College of Saint Benedict and Saint John's University

## DigitalCommons@CSB/SJU

1998

# The Effect of Temperature on Post Feeding Metabolism in House Finches (*Carpodacus mexicanus*)

Shana L. Vifian
*College of Saint Benedict/Saint John's University*

Follow this and additional works at: https://digitalcommons.csbsju.edu/honors_theses

Part of the Biology Commons

# The Effect of Temperature on
# Post Feeding Metabolism
# in House Finches
# *(Carpodacus mexicanus)*

by Shana Vifian
College of St. Benedict and St. John's University
May 1998

# The Effect of Temperature on Post Feeding Metabolism in House Finches
## (*Carpodacus mexicanus*)

Approved By:
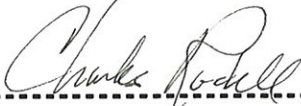
Thesis Advisor: ....................................................
Dr. Marcus Webster, Associate Professor of Biology
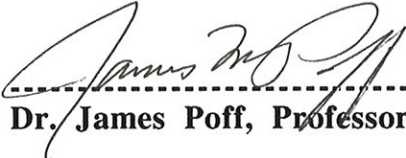
Thesis Reader: ....................................................
Dr. Amy Olson, Professor of Nutrition

Thesis Reader: ....................................................
Dr. Charles Rodell, Professor of Biology

Department Chair: ....................................................
Dr. James Poff, Professor of Biology

# The Effect of Temperature on Post Feeding Metabolism in House Finches (*Carpodacus mexicanus*)

## Abstract

In this study I measured resting metabolic rate (RMR), costs of thermoregulation, and specific dynamic action (SDA) in House Finches and I tested the hypothesis that SDA heat substitutes for costs of thermoregulation at low temperatures in House Finches. Oxygen consumption of both fed and fasted birds was measured at three different temperatures (5 C, 15 C, and 30 C) using an open flow metabolism system and converted to metabolic rate (kJ/hr) using caloric equivalents. RMR, obtained from the fasted metabolic rate within the thermoneutral zone (30 C) was $1.65 \pm .10$ kJ/hr. Costs of thermoregulation at 15 C and 5 C were measured as the difference between fasted metabolic rate at these temperatures and the RMR. At 15 C costs of thermoregulation were .41 kJ/hr and at 5 C were .59 kJ/hr. SDA was calculated from the difference in the metabolic rates between fed and fasted birds and expressed as a percent of gross energy intake (GEI). SDA averaged 3-5% of GEI. No effect of temperature on SDA was observed. At 30 C SDA was 4.12% of GEI and at 5 C was 3.68% of GEI. SDA is expected to decrease with decreasing temperature if it substitutes for the costs of thermoregulation at low temperatures. I found no evidence to support the substitution hypothesis. High variability, however, made it difficult to draw solid conclusions.

Introduction

All animals require chemical energy to do work and to maintain their structural integrity. Some of the energy requiring activities in animals include muscle contraction, ciliary movement, and active transport of molecules across membranes. In addition to these basic requirements, animals also need energy to synthesize complex biological molecules from simple chemical building blocks and to organize these molecules into organelles, cells, tissues, organs, organ systems, and complete organisms (Eckert 1988). To meet these energy needs animals must take in fuel and continuously expend the energy it contains. Metabolism refers to the sum total of all the chemical reactions that take place within an organism in the process of utilizing energy.

Metabolism can be measured in a number of different ways. All of the methods of measuring metabolism make use of the relationship between metabolism and heat. When external work and storage are not factors all of the energy released in metabolic processes appears eventually as heat (Eckert 1988). Heat can be measured directly or indirectly. In direct calorimetry, heat lost by an animal in a closed chamber is trapped by a known mass of water surrounding the chamber (Eckert 1988). The amount of heat lost is calculated from the increase in the temperature of the water. In indirect calorimetry, oxygen consumption and carbon dioxide production are measured to determine metabolic rate. The oxidation of food to yield energy consumes oxygen and produces carbon dioxide in amounts proportional to the heat released. There are well established caloric equivalents that convert oxygen consumption and carbon dioxide into metabolic rate (Kleiber 1975). $O_2$ consumption and $CO_2$ production can be measured with closed or open system respirometry. In closed system calorimetry, $O_2$ depletion and $CO_2$ production within a closed chamber containing an animal are monitored (Eckert 1988). In open system respirometry, $O_2$ and $CO_2$ concentrations of chamber influx and efflux air are compared

using mass-flow analysis (Eckert 1988). The difference in influx and efflux concentrations is used to calculate respiratory exchange.

When an animal is at rest under conditions of minimal environmental and physiological stress and is not processing a meal, metabolism occurs at a steady rate. This is called the basal (or resting) metabolic rate (BMR or RMR). It represents the the cost of maintenance for an animal. BMR is a constant component of the overall metabolic rate. It is a baseline against which comparisons between different species and between different physiological conditions can be made. BMR is highly correlated with body mass and can be estimated simply from body mass (Aschoff 1970). In addition to BMR, other energy requirements contributing to the overall metabolic rate include tissue growth, reproduction (growth of eggs or fetus), activity, and thermoregulation. Field metabolic rate (FMR) includes all of the energy costs an animal encounters in the wild throughout the day. FMR is expressed in kJ/day and can also be calculated from body mass (Nagy 1987).

The cost of thermoregulation is one of the most significant components of the energy budget of animals, particularly in cold temperatures. In Eurasian Kestrels (*Falco tinnunculus*), 31-38% of daily energy expenditures are devoted to the costs of thermoregulation in winter compared to 10% in the summer (Dawson and O'Connor 1996). On average, thermoregulatory costs in birds range from 10-60% of the daily expenditures depending on the season (Dawson and O'Connor 1996). Another source reported that the costs of thermoregulation account for 20-40% of the daily energy expenditure in small birds (Webster and Weathers 1990).

Both birds and mammals are homeothermic endotherms. This means that they regulate their body temperature within a narrow limit (37-40 C for mammals and 37-41 C for birds) by mechanisms that control rates of heat production and heat loss (Eckert 1988). Mammals and birds have a characteristic thermoneutral zone defined by an upper and lower critical temperature (Figure 1). Within this range the basal metabolic rate is sufficient to balance heat loss to the environment in cooperation with adjustments in thermal

conductance of the body surface (Eckert 1988). Thermal conductance can be adjusted through vasomotor responses, postural changes to reduce surface area, and pilomotor regulation of fur or feathers. When the ambient temperature falls below the lower critical temperature, basal metabolism and thermal conductance regulation are not able to balance heat loss to the environment. The animal must increase its heat production by thermogenesis to meet the increased cost of maintaining body temperature. Thermogenesis involves converting chemical energy into heat. This is accomplished by decreasing the efficiency of normal energy-converting pathways so that most of the energy is given off as heat rather than used to perform work. Shivering and nonshivering thermogenesis are two types of heat production. Below the lower critical temperature heat production increases linearly with a decrease in ambient temperature (Figure 1). This is called the zone of metabolic regulation. At temperatures below this zone, thermogenesis mechanisms cannot generate enough heat to maintain body temperature and hypothermia sets in. If the ambient temperature remains below the zone of metabolic regulation the animal will eventually die.
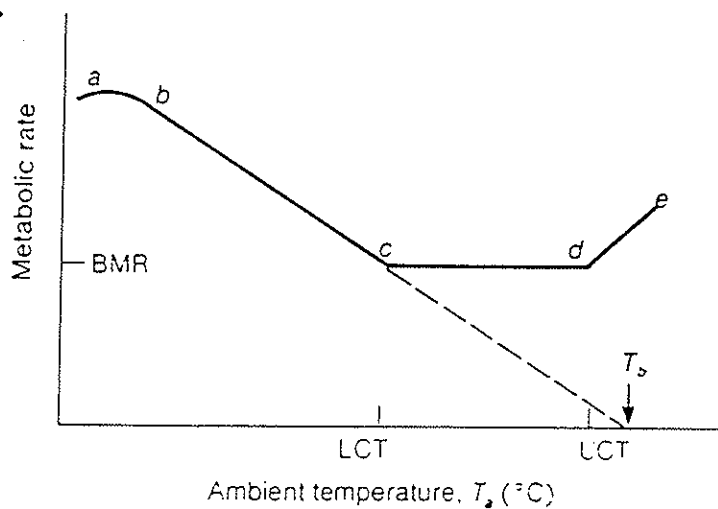


Figure 1. Metabolic rate of an homeothermic endotherm at different ambient temperatures. The thermoneutral zone extends from the lower to the upper critical temperature, points c and d, respectively. Above and below this range, the metabolic rate must rise to either increase heat production in the zone of metabolic regulation (segment b-c) or increase active dissipation of heat by evaporative cooling (segment d-e) if body temperature is to remain essentially constant. (Eckert 1988)

In addition to these mechanisms of thermogenesis, it has been suggested that specific dynamic action may also help an animal meet the costs of thermoregulation. Specific dynamic action (SDA) is defined as the increase in metabolism an animal experiences following feeding due to the costs of digesting and assimilating the meal (Rubner 1902, Brody 1945). The energy associated with the costs of processing a meal become unavailable to the animal to do work. Animals, like all machines, do not operate at 100% efficiency. They are not able to utilize all of the energy they consume. Figure 2 illustrates the path from gross energy to net energy in animals. As the figure indicates, some of the energy is indigestible and is lost as fecal waste. Of the digestible energy some is lost as urinary waste. The remaining energy is called metabolizable energy. Not all of the metabolizable energy is available for use by the animal. Some of the metabolizable energy is lost in the form of heat during the energy conversions that occur in metabolic processes. The heat by-product is SDA. The remaining energy available for subsequent physiological uses is the net energy (Figure 2).

Gross energy
|
|————————————→ *Fecal waste*
|
Digestible energy
|
|————————————→ *Urinary waste*
|
Metabolizable energy
(metabolized)
|
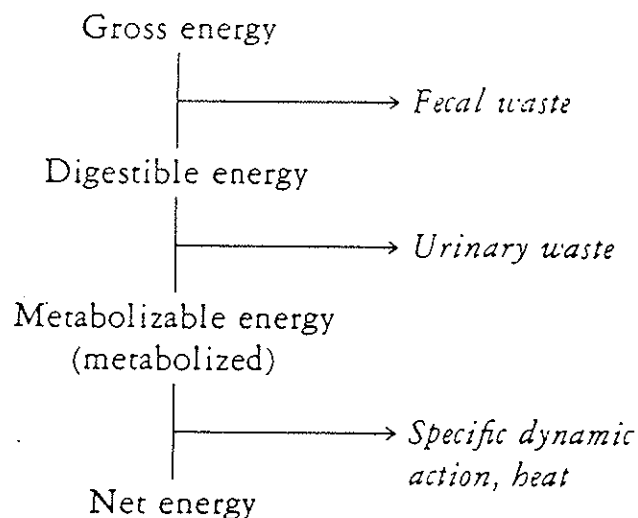|————————————→ *Specific dynamic action, heat*
|
Net energy

Figure 2. Path from gross energy to net energy.

Lavoisier and Laplace were the first scientists to quantify the increase in metabolism following feeding in 1780 (Brown and Cameron 1990). Rubner coined the term specific dynamic action in 1902. Since then SDA has been documented in a variety of animals ranging from invertebrates such as insects and mollusks to vertebrates such as fish, reptiles, birds, and mammals (reviewed by Brown and Cameron 1990). Despite the long history of the study of SDA, the mechanisms involved are still incompletely understood. SDA is measured as the total excess metabolic heat produced by a meal from the time metabolism first rises to the time it returns to the baseline level (Eckert 1988) (Figure 3). The loss of energy as heat is attributed to the costs of digestion and assimilation. Assimilation is the process of preparing digestion products for entry into metabolic pathways (Eckert 1988). It is thought that assimilation, particularly of protein, is responsible for the bulk of SDA with digestion contributing only slightly (Brody 1945). This claim is supported by the fact that amino acids injected directly into the blood stream cause an increase in metabolism similar to that resulting from ingested proteins (Brody 1945).
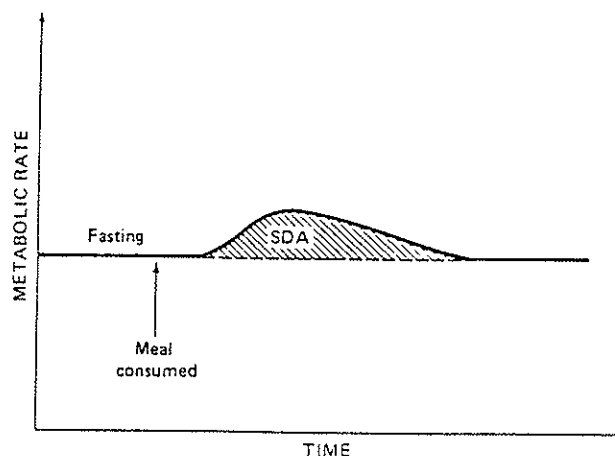
Figure 3. A schematic view of SDA. A resting fasting animal is fed at the time marked and then remains at rest. The solid line shows the animal's actual metabolic rate. The dashed line depicts what the rate would have been, had the meal not been consumed. The shaded area is the magnitude of the SDA. (Eckert 1988)

The magnitude of the SDA depends on the amount and type of food eaten. In other words, the SDA is equal to a certain percentage of the total energy of the food ingested that varies with different types of food from 6% for simple carbohydrates to 31% for proteins (Ricklefs 1974). Complex carbohydrates and fats have intermediate values (Ricklefs 1974). Standard metabolism is elevated by a range of 6% for simple carbohydrates to 45% for proteins (Ricklefs 1974). Complex carbohydrates and fats again have intermediate values (Ricklefs 1974) (Table 1).

It has been suggested that SDA may not just be the unavoidable costs of processing a meal, but instead may substitute for the costs of thermoregulation. The energy lost as heat in processing a meal may be used to help an animal to keep warm at cold temperatures. If SDA is not simply an unavoidable energy cost of assimilating a meal, but can be used to fulfill part of an animal's thermoregulatory costs the animal would have a significant advantage in utilizing its energy sources. The energy stores used in thermogenesis to meet thermoregulatory costs at low temperatures could be conserved for other uses if SDA is able to satisfy part of the costs of thermoregulation. Less food would have to be taken in because the animal would be more efficient at using the energy contained in the food (Webster and Weathers 1990). The increase in its utilization efficiency as a result of SDA substitution would enable the animal to better survive winter, potentially increasing its overall fitness.

If SDA does substitute for thermoregulatory costs, SDA is expected to decrease with decreasing temperatures below the lower critical temperature as more of it is utilized to meet the increasing costs of thermoregulation. Substitution has been documented in mammals (Simek 1975, Costa and Kooyman 1984), but has not been studied extensively in birds (Chappell et al 1997). Substitution has been reported in domestic fowl (*Gallus gallus*) (Berman and Snapir 1965), European Starlings (*Sturnus vulgaris*) (Biebach 1984), House Wren chicks (*Troglodytes aedon*) (Chappell et al 1997), and Verdins (*Auriparus flaviceps*) (Webster and Weathers 1990). But substitution may not occur in all birds. Klassen et al

(1989) found that SDA did not appear to substitute for regulatory thermogenesis in chicks of the Arctic Tern (*Sterna paradisea*). The lack of extensive data on SDA substitution in birds is due in part to the difficulty in measuring SDA because of the high activity of birds and their tendency to eat small frequent meals.

In this study I measured the metabolic response to feeding in House Finches (*Carpodacus mexicanus*). House Finches are small (15-25 g) cardueline finches found throughout most of the U.S. and parts of Canada and Mexico. House Finches are native to western North America from the Pacific Coast to the Great Plains. In 1940 a number of House Finches were released on Long Island, New York. Since then they have spread along the Eastern Seaboard from New England to South Carolina and through the Midwest (O'Connor 1995a). House Finches are generally sedentary but some dispersal occurs in the winter (Hill 1993). They are found in and around suburban towns, farmland, ranches, orchards, scrub, canyons, or semi-dry brush country (Hill 1993). They feed primarily on seeds such as thistle, dandelion, and sunflower, but also eat blossoms, buds, and fruit (Hill 1993). House Finches were chosen for this study primarily because of their abundance in this area, their success in captivity, and the baseline physiological data available on them (O'Connor 1995a and Carrey et al 1989).

I tested the hypothesis that SDA does substitute for the costs of thermoregulation at low temperatures in House Finches. To test this hypothesis I measured the metabolic rates at three different temperatures (5, 15, and 30 C) and compared the metabolic rates of fed and fasted birds at each of these temperatures. I predicted there would be a decrease in the ratio of the metabolic rates for fed to fasted birds as the temperature decreased if substitution occurs (Figure 4).
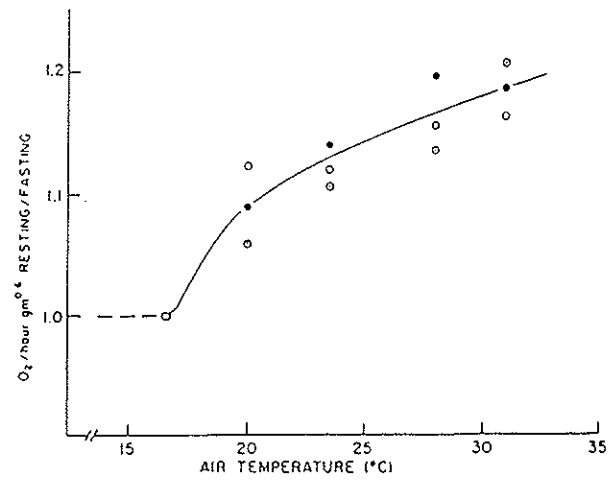
Figure 4. Ratio of mean oxygen consumption in resting (but fed) and fasting chickens (three breeds indicated by symbols) in relation to air temperature. Body weight (in grams) is raised to the 0.6 power to correct for differences in body weight among breeds. (Data from Berman and Snapir, 1965)

## Methods

### Experimental Animals

I used five House Finches in this experiment (4 females and 1 male) ranging in mass from 17-24 g. I captured the birds from the St. John's University and College of St. Benedict campuses and at a private residence near Brainerd, MN in accordance with the guidelines issued by the IACUC. The birds were trained to eat two large meals of sunflower meats daily by allowing them access to the food for a limited time twice a day. In order to get enough food the birds had to eat their fill when the food was available. Fresh water was supplied daily. The birds were housed in individual cages at $20 \pm 1$ C with a reversed photoperiod (12:12 LD) so that the lights went out at 12:00 noon and came on at 12:00 midnight.

### Apparatus

I used an open flow metabolism system to measure the metabolism of two birds contained in two separate chambers. The chambers were approximately 5.5 L polycarbonate boxes with two perches and a food box. The two chambers were located inside a Freas 815 incubator that maintained temperature within $\pm.5$ C. A length of fishing line was attached to the lid of the food box that led outside the incubator allowing me to open the food dish remotely. Each chamber contained a Cu-Cn thermocouple that I used to monitor chamber air temperature.

Air flowed into the chambers at 540-560 ml/min. Flow rate was measured with flowmeters that had been previously calibrated to $\pm1\%$ of actual air flow using a bubble meter. Water and carbon dioxide were removed by drierite and soda lime, respectively, before and after air passed through the chambers. Percent $O_2$ was measured using an Ametek S3A $O_2$ analyzer. Percent $O_2$ was recorded on a microcomputer equipped with an ADC-1 analog-to-digital converter and Sable Systems Datacan software. The program,

Datacan, measured percent $O_2$ in 53.31 segments with readings taken every 5 seconds. Each experimental run consisted of seven segments. I had a switch system that allowed me to alternate the source of air entering the $O_2$ analyzer between inlet air, outlet air from chamber A, and outlet air from chamber B. At the beginning of each 53.31 minute segment inlet air was measured for 5-10 minutes. I then alternated the recording between chamber A outlet air and chamber B outlet air until 5-10 minutes remained. For the last 5-10 minutes of the segment inlet air was recorded. Signal drift, obtained from the inlet air measurements taken at the beginning and end of each segment, was assumed to be linear and averaged about .005%. Within each 10-15 minute period I selected a 3-5 minute interval that appeared to be stable, to represent the period. My aim was to get a reading for each half hour block of time for each bird. Temperature readings for each chamber and the incubator were also taken every 10-15 minutes. A video/infrared system was used to monitor the birds in the light and dark.

## Protocol

I measured the oxygen consumption of each bird in both a fed and fasted state at three temperatures: 5 C, 15 C, and 30 C. Each run began at approximately 10:00 which was about 4-6 hours after the birds' last meal. Each bird was weighed prior to the run. Five grams of food were placed in the food dishes of the birds to be fed. The food dishes remained empty for the birds to be fasted. At 10:00 the birds were placed inside the chambers under fluorescent lights. At 11:00 the food dishes were opened remotely. At 12:00 noon the fluorescent lights were turned off ending the feeding period. (House Finches do not eat in the dark.) Infrared lights were turned on allowing me to to view the birds on the video monitor in the dark. Percent $O_2$ was measured for four more hours. At approximately 16:00 the birds were removed from the chambers, weighed, and returned to their cages. The food from the chambers of the fed birds was weighed and recorded.

<u>Calculation</u>

I calculated oxygen consumption in ml/hr using Equation 2 of Hill for metered $CO_2$

free, dry inlet air with the program Datacan (Sable Systems). For each 10-15 minute period

I selected the 3-5 minute interval that appeared most stable and recorded the oxygen

consumption and the time for this interval. All of these data points were grouped into 13

half hour intervals from 10:00 to 16:00. For example, the first interval was 9:45-10:15, the

second interval was 10:16-10:45, the third 10:46 -11:15 and so on. In cases where more

than one data point from a trial fit into a single interval I took the average. For these 13

points I converted oxygen consumption in ml/hr to metabolic rate in kJ/hr using caloric

equivalents as follows: during and up to 1 hour post feeding, 19.8 kJ/$LO_2$ (RQ=0.75); 1-3

hours post feeding, 19.7 kJ/$LO_2$ (RQ=0.73); and 3-4 hours post feeding, 19.6 kJ/$LO_2$

(RQ=0.71). The RQ for sunflower meats is .75. It was assumed that immediately following

feeding mostly sunflower meats were being oxidized and with time oxidation of sunflower

meats decreased until mostly fat was being oxidized.

To determine the costs of thermoregulation I compared the metabolic rate of the

fasted birds at 15:00 at each of the three temperatures. The temperature of 30 C was

assumed to have no cost of thermoregulation because it is within the thermoneutral zone

(O'Connor 1995a). I calculated the difference in the average metabolic rate for the three

birds at 15:00 between 15 C and 30 C and between 5 C and 30 C. These differences were

divided by the average metabolic rate at 30 C (15:00) to give the cost of thermoregulation at

15 C and 5 C as a percent of the basal metabolic rate at 30 C.

I obtained SDA estimates for each bird at each temperature from graphs of

metabolic rate versus time. The fed and fasted metabolic rates for a single bird at one

temperature were included on the same graph. To estimate SDA I located the area of

elevation of the fed metabolic rate above the fasted metabolic rate after 12:00. The area

between the two graphs represents the SDA. I averaged the fasted metabolic rates for this

period and subtracted them from the average of the fed metabolic rates for the period. I multiplied this difference in kJ/hr by the period of time in hr that the fed metabolic rate was elevated above the fasted. This value represents the SDA in kJ. The SDA in kJ was divided by the total energy in kJ that the bird ingested to give SDA as a percent of gross energy intake. I estimated the gross energy intake using the % composition of sunflower seeds and the energy equivalents of each component (Schmidt-Nielson 1975).

Results

        I measured metabolic rate at 15:00 hours for each of the birds in the fasted state at 30 C to determine the resting metabolic rate (RMR). Average RMR was $1.65 \pm .10$ kJ/hr (Figure 5, Table 2). I averaged the metabolic rates at 15:00 hours for fasted birds at 15 C and 5 C. Average metabolic rate at 15 C at 15:00 was $2.06 \pm .14$ kJ/hr (Figure 5, Table 2). Subtracting the RMR at 30 C gives .41 kJ/hr. This represents a 24.8% increase in RMR. At 5 C average fasted metabolic rate at 15:00 was $2.24 \pm 15$ kJ/hr (Figure 5, Table 2). Subtracting the RMR at 30 C gives .59 kJ/hr. This represents a 35.8% increase in RMR. The margins of error ($\pm$ standard error) overlap between 5 C and 15 C but not between 5 C and 30 C or between 15 C and 30 C (Figure 5, Table 2).

        FMR was calculated using body mass to be 83 kJ/day (Nagy 1987). The amount of food consumed in the experiments ranged from .26-1.14 g with the average being .39 g. This corresponds to 6.79% to 24.69% of the FMR with the average being 8.45%.

        Metabolic rate versus time was graphed for each bird at each temperature (5 C, 15 C , and 30 C). Fed and fasted rates were included on the same graph to allow comparison. At 30 C SDA was measured in RO to be .25 kJ which represents 2.93% of the gross energy intake (GEI) (Figure 6 Table 3). SDA was measured as .58 kJ in RY at 30 C which represents 5.30% of GEI. (Figure 7, Table 3). RB had no measurable SDA at 30 C. Its fasted metabolic rate at 30 C was higher than its fed metabolic rate (Figure 8). This data is suspect and was not included in average SDA. Average SDA at 30 C was 4.12% of GEI

(Figure 9, Table 3).

At 15 C I measured SDA in RO as .57 kJ which is 23.8% of GEI (Figure 10, Table 3). RY had an SDA of .05 kJ which is .4% of GEI. (Figure 11, Table 3). RB had an SDA of .04 kJ which represents .6% of GEI (Figure 12, Table 3). Average SDA at 15 C was 8.1% if RO is included and .5% if it is not (Figure 9, Table 3). At 5 C RO had an SDA of .29 kJ which represents 2.62% of GEI (Figure 13, Table 3). RY had an SDA of .27 kJ which represents 4.75 % of GEI (Figure 14, Table 3). Average SDA at 5 C was 3.68% (Figure 9, Table 3).

Figure 9 shows average SDA as a function of temperature. At 30 C SDA averaged 4.12%. At 15 C it was 8.1% or .5%. The closed circle represents average SDA including RO. The open circle represents average SDA without RO. At 5 C average SDA was 3.68%. There is not a consistent decreasing trend with decreasing temperature. Average SDA at 5 C is almost the same as it is at 30 C.

Table 1 SDA of Sunflower Seeds

| Food | % Composition of Sunflower Seeds | SDA (% of GEI) |
|---|---|---|
| Protein | 42 | 31 |
| Starch | 15 | 23 |
| Fat | 41 | 17 |
| Sunflower Seeds | | 23 |

Table 2 Metabolic Rate (kJ/hr) of House Finches resting in the dark during the rest phase of the daily cycle. Values are expressed as mean $\pm$ SE.

| Temperature | Average Metabolic Rate (kJ/hr) |
|---|---|
| 30 C | $1.65 \pm .10$ |
| 15 C | $2.06 \pm .14$ |
| 5 C | $2.24 \pm .15$ |

Table 3 SDA as % GEI. SDA (in kJ) was measured as the area between graphs of fed and fasted metabolic rate vs. time between 12:30 and 15:00. Average meal was 8.45 kJ.

SDA (% of GEI)

| Temperature | RO | RY | RB | Average |
|---|---|---|---|---|
| 30 C | 2.93 | 5.30 | --- | 4.12 |
| 15 C | 23.8 | 0.40 | 0.60 | 8.10 * |
| 5 C | 2.62 | 4.75 | --- | 3.68 |

* Average SDA when RO is included. When RO is not included average SDA is 0.50%.
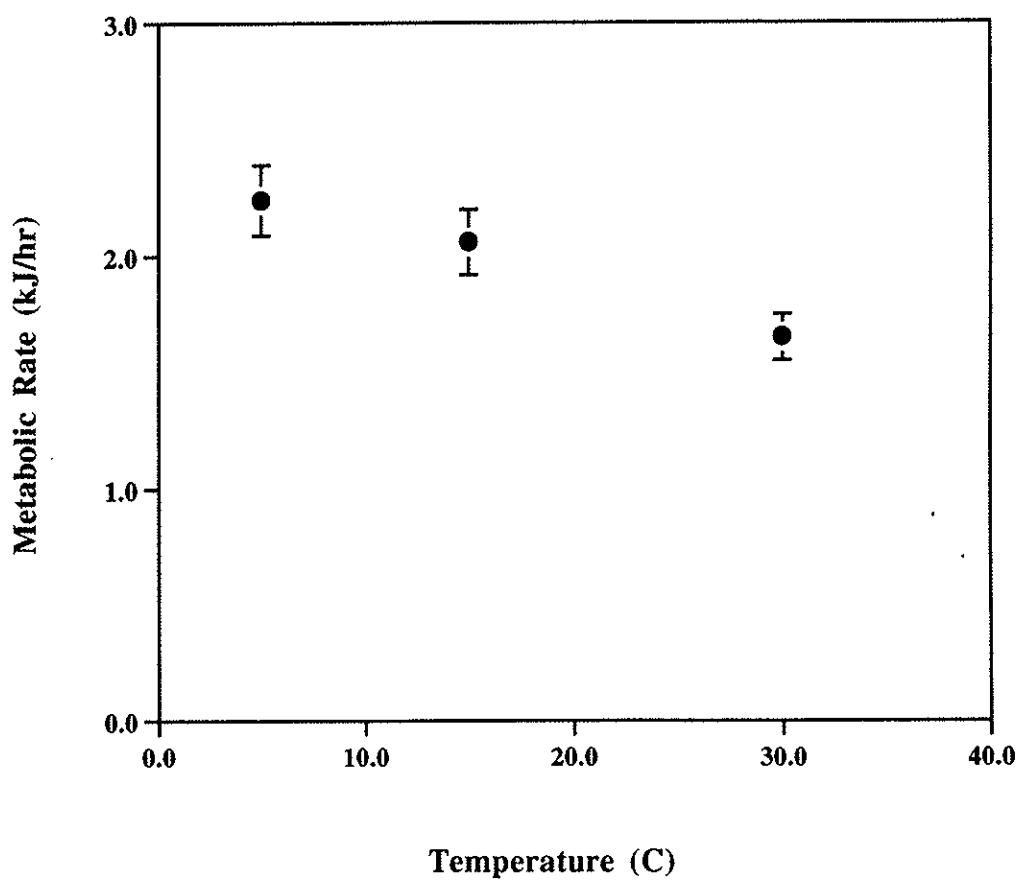
Figure 5. Metabolic rate of House Finches
resting in the dark during the rest phase of
their daily cycle as a function of temperature.
Error bars represent ± standard error.
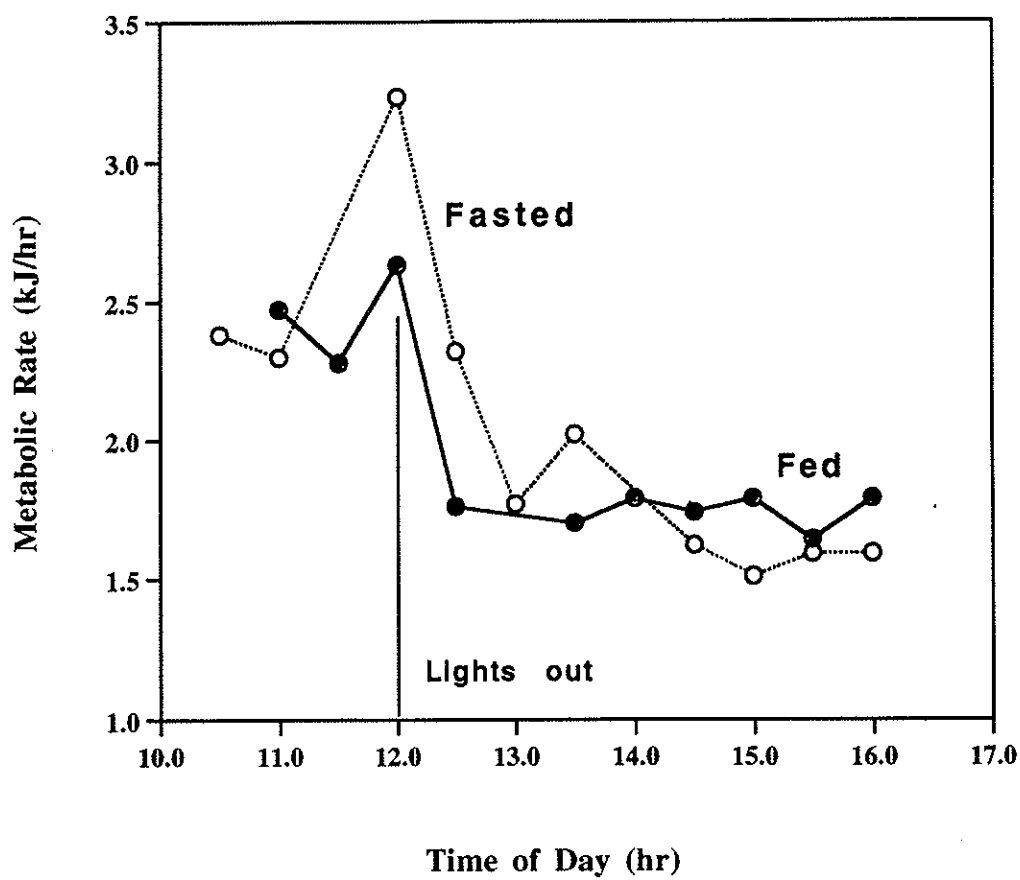
Figure 6. Metabolic rate for RO at 30 C. Closed circles represent fed state and open circles represent fasted state.
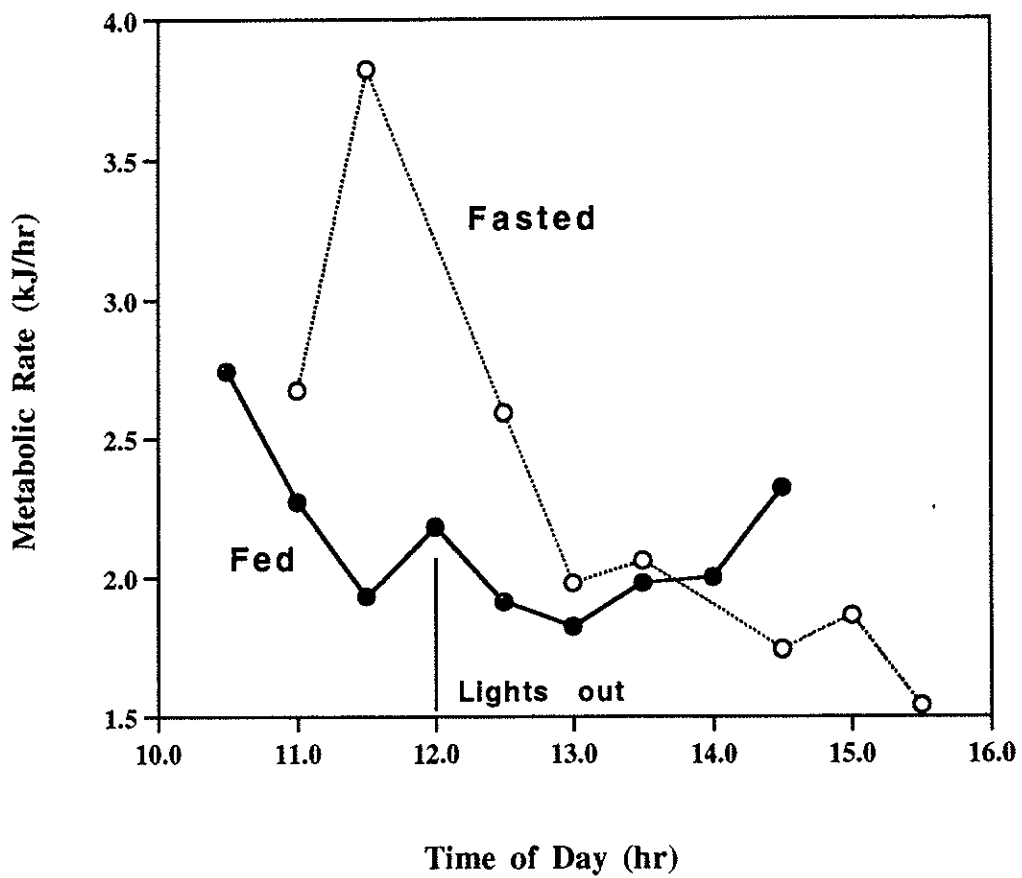
Figure 7. Metabolic rate for RY at 30 C. Closed circles represent fed state and open circles represent fasted state.
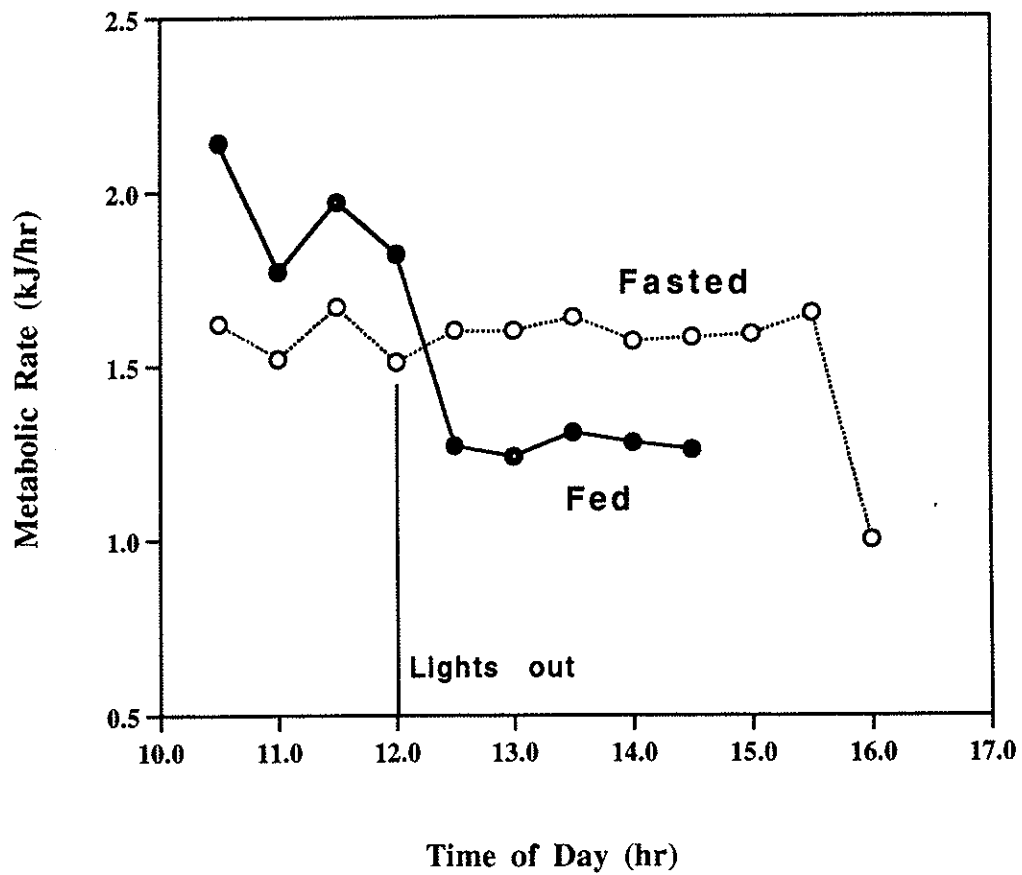
**Figure 8. Metabolic Rate for RB at 30 C. Closed circles represent fed state and open circles represent fasted state.**

Figure 9. Average SDA as %GEI as a function of air temperature. SDA was measured as the area between the graphs of fed and fasted metabolic rates vs. time from 12:30 to 15:00. Average meal was 8.45 kJ. Open circle at 15 C represents average without RO. Closed circle represents average with RO.

Figure 10. Metabolic rate for RO at 15 C. Closed circles represent fed state and open circles represent fasted state.
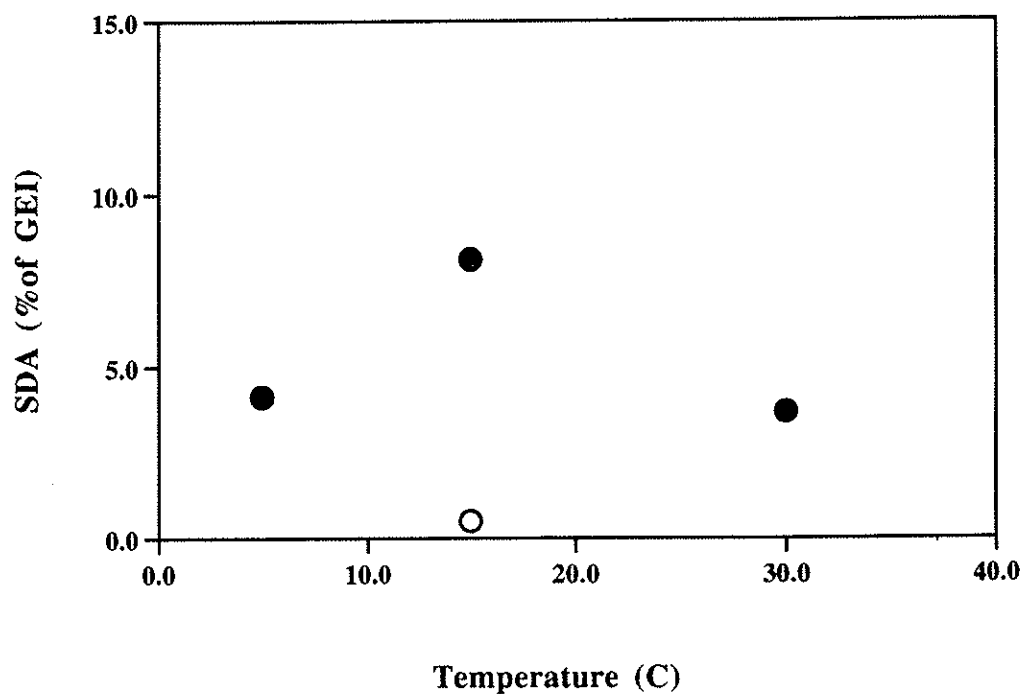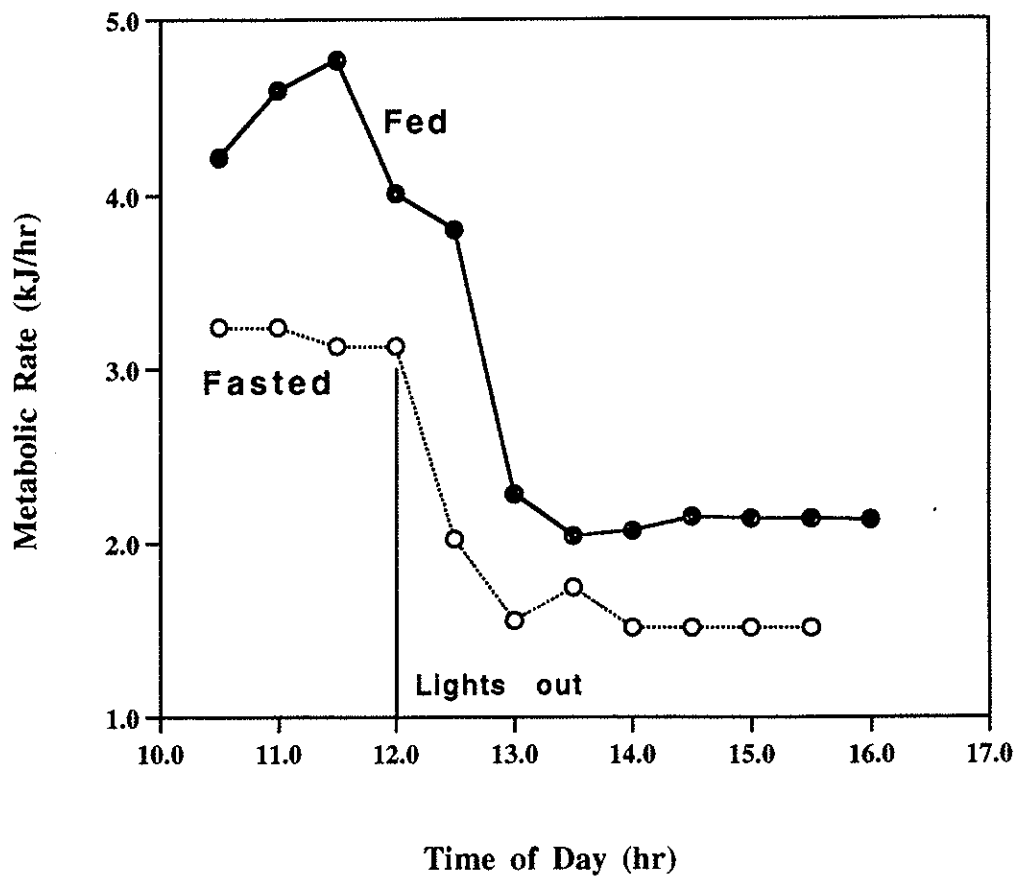
Figure 11. Metabolic rate for RY at 15 C. Closed circles represent fed state and open circles represent fasted state.

**Figure 12. Metabolic rate for RB at 15 C. Closed circles represent fed state. Open circles represent fasted state.**
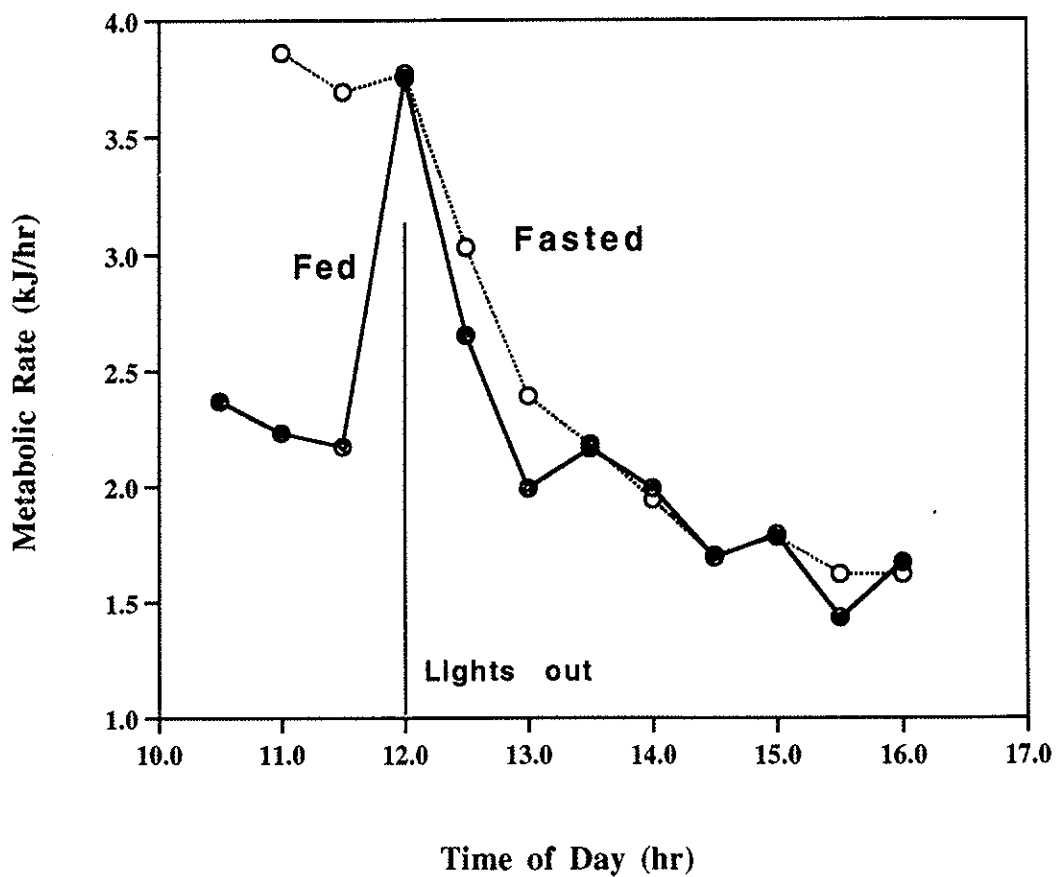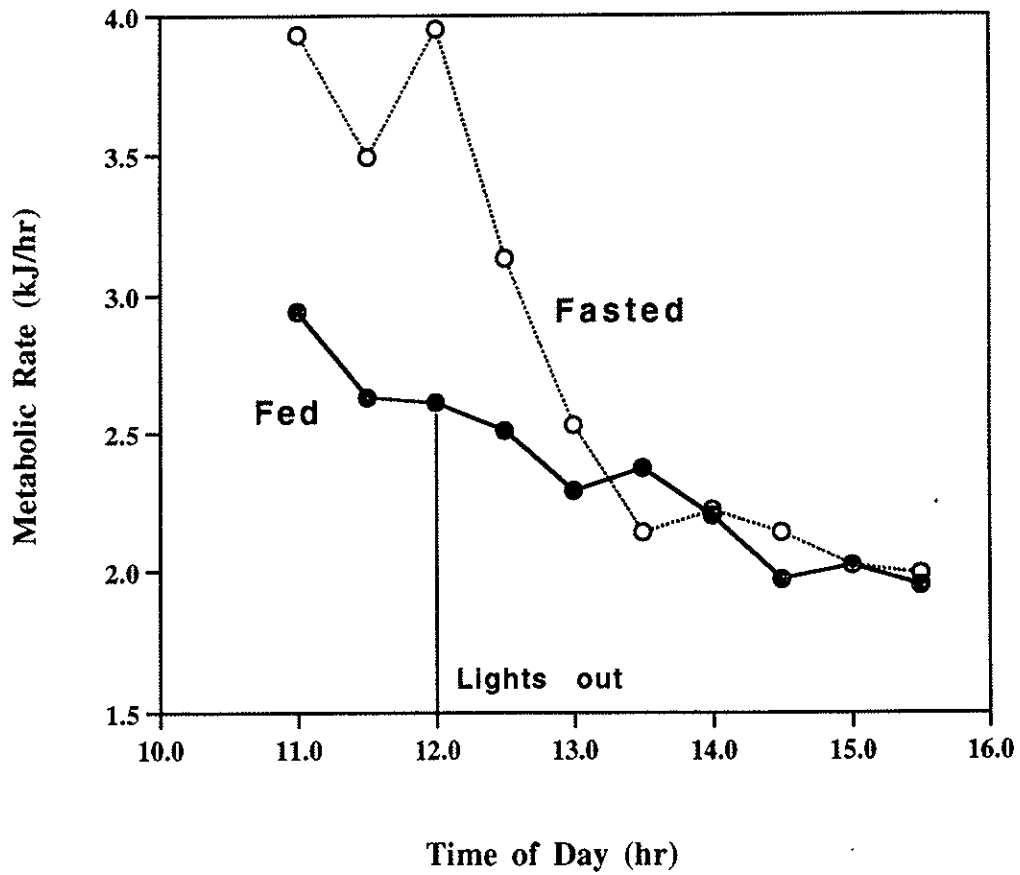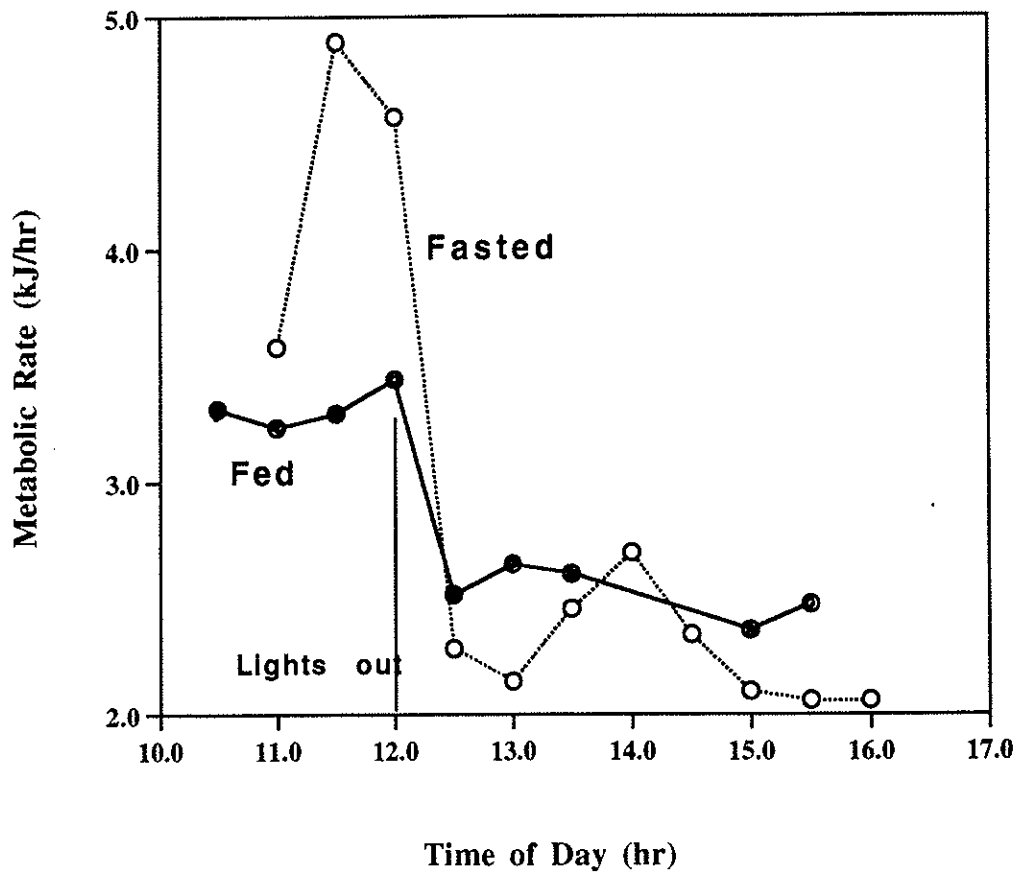
**Figure 13. Metabolic rate for RO at 5 C. Closed circles represent fed state and open circles represent fasted state.**
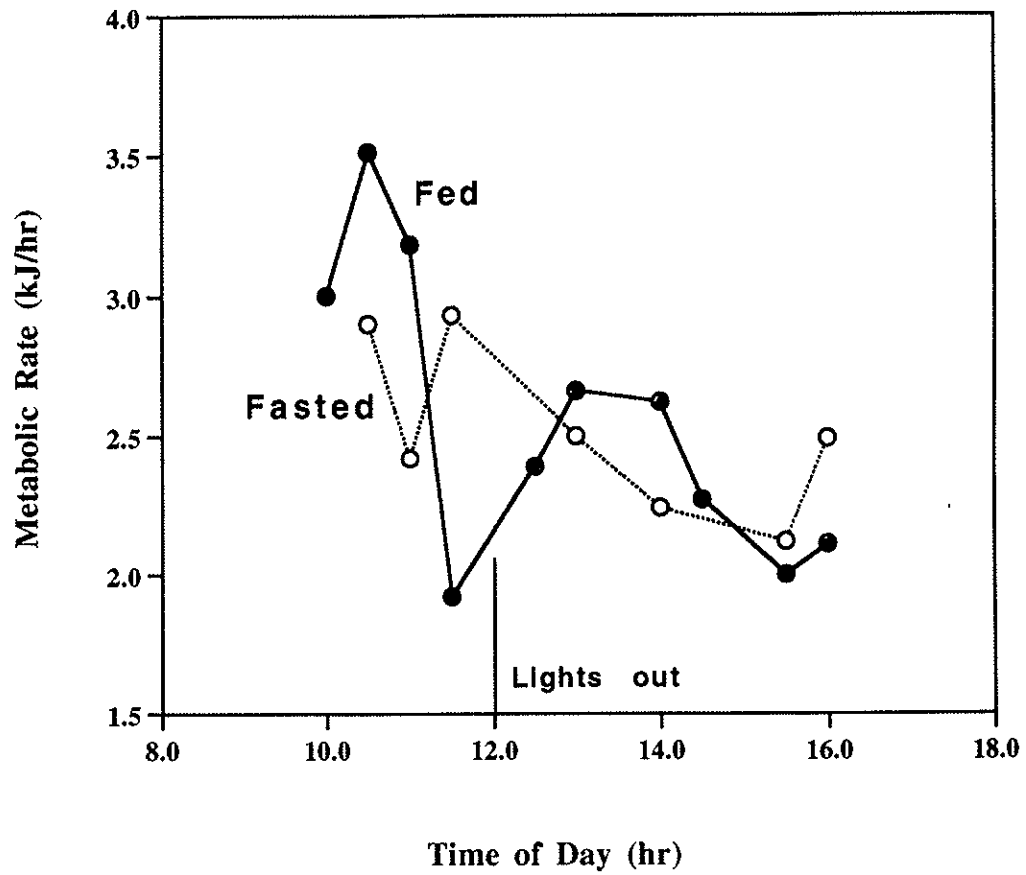
**Figure 14. Metabolic rate for RY at 5 C. Closed circles represent fed state and open circles represent fasted state.**

Discussion

## Basal Metabolic Rate

Resting metabolic rate (RMR) is the metabolic rate of a resting fasting animal in its thermoneutral zone (Calder and King 1974). RMR is a baseline against which comparisons can be made between different species of animals and between different physiological conditions. RMR measured in this study (1.65 kJ/hr or 4.06 ml/ghr) (Figure 5, Table 3) was somewhat higher than that reported by O'Connor (3.12 ml/ghr) for House Finches (1995a). The average mass of the birds in this study was almost 3 grams less than those in the O'Connor study. RMR is highly dependent on body mass. Mass specific RMR is greater in smaller birds than larger birds (Dawson and O'Connor 1996). This is one possible reason for the higher RMR we observed. (Dawson and O'Connor 1996).

In addition to body mass, a number of other factors correlate with RMR. Some of these factors include diet, flight patterns, and feeding behavior (Dawson and O'Connor 1996). For example, significant positive correlation between mass independent RMR and latitude has been observed in both terrestrial and marine birds (Dawson and O'Connor 1996 ). Several tropical bird orders showed lower RMR compared to species from higher latitudes, consistent with the correlation between latitude and RMR. Lower RMR was observed for terrestrial birds that forage in the open than those that forage in the shade (Weathers 1979, Hails 1983). Plumage color may also affect RMR. Dark plumage species of herons and seabirds exposed to intense solar radiation at low altitudes have a somewhat lower RMR than light colored species (Ellis 1980).

Variation of RMR with climate has also been demonstrated. For example, the RMR of the Black-bellied Sandgrouse (*Ptericles orientalis*) is similar to that expected for a bird of its mass (Dawson and O'Connor 1996). The Pintail Sandgrouse (*P. alchata* ) which encounters hotter and more arid regions has an RMR that is only 62% of the mass predicted value (Dawson and O'Connor 1996). House Sparrows (*Passer domesticas*) living

in a warmer climate tend to have a lower RMR than those living in a cooler climate (Hudson and Kimzey 1966). This effect is also seen in House Finches. House Finches from Colorado and Michigan - Ohio experiencing cold winter temperatures, have a higher RMR than House Finches from southern California which experience only mild winter temperatures (Root et al 1991).

In addition to varying with climate, RMR also varies with the season in some species. For Example, the European Goldfinch (*Carduelis carduelis*), Common Redpoll (*C. flammea*), Mute Swan (*Argnus olor*), Dark-eyed Junco (*Junco hyemelis*), and Black-capped Chickadee (*Parus atricapillus*) all have higher RMR's in the winter than in the summer (Dawson and O'Connor 1996). Not all birds demonstrate seasonal variation of RMR, however. The RMR of the House Finch for example, does not differ between winter and late spring within the California and Michigan-Ohio populations, in spite of the RMR noted above (Dawson and O'Connor 1996). The RMR of the Eurasian Kestrel also does not vary with the season (Masman 1989 et al).

Birds may also be conditioned to raise or lower their RMR. Birds transferred from their thermoneutral zone to temperatures of 22 to -14 C may show an increase in RMR of 10-55% after a period of one to two weeks (Dawson and O'Connor 1996). If the birds are transferred in the opposite direction, from cold to warm, the birds will show a corresponding decrease in RMR (Dawson and O'Connor 1996).

A reduced RMR in hot weather could be very advantageous. A lower RMR would reduce a bird's endogenous heat burden and its need for evaporative cooling thus decreasing water loss (Dawson and O'Connor 1996). An elevated RMR in cold temperatures could also be advantageous, though it is not as obvious because of the bird's ability for regulatory thermogenesis (O'Connor 1995a). An elevated RMR may lower the ambient temperature threshold for shivering, extending the thermoneutral zone. Increased thermogenic capacity and increased cold resistance may occur with increases in RMR (O'Connor 1995a). The exact relationship between cold induced RMR and these

improvements is not clear; the increased RMR may contribute to the improvements, it may be a by-product of them or it may be a separate response (O'Connor 1995a).

## Costs of Thermoregulation

In spite of these uncertainties the RMR is still useful in serving as a baseline that can be used to evaluate the relative costs of heat or cold defense. I compared the baseline of 1.65 kJ/hr at 15:00 at 30 C for a fasted bird at rest to the fasting resting levels at 15:00 for temperatures of 15 C and 5 C (Figure 5, Table 2). The difference between these metabolic rates and the RMR at 30 C represents the costs of thermoregulation at these two temperatures. At 15 C the metabolic rate at 15:00 was 2.06 kJ/hr (Figure 5, Table 2). The difference between this rate and the RMR was .41 kJ/hr which is 24.8% of the RMR. At 5 C the metabolic rate was 2.24 kJ/hr (Figure 5, Table 2). The difference between this rate and the RMR was .59 kJ/hr which is 35.8% of the RMR. In other words, at 15 C the thermoregulatory costs are .41 kJ/hr requiring a 24.8% increase in RMR (Figure 5, Table 2). At 5 C the thermoregulatory costs are .59 kJ/hr which requires a 35.8% increase in RMR (Figure 5, Table 2). These values were compared with the FMR of 83 kJ/day calculated from body mass (Nagy 1987). At 15 C thermostatic costs represent 12% of the daily energy requirement. At 5 C thermostatic costs are 17% of the FMR. These values are less than the 20-40% reported for free-living birds by Webster and Weathers (1990).

## Mechanisms of Thermoregulation

Birds have a number of responses to maintain body temperature with decreasing ambient temperature. Within the thermoneutral zone these include reducing body heat loss through vasomotor responses, postural changes to reduce exposed surface area, and pilomotor regulation of the insulating effectiveness of the plumage (Eckert 1988). Outside the thermoneutral zone these responses are not sufficient to maintain body temperature.

When the ambient temperature drops below the lower critical temperature (the lower

boundary of the thermoneutral zone) the bird must initiate heat production to balance the

heat lost to the environment. This is achieved by increasing the metabolic rate above basal

(resting) levels through thermogenesis. (Eckert 1988)

Shivering Thermogenesis Shivering uses muscle contractions to produce heat.

Shivering is the primary means by which birds generate heat in the cold (O'Connor 1995a).

In most birds the greatest fraction of skeletal muscles are flight muscles; the pectoralis and

supracoracoideus muscles account for 15-25% of total body mass (O'Connor 1995a). In

House Finches the pectoralis alone accounts for 20% of total body mass (O'Connor

1995a). The pectoralis and supracoracoideus muscles seem to be the primary site for

shivering thermogenesis (Dawson and O'Connor 1996).

Acclimation to the cold in House Finches involves a 20% increase in pectoralis lean

dry mass (O'Connor 1995a). It is thought that increases in winter muscle mass may result

in greater cold tolerance by elevating the capacity for shivering thermogenesis (O'Connor

1995a). Acclimation to cold also involves increasing lipid stores. Lipids are the primary fuel

for shivering thermogenesis. O'Connor (1995a) found that House Finches captured in the

winter had more than twice the amount of fat as those captured in late spring (2.37 g vs.

1.12 g). In addition to the increased lipid stores it is thought that acclimation to the cold

also involves an increased capacity to catabolize lipids in shivering muscles in the winter

through increased citrate synthase and B-hydroxyacyl CoA dehydrogenase activity and

improved ability to mobilize free fatty acids from adipose tissue during winter possibly

through changes in glucagon/insulin ratios (O'Connor 1995b).

There are two types of shivering patterns in birds: bursting and continuous. The

bursting pattern involves alternating periods of electrical silence and bursts of motor unit

activity (Dawson and O'Connor 1996). Sustained electrical activity with changes in

intensity are characteristic of the continuous pattern of shivering (Dawson and O'Connor

1996). The difference between bursting and continuous patterns may be a genetic

distinction. Hohtola and Stevens (1986) observed that changes in acclimation states, age, and ambient temperature did not cause a shift from one pattern to the other in the birds in this study.

The increase in electomyographic (EMG) activity with decreasing ambient temperatures has been demonstrated in numerous studies (Dawson and O'Connor 1996). Mean rectified voltage calculated from electromyograms was used in studies of domestic pigeons (*Columba livia*) and is thought to be the best indication of heat production (Hohtola 1982). EMG activity changes accounted for only half of the change in heat production, however. In addition, at low temperatures where shivering was high the relationship between EMG activity and ambient temperature was less apparent (Dawson and O'Connor 1996). Nonshivering thermogenesis could account for the remaining heat production (Dawson and O'Connor 1996). Or the EMG electrodes may not have detected the recruitment of motor units farther away from electrode sites in the same muscle or different muscles (Dawson and O'Connor 1996). For example, in House Finches the pectoralis muscles started shivering much sooner than the leg muscles (Carey et al 1989). In bantam hens of domestic fowl (*Gallus gallus*) the leg muscles started shivering sooner than the pectoralis (Dawson and O'Connor 1996). Recruitment of shivering muscles with decreasing temperature is an area that warrants additional study.

Nonshivering Thermogenesis  Nonshivering thermogenesis (NST) is a less understood process in birds. NST in mammals has been studied much more extensively. NST involves the activation of enzymes involved in fat metabolism to catalyze the breakdown and oxidation of fats to produce heat (Eckert 1988). Only a small amount of the energy released is conserved as ATP. Most of it is given off as heat. The site of NST in mammals is brown adipose tissue (BAT) which is usually deposited between the shoulders and the neck. It is highly vascularized tissue rich in mitochondria that is specially adapted to rapidly produce large amounts of heat (Eckert 1988).

Prolonged exposure to cold results in a change in the amount of several circulating

hormones and transmitters (Rothwell et al 1990). Increased sympathetic activity seems to be the direct cause of NST. Norepinephrine, released by the sympathetic nervous system, binds to receptors on the adipose cells of BAT. Thermogenesis is triggered by a second messenger system, and proceeds by two different mechanisms (Eckert 1988). The first mechanism involves normal mitochondrial function and increased breakdown of ATP (Rothwell et al 1990). In response to the sympathetic signal ATP utilization for cellular functions increases in BAT fat cells. Horwitz (1979) indicates that the enzyme sodium-potassium ATPase, a membrane bound sodium potassium adenosine triphosphatase, increased in activity in response to noradrenaline in cold adapted animals and is likely involved in BAT thermogenesis. Increased activity of this enzyme increases the rate of ion pumping by the cell membrane which increases the rate of ATP hydrolysis for work and heat (Horwitz 1979).

The second mechanism of NST involves the uncoupling of ATP synthesis from respiratory chain oxidation. In oxidative phosphorylation, as electrons are moved down the electron transport chain a proton gradient is formed across the inner mitochondrial membrane. These protons then flow down their concentration gradient through specific channels and the energy released is used to synthesize ATP from ADP and Pi. In BAT there is a separate pathway for the protons allowing them to leak across the membrane (Rothwell et al 1990). When protons travel down this alternate pathway their energy is not utilized to synthesize ATP. After crossing the membrane the protons oxidize substrate oxygen in the mitochondria to produce water and heat (Eckert 1988). BAT temperature increases significantly during thermogenesis. The extensive vascularization in BAT distributes the heat to the rest of the body. Acclimation to cold in mammals involves an increase in BAT deposits causing a gradual shift from shivering to nonshivering thermogenesis at low ambient temperatures (Eckert 1988).

In birds the mechanism for nonshivering thermogenesis is not thoroughly understood. Brown adipose tissue is not present in birds so NST must occur at another

site, if it in fact occurs in birds (Dawson and O'Connor 1996). In addition, catacholamines, such as epinephrine, norepinephrine, and dopamine, do not increase during chronic cold exposure and are not thought to be involved in thermogenesis (Dawson and O'Connor 1996). So to understand the mechanism of NST in birds the site and mediator need to be identified. Several studies involving Japanese quail (*Corturnix* sp.), Muscovy ducklings (*Cairina moschata*), and King Penguin chicks (*Aptenodytes patagonicus*) have indicated that glucagon rather than catacholamines is the mediator of NST in birds (Dawson and O'Connor 1996). This conclusion is based on the greater increase in metabolic rate observed in the cold acclimated birds than the warm acclimated birds after ingestion with glucagon. To determine if glucagon has this same effect in other birds species more tests need to be carried out. Another study involving Muscovy ducklings and King Penguin chicks indicated that skeletal muscle may be a potential site for NST (Dawson and O'Connor 1996). Increased capacity for oxidative metabolism, mitochondrial calcium recycling and reduced coupling of oxidation and phosphorylation were observed in the skeletal muscle of cold-acclimated birds (Dawson and O'Connor 1996).

Barre et al (1989) proposed a mechanism for NST that resembles the mechanism involving BAT in mammals though the mediator and site are different in avian NST. It is thought that a release of free fatty acids triggered by the lipolytic action of glucagon may lead to uncoupling of oxidative phosphorylation and electron transport in skeletal muscle mitochondria, producing metabolic heat without contraction (Dawson and O'Connor 1996). The thermogenic action of skeletal muscle might conflict with the contractile action. To be effective there must be a way for skeletal muscles to produce heat by NST in the cold and to maintain the ability to contract. Duchamp et al (1991) proposed a method that involves two distinct populations of muscle mitochondria with two different functions. Barre et al (1989) observed that one population, subsarcolemmal mitochondria, was more affected by the cold than the second population, intermyofibrillar mitochondria. Subsarcolemmal mitochondria are involved in supplying the energy needs of

phosphorylating substrates and sarcolemmal proteins and may be the source of NST
(Dawson and O'Connor 1996). Intermyofibrillar mitochondria are more involved in the
maintenance of the contraction-relaxation cycle. The separation of these mitochondrial
populations would allow NST to occur within skeletal muscle without affecting its
mechanical capacity (Dawson and O'Connor 1996). These studies are certainly not
conclusive and more research is needed to determine the mediator, site, and mechanism of
avian NST.

## SDA

SDA is an increase in metabolism following feeding and is often considered as the
cost of processing a meal which includes the mechanical and biochemical processes
associated with digestion and assimilation such as peristalsis, digestive enzyme synthesis,
and active transport (Rubner 1902, Brody 1945). SDA has been documented in insects,
mollusks, fish, reptiles, mammals, and birds (reviewed by Chappell et al 1997).

Recent studies indicate that protein synthesis is the most important cause of SDA.
One of these studies by Brown and Cameron (1990) involved the infusion of essential
amino acids into channel catfish to determine the contribution of protein synthesis to SDA.
Protein synthesis requires four ATP to bond one amino acid to the next. Brown and
Cameron predicted a relationship between oxygen consumption and the disappearance of
amino acids. In fish amino acids in the plasma increase after ingestion of a meal (Brown
and Cameron 1990). Energy is required to incorporate the amino acids into proteins. To
test for the relationship between SDA and protein synthesis Brown and Cameron (1990)
induced SDA while simultaneously inhibiting protein synthesis. They used the translational
inhibitor, cyclohexamide, which prevents protein synthesis without seriously altering whole
body metabolism when applied in vivo (Brown and Cameron 1990). Following infusion of
amino acids oxygen consumption rose from 34 ml/kgh to a peak of 53 ml/kgh (Brown and
Cameron 1990). When cyclohexamide was administered before amino acid infusion this

increase in oxygen consumption was not observed. Cyclohexamide prevents protein synthesis at the stage where amino acids are being used to form new tissue (Brown and Cameron 1990) All other processes leading to peptide bond formation are unaffected. Therefore, the disappearance of SDA with administration of cyclohexamide provides strong evidence that protein synthesis is responsible for the majority of SDA (Brown and Cameron 1990).

It follows then that animals eating high protein foods would experience a greater SDA than those eating lipid or carbohydrate rich foods. In mammals, SDA as a percent of gross energy intake (GEI) for proteins, complex carbohydrates, lipids, and simple carbohydrates is 31%, 23%, 17%, and 6%, respectively (Ricklefs 1974). Using these values and the fraction of these components in the sunflower meats I calculated an expected SDA of 23% of GEI (Table 1). The actual values from my data were significantly less. At a temperature of 30 C which is within the thermoneutral zone where the total SDA should be apparent, I observed an SDA in two birds, RY and RO, of only .25 kJ and .58 kJ respectively, which represented 2.93% and 5.30% of GEI (Figures 6,7, Table 3). The third bird in my study, RB, had a greater metabolic rate when it was fasted than when it was fed (Figure 8). These values are significantly less than the 23% of GEI predicted by Ricklefs (1974).

SDA is dependent on the amount of food eaten as well as the type of food. The more food ingested the greater the cost of processing it and thus the greater the magnitude of the SDA. The SDA of the small meals eaten by the birds may have been too small to be accurately measured, resulting in the discrepancy between my data and Ricklefs'. I calculated the field metabolic rate according to body mass to be 83 kJ/day (Nagy 1987). In this experiment the meals eaten by the birds averaged .39 g which accounts for 8.4% of the daily energy requirements.

Birds typically eat small frequent meals and they often store their food in a crop where it is released slowly and incrementally for actual digestion (Chappell et al 1997). This

makes it difficult to distinguish between SDA and metabolic increases due to activity and is one of the reasons that SDA has not been studied extensively in birds. I attempted to train the birds in my study to eat two large meals a day. I allowed them access to the food for only short periods of time hoping they would be hungry enough between meals to eat a large portion when the food was available. When I conducted a run birds were fasted approximately four hours before a run. It was assumed that their crops were empty after this time. The fasted birds were denied one meal and the fed birds were given access to 5 grams of food for a period of one hour during the run. My training methods were not entirely effective. A number of runs had to be repeated because the birds to be fed did not eat at all during the one hour period that the food was available and those that did eat ate only .26 - 1 g. The SDA of these small meals may not have been significant enough to be distinguished from the fluctuations in metabolic rate due to activity or other factors. These fluctuations may have interfered in the measurements of SDA accounting for the inconsistency between the Ricklefs' predicted SDA as %of ingested energy according to composition and my observed SDA. I am also unsure of the effects of meal training on the birds. In training the birds to eat large meals instead of nibbling I was altering their normal feeding patterns. The effect of this change on their metabolic processes is unknown. It was assumed that meal feeding would simply concentrate SDA in a shorter period making it easier to measure. The unknown effects of this is a potential source of error in this study.

Though the average SDA of 4.12% of GEI was significantly less than that predicted by Ricklefs it was not far off from those reported in a number of more recent studies of SDA. In House Wren chicks eating crickets the SDA was 6.3% of ingested energy (Chappell et al 1997). Harbor seals (*Phoca vitulina*) eating fish had an SDA that was 4.7% (Ashwell-Erickson et al 1981) and 5.1-9.0% (Markussen et al 1994) of ingested energy. Sea otters (*Enhydra lutris*) eating squid and clam had SDA's that were 13.2% and 10%, respectively, of ingested energy (Costa and Kooyman 1984). Eurasian Kestrels eating mice showed an SDA that was 12.9% of ingested energy (Masman et al 1989). Adelei Penguin

chicks (*Pygocelis adeliae*) eating euphasid crustacean had as SDA that was 10% of ingested energy (Janes and Chappell 1995). These smaller SDA values may be a reflection of the type of food eaten or it may be that Ricklefs' data, based on rabbits, dogs, and humans, are not accurate predictions of SDA in these animals.

## Substitution of Heat for Thermoregulatory Costs

A number of studies have indicated that heat produced by activity and SDA may replace the heat production of shivering and nonshivering thermogenesis in some animals at low temperatures (Dawson and O'Connor 1996). In theory, substitution makes excellent sense. It would allow an animal to increase its efficiency in utilizing energy. Energy that would otherwise be lost as a by-product of activity or as an unavoidable cost of processing a meal could be put to use to help the animal keep warm at low temperatures. Substitution of SDA for thermoregulatory costs would allow the animal to conserve the energy sources it would otherwise have to mobilize for thermogenesis. The animal would have to spend less time and energy foraging for food and could devote more time and energy to activities such as reproduction, thus potentially allowing the animal to increase its overall fitness.

Activity Metabolism Avian activities of running and flying often involve a five to ten fold increase in metabolic rates (Dawson and O'Connor 1996). Studies done on the potential role of heat generated from activity in substitution for the cost of thermoregulation at low temperatures are not consistent across species or temperatures. No substitution was evident in the Common Redpoll at temperatures 0 to -30 C, but complete substitution was observed at -45 C (Pohl and West 1973). In the White Crowned Sparrow (*Zonotrichia leucophrys gambelii*), substitution was observed to increase with a decrease in temperature until -10 C where substitution was complete (Paladino and King 1984). Substitution was also observed in Verdins (*Auriparus flaviceps*) (Webster and Weathers 1990), Yellow-eyed Juncos (*Junco phaenotus*), and Dark-eyed Juncos (Weathers and Sullivan 1993).

SDA Studies done on the possible substitution of SDA for regulatory thermogenesis show similar inconsistencies across species and temperatures, particularly in birds. Studies on mammals have yielded good evidence of substitution. In golden hamsters SDA decreases with decreasing temperature pointing to substitution of SDA for thermoregulatory costs (Simek 1975). SDA is apparent in muskrats (*Ondatra zibethicus*) in a dry thermoneutral environment, but not in cold water (MacArthur and Campbell 1994). Substitution of SDA for exercise heat production appears to occur in sea otters following a meal (Costa and Kooyman 1984). Evidence of substitution of SDA for thermoregulatory costs is not as clear in birds. Substitution was reported in Eurasian Kestrels (Masman et al 1989), domestic fowl (Berman and Snapir 1965), European Starlings (Biebach 1984), and House Wren chicks (Chappell et al 1997). The European Starlings did not show substitution at some temperatures (10 C) below the lower critical temperature, however (Biebach 1984). In addition, no substitution was observed in Arctic Tern chicks (Klassen et al 1989).

I did not observe substitution in House Finches. There was not a consistent decline in SDA with a decrease in ambient temperature (Figure 9). Average SDA at 30 C was 4.12% of GEI (Table 3). At 15 C it was 8.1% if RO is included and .5% if RO is not included (Table 3). At 5 C average SDA was 3.68% at 5 C (Table 3). Though there doesn't seem to be a consistent trend across all three temperatures I think that an important comparison can be made between the SDA values for two of the birds, RO an RY, at 30 C and 5 C. At 30 C these two birds show an SDA of 2.93% and 5.30% of ingested energy and at 5 C they show an SDA of 2.62% and 4.75% of ingested energy, respectively (Table 3). SDA and RMR at these two temperatures were almost identical. The average SDA at 30 C and 5 C is also similar, 4.12% and 3.68%, respectively. This supports my null hypothesis, SDA is not utilized to aid in thermoregulation.

Based strictly on my data, SDA does not appear to substitute for the costs of thermoregulation in House Finches. Heat production is supplied primarily by shivering thermogenesis and perhaps nonshivering thermogenesis and the energy of SDA can be

considered an unavoidable cost of processing a meal.

One factor that may have affected my test for substitution, however, is that the birds were housed at 20 C. They were not acclimated to cold temperatures as in the wild during winter. It is possible that the mechanism responsible for substituting SDA for thermoregulatory costs is not activated unless the birds are acclimated to the cold. I caught the birds during the summer and while in captivity they were never exposed to prolonged periods of cold.

As discussed earlier, the SDA measurements at 30 C (in the thermoneutral zone) were lower than I expected. The small meals eaten by the birds may not have been enough to distinguish the SDA from fluctuations in the metabolic rate due to activity or other factors. This could have masked any role of SDA in regulatory thermogenesis.

This study only included three birds. My goal was to have 6 to 8 birds, but I lost a few birds and I was not able to catch any more. A larger sample size would have given a more accurate representation of SDA and of the effect of temperature on SDA. I may have been able to observe more consistent trends if I had included more birds in my study.


## Conclusions

The cost of thermoregulation in House Finches at 5 C was measured as .59 kJ/hr requiring a 35.8% increase in metabolism and accounting for 17% of FMR. This is somewhat less than the 20-40% reported for free-living birds by Webster and Weathers (1990), but it was measured in captivity when the birds were not cold acclimated. SDA averaged 3-5% of GEI. No effect of temperature on SDA was observed. No evidence was found to support the substitution of SDA for costs of thermoregulation in House Finches. Potential sources of error include the small meals eaten by the birds, the unknown effects of altering the feeding pattern of the birds, the possible storage of food in the crop during a run, and the unacclimated state of the birds.

## References

Aschoff, J. and H. Pohl. 1970. Der ruheumsatz von Vogeln als funktion der tageszeit und der korergrosse. J. Ornithol. 111:38-47.

Ashwell-Erickson, S. and R. Elsner. 1981. The energy cost of free existence for Bearing Sea harbor and spotted seals. *In*: Eastern Bearing Sea shelf: oceanography and resources. vol. 2., eds. D. Hood, and J. Calder, pp. 869-899. University of Washington Press, Seattle.

Barre, H., C. Duchamp, J. Rouanet, A. Dittmar, and G. Delhomme. 1989. Muscular nonshivering thermogenesis in cold acclimated ducklings. *In:* Physiology of cold adaptation in Birds, eds. C. Bech and R. Reinertsen, pp. 95-104. New York: Phlenum.

Berman, A. and N. Snapir. 1965. The relation of fasting and resting metabolic rates to heat tolerance in the domestic fowl. Brit. Poultry Sci. 6:207-216.

Biebach, H. 1984. Effect of clutch size and time of day on energy expenditure of incubating starlings, *Sturnus vulgaris*. Physiol. Zool. 57:26-31.

Brody, S. 1945. Specific dynamic action and efficiency of productive processes. *In:* Bioenegetics and growth. New York: Reinhold.

Brown, C. and J. Cameron. 1990. The induction of specific dynamic action in channel catfish by infusion of essential amino acids. Physiol. Zool. 64:276-297.

Calder, W. and J. King. 1974. Thermal and caloric relations of birds. *In*: Avian · Biology Vol. IV, eds. J.R. King and D.S. Farmer. New York: Academic.

Carrey, C., R. Marsh, A. Bechoff, R. Johnston, and A. Olin. 1989. Enzyme activities in muscles of seasonally acclimated House Finches. *In:* Physiology of Cold Adaptation in Birds, eds. C. Bech and R. Reinertsen, pp. 95-104. New York: Phlenum.

Chappell, Mark A., G. Bachman, and A. Hammond. 1997. The heat increment of feeding in house wren chicks: magnitude, duration, and substitution for thermostatic costs. J. Comp. Physiol. 167:313-318.

Costa, D. and G. Kooyman. 1984. Contribution of specific dynamic action to heat balance and thermoregulation in the sea otter *Enhydra lutris*. Physiol. Zool. 57:199-203.

Dawson, W. and T. O'Connor. 1996. Energetic features of avian thermoregulatory responses. In: Avian energetics and Nutritional Ecology, ed. Carey C., Pp. 85-124. New York: Chapman & Hall.

Eckert, R. 1988. Animal physiology: mechanisms and adaptations. 3rd edn. W.H. New York: Freeman.

Ellis, H. 1980. Metabolism and solar radiation in dark and white herons in hot climates. Physiol. Zool. 53:358-372.

Hails, C. 1983. The metabolic rate of tropical birds. Condor 85:61-65.

Hill, G.E. 1993. House Finch, *Carpodacus mexicanus. In:* The birds of North America, No. 46, eds. A. Poole and F. Gill. Philadelphia: The Academy of Natural Sciences; Washington, D.C.: The American Ornithologists' Union.

Hotohla, E. 1982. Thermal and electromyographic correlates of shivering thermogenesis in the pigeon. Comp. Biochem. Physiol. 73A:159-166.

Hohtola, E. and E. Stevens. 1986. The relationship of muscle electric activity, tremor and heat production to shivering thermogenesis in Japanese Quail. J. Exp. Biol. 125:119-135.

Horwitz, B. 1979. Cellular events underlying catecholamine-induced thermogenesis: cation transport in brown adipocytes. Fed. Proc. 38:2170-76.

Hudson, J. and S. Kimzey. 1966. Temperature regulation and metabolic rhythms in populations of the House Sparrow, *Passer domesticus.* Comp. Biochem. Physiol. 17:203-217.

Janes, D. and M. Chappell. 1995. The effect of ration size and body size on specific dynamic action in Adelei Penguin chicks, *Pygocelis adeliae* . Physiological Zoology. 68:1029-1044.

Klassen, M., C. Bech, and G. Slagsvold. 1989. Basal metabolic rate and thermal conductance in Arctic tern chicks and the effect of the heat increment of feeding on thermoregulatory expenses. Ardea 77:193-200.

Kleiber, M. 1975. The fire of life: an introduction to animal energetics. Huntington: Kreiger Publishing.

MacArthur R. and K. Campbell. 1994. Heat increment of feeding and its thermoregulatory benefit in the muskrat, *Ondatra zibethicus.* J. Comp. Physiol. B. 164:141-146.

Markussen, H. and M. Ryg, and N. Oritsland. 1994. The effect of feeding on the metabolic rate in harbour seals, *Phoca vitulina.* J. Comm. Physiol. B. 164:89-93.

Masman, D., S. Daan, and M. Dietz. 1989. Heat increment of feeding in the Kestrel, *Falco tinnunculus,* and its natural seasonal variation. *In:* Physiology of cold adaptation in birds, eds. C. Bech and R. Reinertsen, Pp. 123-135. New York: Phlenum.

Nagy, K. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecological Monographs. 57:111-128.

O'Connor, T. 1995a. Metabolic characteristics and body composition in house finches: effects of seasonal acclimation. J. Comp. Physiol. 165:298-305.

O'Connor, T. 1995b. Seasonal acclimatization of lipid mobilization and catabolism in house finches, *Carpodacus mexicanus*. Physiol. Zool. 68:985-1005.

Paladino, F. and J. King. 1984. Thermoregulation and oxygen consumption during terrestrial locomotion by White-crowned Sparrows, *Zonotrichia leucophrys gambelii*. Physiol. Zool. 57:226-236.

Pohl, H. and G. West. 1973. Daily and seasonal variation in metabolic response to cold during rest and forced exercise in the Common Redpoll. Comp. Biochem. Physiol. 45A:851-867.

Ricklefs, R. 1974. Energetics of reproduction in birds. *In*: Avian energetics, ed. R. Paytner. Cambridge: Nuthall Ornithological Club.

Root, T., T. O'Connor, and W. Dawson. 1991. Standard metabolic level and insulative characteristics of eastern House Finches, *Carpodacus mexicanus* (Muller). Physiol Zool. 64:1279-1295.

Rothwell, N., M. Stock, and D. Stribling. 1990. Diet induced thermogenesis. *In*: Thermoregulation: Physiology and Biochemistry. New York: Pergamon Press.

Rubner, M. 1902. Die gesetze des energieverbrauschs bei der ernahrung. Franz Deuticke, Leipzig and Vienna.

Schmidt-Nielson, K. 1975. Animal physiology, adaptation and environment. New York: Cambridge UP

Simek, V. 1975. Specific dynamic action of a high-protein diet and its significance for thermoregulation in the golden hamster. Physiol. Bohemoslav. 24:421-424.

Weathers, W. 1979. Climatic adaptation in avian standard metabolic rate. Oecologia 42:81-89.

Weathers, W. and K. Sullivan. 1993. Seasonal patterns of time and and energy allocation by birds. Physiol. Zool. 66:511-536.

Webster, M. and W. Weathers. 1990. Heat produced as a by-product of foraging activity contributes to thermoregulation by verdins, *Auriparus flaviceps*. Physiol. Zool. 63:777-794.