one-time search and found 69 dead or debilitated individuals, including birds of prey in a 32 ha wheat field treated with monocrotophos[19]. Kill reports were coming from many parts of the globe in a wide variety of croplands. Before the aforementioned incident in Argentina, the largest kill on record had been an estimated 10,000 wintering American robins (*Turdus migratorius*) following application of monocrotophos to two Florida potato fields[20]. The manufacturer determined that the pesticide had contaminated berry-producing shrubs in the field edge.

After monocrotophos ceased to be registered or used in North America or Europe, it fell below our radar. We were not thinking globally. In 1994, monocrotophos was said to be one of the most widely used insecticides in

Figure 3. Agricultural workers sprinkling insecticide granules on maize plants *Photo: FAO 1994*

Figure 4. What people imagine a bird kill should look like. Swainson's hawks killed by monocrotophos were gathered from surrounding fields for the photograph
Photo: Brian Woodbridge

the world and 15 different manufacturers were making it. According to the latest information available, there are now 19 listed manufacturers although I would like to believe the overall use of the chemical has declined[21].

The Swainson's hawk slaughter could just as easily have remained an idle topic of conversation among Argentine farmers over a shared yerba-mate. The reason the world found out was because the species was declining and US researchers were following radio-marked birds. By chance, the kills happened in close proximity to a farm where these researchers had been billeted previously and the farmer therefore knew exactly who to contact to report the kills.

The incident attracted substantial attention from newspapers and popular periodicals such as the National Geographic. When the Canadian government sent me to Buenos Aires to help the Argentine government develop a regulatory response to the events, my task was made a lot easier by the publicity the issue had received. I was able to work with dedicated and competent officials from the Argentine Ministry of Agriculture who clearly understood the possible impact on the reputation of their country's agriculture. Under a harsh international spotlight, Argentina immediately altered its regulations to control the use of monocrotophos. A few years later, they followed through with a total ban on the product in Argentina. The events in Argentina proved to be a watershed for that country and led to bird monitoring programmes and farmer outreach programs to reduce the footprint of agriculture on the pampas[22].

It is now obvious that insecticides such as carbofuran and monocrotophos continue to kill birds regularly and predictably wherever and whenever they are used. The latter are but two of the worst offenders but other products in current use in North America also carry a significant risk of causing mortality when used according to label directions. Based on mortality models and knowledge of pesticide use patterns, we can plot the likelihood of bird mortality across the agricultural landscape. This is shown here for the US on a State by State basis for 1997, the last year for which a comprehensive pesticide use survey was available (Figure 5). In several States, we expect more than 20% of the total farmland area to be causing bird mortality by virtue of the pesticide choices that were made.

Even for a developed country such as the US, pesticide use leads to a constant drain of bird life from cropped fields. A key question that emerged from the Swainson's hawk incident was: How many bird species like robins or Swainson's hawks are packed into a few hectares of farmland where a single farmer strictly following pesticide label instructions can kill thousands of birds at a time? It is a frightening reality that a single Argentine farmer could have as much impact on the Swainson's hawk population as several hundred farmers applying the same pesticide in the northern hemisphere where the birds are breeding, and therefore widely dispersed over farmland.

Today, carbofuran, monocrotophos and other well-known problem insecticides continue to be mainstays of the pesticide market in many countries, especially the developing nations of the southern hemisphere. The Canadian academic Bridget Stutchbury, in her popular book *Silence of the Songbirds*, recently revived the issue of the risk to migrant birds from toxic pesticides in the farmland where these birds overwinter. She describes the profligate and increasing use of bird-toxic compounds in Central and South American countries where North American migrants fly in the winter. She documents pesticide use that is four times higher than the level normally used on similar crops in the US, a level already considered far too high if bird losses are to be avoided.

In a 2001 *Pesticide News* article[24], Catharina Wesseling documented increasing pesticide imports into Central America, notably Costa Rica and Guatemala. Pesticides that made it to the top ten in one or more countries of the region included aldicarb, carbofuran, methamidophos and terbufos – all cholinesterase-inhibiting compounds of extreme toxicity to birds. Two years ago, Dr Rosalyn Renfrew, who was working on bobolinks (*Dolichonys oryzivorus*) in Bolivia, was able to confirm that monocrotophos was the dominant insecticide in rice cultivation in that country and was likely affecting a lot of birds.

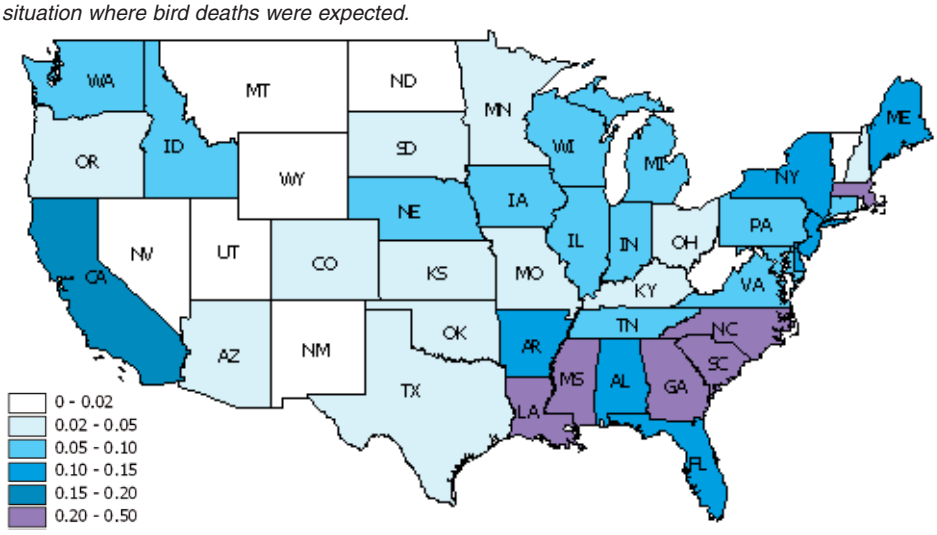
Why do people today not care about bird mortality?

Rachel Carson would have decried these continuing bird losses. But someone who is a little more hard-nosed might ask whether these pesticide-related kills really matter in the overall scheme of things. After all, birds die of many causes.

Some of our own work in North America suggests that pesticide-induced mortality has been an important contributor to grassland and farmland bird declines. For example, regional declines have been correlated with the use of toxic granular insecticides in rapeseed (canola) fields[25]. On the continental scale, my analyses suggest that the killing potential of the insecticides used is a better correlate of species declines than the simple intensity of farming. As argued in the previous section, kills on the wintering grounds can affect large numbers of birds. When bird populations are in decline because of a multitude of factors, is it wise to keep ignoring such a highly preventable cause of mortality?

The speed with which the Argentine government dealt with the monocrotophos issue remains a hard act to follow. Based on over 25 years of observation, I believe that pesticide regulation systems in different countries around the world are more or less in step with national public opinion, whatever that may be. The UK, for example, is a land of bird lovers and bird watchers. When it comes to pesticides and their impact on birds, Britons have been living in a bird-friendly bubble. Very early on, it was decided in the UK that high bird mortality in fields was unacceptable.

Figure 5. Proportion of the total 1997 US farmland area where the use of pesticides created a situation where bird deaths were expected.



Therefore, most of the pesticides that continue to kill birds around the world have been severely restricted in the UK. The UK Wildlife Incident Investigation Scheme[26] has been the envy of the rest of the world for as long as I have been studying pesticides. Current declines of common birds in farmland are better understood in the UK than they are anywhere in the world; the British are therefore in a much better position to address these declines.

The removal of many products with a direct lethal effect on birds has shifted concerns to how pesticide effects on plants and invertebrates, whether targeted or not, may indirectly affect birds. In the most general way, the increased use of herbicides has reduced the need for crop rotation and allowed for larger fields and extensive monoculture. The use of herbicides in fields and field margins has directly affected the quantity and availability of nesting habitat as well as the capacity of that habitat to support a rich variety of insects, which is essential for birds. Some herbicides and fungicides and especially insecticides can directly reduce the quantity and variety of insect life available to birds. The potential for these indirect effects is still not formally considered at the time of pesticide registration – but at least British regulators are aware of it. In North America, we are still too busy dealing with acute direct effects to pay attention to indirect effects.

The North American public is generally apathetic about the loss of birdlife on farmland, and there is a long-standing belief in rural North America that farmland birds are crop pests. For these reasons, regulation to reduce pesticide-induced bird deaths moves at a glacial pace. This is all the more surprising because, in both Canada and the United States, most birds are 'protected' under treaty obligation and strict liability provisions and laws against killing birds have been in place since 1916[27]. Ironically, the 'Migratory Bird' treaty or convention was initially signed as a result of concerns about declines in bird species considered beneficial to farming interests because of their role in insect control. Yet, it took 47 years between the first observations of mass bird mortality from the use of parathion to its eventual ban in the US. And when the restrictions were finally imposed, human health was invoked rather than the safety of the environment.

The US Environmental Protection Agency launched its re-evaluation of the granular formulations of carbofuran in 1985, following a risk assessment that confirmed how dangerous the product really was. Nevertheless, it took six years to reach a negotiated settlement with FMC Corporation before the principal uses of the pesticide could be gradually phased out. As recently as 2006, the State of Louisiana was still petitioning the EPA to allow granular carbofuran to be used again in rice cultivation, a move fiercely contested by environmental groups and wildlife agencies. Perhaps more surprising is the fact that the use of carbofuran as a liquid spray has continued more or less unabated despite ample evidence from industry field studies dating back to the 1980s that the use of this product causes regular and predictable avian mortality.

As an employee of the Canadian Wildlife Service and responsible for the review of both new and existing pesticides in Canada, I built and submitted the case for cancelling the registration of granular carbofuran in 1991. At the time, my department presented a strong scientific case against *all* formulations and *all* uses of this product. In our opinion, there was no possible application of carbofuran (short of glasshouse use) that did not place birds at risk. The US Fish and Wildlife Service went on record with a similar evaluation. Both Canada and the US have imposed restrictions on the granular products and on uses that place endangered species at risk. However, as of 2009, the fate of carbofuran is still being debated in both Canada and the US and the manufacturer has vowed to keep fighting the EPA's proposed cancellation of the product. The presence of carbofuran residues in drinking water, rather than bird deaths, is now the main reason cited by the EPA for proposing cancellation.

Except in the UK and some parts of Europe, it is fair to say that the interests of birds have not weighed heavily in decisions about which pesticides should be registered and where. The products responsible for much of the avian mortality seen around the globe tend to be the same depressingly familiar ones: carbofuran, monocrotophos, diazinon, parathion, phorate, terbufos, fenthion, and a few others of lesser toxicity such as fenitrothion or chlorpyrifos.

Several of these products are in the process of being curtailed in North America because of their threat to human health (that of children especially) and, as a fortunate side-effect, bird mortality in fields is decreasing[28]. However, many uses potentially damaging to birdlife persist and important regulatory decisions affecting bird welfare can take decades to be settled as illustrated earlier. The patents for all of these pesticides have now expired, allowing a host of small (or not so small) companies to manufacture and sell them to any country that has not limited their use.

Have we made progress?

Certainly, our understanding of pesticides has improved dramatically over the past five decades. Whether you believe that we have acted quickly enough to deal with pesticide problems may depend on whether you are a 'glass half full,' or 'glass half empty' sort of person. Certainly, when I look at our own forestry situation in Canada, I now see greater emphasis on biological control of forest pests than there was a few decades ago. Similarly, the promotion of products with a better environmental profile is now part-and-parcel of project planning by the World Bank[29] and other international development agencies. The use of monocrotophos has recently declined in Central and South America following the listing of all monocrotophos formulations in Annex III of the Rotterdam Convention on the trade in hazardous chemicals. The American Bird Conservancy, the only non-governmental organisation that maintains an interest in bird and pesticide issues in North America, is tracking these changes and encouraging the remaining user countries to find safer alternatives.

The last major desert locust outbreak in the Sahel region of Africa between late 2003 and early 2005 saw 13 million hectares sprayed with toxic organophosphorous insecticides, notably chlorpyrifos and malathion and to a lesser extent fenitrothion, putting millions of birds (not to mention people) at risk of poisoning. Incidentally, a number of European bird species migrate to and through the Sahel, so what takes place in that region should be of interest to European conservationists. A large study published in September this year[30] reported that a staggering 59% of all trans-Saharan migrating birds (75 out of 127 species) have been declining since 1970 due to low rainfall, shrinking floodplains and massive changes in land use, making these bird species more vulnerable to additional stressors. I suggest that the treatment of 13 million hectares with toxic organophosphorous insecticides is an additional stressor those birds could do without!

Recently, the Food and Agriculture Organization (FAO) and some national governments have been promoting the switch to biological control agents such as the fungus *Metarhizium anisopliae* (commercialized as Green Muscle™). Pioneering work on behalf of the Government of Senegal is showing the world that biological control of locusts is not only possible but cost effective for a developing country. In 2009, almost 100% of grasshopper infestations in Senegal were treated with *Metarhizium* early in the season, preventing the second and third generations from developing and completely eliminating the need for any chemical interventions later in the season, resulting in major environmental and economic savings for that country[31].

The adoption of better laws and regulations governing pesticide registration make it possible to oppose new products that have a poor environmental profile. This continues to be easier than removing older, well-established products from circulation and this is true regardless of how bad the older products are.

For example, a few years ago, the American Bird Conservancy led the fight against registration of chlorfenapyr, a novel insecticide that showed promise against pests already resistant to existing insecticides in cotton. Unfortunately, the pesticide was *extremely* harmful to birds, in terms of both acute and reproductive toxicity. In addition, it had very high toxicity to aquatic organisms and was quite persistent. Through a coordinated campaign, the American Bird Conservancy was able to show US regulators that the American public *did* care about high risks to birds and this gave the EPA the necessary support to make a science-based decision and deny registration in the US[32]

although chlorfenapyr has now been registered in many other countries.

Over the span of my career, I have seen an increased acceptance in government circles of the concept of relative risk assessment. This replaces the old 'ostrich with its head in the sand' policy that could be paraphrased as: *'If it's registered, it's safe!'* There is a grudging acknowledgment that pesticide registration decisions involve a difficult and value-laden balancing of risks and benefits. Having done the work myself, I have a genuine appreciation for the scientists who have to weigh the pros and cons of the pesticides submitted for registration. This is not an easy job. For example, you may recall that in last year's Rachel Carson Lecture, Dr Charles Benbrook discussed the increasing use of systemic insecticides including the new family of products known as the neonicotinoids. These products pose a whole new set of problems. It is becoming clear that we may now be putting pollinators like bees at greater risk even as the farm landscape gradually becomes safer for birds.

For several years now, I have been involved in the design of an unbiased and objective pesticide risk measurement system that will be used to inform farmers and crop advisors about these trade-offs in risks and how best to tailor their pest control strategies to the local environment. This web-based system, called PRiME (for Pesticide Risk Mitigation Engine)[33] has been developed through a grant from the Natural Resources Conservation Service (of the US Department of Agriculture) to the not-for-profit IPM Institute and is about to be rolled out in the US, with Canada ideally to follow soon.

Are we better at predicting new problems?

Undoubtedly, Rachel Carson would be pleased to see that we now ask about bird toxicity and other potential environmental effects before new pesticides are put on the market. It will continue to be necessary to test new pesticides because surprises are always possible. One recent surprise for avian scientists was the fact that the usual inter-species sensitivity profile was completely reversed for the new insecticide fipronil. For some reason, galliform species that are usually reasonably resistant to most of the cholinesterase-inhibiting products are exquisitely sensitive to this particular molecule.

Without wishing to enter into a discussion on the ethics of animal testing, I have to open a small parenthesis here. Many of my colleagues who are involved with predicting the risk to birds are becoming increasingly concerned about the current trend to cut back drastically on toxicity testing on live organisms. Many of us believe that already too few species are tested to provide an accurate picture of potential ecological harm. I am afraid it will be a very long time indeed before we see *in vitro* systems that will allow us to detect a specific sensitivity issue such as we saw in the case of fipronil and galliform bird species.

With the years of experience we have with bird impacts, it would be logical to assume that we now can predict which pesticides are likely to cause a problem for birds and when. Certainly, we now understand about persistence and bio-accumulation. And some of my own modeling of pesticide field studies has shown that bird mortality is somewhat predictable. However, the same work suggests that the way we assess the safety of new pesticides for birds is fundamentally flawed and most definitely out of date. Relying on long-established methods that were devised in the organochlorine era, pesticide regulators around the world still assume that birds are only exposed to pesticides through the ingestion of contaminated foods. And this, despite early research results that unequivocally showed the importance of non-dietary routes of exposure such as absorption through the feet and skin, preening, and inhalation. Research we are currently conducting supports these early findings.

So far in this article, I have placed a great deal of emphasis on bird mortality and the reader may come away with the impression that this is the only issue that concerns us. If the truth be told, the issue of lethal effects still dominates the registration review of pesticides. It is hard to argue that lethal effects do not represent an important endpoint. Put more simply, dead birds do not breed very well nor do they exhibit normal behaviours!

However, an increasing concern for regulators is that a high proportion of pesticides currently registered, or submitted for registration, is predicted to cause reproductive effects based on current testing and assessment procedures. Our analysis showed over 15 years ago that, even with modern pesticides, effects on the development and survival of eggs and chicks often occur without visible signs of toxicity in the parents[34]. Reproductive tests, like the requirements for acute and dietary toxicity testing, are carried out in precocial species – that is, birds that do not require much parental care after hatching. The two species most often used are the mallard (*Anas platyrhynchos*) and a quail species, the northern bobwhite (*Colinus virginianus*). The design of the test harks back to the organochlorine period. The test subjects are exposed to the pesticide for a long time (10 weeks) before they lay eggs, and then for another 10 weeks of egg-laying. The eggs are collected immediately after they are laid and artificially incubated. Endpoints include parental weights, egg production, eggshell thickness, and hatching and survival rates of the chicks. The test therefore is based on a very truncated version of avian reproduction with minimal behavioural input on the part of the parent birds.

Nevertheless, the obvious question is whether effects seen following such prolonged feeding on the pesticide would also be seen in the course of the much shorter exposure periods more typical of current pesticides. It does not help that there currently is no requirement to report pesticide concentrations in the eggs of the birds on test – thereby precluding any attempt to link the laboratory with field conditions. The jury is still out as to whether effects seen in the laboratory are occurring in wild bird populations.

Many readers will be familiar with *Our Stolen Future* by Theo Colborn and colleagues, first published in 1996. This book has been compared to *Silent Spring* in terms of the impact it has had on the public's awareness of chemical hazards and on chemical regulators worldwide. The thesis of the book is that minute amounts of some chemicals can disrupt the delicate hormonal balance of developing embryos and foetuses and have long-term effects that are sometimes not evident until the exposed individuals reach sexual maturity. The authors questioned some of the basic tenets of toxicology such as the idea that a toxic response is always proportional to the size of the dose. At this point, our meagre understanding of how pesticides affect birds sub-lethally in the wild does not allow us to conclude whether pesticide effects on hormones lead to further losses of birds.

As part of a group of ecotoxicology experts and veterinarians convened by the Smithsonian Institute in 2007, I was able to add to the chorus of voices that lamented our continued ignorance of the consequences of sub-lethal pesticide exposure in wildlife. As a group, we rated this lack of understanding as the most vexing problem in ecotoxicology[35]. As mentioned earlier, cholinesterase inhibitors can have a broad range of physiological effects at less than lethal exposure levels. Poisoned individuals may be seriously compromised physiologically even if they initially survive an acute exposure.

Some of the factors that may contribute to later death in the case of birds exposed to cholinesterase inhibitors include injuries from hitting moving or stationary objects while in a state of intoxication, weakness from anorexia, a reduced ability to regulate internal temperature, disorientation during migration, disruption of normal circadian (24-hour) patterns, as well as delayed muscular weakness, to name a few[36]. There have been several reviews of the behavioural effects of pesticides[37]. Some of the on-going debates have been whether behavioural impairments are expected at low levels of exposure relative to levels that cause visible impairment or death and whether these subtle (or not so subtle) effects contribute to reduced survival and should be factored into the assessment of pesticide safety to birds more than they currently are. When it comes to newer pesticides, our ignorance of sub-lethal effects is almost complete.

What should we do?

Above all else, I believe that Rachel Carson would urge people who care about the environment to get more involved in pesticide issues. Specifically, she would probably counsel that the most effective action organisations and individuals can take is to let regulatory authorities know they care about the impact of pesticides on people, birds, and the environment in general. Good science-based advocacy from responsible NGOs is needed to provide a counterweight to the pesticide industry lobby. Initiatives to make pesticide informa-

tion more generally available, such as the Pesticide Action Network Pesticide Database[38] or the more recent European Footprint initiative[39], are critical to our understanding of pesticide risks. Unfortunately, the latter even with official EU backing appears to be running out of funds and may not be updated.

Rachel Carson would clearly be pushing the system to move to more Integrated Pest Management and intelligent pest control. Rachel Carson was not against pesticides – she was against a blind reliance on poorly thought out chemical solutions replacing common sense. The pesticide industry is in the business of pest control to make a profit and this profit is dependent on high volume sales. It is illogical to expect those industries to advocate rational pesticide use and to promote non-pesticide alternatives. This is why Rachel Carson would continue to push government to take its rightful place in the design of pest control strategies in order to ensure our food supply while minimizing and mitigating the impact on the natural environment and on our health and well-being.

Concerns over the well-being of migratory birds must transcend national boundaries. We need to think more globally. In the US, the American Bird Conservancy has recently petitioned the EPA to prohibit the import of foodstuffs with residues of pesticides that have been restricted domestically because of their impact on birds and the environment. If the EPA were to adopt this policy, it would greatly curtail the use of environmentally-damaging pesticides and hasten the advent of lower-risk alternatives in countries that do not have the infrastructure necessary to oppose powerful agro-chemical interests.

The pesticides most responsible for bird mortality around the globe can easily be replaced by better alternatives without risk to the livelihood of farmers. Despite corporate pronouncements to the contrary, environmentally-damaging pesticides are rarely essential to food security. Removing them from the market should be the next phase in the continued evolution of our pest control strategy.

That would be a legacy that Rachel Carson would be proud of.

References

1. One notable exception was VB Wigglesworth. In an incredibly prescient article in the Atlantic Monthly in 1945, Wigglesworth warned about DDT's potential to disrupt the 'balance of nature.'

2. Peakall DB. 1993. DDE-induced eggshell thinning: an environmental detective story. Enviromental Review 1:13-20; LJ Blus, Organochlorine Pesticides, in Handbook of Ecotoxicology, DJ Hoffman, BA Rattner, GA Burton, J Cairns, Eds. (Lewis Publishers, Boca Raton, 1995) chap.13.

3. Harris, ML, LK Wilson, JE Elliott, CA Bishop, AD Tomlin, KV Henning. 2000. Transfer of DDT and Metabolites from Fruit Orchard Soils to American Robins (Turdus migratorius) Twenty Years After Agricultural Use of DDT in Canada. Archives of Environmental Contamation and Toxicology 39: 205–220.

4. Sánchez-Bayo F, 1999. Ward R, Beasley H. A new technique to measure bird's dietary exposure to pesticides. Anal.Chim. Acta :399: 173-183.

5. Necessary inputs into the calculation: Each tablet weighs approximately 392 mg. Average sensitivity is determined by the median lethal dose. The typical agricultural bird chosen for the index would have the weight of a Eurasian tree sparrow (Passer montanus) or 22g.

6. Assuming an application rate of 1.5 kg of active ingredient per hectare.

7. Mineau, P. 2002. Estimating the probability of bird mortality from pesticide sprays on the basis of the field study record. Environmental Toxicology and Chemistry 24(7): 1497-1506.

8. Kirk, DA, MD Evenden and P Mineau. 1996. Past and current attempts to evaluate the role of birds as predators of insect pests in temperate agriculture. Nolan and Ketterson (eds.) Current Ornithology 13: 175-269.

9. Of course, granular formulations can often be safer to apply by hand with a shaker than a liquid formulation of the same pesticide with a back-pack sprayer. The cost-benefit ratio of these formulations may therefore shift in developing countries where hand application is the norm.

10. Best, LB and DL Fischer. 1992. Granular insecticides and birds: factors to be considered in understanding exposure and reducing risk. Environmental Toxicology and Chemistry 11(10):1495–1508

11. Mineau, P. and A Clark. 2008. Granule incorporation. Appendix 21; Scientific Opinion of the Panel on Plant Protection Products and their Residues on Risk Assessment for Birds and Mammals, EFSA Journal (2008) 734, 12pp.

12. These studies were required by the US Environmental Protection Agency as a condition of continued registration.

13. Mineau P. 2005. Direct losses of birds to pesticides - Beginnings of a quantification. In Bird Conservation Implementation and Integration in the Americas: Proceedings of the Third International Partners in Flight Conference 2002 (C.J. Ralph and T.D. Rich, eds.), U.S.D.A. Forest Service, GTR-PSW-191, Albany, CA; Vol. 2, pp. 1065-1070.

14. Mineau, P. 1993. The hazard of carbofuran to birds and other vertebrate wildlife. Technical Report Series. No. 177. Environment Canada, Canadian Wildlife Service, Ottawa. XXii+96 pp.

15. FMC Corporation 2009. http://www.furadanfacts.com/ Accessed 2 October 2009.

16. For a review of the case and original references see Hooper, M, P Mineau, ME Zaccagnini and B Woodbridge. 2003. Pesticides and international migratory bird conservation, In: Handbook of Ecotoxicology, 2nd Edition, DJ Hoffman, B.A. Rattner, G.A. Burton and J. Cairns Jr. [eds] Lewis Publishers, Boca Raton, Florida, pp. 737-754.

17. Ciba-Geigy, now Syngenta and Shell, now BASF.

18. The published account of this study says that ca. 100 birds were killed and more intoxicated. See: Guth. JA 1994. Monocrotophos - Environmental Fate and Toxicity in Reviews of Environmental Contamination and Toxicology, edited by Ware, G. New York: Springer-Verlag, pp. 75 - 136.

19. Benson, DE and Baker, DL. 1971.Effects of a mid-April application of Azodrin-5 on wildlife populations in northeastern Colorado wheatlands. Unpublished report.

20. Lee, C. 1972. Death in the Potato Fields. The Florida Naturalist 45:60-61.

21. According to the 2009-2010 Pesticide Manual, British Crop Protection Council, current manufacturers are: ACA; Agro Chemicals India; Cheminova; Comlets; Coromandel; Crystal; DE-NOCIL; Gujarat Pesticides; Hindustan; Hui Kwang; India Pesticides; Makhteshim-Agan; Nagarjuna Agrichem; Nantong Jiangshan; Ralchem; Sabero; Sharda; Taiwan Tainan Giant and United Phosphorus.

22.Maria-Elena Zaccagnini, personal communication

23. Mineau, P. and M Whiteside. 2006. The lethal risk to birds from insecticide use in the U.S. – A spatial and temporal analysis. Environmental Toxicology and Chemistry 25(5):1214-1222.

24. Wesseling C. 2001. Dangerous pesticide use in Central America - Wanted: a new approach. Pesticides News 54, December 2001, pages 12-14.

25. Mineau, P, CM Downes, DA Kirk, E. Bayne and M Csizy. 2005. Patterns of bird species abundance in relation to granular insecticide use in the Canadian prairies. Ecoscience 12(2):267-278.

26. http://www.pesticides.gov.uk/environment.asp?id=58

27. Mineau, P. 2004. Birds and pesticides: Are pesticide regulatory decisions consistent with the protection afforded migratory bird species under the Migratory Bird Treaty Act? The William and Mary Environmental Law and Policy Review 28(2): 313-338.

28. Mineau, P. and M Whiteside. 2006. The lethal risk to birds from insecticide use in the U.S. – A spatial and temporal analysis. Environmental Toxicology and Chemistry 25(5): 1214-1222.

29. For example, the World Bank prohibits the use of certain classes of pesticides in their funded projects in the interests of protecting human health.

30. Zwarts L., Bijlsma RG, van der Kamp J. and Wymenga E. 2009. Living on the edge: Wetlands and birds in a changing Sahel. KNNV Publishing, Zeist, The Netherlands.

31. Wim Mullié; personal communication

32.http://www.epa.gov/opprd001/chlorfenapyr/

33. http://www.ipminstitute.org/prime/

34. Mineau, P, DC Boersma, and B Collins. 1994. An analysis of avian reproduction studies submitted for pesticide registration. Ecotoxicology and Environmental Safety 29:304-329.

35. Aguirre, AA, T Augspurger, V Beasley, S Bursian, J Elliott, A Fairbrother, J French, Jr., M Fry, S Gerould, N Golden, R Helm, M Hooper, M Jankowski, MS Johnson, E Little, J Meiller, S Miller, P Mineau, S Monfort, C Orazio, M Pokras, BA Rattner, T Rowles, J Sevin, K Skrabis, D Sparling, R Stroud, Y Tondeur, L Touart. In Press. Summary of the Wildlife Toxicology Workshop. Ecotoxicology.

36. Mineau, P. 1991. Difficulties in the regulatory assessment of cholinesterase-inhibiting insecticides. In: P. Mineau (Ed.) Cholinesterase Inhibiting Insecticides - Their Impact on Wildlife and the Environment, Elsevier, Amsterdam, pp. 277-299.

37. Grue, CE, PL Gibert, and ME Seeley. 1997. Neurophysiological and behavioral changes in non-target wildlife exposed to organophosphate and carbamate pesticides: thermoregulation, food consumption, and reproduction. Amer. Zool. 37:369-388.

38. http://www.pesticideinfo.org/

39. http://sitem.herts.ac.uk/aeru/footprint/en/index.htm