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Anticoagulant rodenticides in red kites (*Milvus milvus*) in Britain

ABSTRACT: In the first GB-wide assessment of second generation anticoagulant rodenticide (SGAR) exposure and poisoning in red kites (*Milvus milvus*), 29/32 birds found dead in 2015 had detectable liver residues of at least two SGARs. Summed SGAR residues in kites with detected residues ranged between 50 and 1266 ng/g wet weight. Ten kites (31% of the sample) were diagnosed as poisoned by SGARs.

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Introduction

Second generation anticoagulant rodenticides (SGARs) can be toxic to all mammals and birds. Studies around the world have shown widespread exposure in a diverse range of species.

Red kites (*Milvus milvus*) may be particularly at risk as they scavenge dead rats, a target species for rodent control. In Britain, SGAR exposure in kites is monitored by multiple independent laboratories. The resultant data has not previously been collated, hampering our ability to gain a national overview.

Recently, the WILDCOMS network (www.wildcoms.org.uk/) has brought together these disparate data sources. We report the first national scale set of exposure measurements in red kites which were conducted for birds found dead in 2015. .

Exposure of red kites to SGARs

Carcasses were typically found by members of the public, sent to an investigating laboratory, necropsied and exposure was determined from the presence of liver SGARs as determined by Liquid Chromatography Mass Spectrometry.

Twenty-six red kites from England & Wales were analysed. All had detectable liver residues of difenacoum and brodifacoum; most also contained bromadiolone. Difethialone was less frequently detected and flocoumafen was not detected at all. Sum SGAR concentrations ranged from 50 to 1266 ng/g wet wt. (Fig. 1).

Six red kites from Scotland were also analysed. Three had liver residues of at least two SGARs (bromadiolone and difenacoum); brodifacoum was also detected in one of these kites. Sum liver SGARs ranged from non-detected to 280 ng/g wet wt. (Fig. 1).

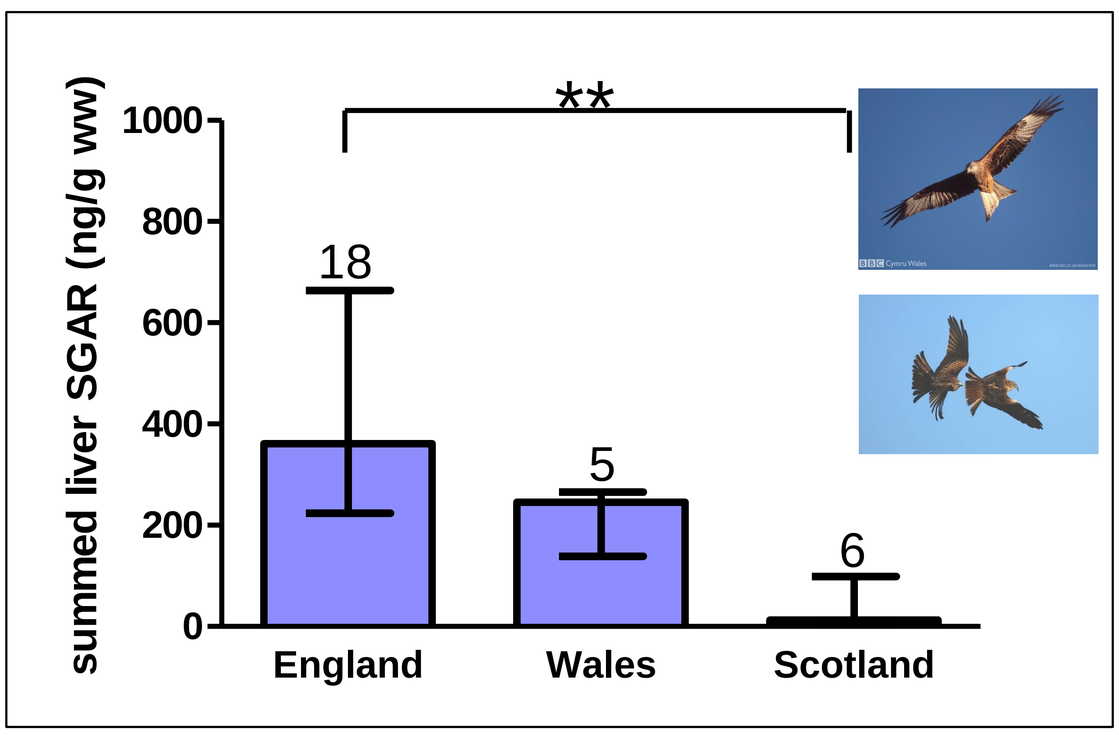

Figure 1. Median (± interquartile range) summed SGAR liver residue in red kites found dead in 2015. Numbers above columns indicate sample sizes. Stars indicate significant (P<0.01) difference between columns (Kruskal-Wallis test and *post-hoc* Dunn's multiple comparison tests).

Although the data are limited, they suggest that exposure of red kites in 2015 may have been less marked in Scotland than in England or probably Wales. This is consistent with regional differences in SGAR exposure that have been previously observed in barn owls *Tyto alba*[1].

SGAR poisoning in red kites

Post-mortems indicated that 9 (35%) of the kites from England & Wales and one of the six kites from Scotland had internal haemorrhaging that was not associated with detectable trauma, and detectable liver SGAR residues. It was considered probable that SGARs were a contributory cause of death in these birds.

Most of the kites diagnosed as SGARs-poisoned had elevated sum SGAR liver concentrations (Fig. 2).

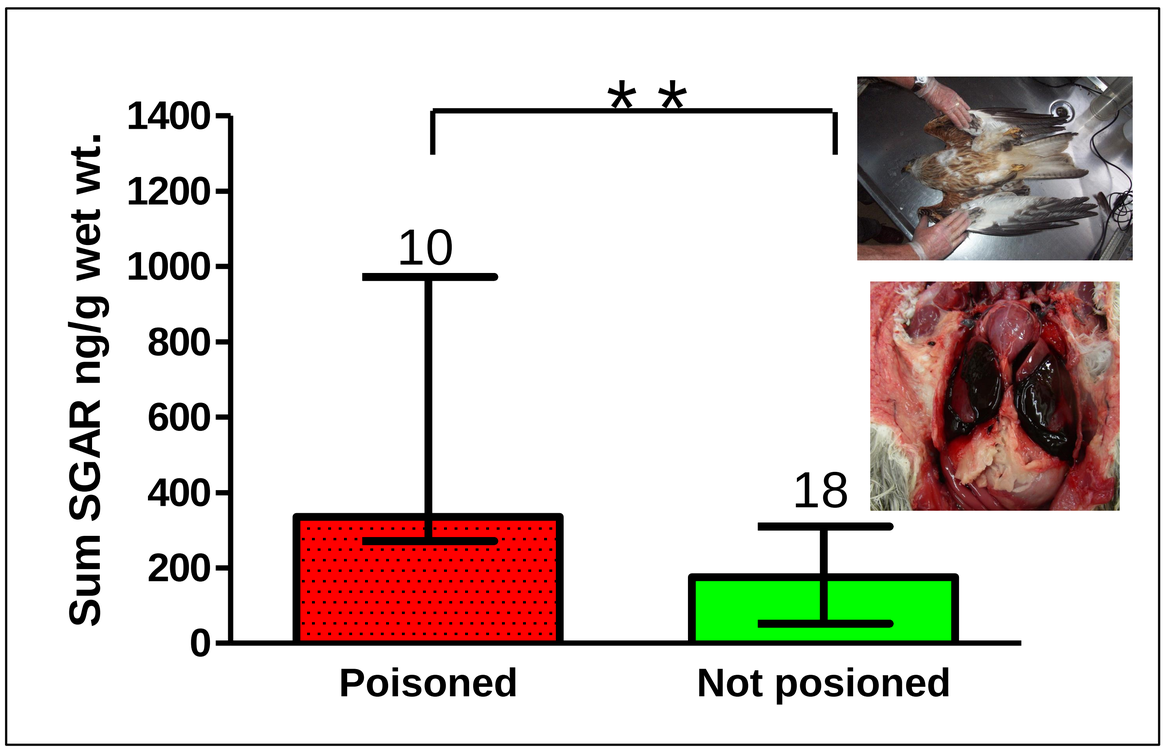

Figure 2. Median (± interquartile range) summed SGAR liver residue in red kites diagnosed as poisoned and not poisoned by SGARs. Numbers above columns indicate sample sizes. Stars indicate significant (P<0.01) difference between columns (Mann-Whitney U test).

Conclusions

- About one third of red kites from England & Wales analysed were considered likely to have been poisoned by SGARs.
- This already high incidence of poisoning does not include any other type of poisoning that may occur (illegal use of other pesticides, lead, etc)
- Poisoning thus appears to be a significant mortality factor in red kite populations
- Despite this, the red kite population has greatly expanded in Britain as birds recolonise former haunts.
- The extent to which exposure to SGARs may affect future population growth merits further investigation

[1]Shore, R.F., et al., 2015. Monitoring rodenticide residues in wildlife. In *Rodent pests and their Control: 2nd edition*. (Eds, A.P. Buckle and R.H. Smith), CAB International, Wallingford Oxfordshire, , pp. 346-365. ISBN: 978 1 84593 817 8.

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