Additional mortality rate of wildfowl caused by ingestion of lead shotgun pellets: a re-analysis of data from a 70-year-old field experiment on wild Mallards *Anas platyrhynchos*

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Abstract

A widely-used method for the estimation of numbers of deaths of wildfowl caused by the ingestion of spent lead gunshot pellets depends entirely upon field experiments on wild Mallards *Anas platyrhynchos* conducted 70 years ago in the USA by Frank C. Bellrose. The validity of this method has recently been questioned because of the simple method used to estimate the annual mortality caused by the experimental administration of lead gunshot and because the administration of shot appeared to increase the risk that treated birds would be shot by hunters, which might bias the mortality estimates. In this paper, we report a re-analysis of Bellrose's data using a different method which removes the possible effect on mortality estimates of differences in the proportion of birds killed by hunters in the first year after release. Our results indicate that the additional mortality caused by the administration of lead gunshot in the first year after treatment was substantial and dependent upon the dose of gunshot administered, to a similar extent as was found by Bellrose.

Key words: ammunition, Bellrose, death rate, lead poisoning, ring recovery.

It is 70 years since Frank C. Bellrose conducted controlled experiments on the effects of ingested lead gunshot on wild Mallards *Anas platyrhynchos*. Bellrose's experimental results, together with much other work by him on the effects of ammunition-derived lead on ducks, were published in the Illinois Natural History Survey Bulletin in 1959 (Bellrose 1959). Bellrose's study has continued to be much used and cited worldwide. For example, his experimental study has recently been used to estimate annual numbers of deaths and annual additional mortality rates of European waterfowl caused by the ingestion of spent lead gunshot (Mateo 2009; Pain *et al.* 2015; Green & Pain 2016) and the economic value of waterfowl hunting

opportunities foregone in Europe because of this mortality (Andreotti *et al.* 2018).

However, the validity of these applications of Bellrose's experimental results has recently been called into question. The European Chemicals Agency (ECHA) submitted a restriction proposal in April 2017 on lead in gunshot used in or over wetlands. Restrictions are a tool that exists in European Union (EU) law to protect human health and the environment from unacceptable risks posed by chemicals. They may limit or ban the manufacture, placing on the market or use of a substance. ECHA works with experts from EU Member States to provide scientific opinions on any proposed restriction to help the European Commission and the Member States to take the final decision on whether to apply the restriction. Its proposals about restrictions on lead ammunition have been evaluated in detail (Treu *et al.* 2020).

During evaluations of this restriction proposal, the ECHA Committee for Risk Assessment (RAC) examined the Bellrose (1959) calculations and questioned the appropriateness and reliability of the methods (RAC & SEAC 2018). Bellrose estimated the additional *per capita* annual mortality of waterfowl caused by the ingestion of spent lead shotgun pellets using recoveries of wild Mallards captured, dosed experimentally with lead gunshot, ringed (banded) and released. His principal findings were: 1) mortality of wild Mallards experimentally dosed with lead gunshot pellets and then released was higher in the year after dosing than the mortality during the same period of control animals that were not dosed, and 2) the proportion of dosed birds reported to have been shot by hunters in the year after dosing was higher than for the control untreated ducks during the same period, indicating that dosed ducks were more vulnerable to shooting. Both effects were dose-dependent: ducks given larger numbers of shot were affected to a greater degree than those given just one shot. ECHA wished to know (Question 1) whether Bellrose's estimate of the total additional mortality of dosed ducks remained valid when more recent statistical methods were used and also (Question 2) how much of the additional mortality was attributable to the greater vulnerability of dosed birds to shooting, rather than to death caused by lead poisoning.

In this paper, we use the original experimental data of Bellrose (1959) to attempt to resolve the first of these questions by estimating the additional mortality caused by experimental dosing by a new method, which we argue is less likely to be biased than the simple method used by Bellrose.

Methods

The experimental data

The data relevant to this paper are all given in Table 27 of Bellrose (1959) which reports results of field experiments done on wild Mallard ducks in 1949, 1950 and 1951 at the Chautauqua National Wildlife Refuge, Illinois, USA. In each experiment, wild Mallards were captured and fitted with numbered metal bands. Treated birds were given one, two or four #6 lead shotgun pellets via the oesophagus before being released. Similar numbers of ducks were banded and released without administration

of shotgun pellets. Experiments varied in the age class (juvenile or older) of the birds at capture and in the number of shot administered to the treatment group or groups, but in all cases there were matched cohorts of treated and untreated birds of comparable age, banded and released in the same season. In 1950 and 1951 there were separate experiments on juveniles and adults. In four cases, birds of the same age class in the same banding year were divided into three groups; a control group and two treatment groups given different numbers of shot. We treated these cases as two separate experiments, though they shared the same control group. This gave nine experiments in all (Table 1). All of the experiments we analyse here were conducted on male Mallards. Bellrose also reported a study on female Mallards in his Table 27, but we do not use the results from that because we do not regard it as an adequately controlled experiment. This is because the ages of the treatment and control groups of females were not known and the groups were banded and released at different times

Table 1. Numbers of wild male Mallards banded and recovered in experiments in which different numbers of lead gunshot pellets were administered when the birds were banded. Each row of the table represents the comparison between an experimental treatment group and a control group. Experiments with the same prefix letter in the code had two treatments which shared the same control group. The age classes of birds at banding were juvenile (J) or adult (A). The additional annual mortality caused by the treatment in the first year after banding M_{1a} was calculated for each experiment as described in the text. Data are from Table 27 of Bellrose (1959).

				Control		**Treated**		
Code	Banding year	Age class	Number of shot	Number banded	Recoveries in years 2–4	Number banded	Recoveries in years 2–4	M_{1a}
A	1949	A	1	560	124	559	120	0.031
B1	1950	A	1	278	73	274	58	0.194
B2	1950	A	2	278	73	277	25	0.656
C1	1950	J	1	111	26	117	20	0.270
C2	1950	J	2	111	26	115	28	–0.039
D1	1951	A	1	300	53	324	49	0.144
D2	1951	A	4	300	53	284	22	0.562
E1	1951	J	1	207	50	180	42	0.034
E2	1951	J	4	207	50	220	24	0.548

(1951 for treated females; 1939–1943 for controls).

Bellrose reported the number of band recoveries in the year after release and the first four years combined after release for each group of birds. In the USA, recoveries of banded ducks are mostly made by the hunters who shoot them. Few banded birds that die from other causes are reported. Not all banded ducks that hunters kill are reported to the authorities, but Bellrose used reward bands in some experiments to increase this proportion, though treated and control ducks were matched in this regard. Reward bands have an inscription offering a payment to the hunter if the band is reported, whereas the inscription on standard bands does not. In the 1949 experiment re-analysed here, standard bands were used on one group of Mallards and reward bands were used on another group. The reward bands were federal ones that gave no indication of the nature of the reward. Within each reward/no-reward group, bands were alternately attached to ducks with and without experimental administration of one shot pellet, which was the only experimental treatment in 1949. In 1950 and 1951, all ducks were marked with a reward band that specified a reward of $2 (Bellrose 1955, 1959).

Analyses by Bellrose

Bellrose (1959) presented two types of analysis of the experimental results. Firstly, the proportion of banded birds recovered in the year following banding was calculated for each group of birds. The ratio of these proportions for treated relative to control birds for a given experiment was used as a measure of the effect of treatment on vulnerability to shooting. Secondly, the *per capita* mortality rate in the year after banding was calculated by dividing the number of recoveries in the first year by the number in the first four years after banding. This was done separately for each group of birds. The difference in these first-year mortality rates between treatment and control groups in the same experiment was calculated as a measure of the effect of treatment with ingested lead gunshot.

Our method for estimating the total additional per capita mortality rate of treated ducks compared with controls

Our re-analysis addresses the second of the two analyses by Bellrose described above. Bellrose conducted his study at a time when methods available for the analysis of band recovery data were simple and did not take into account differences among age classes and time periods in the proportion of those banded birds that die in a given period that are reported (the reporting rate). The assumption of constant reporting rate underlies the methods developed and used by Lack (1943) and Haldane (1955). The method used by Bellrose to calculate additional mortality would have been reliable if all banded birds had died by the end of the recovery period (4 years) and if the reporting rate had been the same for treated and untreated groups and in all of the first four years after banding, but neither of these assumptions is likely to have been correct. Whilst annual mortality rates of Mallards in the USA are usually sufficiently high that most birds would be expected to have died after four years, some would still have been alive and this proportion was not

established in this study. Band reporting rates were not estimated and may have varied among years and groups. Seber (1973) and Brownie *et al.* (1985) provide accounts of the subsequent development of statistical methods used to estimate mortality rates from band recovery data which did not assume that reporting rate was constant and allowed it to vary with time and characteristics of the bird, such as its age and sex. Hence, the estimates by Bellrose of differences in first-year mortality between experimentally dosed and control birds can now be improved upon by allowing for possible differences in reporting rate in the first and subsequent years after banding and the truncation of the recovery period at four years.

Fortunately, the design of the experiments means that more detailed recovery data separated by year and sophisticated analytical methods are not needed to obtain more robust estimates of the total additional annual mortality caused by treatments with ingested lead gunshot. A comparatively simple procedure is sufficient to avoid the problems described above. We suggest that it is reasonable to assume that the effects of gunshot administration on mortality rate and vulnerability to shooting would have been absent or minimal more than a year after treatment. Experiments on captive Mallards show that lead shot are generally voided from the digestive tract and/or ground down and absorbed, or kill birds within weeks of being administered (*e.g.* Bellrose 1959; Longcore *et al.* 1974), although under certain conditions shot can be retained for several months (Finley & Dieter 1978a). Consequently the experimentally treated wild birds were very unlikely to have retained any of the administered shot beyond the first year after treatment. Absorbed lead is excreted or incorporated into bone and feathers. Lead incorporated into feathers remains there and is not thought to have any effect on the bird's health. Once lead has been deposited in bone, relatively little of it is remobilised (Scheuhammer 1987; Franson & Pain 2011; Krone 2018). Medullary bone in female birds acts as a labile reservoir for the supply of eggshell calcium during egg-laying (Dacke *et al.* 1993). Birds store and remobilise lead in bone in a similar way to calcium and Finley & Dieter (1978b) found the medullary bones of egg-laying female Mallards dosed with lead shot had much higher lead residues than the bones of non-laying females and males. Female birds remobilise some lead from the skeleton when they form eggshells (Finley & Dieter 1978b). While it is possible that some lead from experimentally-administered shot previously deposited in bone could be mobilised in a subsequent year, this seems likely to be a small effect, even for in female birds during egg-laying. In male Mallards, which are the subject of all of our analyses, we would expect any residual effect of treatment more than one year later to be negligible.

Hence, for a given experiment, the fraction of banded birds recovered during years 2, 3 and 4 after banding combined should be directly proportional to the proportion of banded birds that survived beyond one year after banding in both the treatment and control groups of a given experiment. That is because the control and treated groups in an experiment were

matched for band type (reward or not), age, sex and the calendar years in which band recoveries were obtained. The two groups only differed in conditions in the first year after banding, when the birds in the treatment groups were exposed to the effects of the administered gunshot whilst those in the control group were not. We therefore expect that the conditions that the two groups of ducks experienced in years 2–4 after banding, and hence the probability that a banded duck still alive at the end of the first year would die and be reported, would be the same for treated and control birds. Hence, for a single experiment, we argue that the additional mortality rate in the first year due to experimental exposure to ingested gunshot M_{1a} is given by

$$M_{1a} = 1 - ((R_{2\text{–}4T}\, N_C)/(R_{2\text{–}4C}\, N_T)),$$

where $R_{2\text{–}4T}$ and $R_{2\text{–}4C}$ are the numbers of band recoveries in years 2, 3 and 4 after banding combined for a treatment group and the control group, respectively, and N_T and N_C are the numbers of birds initially banded for a treatment group and the control group, respectively. The logic underlying the use of this expression is explained in the Appendix. We note here that M_{1a} is an estimate of the annual probability of death caused by the experimental treatment during the first year after banding, *conditional* on the individual being exposed to the risk of death caused by treatment throughout the year and not having died during that year for reasons unrelated to the treatment. In other words, it is the *proportion* by which the survival of banded birds to one year after banding is reduced by the treatment. This is not the same as the proportion of birds in the treatment group that died because of the treatment. Causes of death attributable and not attributable to dosing can be thought of as competing with each other to kill the birds.

For a given experiment, the magnitude of M_{1a} depends upon how much the proportion of banded birds recovered in years 2–4 was lower for the treated group than the control group. If experimental treatment with shot lowered survival in the first year after banding, we would expect the number of birds recovered in years 2–4, as a proportion of the number banded, to be lower for the treated group than for the control group and for M_{1a} therefore to be positive. Negative values of M_{1a} would occur if the proportion of banded birds recovered in years 2–4 happened to be lower for control than for treated birds. If there was no effect of treatment on survival we would expect approximately equal numbers of positive and negative M_{1a} values by chance. We counted the number of experiments for which M_{1a} was positive and negative and performed a Sign Test (Seigel & Castellan 1988) to assess the statistical significance of any excess of positive or negative values. The experiments involved dosing with three different numbers of shot (1, 2 and 4). We calculated the Pearson correlation coefficient for the M_{1a} for individual experiments ($n = 9$) in relation to the number of shot administered. We also calculated the least-squares regression slope of M_{1a} on the number of shot administered, constrained to pass through the origin.

Because the numbers of band recoveries from individual experiments were small, we also wished to calculate the additional mortality when a given number of shot were administered from data for all the

experiments with the same treatment, allowing for differences among experiments in sample sizes. We did this by calculating the binomial log-likelihood

$$R_{2–4T} \log_e(R_{2–4C} (1 – M_{1a})/N_C) + (N_T – R_{2–4T}) \log_e(1 – (R_{2–4C} (1 – M_{1a})/N_C)),$$

for each experiment and then summing them across all experiments for a given dose. The resulting sum depends only upon the observed values of $R_{2–4C}$, N_C, $R_{2–4T}$ and N_T for each experiment and the unknown parameter of interest M_{1a}, which we assumed was the same for all experiments with the same number of administered shot. We used an iterative bisection search algorithm (Kalbfleisch 1985), implemented in a BBCBASIC program, to find the value of M_{1a} at which the quantity above was maximised. We determined the 95% confidence limits for these estimates of M_{1a} by drawing 10,000 bootstrap samples of band recovery outcomes at 2–4 years of size N_c, with replacement, from the observed set of N_c outcomes for the control group in each experiment. The equivalent bootstrap samples were also obtained from the treatment group. We then used the procedure described above to obtain 10,000 bootstrap estimates of M_{1a} for a given number of shot administered. We ranked the bootstrap estimates and took the bounds of the central 9,500 of them to be the 95% confidence interval of M_{1a}.

We used the same approach to estimate the mean increment K by which M_{1a} increased for each additional shot administered. We calculated the log-likelihood

$$R_{2–4T} \log_e(R_{2–4C} (1 – S\ K)/N_C) + (N_T – R_{2–4T}) \log_e(1 – (R_{2–4C} (1 – S\ K)/N_C)),$$

for each experiment and then summed them across all nine experiments, where S is the number of shot administered to each treated bird in a given experiment. This sum depends only upon the observed values of S, $R_{2–4C}$, N_C, $R_{2–4T}$ and N_T for each experiment and the unknown parameter of interest K, which we assumed was the same for all experiments. This formulation of the log-likelihood as the same as that for the estimation above of M_{1a} for a single dose level, except that M_{1a} is taken to be directly proportional to the number of shot administered, *i.e.* $M_{1a} = S\ K$. We used the same procedure as that described above to estimate K and its bootstrap confidence limits.

Results

We made nine estimates of the total additional annual mortality M_{1a} in the year after treatment caused by administering various numbers of shotgun pellets from each of the experiments for which data are shown in Table 1. Eight of the M_{1a} values (89%) were greater than zero, which is the case when the treatment increased mortality. This number of positive values is significantly higher than that expected under a null hypothesis of no effect of the treatments (Sign Test: two-tailed $P = 0.04$). The total additional annual mortality M_{1a} values calculated from data for individual experiments were significantly positively correlated with the dose of shotgun pellets administered to each treated bird ($r = 0.686$, $t_7 = 2.50$, two-tailed $P = 0.041$).

Our estimate of the total additional annual mortality M_{1a} caused by administering one shotgun pellet used data from five

experiments in the three years 1949, 1950 and 1951. The estimate of the mean total additional annual mortality rate in the year after administration of one pellet was 0.108 (Table 2). The confidence interval of M_{1a} overlapped zero, but only a small proportion of the 10,000 bootstrap M_{1a} values (0.050) were of opposite sign (negative) to that expected, indicating that a mean value as positive as that observed is unlikely to have occurred by chance. The estimate of the total additional annual mortality caused by administering two and four shotgun pellets each used data from two experiments and were 0.471 and 0.555, respectively (Table 2). In both cases, the 95% confidence intervals of these estimates did not overlap zero.

The estimates of mean total additional mortality due to experimental administration of ingested lead gunshot pellets, shown in Table 2, indicate that the total additional mortality rate was dose-dependent and substantial. The maximum-likelihood estimate of K, the mean increase in M_{1a} for an increase in dose of one pellet, was 0.148 and its 95% confidence interval did not overlap zero (0.115–0.176). An alternative method for estimating this increment is to take the slope of the least-squares regression, constrained through the origin, of the nine values of M_{1a}, given in Table 1, calculated for individual experiments, on the number of pellets administered. The value of this regression slope was 0.141, which is similar to the maximum-likelihood estimate (0.148). Its 95% confidence interval did not overlap zero (0.070–0.212).

We plotted the mean M_{1a} values for each dose level from Table 2 against the number of shotgun pellets administered per dose (Fig. 1). Inspection of this plot suggests that there might be some levelling off in the relationship of M_{1a} with the number of pellets administered, but the 95% confidence limits of the estimates for each dose overlap the expected values from the simple model in which M_{1a} is assumed to increase in direct proportion to the number of pellets.

Discussion

We suggest that our new method for estimating total additional mortality due

Table 2. Maximum-likelihood estimates of the mean additional annual mortality caused by the treatment in the first year after banding M_{1a} calculated for all experiments with the same dose level (number of pellets administered). Data are from Table 1. The experiments whose results were used in each calculation are shown by the codes in the left-hand column. 95% bootstrap confidence limits for the M_{1a} estimates are given.

Experiments included	Number of shot	M_{1a} estimate	95% C.L.
A, B1, C1, D1, E1	1	0.108	–0.017–0.219
B2, C2	2	0.471	0.294–0.614
D2, E2	4	0.555	0.390–0.683

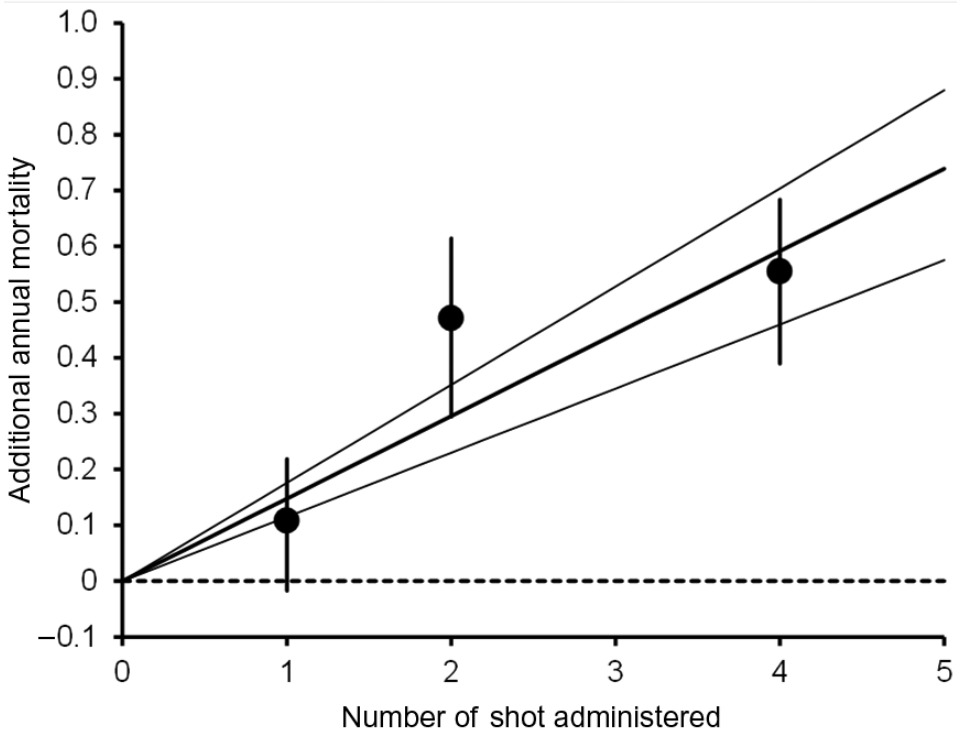

Figure 1. Mean **a**dditional mortality M_{1a} of wild male Mallard ducks in the year after treatment, attributable to experimental dosing with various numbers of lead gunshot pellets via the oesophagus (filled circles). Vertical lines represent 95% confidence limits. The horizontal dashed line represents no additional mortality (M_{1a} = 0). The thick line represents results from a simple model in which each additional pellet increases M_{1a} by a fixed amount (K = 0.148). The thin lines represent 95% confidence limits of expected values from that model.

to the treatments is likely to be more reliable than the simple calculations used by Bellrose (1959) because we did not use band recoveries from the first year. Avoiding the use of the first-year recoveries is important because their numbers are affected by the influence of the gunshot treatments on the number of actual deaths, the proportion of those deaths caused by shooting and the proportion of deaths which are reported, all of which are likely to differ between treatment and control groups. Our new method uses only recoveries of birds made 2–4 years after banding, for which we suggest that the annual mortality rate, the proportion of deaths caused by shooting and the proportion of deaths which are reported are unlikely to differ between treatment and control groups.

Our results agree with those of Bellrose (1959) in indicating a strong dose-response relationship between total additional annual mortality rate and the number of lead

shotgun pellets administered. The data available are insufficient to establish whether the additional mortality rate is directly proportional to the number of pellets administered. The results suggest the possibility that additional mortality might increase more slowly with the number of pellets as the dose is increased, but the simple model appears to give an adequate description of the limited quantity of experimental data available.

Many experimental studies in which captive ducks (mainly Mallards but also other species) were dosed with commercial lead shot were conducted in the decades after Bellrose's publication. These studies illustrated that, under controlled experimental conditions, many factors influence the toxicity of ingested lead shot. Both physical and chemical dietary components are important. The availability and type of grit is particularly significant and can influence both shot erosion and retention rates, whereas the chemical composition of the diet can mediate lead toxicity (see Eisler 1988; Pain 1996 for reviews). However, under similar experimental conditions, mortality increases with the number of shot ingested. For example, Finley & Dieter (1978a) found that the percentage mortality in each of four groups of 20 Mallards dosed with lead shot (73 mg; 205 mg; 292 mg; 1,025 mg Pb) was 35%, 50%, 90% and 100%, respectively after four weeks. In this study, the 205 mg dosage was one #4 shot and the 292 mg Pb dosage was two #6 shot. Mortality also occurred more rapidly with higher lead shot doses. In another experimental dosing study Longcore *et al.* (1974) found 19% mortality within an average of 20 days in 80 Mallards (40 males, 40 females) dosed with one #4 lead shot; other authors have found higher or lower levels of mortality with this dosage depending upon experimental conditions. Longcore *et al.* (1974) found 94% mortality in an average of 10–15 days in 80 Mallards (four groups containing 20 each adults and first year birds, males and females) dosed with eight #6 lead shot (*c.* 1000 mg). While annual mortality of wild birds from lead poisoning will undoubtedly vary with environmental conditions, it would nonetheless be expected to relate to the number of shot ingested in a broadly linear pattern.

We note that our method does not estimate the absolute proportion of banded birds that died because of experimental dosing. Instead, it estimates the proportion by which survival to one year after treatment was reduced by treatment, which is the annual additional mortality rate conditional on exposure for one year to the risk posed by the treatment. To obtain the absolute proportion of birds that die because of experimental dosing it would be necessary also to estimate the true annual survival rate for the control groups, which we do not think can be done with the data available, without making further assumptions about reporting rates. However, if we accept as correct the mean survival rate in the year after banding for the control groups in Bellrose's experiment, as calculated by Bellrose's method, we can compare the survival rates of treatment groups in the year after banding obtained by our new method with those calculated by Bellrose. Bellrose's method gives a mean survival rate in the year after banding across the five

control groups of 0.707. Applying our estimate of K, the increment in M_{1a} for each additional shot administered, to this survival rate gives expected annual survival rates for dosed birds in the year after banding of 0.602, 0.498 and 0.288, for birds dosed with 1, 2 and 4 shot, respectively. This compares with mean first-year survival rates for dosed birds calculated by Bellrose's method of 0.617, 0.412 and 0.322, for birds dosed with 1, 2 and 4 shot, respectively. These survival estimates made using Bellrose's method lead to additional mortality values M_{1a} of 0.127, 0.417 and 0.544 for birds given 1, 2 and 4 shot, respectively. These M_{1a} values are similar to those we obtained using our new method (0.108, 0.471 and 0.555, respectively). Hence, we conclude that our new approach and that of Bellrose give broadly similar values for the effects of dosing on annual survival. Therefore, the recent use of Bellrose's method by Mateo (2009), Pain *et al.* (2015), Green & Pain (2016) and Andreotti *et al.* (2018) in calculating numbers of deaths for European waterfowl is likely to have given reliable estimates.

Our study does not address the second question about Bellrose's results posed by the ECHA RAC (RAC & SEAC 2018): how much of the additional mortality of the dosed ducks in the experiments was attributable to their greater vulnerability to shooting, rather than to death caused by lead poisoning? Answering this question is not straightforward because it is necessary to make and justify several assumptions about the reporting rates that apply to the ordinary bands and reward bands used in Bellrose's experiments. These rates cannot be estimated directly from Bellrose's data, so values from other studies, such as that of Henny & Burnham (1976), would need to be used. We are preparing to address this problem in a follow-up paper.

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Appendix 1. An explanation of the new calculations of total additional mortality caused by experimental dosing with lead, based upon the Bellrose (1959) duck band recovery data.

Notation

M_1 = *per capita* death rate in year 1 of the 4-year period for control (untreated) ducks.

M_1* = *per capita* death rate in year 1 of the 4-year period for experimentally dosed ducks.

M_{1a} = total additional per capita death rate in year 1 of the 4-year period caused by experimental dosing, conditional on exposure to the risk of death caused by dosing for one year.

M_2 = *per capita* death rate in year 2 of the 4-year period for control and dosed ducks (assumed not to differ between control and treatment after year 1).

M_3 = *per capita* death rate in year 3 of the 4-year period for control and dosed ducks (assumed not to differ between control and treatment after year 1).

M_4 = *per capita* death rate in year 4 of the 4-year period for control and dosed ducks (assumed not to differ between control and treatment after year 1).

L_1 = the proportion of those control ducks that die from any cause in year 1 that are reported to the authorities.

L_1* = the proportion of those dosed ducks that die from any cause in year 1 that are reported to the authorities.

L_2 = the proportion of those control and dosed ducks that die from any cause in year 2 that are reported to the authorities (assumed not to differ between control and treatment after year 1).

L_3 = the proportion of those control and dosed ducks that die from any cause in year 3 that are reported to the authorities (assumed not to differ between control and treatment after year 1).

L_4 = the proportion of those control and dosed ducks that die from any cause in year 4 that are reported to the authorities (assumed not to differ between control and treatment after year 1).

N_C = number of ducks banded and released in the control group.

N_T = number of ducks banded and released in the dosed group.

The proportion of dosed birds that survive year 1 is $(1 - M_1^*)$ which is equal to

$$(1 - M_1) \times (1 - M_{1a})$$

i.e. the product of the first-year survival of untreated control ducks and one minus the additional *per capita* death rate caused by the experimental treatment. Note that × means multiplied by in the above expression and all later expressions.

Expected numbers of band recoveries in each of years 1 to 4.

CONTROL GROUP	DOSED GROUP
Expected recoveries in Year 1	
$R_{1C} = N_C \times M_1 \times L_1$	$R_{1T} = N_T \times M_1{*} \times L_1{*}$
Expected recoveries in Year 2	
$R_{2C} = N_C \times (1 - M_1) \times M_2 \times L_2$	$R_{2T} = N_T \times (1 - M_1{*}) \times M_2 \times L_2$
Expected recoveries in Year 3	
$R_{3C} = N_C \times (1 - M_1) \times (1 - M_2) \times M_3 \times L_3$	$R_{3T} = N_T \times (1 - M_1{*}) \times (1 - M_2) \times M_3 \times L_3$
Expected recoveries in Year 4	
$R_{4C} = N_C \times (1 - M_1) \times (1 - M_2) \times (1 - M_3) \times M_4 \times L_4$	$R_{4T} = N_T \times (1 - M_1{*}) \times (1 - M_2) \times (1 - M_3) \times M_4 \times L_4$

Expected numbers of band recoveries in years 2 to 4 combined (the sum of expressions in rows 2–4 above).

CONTROL GROUP

$$R_{2\text{–}4C} = N_C \times (1 - M_1) \times [(M_2 \times L_2) + ((1 - M_2) \times M_3 \times L_3) + ((1 - M_2) \times (1 - M_3) \times M_4 \times L_4)]$$

DOSED GROUP

$$R_{2\text{–}4T} = N_T \times (1 - M_1{*}) \times [(M_2 \times L_2) + ((1 - M_2) \times M_3 \times L_3) + ((1 - M_2) \times (1 - M_3) \times M_4 \times L_4)]$$

Divide the expected number of band recoveries in dosed group in years 2–4 by the expected number of band recoveries in dosed group in years 2–4 to give a ratio Q which is the expected ratio of the number of band recoveries in years 2–4 for dosed birds relative to the number of band recoveries in years 2–4 for control birds.

$$Q = R_{2\text{–}4T} / R_{2\text{–}4C} = \frac{N_T \times (1 - M_1{*}) \times [(M_2 \times L_2) + ((1 - M_2) \times M_3 \times L_3) + ((1 - M_2) \times (1 - M_3) \times M_4 \times L_4)]}{N_C \times (1 - M_1) \times [(M_2 \times L_2) + ((1 - M_2) \times M_3 \times L_3) + ((1 - M_2) \times (1 - M_3) \times M_4 \times L_4)]}$$

Cancel out factors that occur on both the top and bottom of the expression (shown in square brackets).

$$Q = \frac{N_T \times (1 - M_1{*}) \times [(M_2 \times L_2) + ((1 - M_2) \times M_3 \times L_3) + ((1 - M_2) \times (1 - M_3) \times M_4 \times L_4)]}{N_C \times (1 - M_1) \times [(M_2 \times L_2) + ((1 - M_2) \times M_3 \times L_3) + ((1 - M_2) \times (1 - M_3) \times M_4 \times L_4)]}$$

Which leaves

$$Q = (N_T \times (1 - M_1{*}))/(N_C \times (1 - M_1))$$

Rearranging gives

$$(1 - M_1{*})/(1 - M_1) = N_C \times Q/N_T$$

Remember that

$$(1 - M_1^*) = (1 - M_1) \times (1 - M_{1a})$$

Substitution gives

$$[(1 - M_1) \times (1 - M_{1a})]/(1 - M_1) = (N_C \times Q)/N_T$$

Cancel out factors that occur on both the top and bottom of the left-hand part of the expression

$$[(1 - M_1) \times (1 - M_{1a})]/(1 - M_1) = (N_C \times Q)/N_T$$

to give

$$(1 - M_{1a}) = (N_C \times Q)/N_T$$

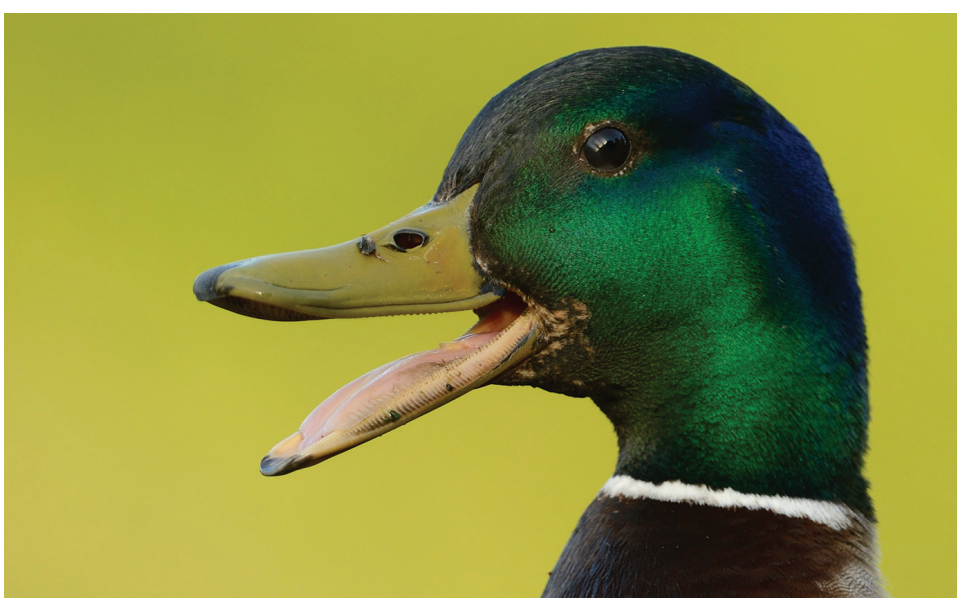
Rearrange to give the additional per capita total annual mortality rate caused by experimental dosing

$$M_{1a} = 1–[(N_C \times Q)/N_T].$$

Substituting $R_{2–4T}/R_{2–4C}$ for Q gives

$$M_{1a} = 1–[(R_{2–4T} \times N_C)/(R_{2–4C} \times N_T)].$$

Hence, an estimate of the additional *per capita* death rate in the first year after release attributable to the experimental treatment can be obtained from the band recovery totals for years 2–4 after release and the numbers of ducks banded. Note that the numbers of recoveries in the first year after banding are not used in this calculation.



Photograph: Male Mallard, by Ben Andrew/rspb.images.com.