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RISK FACTORS ASSOCIATED WITH CAPTURE-RELATED DEATH IN EASTERN WILD TURKEY HENS

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ABSTRACT: Capture-related mortality has been a notable risk in the handling of eastern wild turkey (*Meleagris gallopavo silvestris*). Our objective was to evaluate how environmental factors influence risk and identify physiological correlates that could be used to identify susceptible birds. During winter (January–March) 1995–97, 130 eastern wild turkey hens were captured in southeastern Oklahoma and radiocollared. Of those, 20 hens died ≤14 days of capture. Serum creatine kinase activity (CK; $P < 0.01$), body temperature ($P < 0.01$), processing time ($P = 0.02$), and ambient temperature ($P < 0.01$) showed a positive relationship with mortality that occurred within 14 days of capture. Plasma corticosterone concentration ($P = 0.08$) and relative humidity ($P < 0.01$) showed a negative relationship with mortalities that occurred within 14 days post-capture. Stepwise logistic regression selected CK activity, relative humidity, and ambient temperature as the best predictors of mortality within 14 days post-capture. Our data suggest that susceptible individuals may be identified from CK activity and that capture-related mortality may be minimized by establishing guidelines of when to curtail capture operations based on various weather conditions.

Key words: Aspartate aminotransferase, capture mortality, capture myopathy, creatine kinase, *Meleagris gallopavo,* plasma corticosterone, relative humidity, stress, temperature, wild turkey.

INTRODUCTION

Wild turkeys (*Meleagris gallopavo*) are routinely captured for trap and transplant programs and research purposes. With many capture routines, complications may occur during capture, which may lead to losses from capture myopathy (CM). Losses may occur during capture, transport, or after release, thereby influencing short-term survival. In some instances, mortalities that occur within 1 to 2 wk after release go undetected, ultimately influencing the success of some trap and transplant programs. In cases where mortalities are known, deaths of birds within 1 to 2 wk of capture may be related to capture (Campo et al., 1984; Kurzejeski et al., 1987; Godwin et al., 1991; Palmer et al., 1993; Chamberlain et al., 1996; Johnson et al., 1996; Miller et al., 1996), although the direct relationship between capture and death are often unknown.

Capture myopathy has been studied widely in mammals (Chalmers and Barrett, 1982; Beringer et al., 1996); however, relatively few studies have been conducted with birds (Bollinger et al., 1989; Dabbert and Powell, 1993), although capture myopathy has been documented in several avian species (Young, 1967; Windingstad et al., 1983; Carpenter et al., 1991), including wild turkeys (Spraker et al., 1987). Capture myopathy is a condition resulting from isotonic muscle contraction during restraint and handling that causes reduced blood flow to affected muscles (Spraker, 1982). It can lead to anaerobic metabolism and buildup of lactic acid within muscles that may result in lactic acidosis and cellular death. With increased cell permeability and cell lysis, increases in enzyme activity of creatine kinase (CK) and aspartate aminotransferase (AST) are often observed in serum, relative to skeletal and cardiac muscle necrosis (Chalmers and Barrett, 1982; Bollinger et al., 1989; Dab-

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bert and Powell, 1993), with the activity of CK being the most sensitive indicator of muscle damage in mammals (Chalmers and Barrett, 1982) and birds (Franson et al., 1985; Bollinger et al., 1989; Dabbert and Powell, 1993). However, this relationship has not been documented in wild turkeys.

Spraker et al. (1987) found that only 13 (22%) of 60 wild turkeys, captured and necropsied between 1980 and 1983, showed gross lesions characteristic of capture myopathy. However, upon microscopic examination, 30% of the birds had muscle lesions, with 96% of the 46 examined birds showing signs of microscopic skeletal muscle lesions. Of the birds with gross lesions, 73% were juveniles and 17% were adults, suggesting that juvenile turkeys may be more susceptible to capture myopathy. Spraker et al. (1987) noted that although many of those birds may have recovered following release, some may have been more susceptible to predation for several weeks following release.

Our objective was to identify physiological and climatic factors that may help to predict the incidence of capture-related death in eastern wild turkey hens. We hypothesized that enzyme activity of CK and AST, and plasma corticosterone concentrations in the blood of turkeys at the time of capture would be predictive of risk of mortality within 14 days of capture.

MATERIALS AND METHODS

The study was conducted on the Pushmataha Wildlife Management Area (PWMA, Pushmataha County, Oklahoma, USA; 34°32′N, 95°21′W) located about 6 km south of Clayton, Oklahoma. The study area was in mountainous terrain along the western edge of the Ouachita Highland Province, and habitat types were similar to those throughout most of southeastern Oklahoma (Duck and Fletcher, 1945). A detailed description of the study area was given by Masters (1991).

Wild turkey hens were captured using rocket nets at pre-baited sites during winter (January–March) 1995–97. On all but three trapping occasions, hens were placed in cardboard boxes and placed in the shade until they could be processed. When handling birds, a sock was placed over the head to calm the bird. Captured hens were fitted with a 90 g radio transmitter with a mortality sensor (3 to 4 hr delay; Lotek Engineering Inc., Ontario, Canada) that was attached by a backpack harness. Individually numbered leg-bands were attached to each bird. Turkeys were classified as juvenile or adult (Pelham and Dickson, 1992). Body mass (nearest 0.1 kg), body temperature (nearest 0.1 C), handling time (min), and ambient temperature (nearest 0.1 C) were recorded subsequent to release. We defined handling time as the elapsed time between firing of the net and release of the bird. Relative humidity at the time of capture was obtained from the Mesonet weather station (Oklahoma Climatological Survey, Norman, Oklahoma, USA) located about 9 km northeast of the study area (34°39′20″N, 95°19′33″W) where weather measurements were taken at 15 min intervals.

Blood samples were taken from the cutaneous ulnar vein using a 20 gauge needle and vacutainer (Becton Dickinson Vacutainer Systems, Rutherford, New Jersey, USA). Blood was collected into a 3-ml evacuated EDTA-K collection vial (Sherwood Medical, St. Louis, Missouri, USA) and a 10-ml evacuated serum-separating tube (Becton Dickinson Vacutainer Systems, Rutherford, New Jersey, USA). The birds were released at the capture site. Serum-separating tubes were centrifuged for 10 min at 1,000 rpm within 5 hr of capture, and serum was poured off into separate aliquots and stored at −80 C for future analysis. Serum samples showing marked signs of hemolysis were excluded from analysis. Activity of CK and AST in serum were determined by Vet Pro Laboratories (Tulsa, Oklahoma, USA) using a Technicon RA 1000 chemistry analyzer (Bayer Diagnostics, Tarrytown, New York, USA), and plasma corticosterone concentrations were determined by Texas Veterinary Medical Diagnostic Laboratory (College Station, Texas, USA).

Hens were monitored daily following their release using a hand-held 3-element yagi antennae and portable scanning receiver (Lotek Engineering Inc., Ontario, Canada). Upon receiving a mortality signal, cause of death was determined as soon as possible (usually <6 hr). Hens dying ≤ 14 days of capture were assumed to have died from capture-related stressors.

Comparisons were made to determine if there was differential susceptibility between adults and subadults to capture-related mortality using a likelihood ratio chi-square test (PROC UNIVARIATE; SAS Institute, Inc., 1990). Because of non-normal distributions, serum activity of CK and AST between hens that died ≤ 14 days of capture to those surviving >14 days of capture were compared using a

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TABLE 1. Differences in selected factors associated with mortality and survival ≤14 days of capture for eastern wild turkey hens at Pushmataha Wildlife Management Area, Oklahoma, 1995–97. Differences were tested using Wilcoxon rank sum tests (CK and AST) and analysis of variance (all other variables).

Variable	Capture deaths			Survivors			*P*-value
	n	$\bar{x}$	SE	*n*	$\bar{x}$	SE	
AST (IU/L)[a]	17	316	13	102	294	12	0.15
CK (IU/L)[b]	17	4,807	951	102	1,986	114	<0.01
Corticosterone (ng/ml)	16	135.8	14.4	99	161.6	6.0	0.11
Body temperature (C)	18	42.4	0.4	95	41.0	0.2	<0.01
Handling time (min)[c]	20	98.1	6.3	102	72.2	3.7	<0.01
Ambient temperature (C)	20	6.9	1.7	107	0.8	0.7	<0.01
Relative humidity	20	46.9	3.7	107	65.6	1.9	<0.01

[a] Aspartate aminotransferase.
[b] Creatine kinase.
[c] Time elapsed from when the net was fired until the bird was released.

Wilcoxon rank sum test (PROC NPARIWAY; SAS Institute, Inc., 1990). Differences in plasma corticosterone concentrations, processing time, body temperature, ambient temperature, and relative humidity between groups were tested using analysis of variance (PROC GLM; SAS Institute, Inc., 1990). Univariate logistic regression procedures (PROC LOGISTIC; SAS Institute, Inc., 1990) were used to determine if selected variables were significant predictors of the probability of mortality ≤14 days of capture (P_m). We then developed a multiple logistic regression model using stepwise forward selection of variables to determine the model that best predicted mortality. Variables were allowed to enter the model when the $\log_e$ likelihood was deemed appropriate ($P < 0.15$). Because observations with missing values were omitted by logistic regression procedures, initial analysis included all variables, and then variables that were not significant in the model and contained missing values were omitted and the analysis was repeated until the maximum number of observations was obtained.

RESULTS

During the three years of study, 130 hens were captured (111 adult, 19 subadult). Of the 130 hens captured, 20 (15%) died ≤14 days of capture (16 adult, 14%, and four subadult, 21%). Susceptibility of adults to mortality ≤14 days of capture did not differ from subadults ($\chi^2 = 0.511$, df = 1, $P = 0.48$); therefore, ages were pooled for further analyses. Of the hens that died ≤14 days of capture, mean number of days survived was 2.80 ± 0.62 (SE) and ranged from 0 to 9 days. Enzyme activity of CK was significantly higher for hens dying ≤14 days of capture compared with those surviving >14 days of capture ($P < 0.01$; Table 1). Enzyme activity of AST ($P = 0.15$) and plasma corticosterone concentration ($P = 0.11$) did not differ between groups (Table 1). Handling time was longer ($F = 8.78$; df = 1, 120; $P < 0.01$), body temperature greater ($F = 9.57$; df = 1, 111; $P < 0.01$), ambient temperature greater ($F = 12.69$; df = 1, 125; $P < 0.01$), and relative humidity lower ($F = 15.92$; df = 1, 125; $P < 0.01$) for hens dying ≤14 days of capture (Table 1).

Univariate logistic regression indicated a positive relationship ($\chi^2 = 13.02$, df = 1, $P < 0.01$) between CK activity and $P_m \leq 14$ days of capture (Fig. 1). No relationship was found between AST activity and P_m ($\chi^2 = 0.47$, df = 1, $P = 0.49$). Concentrations of plasma corticosterone demonstrated a weak negative relationship with P_m ($\chi^2 = 3.02$, df = 1, $P = 0.08$; Fig. 1). Body temperature ($\chi^2 = 12.95$, df = 1, $P < 0.01$), processing time ($\chi^2 = 5.73$, df = 1, $P = 0.02$), and ambient temperature (χ^2 10.55, df = 1, $P < 0.01$) were related positively to P_m (Fig. 1). Relative humidity demonstrated a strong negative relationship with P_m ($\chi^2 = 10.80$, df = 1, $P < 0.01$; Fig. 1).

Stepwise logistic regression selected CK activity ($\chi^2 = 3.32$, $P = 0.07$), relative humidity ($\chi^2 = 4.85$, $P = 0.03$), and ambient

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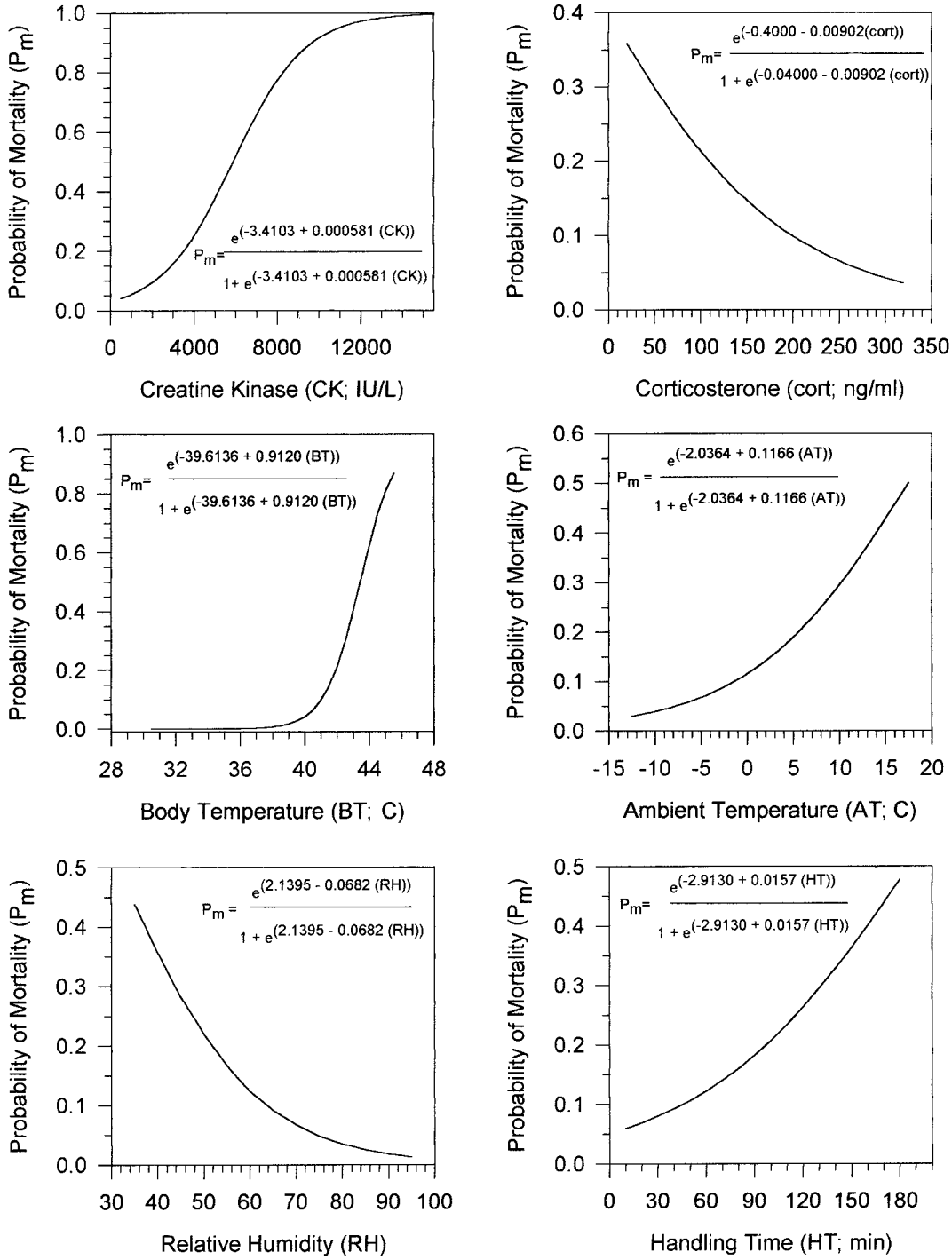


FIGURE 1. Relationships between creatine kinase (CK), plasma corticosterone (cort), body temperature (BT), handling time (PT), ambient temperature (AT), and relative humidity (RH) as related to mortality ≤14 days post-capture for eastern wild turkey hens at Pushmataha Wildlife Management Area Oklahoma, 1995–97. Values calculated using predictive equations derived from univariate logistic regression models ($P \leq 0.08$).

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TABLE 2. Stepwise logistic regression model[a] accuracy for predicting mortalities ≤14 days of capture for eastern wild turkey hens at Pushmataha Wildlife Management Area, Oklahoma, 1995–97. Variables included in the model were relative humidity, ambient temperature, body temperature, and processing time. Accuracies were determined when the probability of mortality (P_m) was ≥0.5 and ≤0.4. Creatine kinase (CK) was excluded from analysis because of correlations with other variables.

	$P_m > 0.5$ predicts mortality		$P_m > 0.4$ predicts mortality	
Outcome	Mortalities	Survivors	Mortalities	Survivors
Actual observation	18	86	18	86
Predicted				
Mortalities	11	1	12	2
Survivors	7	100	6	99
Sensitivity (%)[b]	61.1		66.7	
Specificity (%)[c]	97.7		97.7	

[a] $P_m = e^a/1 + e^a$, where a = [−17.3651 + 0.4241 (body temperature) + 0.0659 (ambient temperature) − 0.0577 (relative humidity) + 0.0166 (handling time)].
[b] Proportion of mortalities ≤14 days of capture that are predicted to be mortalities.
[c] Proportion of hens surviving >14 days that are predicted to survive.

temperature ($\chi^2 = 3.50$, $P = 0.06$) as the best predictors of $P_m \leq 14$ days of capture. The selected model ($P_m = e^a/1 + e^a$, where a = [−0.2466 + 0.000342 (CK) + 0.0870 (ambient temperature) − 0.0464 (relative humidity)]) predicted 52.9% of the deaths and 97.8% of the survivors correctly at a $P_m \geq 0.5$, with the sensitivity of the model increasing to 58.8% at a $P_m \geq 0.4$. Based on the predictive equation, P_m of hens dying ≤14 days of capture ranged from 0.019 to 0.988 ($\bar{x} = 0.487 \pm 0.086$; $n = 17$), and that of those surviving ranged from 0.01 to 0.65 ($\bar{x} = 0.097 \pm 0.010$; $n = 95$). Because of a strong correlation between CK activity and other capture-related variables, we removed CK activity from the model to determine which of the other variables were predictors of mortality within 14 days of capture. With CK excluded, relative humidity ($\chi^2 = 7.99$, $P < 0.01$), ambient temperature ($\chi^2 = 1.48$, $P = 0.22$), body temperature ($\chi^2 = 1.80$, $P = 0.18$), and handling time ($\chi^2 = 2.68$, $P = 0.10$) were included in the model. The model accurately predicted 61% of the deaths and 98% of the survivors at a $P_m \geq 0.5$ with the sensitivity of the model increasing to 67% at a $P_m \geq 0.4$ (Table 2). Based on the predictive equation, P_m of hens dying ≤14 days of capture ranged from 0.23 to 1.00 ($\bar{x} = 0.84 \pm 0.06$; $n = 18$) and that of surviving hens ranged from 0.06 to 0.98 ($\bar{x} = 0.55 \pm 0.03$; $n = 86$).

DISCUSSION

Previous studies indicated that juvenile turkeys may be more susceptible to capture-related death in winter (Spraker et al., 1987; Miller et al., 1996). Miller et al. (1996) reported that during winter capture (7 January to 4 March), 17% of the juvenile hens and 7% of adult hens died as a result of capture stress. We observed that 14% of adults and 21% of juveniles experienced mortality as a result of capture. Although a higher percentage of juveniles died within 14 days of capture, the difference was not significant in our study.

Environmental conditions have been linked to capture-related deaths in previous studies; however, these claims were not thoroughly tested. Bailey (1976) suggested that turkeys should not be trapped with temperatures >21.1 C, and Miller et al. (1996) suggested that winter trapping should only be conducted when temperatures are >15 C in Mississippi. In our study, a hen trapped at 15 C or 21.1 C had a P_m of 0.43 and 0.60, respectively, and at colder temperatures, P_m was lower. Therefore, we recommend not trapping turkeys when winter temperatures are >10 C (30% P_m) in southeastern Oklahoma. In

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addition to ambient temperature, relative humidity and handling time should be considered important determinants of risk of mortality within 14 days of capture. Hens captured when relative humidity is <40% are more susceptible to capture mortality, possibly as a result of more rapid dehydration, especially when ambient temperatures are elevated. Handling time should be kept to a minimum; P_m of hens released ≤1 hr of capture was ≤0.12. Many capture-related mortalities may be prevented by monitoring ambient temperature and relative humidity and adhering to guidelines of when to curtail trapping operations based on environmental variables. Additionally, adequate personnel should be made available to minimize the subsequent handling time of wild turkeys after capture.

Although serum activities of CK and AST have been shown to be indicators of capture stress in mallards (*Anas platyrhynchos*) (Bollinger et al., 1989; Dabbert and Powell, 1993), their usefulness in turkeys has not been evaluated relative to capture myopathy. With respect to eastern wild turkeys, AST activity was not a good indicator of mortality risk. In our study, there may not have been adequate time for AST activity to become significantly elevated as mean handling time for all birds was 76 ± 37.7 min compared with a mean handling time of 106.1 min reported by Bollinger et al. (1989). Dabbert and Powell (1993) reported handling times of about 45 min that included transport from 4.8 to 12.9 km in a truck, which may have added to the elevated AST activities.

The activities of CK and AST are not thought to have diminished appreciably during the course of the collection, handling, and storage of serum in our study. Jones (1985b) noted no loss of CK activity and only 7% loss of AST activity in ovine plasma after four months of storage at −20 C. Similar observations were noted for blood plasma of cattle stored under similar conditions (Jones, 1985a). Given that we stored samples for an average of 199 days (range 166–244 days) at −80 C and all samples were treated in a similar fashion during the study, we do not feel that loss of enzyme activity influenced the results of our study.

Plasma corticosterone has been demonstrated to be an effective measure of stress levels in domestic turkeys (Brown, 1961) and wild turkeys (Whatley et al., 1977). However, no previous work has been done on corticosterone levels and the incidence of capture-related death in wild turkeys. There was a slight tendency for lower levels of plasma corticosterone to be associated with an increased risk of mortality within 14 days of capture, in this study. Although this relationship was not significant, it deserves further investigation. Our initial hypothesis was that corticosterone levels would behave similarly to CK activity. The most logical explanation for this relationship is that hens that experience high levels of stress (high levels of plasma corticosterone) reach a state of shock and "freeze," thus minimizing the amount of skeletal and muscle damage (low CK activity). Conversely, those hens that experience lower levels of stress (low levels of plasma corticosterone), struggle more violently and therefore cause more muscle and skeletal damage (high CK activity). However, further study should be conducted to determine the direct relationship of plasma corticosterone and CK activity under these conditions.

Enzymatic profiles from this study could be useful in identifying individuals that may be at risk of post-release death from handling. Such information could be useful in planning and operating transplant programs, especially when birds have been obtained from other state agencies and a sizeable investment has been made in the birds. Blood is often collected for disease screening, therefore, a CK analysis could be easily performed at the same time for minimal cost. To help minimize the loss of wild turkeys as a result of capture, we suggest trapping turkeys when the ambient temperature is below 10 C and relative hu-

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midity is above 40%, in southeastern Oklahoma. Additionally, adequate personnel should be available to assist with handling birds such that handling time is minimized (preferably <1 hr). Similar studies should be conducted from other geographic regions to determine critical environmental values such that capture-related deaths are minimized.

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