Bull. Environm. Contam. Toxicol. 28, 225–229 (1982)

Brain Cholinesterase Inhibition in Forest Passerines Exposed to Experimental Aminocarb Spraying

D. G. Busby, P. A. Pearce, and N. R. Garrity

Canadian Wildlife Service, P.O. Box 400, Fredericton, New Brunswick, E3B 4Z9 Canada

Continued spruce budworm (Choristoneura fumiferana) infestation of the fir-spruce forest of eastern Canada has been marked by a constant search for control strategies that are effective, environmentally acceptable, and that pose no threat to human health. In New Brunswick, forest protection has relied heavily on the use of chemical insecticides for almost three decades, a practice for which there is apparently no viable alternative at present. Testing of the efficacy and safety of new chemicals, old chemicals in new formulations, and of developments in spraying technology have been important elements in the province's approach to management of the budworm problem.

This study was carried out in the context of a series of spray trials of a new formulation of aminocarb (4-(dimethylamino)-3-methylphenol methylcarbamate) known as Matacil® 180 Flowable (180F), conducted by the Forest Pest Management Institute, Canadian Forestry Service, primarily to determine efficacy against spruce budworm. The trials were of particular interest to the Canadian Wildlife Service since there is little published information on the effects of aminocarb on brain cholinesterase (ChE) activity in forest songbirds and the logistical problems normally encountered in monitoring bird responses under operational spraying conditions were minimized by the relatively small scale of the project.

MATERIALS AND METHODS

Spray plots were located in predominantly fir-spruce forest about 25 km west of Bathurst, in northern New Brunswick. Each was 50 ha (1.0 km x 0.5 km) in area, and was sprayed by a Cessna 188 Ag-Truck fitted with four Micronair AU3000 rotary atomizers.

The two plots of interest to us were both sprayed at a stated emission dosage of 70 g/ha of active ingredient. By volume, the formulation consisted of 25.93% Matacil® 180F, 1.27% Atlox® (emulsifier), 72.27% water (carrier) and 0.53% Rhodamine Red dye. The emulsion was emitted at a rate of 1.5 L/ha. Droplet VMD and NMD at first

0007-4861/82/0028-0225 $01.00

application were 36 and 28 μm, respectively, and at second application 33 and 23 μm, respectively. Both plots were sprayed twice, first at 1945-2015 on June 12 and again at 0622-0645 six days later.

Birds were collected in the plots with a .410 shotgun 2-48 h after spraying. Birds were taken as controls from areas well outside the target zone during the treatment period. Individuals of the following five most abundant passerine species were collected: Tennessee Warbler (Vermivora peregrina), Yellow-rumped Warbler (Dendroica coronata), Blackburnian Warbler (D. fusca), Bay-breasted Warbler (D. castanea) and American Redstart (Setophaga ruticilla).

Brains were immediately removed, placed in polyethylene serum vials and stored in liquid nitrogen (-196°C) until assayed, a maximum of 12 days later. ChE activity was determined using the technique of ELLMAN et al. (1961), as modified by HILL and FLEMING (in press).

RESULTS AND DISCUSSION

None of the species sampled showed depression of ChE after spraying (TABLE 1). There was no significant difference (t-test) between ChE activity in birds exposed to a morning spray and in those exposed to an evening spray application, nor in birds collected within the first 24 h post-spray and in those collected 24-48 h post-spray. Only four birds from spray plots exhibited ChE activity of more than 20% below the mean control value. They were a Tennessee Warbler, a Bay-breasted Warbler and two American Redstarts, in which ChE depression was 26.6, 26.2, 35.2 and 37.5% respectively. No birds had ChE activity greater than 50% below that of the mean control value. This is in contrast to the results of a similar study (BUSBY et al. 1981) of the impact of the organophosphate insecticide fenitrothion on forest songbird brain ChE activity.

Measurement of ChE activity has recently been widely adopted as a useful tool in determining exposure of birds to organophosphate and carbamate insecticides (e.g. WHITE et al. 1979, ZINKL et al. 1980, BUSBY et al. 1981). The suggestion by LUDKE et al. (1975) that 20% brain ChE inhibition is indicative of exposure and that 50% inhibition is sufficient for diagnosis of death has gained general acceptance (DEWEESE et al. 1979, ZINKL et al. 1980, BUSBY et al. 1981). Under some circumstances, however, ChE inhibition can exceed 50% without mortality occurring (LUDKE et al. 1975, ZINKL et al. 1979, ZINKL et al. 1980, FLEMING 1981).

TABLE 1

Brain ChE activity (mU/mg brain) in warblers (Parulidae) from sprayed and unsprayed forest.

Species	Sprayed Forest[1] n	mean±S.D.	range	Unsprayed Forest n	mean±S.D.	range
Tennessee Warbler	11	32.3±4.2[2]	22.1-37.5	11	30.1±2.6	25.0-33.9
Yellow-rumped Warbler	13	32.9±4.1[2]	25.5-39.8	5	30.9±3.1	29.0-33.4
Blackburnian Warbler	12	32.8±5.7[2]	27.7-49.6	10	32.1±2.2	28.1-35.4
Bay-breasted Warbler	48	32.7±3.4[2]	22.5-39.2	11	30.5±3.1	26.3-37.5
American Redstart	7	30.0±6.6[2]	21.3-39.8	8	34.1±4.6	27.2-42.7

[1]This category includes birds collected after both the first and second sprays from the same plot. Therefore some birds experienced both sprays. There was, however, no statistical difference (t-test) in ChE activities of birds collected after spray 1 and spray 2.

[2]No significant decrease (t-test) in brain ChE activity in birds from sprayed forest.

The effects of ChE inhibition on the reproduction of songbirds are not well understood. Most studies of the influence of insecticide spraying have concentrated on pre- and post-spray singing surveys. However, BEDNAREK and DAVIDSON (1967) and MOULDING (1976) alluded to changes in reproductive biology of birds from forests sprayed with the carbamate insecticide carbaryl. Although in those studies nesting failure did not follow, any interference in the nesting cycle would be regarded as potentially detrimental, especially in years when other unfavorable factors such as adverse weather conditions had already predisposed the birds to failure.

The criteria of LUDKE et al. (1975) indicate the unlikelihood that any of the birds we collected were in danger of death through poisoning. There was no evidence of aberrant bird behavior that could be attributed to the spray. Only the four birds with more than 20% inhibition suggest exposure to the insecticide and potential minor sub-lethal effects. These findings support the conclusions of PEARCE and GARRITY (1981), GERMAIN and TINGLEY (1980), GERMAIN and MORIN (1979) and BUCKNER et al. (1976) that aminocarb spraying at similar dosages in eastern Canadian forests, but in different formulations, caused little or no detrimental effect on the singing and territorial behavior of songbirds. PETERSON (1976) found no significant brain ChE inhibition in forest passerines in Maine in areas sprayed with aminocarb at a dosage of 168 g/ha.

We conclude that aerial spraying of Matacil® 180F at 2 x 70 g/ha over small plots had no significant inhibitory effect on brain ChE in songbirds exposed in the forest canopy. Further examination for potential hazard to birds of Matacil® 180F in other formulations and in the context of larger spray plots would be prudent before translation to full-scale operational use.

ACKNOWLEDGEMENTS

We wish to express our gratitude to B.L. Cadogan, B.F. Zylstra and C. Nystrom of the Forest Pest Management Institute, Canadian Forestry Service, for their interest in and accommodation of our study. K.M.S. Sundaram, of that agency, kindly made available information on spray droplet size. V.L. Edge provided willing assistance throughout the project.

REFERENCES

BEDNAREK, R. and C.S. DAVIDSON: Bird Banding 38, 66 (1967).
BUCKNER, C.H., B.B. MCLEOD, and R.G. LIDSTONE: Can. For. Serv. Chem. Control Res. Inst. Rep. CC-X-135. 141p (1976).

BUSBY, D.G., P.A. PEARCE, and N.R. GARRITY: Bull. Environ. Contam. Toxicol. 26, 401 (1981).
DEWEESE, L.R., C.J. HENNY, F.L. FLOYD, K.A. BOBAL, and A.W. SCHULTZ: U.S. Fish Wildl. Serv. Spec. Sci. Rep. Wildl. 224. 29p (1979).
ELLMAN, G.L., K.D. COURTNEY, V. ANDRES, JR., and R.M. FEATHERSTONE: Biochem. Pharmacol. 1, 88 (1961).
FLEMING, W.J.: Arch. Environ. Contam. Toxicol. 10, 215 (1981).
GERMAIN, P. and G. MORIN: Avifauna Ltée Rep. to For. Prot. Ltd. and N.B. Dept. Natur. Res. 31p (1979).
GERMAIN, P. and S. TINGLEY: Avifauna Ltée Rep. to For. Prot. Ltd. 38p (1980).
HILL, E.F. and W.J. FLEMING: Anticholinesterase Poisoning in Birds: Field Monitoring and Diagnosis of Acute Poisoning. Proc. First Ann. Meeting Soc. Environ. Toxicol. Chem. (in press).
LUDKE, J.L., E.F. HILL, and M.P. DIETER: Arch. Environ. Contam. Toxicol. 3, 1 (1975).
MOULDING, J.D.: Auk 93, 692 (1976).
PEARCE, P.A. and N.R. GARRITY: Can. Wildl. Serv. Prog. Note No. 121. 19p (1981).
PETERSON, J.W.: Rep. For. Insect Dis. Manage. Northeastern Area. State Priv. For. U.S. For. Serv. 92p (1976).
WHITE, D.H., K.A. KING, C.A. MITCHELL, E.F. HILL, and T.G. LAMONT: Bull. Environ. Contam. Toxicol. 23, 281 (1979).
ZINKL, J.G., C.J. HENNY, and P.J. SHEA: In Animals as Monitors of Environmental Pollutants. National Academy of Sciences, Washington D.C. 356p (1979).
ZINKL, J.G., R.B. ROBERTS, C.J. HENNY, and D.J. LENHART: Bull. Environ. Contam. Toxicol. 24, 676 (1980).