The Auk 111(1):170-177, 1994

SUCROSE INTOLERANCE IN BIRDS: SIMPLE NONLETHAL DIAGNOSTIC METHODS AND CONSEQUENCES FOR ASSIMILATION OF COMPLEX CARBOHYDRATES

HYLARY L. MALCARNEY, CARLOS MARTÍNEZ DEL RIO, AND VICTOR APANIUS

Department of Ecology and Evolutionary Biology, Princeton University, Princeton, New Jersey 08544, USA

ABSTRACT.—Gray Catbirds (*Dumetella carolinensis*, Mimidae) and Purple-headed Glossy-Starlings (*Lamprotornis purpureiceps*, Sturnidae) showed depressed ingestion and increased fecal sugar contents when shifted from glucose and fructose to sucrose solutions. These species also exhibited no increases in plasma glucose after ingestion of sucrose, but an increase in plasma glucose after ingestion of equicaloric doses of a mixture of glucose and fructose. *In vitro* measurements of intestinal disaccharidase activities in *D. carolinensis* revealed insignificant sucrase activity, and low levels of maltase activities. These results support the hypothesis that sucrose intolerance is a shared-derived character of the monophyletic lineage that includes starlings, mimids, and thrushes, and indicate that sucrose intolerance in birds can be easily diagnosed with a combination of behavioral and nonlethal physiological measurements. We suggest that, in birds, low intestinal maltase activity is correlated with the lack of sucrase activity. We further hypothesize that sucrose-intolerant birds are poor at assimilating complex carbohydrates. *Received 1 March 1993, accepted 17 November 1993.*

PROXIMATE NUTRIENT ANALYSIS distinguishes between two broad classes of carbohydrates: structural carbohydrates, largely composed of β-1,4 polysaccharides like cellulose, and soluble carbohydrates including mono- and disaccharides and α-1,4 and α-1,6 polysaccharides like starch, amylopectin, and glycogen (Whistler and Daniel 1985). Most vertebrates have endogenous enzymes that can hydrolyze the α linkages in starch and glycogen, but do not possess enzymes capable of breaking the β linkages of cellulose and hemicellulose (Vonk and Western 1984). Vertebrates that assimilate the structural carbohydrate component in food have to rely on time consuming microbial fermentation (Stevens 1988). Structural carbohydrates are generally considered "hard" to assimilate whereas soluble carbohydrates are considered "easy" to assimilate (Prop and Vulink 1992). Here we present data showing that this pattern has exceptions and that some soluble carbohydrates may be extremely difficult or impossible to assimilate by a broad group of birds.

Martínez del Rio et al. (1992) demonstrated that even subtle differences in the chemical structure of soluble carbohydrates can result in differences in bird digestive-utilization efficiency, in preferences among these substances, and presumably in differential utilization by birds of food plants containing different soluble carbohydrate compositions. Nectar and fruit pulp contain sucrose, glucose, and fructose in varying proportions (Baker and Baker 1983, Baker et al. 1993). The chemical differences between these sugars are relatively small. Sucrose is a disaccharide of glucose and fructose, and the monosaccharides glucose and fructose differ only in the position of the carbonyl group. To be assimilated, sucrose has first to be hydrolyzed into its components—glucose and fructose—by the intestinal enzyme sucrase. Glucose and fructose are absorbed directly by the intestine (Alpers 1987). Some frugivorous birds lack intestinal sucrase activity and cannot split the sucrose bond between glucose and fructose (e.g. Martínez del Rio and Stevens 1989). For these species, sucrose is a useless energy source that can cause osmotic diarrhea and a consequent feeding aversion (Brugger and Nelms 1991). These same sucrose-intolerant birds avidly ingest and profit from glucose and fructose.

Martínez del Rio (1990) reported that sucrase is lacking in several species of birds in the families Sturnidae (starlings) and Muscicapidae (thrushes). DNA-DNA hybridization data suggest that these families are part of a monophyletic lineage (Sibley and Ahlquist 1990). These data also suggest that members of the Mimidae (catbirds, mockingbirds and thrashers) are the closest living relatives of starlings (Sibley and

Ahlquist 1984, 1990). Martínez del Rio (1990) hypothesized that lack of sucrase is a shared-derived trait of the monophyletic lineage that includes starlings, thrushes, and catbirds (the sturnid-muscicapid lineage sensu Sibley and Ahlquist 1990). Here we explore behavioral and physiological correlates of sucrose ingestion in two species of fruit-eating birds in this lineage: Gray Catbirds (*Dumetella carolinensis*, Mimidae) and Purple-headed Glossy-Starlings (*Lamprotornis purpureiceps*, Sturnidae). Based on the phylogenetic affinities of Gray Catbirds and Purple-headed Glossy-Starlings, we predicted that they would: (1) lack intestinal sucrase activity; (2) be unable to assimilate sucrose; and (3) reject sucrose in feeding trials.

Maltose and isomaltose are the most abundant products of the hydrolysis of starch by salivary and pancreatic amylases. Maltose is hydrolyzed by two independent enzyme systems: sucrase-isomaltase and maltase-glucoamylase (Semenza and Auricchio 1989). Because sucrase-isomaltase is a powerful maltase, its deficiency in humans is often associated with reduced ability to utilize dietary starch (Auricchio et al. 1963, 1972). Isomaltase is hydrolyzed by sucrase-isomaltase, and to a small degree by maltase-glucoamylase. Martínez del Rio (1990) suggested that birds lacking intestinal sucrase also would be poor hydrolyzers of maltose. We predicted that, if sucrase activity were low or missing in *D. carolinensis*, both maltase and isomaltase activity would be low.

Materials and Methods

Sucrose intolerance in vertebrates is most commonly caused by the absence of intestinal sucrase activity (Gudman-Høyer et al. 1984). Measuring sucrase activity requires biochemical analysis of intestinal tissue (Dahlqvist 1984). Obtaining samples of intestinal tissues from live small birds is not yet practical and, consequently, measuring sucrase activity requires sacrificing birds and extracting the intestine (Martínez del Rio 1990). To diagnose sucrose intolerance in *D. carolinensis* and *L. purpureiceps*, we used two nonlethal and minimally invasive methods. The first method relies on sequentially feeding birds a 1:1 mixture of glucose and fructose, and then offering them sucrose. Sucrose-intolerant birds should show depressed ingestion and increased fecal sugar contents when shifted from glucose and fructose to sucrose. The second method involves measuring the increase in plasma glucose after birds have been challenged with an oral dose of sucrose. Sucrose-intolerant birds should show nil increases in plasma glucose. This latter method is a modification of a test commonly used to diagnose disaccharide intolerance in humans (Isokosi et al. 1972, Krasilnikoff et al. 1975). We validated these methods in *D. carolinensis* by measuring the activity of intestinal disaccharides (including sucrase) in vitro.

Dumetella carolinensis (mean mass = 36.3 ± SD of 4.0 g, $n = 9$) are among the most frugivorous birds in temperate North America (Martin et al. 1951, Bent 1948). The fraction of fruit in Gray Catbird diets can be as high as 95% during the fall and winter (Beal 1897, Blake and Loiselle 1992), and is reduced only during the breeding season when they become mainly insectivorous (White and Stiles 1990, Helmy and Martínez del Rio unpubl. data). *Lamprotornis purpureiceps* (mean mass = 79.4 ± 10.6 g, $n = 4$) are gregarious forest birds widely distributed in West and Central Africa (Nigeria, Gabon, Central African Republic, and Uganda; Mackworth-Praed and Grant 1955, Sibley and Monroe 1990). Their diet apparently also consists largely of fruit (Beecher 1978, Serle et al. 1977).

We captured eight immature *D. carolinensis* with mist nets in early October 1991 at the Hutchinson Memorial Forest, New Jersey, and at a secondary field adjacent to the Sourland Mountain State Park, New Jersey (birds aged following Suthers and Suthers 1990). Four *L. purpureiceps* (two males and two females) were obtained on loan from the Bronx Zoo. Birds were individually housed in 48 × 48 × 48 cm cages at 21°C and on a 12L/12D daily cycle. Food and water were supplied *ad libitum* except during experiments. Birds were fed a mixed diet of Ziegler soft-billed bird diet (Ziegler Bros. Inc, Gardners, Pennsylvania) and banana mash (a mixture of mashed ripe bananas, vegetable oil, soy protein isolate complemented with methionine, and a vitamin supplement in an agar-based gel; Denslow et al. 1987). All experiments were conducted from October 1991 to February 1992. At the end of the experiments, *L. purpureiceps* individuals were returned to the Bronx Zoo. The surviving *D. carolinensis* individuals were released in August 1992.

We conducted all behavioral tests at the onset of the light period (0800 EST). In each trial we removed food and water, and lined the bottom of the cages with teflon-coated plastic to facilitate excreta collection. Each test consisted of four 2-h trials conducted in four successive days. We offered birds sugar solutions in glass tubes consisting of an upper 42-cm section and a lower 7-cm section bent upward at a 45° angle. Birds drank from an elliptical hole (2 × 1.5 cm) at the distal end of the lower portion of the tube. In the first two trials we presented birds with a tube containing a 1:1 mixture of glucose and fructose (15% mass/total volume). On days 3 and 4, birds were presented with an equicaloric solution of sucrose. At the end of each trial we measured the amount of test solution consumed and collected 5 to 10 samples of excreta from the bottom of each cage. We measured fecal sugar from these samples with a temperature

compensated refractometer (Reichter-Jung 10431). Although urates and fecal materials can make refractometer readings innacurate, the refractive index of bird fecal samples provides a consistent relative indicator of sugar excretion (Martínez del Rio et al. 1989, Brugger and Nelms 1991). We report fecal sugar concentration in percent of sucrose (Brix = mass of sugar per volume of solution; Bolten et al. 1979).

We measured the response in plasma glucose levels (PGL) in birds subject to two treatments: a 1:1 glucose : fructose intubation, and a sucrose intubation. Birds were fasted overnight and intubated with 3 g/kg of a 15% (mass/volume) sugar solution. After 30 min we obtained approximately 200 μl of blood by jugular venipuncture using a 0.5-ml syringe with a 28-gauge needle (Hoysak and Weatherhead 1991). We obtained a blood sample after 30 min because preliminary data indicated that plasma glucose peaks between 25 and 35 min after a 1:1 glucose : fructose challenge (see Martínez del Rio et al. 1988: fig. 3). Blood was transferred to chilled heparinized microcapillary tubes and centrifuged for 5 min (IEC microhematocrit centrifuge; see Cohen 1966). We measured plasma glucose after color development (Glucose-Trinder 500 reagent, Sigma Chemical Co.) on a spectrophotometer (Beckman DU-64) set at 505 nm. As an estimate of the response to a glucose : fructose or sucrose challenge we used the difference in PGL 30 min after treatment and PGL in untreated birds fasted overnight. Because we were concerned about stressing birds excessively by repeatedly drawing blood in a short time, we obtained a single fasting plasma measurement per individual. This procedure is justified because in birds fasting PGL remains relatively constant at the temporal scale of our experiments (three weeks; Martínez del Rio and Phillips unpubl. data). Plasma osmolarity was measured on a Wescor 5500 vapor pressure osmometer.

Three *D. carolinensis* individuals were euthanized with a halothane overdose. The small intestine was immediately excised, divided into three sections of equal length, and placed in ice-cold saline (1.02%). We slit each section longitudinally and measured its length and width to obtain an estimate of intestinal nominal area. After weighing, each section was stored in liquid nitrogen. We measured sucrase, maltase, and isomaltase activities in these intestinal samples using a previously described method (Martínez del Rio et al. 1988) from Dahlqvist (1984). Martínez del Rio (1990) provided details of the disaccharide assay. Briefly, tissues were homogenized in 350 mM mannitol in 1 mM Hepes/KOH, pH 7.5 (30 s at OMNI 5000 homogenizer setting 6), and tissue homogenates (100 μl) were incubated at 40°C (Prinzinger et al. 1991) with 100 μl of 56 mM sugar (sucrose, maltose, and isomaltase) solutions in 0.1 M maleate/NaOH buffer, pH 6.5 for 10 min. After incubation, reactions were arrested by adding 3 ml of a stop/develop reagent (one bottle of Glucose-Trinder 500 reagent [Sigma Chemical Co.] in 250 ml 1.0 M Tris/HCl, pH 7, plus 250 ml 0.5 NaH_2PO_4/Na_2HPO_4, pH 7). Glucose standards (0–120 μg in 200 μl 0.1 M sodium maleate buffer, pH 7) were reacted with the stop develop reagent to obtain a standard curve. Protein concentration in tissue homogenates was measured using the Bio-Rad kit (Bio-Rad, Richmond, California) with bovine serum albumin standards. To allow comparison with other intestinal disaccharidase studies we calculated disaccharidase activities using three different standardization procedures: activity per unit intestinal area as $\mu mol \cdot (min)^{-1} \cdot (cm^2 \text{ nominal area})^{-1}$; activity per gram of protein as $\mu mol \cdot (min)^{-1} \cdot (cm^2 \cdot \text{gram of protein})^{-1}$; and total hydrolytic capacity as $\mu mol \cdot (min)^{-1}$.

Statistical analysis.—Although we report descriptive statistics for both species in all experiments, we only report significance values from inferential statistics tests for data on *D. carolinensis* for which sample size was adequate (eight individuals). The number of individuals of *L. purpureiceps* studied was too small (four individuals) to allow use of inferential statistics. For paired data we used sign tests, which are robust albeit conservative (Mosteller and Tukey 1977). To estimate parameters of linear regressions we used standard least-squares methods. Results are given as $\bar{x} \pm$ SD.

RESULTS

Neither *D. carolinensis* nor *L. purpureiceps* changed consumption of glucose and fructose solution from day 1 to day 2 ($P > 0.1$; Fig. 1). Consumption decreased in all individuals of both species from day 2 to day 3, when birds were shifted from glucose and fructose to sucrose ($P < 0.05$). This decline in consumption continued from day 3 to day 4 in all birds but one glossy-starling, which drank very little of the sucrose solution during either days ($P < 0.05$, Fig. 1). Fecal sugar levels over the four-day trials followed a similar pattern in both species (Fig. 1). Fecal sugar remained low (<2% BRIX) the first two days of the trials (Fig. 1), increased on day 3 when birds were shifted to sucrose solutions ($P < 0.05$), and remained high through day 4 ($P > 0.1$, Fig. 1). These results suggest high assimilation of glucose and fructose, but low assimilation of sucrose.

We found the extremely broad range of concentrations of fasting plasma glucose that seems to be typical of bird species (Hazelwood 1984, Groscolas and Rodriguez 1981, Marsh et al. 1984). Fasting plasma glucose ranged from 260 to 335 mg/100 ml in *L. purpureiceps*, and from 287 to 458 mg/100 ml in *D. carolinensis* (Fig. 2). All individuals showed increased PGL relative to fasting levels 30 min after administration of

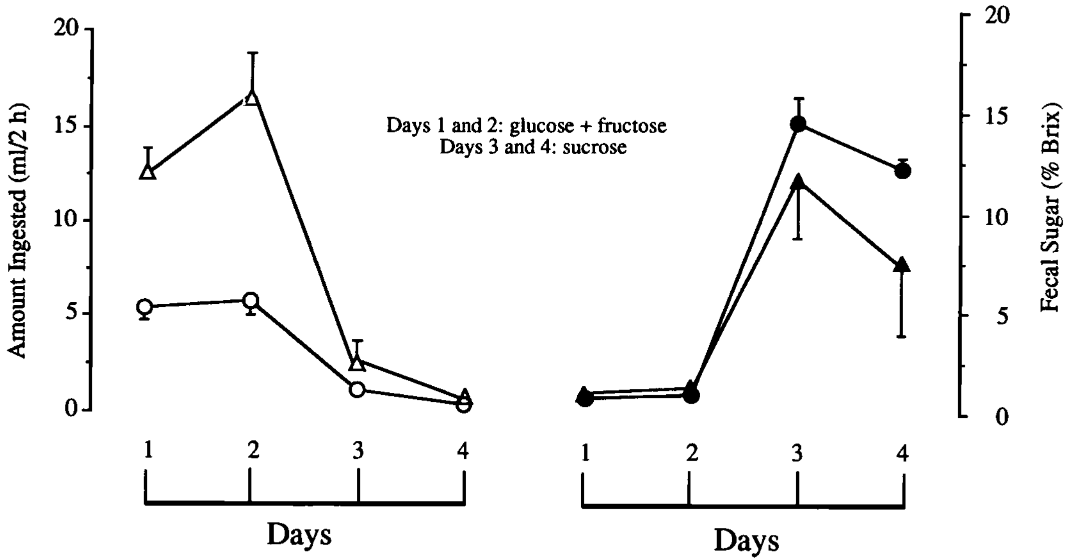

Fig. 1. Ingestion of glucose : fructose (days 1 and 2) and sucrose (days 3 and 4) solutions (open symbols) and fecal sugar concentration after ingestion (closed symbols) in *D. carolinensis* (circles) and *L. purpureiceps* individuals (triangles). Solutions of glucose : fructose and sucrose were equicaloric (15% mass/total volume).

glucose and fructose (Fig. 2; $P < 0.05$). The mean increases above fasting levels in *L. purpureiceps* and *D. carolinensis* were 104.9 ± 19.2 and 191 ± 18.2 mg/100 ml, respectively. In *D. carolinensis* the increase in PGL over fasting levels after administration with glucose and fructose was linearly and negatively correlated with fasting PGL ($Y = 350.9 - 0.7X$; $r = 0.77$, $P < 0.005$), suggesting regulation of maximal plasma glucose concentration. As expected for sucrose-intolerant animals, none of the birds intubated with sucrose showed increased PGL (Fig. 2). Surprisingly, all *D. carolinensis* individuals showed a small (−18.4 ± 3.2 mg/100 ml) but significant (sign test, $P < 0.05$) decrease in PGL 30 min after intubation with sucrose (Fig. 2). Plasma osmolarity did not vary significantly among treatments ($P > 0.05$). Mean plasma osmolarities for *L. purpureiceps* and *D. carolinensis* were 385.2 ± 28.3 and 350.8 ± 8.4 mg/100 ml, respectively.

We found maltase and isomaltase activities in three *D. carolinensis* individuals, but only traces of sucrase activity in two individuals and no detectable sucrase activity in another (Table 1). Sucrase activity was extremely low (less than 0.001 g of sucrose hydrolyzed·h^{-1}·individual^{-1}). Martínez del Rio (1990) reported that maltase was linearly and positively correlated with sucrase activity in a sample of 11 species of passerine birds. As predicted, mean maltase activ-

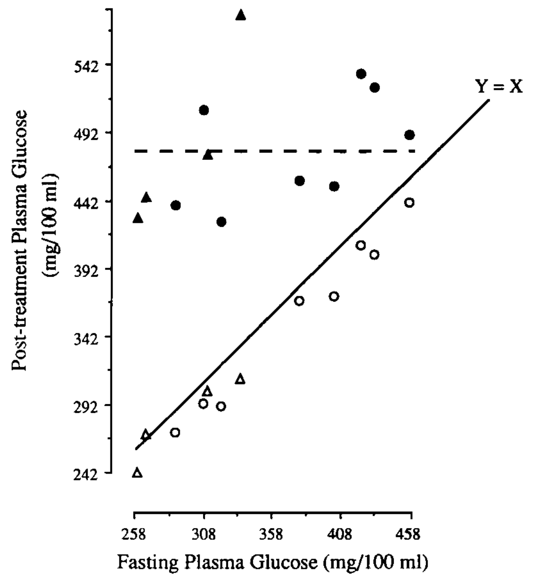

Fig. 2. Plasma glucose concentration after 30 min of glucose : fructose (closed symbols) and sucrose (open symbols) oral doses (3 g/kg in a 15% solution). Circles represent *D. carolinensis* and triangles *L. purpureiceps* individuals. For *D. carolinensis*, mean plasma glucose 30 min after a glucose : fructose dose was 481 ± 14 mg/100 ml and is represented by horizontal dotted line. Identical line ($Y = X$) denoting no posttreatment change in PGL relative to fasting levels is shown as reference.

TABLE 1. Intestinal disaccharidase activities in three individuals of *Dumetella carolinensis* ($\bar{x} \pm$ SD). Disaccharidases measured by glucose liberated after incubating tissue homogenates with 0.28 M substrate at 40°C for 10 min.

	Total activity (μmol·min^{-1})	Activity/area (μmol·min^{-1}·cm^{-2})	Activity/mg of protein (μmol·min^{-1}·g of protein^{-1})
Maltase	28.96 ± 14.12	2.38 ± 0.88	102.03 ± 31.03
Isomaltase	0.29 ± 0.12	0.02 ± 0.01	2.77 ± 1.81
Sucrase[a]	0.04 ± 0.05	0.004 ± 0.001	0.50 ± 0.43

[a] Only two individuals showed detectable sucrase activity.

ity standardized per unit nominal area of intestine ([total maltose hydrolysis]/[total small intestine normal area]) in *D. carolinensis* was low and within the 95% confidence interval of the intercept of the maltase-versus-sucrase regression line for these 11 species (Fig. 3).

DISCUSSION

In analogy with human clinical terminology, we define "sucrose intolerance" in birds as a combination of symptoms including sucrose malabsorption as evidenced by high fecal sugar concentration, aversion to sucrose in sequential feeding trials, and a "flat" response in plasma glucose after sucrose ingestion (Gudman-Høyer et al. 1984). Our results demonstrate that *L. purpureiceps* and *D. carolinensis* are sucrose intolerant, and indicate that in *D. carolinensis* this intolerance is caused by the absence of significant intestinal sucrase activity. The behavioral, fecal sugar, and blood tests appear to be easy nonlethal techniques to diagnose poor sucrose digestion in birds. Care must be exercised in the interpretation of behavioral tests and fecal sugar tests, however. Birds that possess sucrase activity but that are relatively inefficient at digesting sucrose, such as Cedar Waxwings (*Bombycilla cedrorum*), can show sucrose aversion and increased fecal sugar concentration when exposed to sucrose-containing food (Martínez del Rio et al. 1989, K. E. Brugger unpubl. data). Apparently, a reduction in consumption after exposure to sucrose accompanied by increased fecal sugar concentration can be reliably interpreted as caused by inefficient digestion of sucrose, but not necessarily as evidence for lack of intestinal sucrase activity. Table 2 summarizes sugar fecal output in five species of sucrose-fed birds, including Cedar Waxwings. Fecal sugar in these species varies relatively little and does not differ significantly between species lacking sucrase and Cedar Waxwings ($F_{4,28}$ = 1.65, $P > 0.2$), which digest sucrose inefficiently (Martínez del Rio et al. 1989).

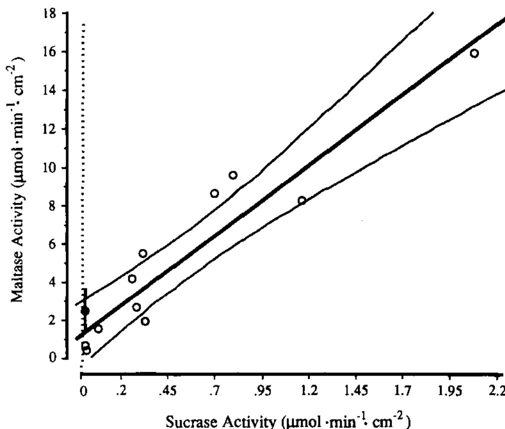

Fig. 3. Linear regression (thick line) and 95% confidence intervals (thin lines) for relationship between intestinal sucrase and maltase activities in 11 species of passerine birds (open circles). Relationship excluding *D. carolinensis* is $Y = 1.4 + 7.4X$; $r = 0.95$; Martínez del Rio 1990). Closed symbol represents average values for *D. carolinensis* (from Table 1). Error bars are SE. Note that point for *D. carolinensis* is within the 95% confidence interval for passerine regression.

Why do these five species exhibit such similar fecal concentrations when fed on sucrose? Assuming that all fecal solutes are undigested sucrose and transforming BRIX% to osmolarity yields a mean fecal osmolarity equal to 380 ± 15 mM. This value is slightly higher than the average value for passerine plasma osmolarity (342 ± 18.5 mM; Skadhauge 1981). Birds with poor or no digestion of sucrose are apparently incapable of concentrating excreta against a concentration gradient to osmolarities much higher than plasma (see Skadhauge 1981:92). Because plasma osmolarity is very similar among small passerine species, fecal sugar after sucrose ingestion should also be similar. A corollary of

TABLE 2. Comparison of fecal sugar in sucrose-fed Gray Catbirds (*Dumetella carolinensis*), Purple-headed Glossy-Starlings (*Lamprotornis purpureiceps*), European Starlings (*Sturnus vulgaris*), American Robins (*Turdus migratorius*), and Cedar Waxwings (*Bombycilla cedrorum*). The latter species exhibits sucrase activity, but is relatively inefficient at digesting sucrose (see Martínez del Rio et al. 1989).

Species	Fecal sugar[a]	Reference
Dumetella carolinensis[b]	14.5 ± 3.4 (8)	This report
Lamprotornis purpureiceps[b]	11.6 ± 2.9 (4)	This report
Sturnus vulgaris	11.5 ± 2.1 (7)	Brugger et al. (1992)
Turdus migratorius	11.6 ± 1.1 (4)	Brugger and Nelms (1991)
Bombycilla cedrorum	12.1 ± 2.6 (10)	Martínez del Rio et al. (1989)

[a] % Brix ± SD (*n*).
[b] Measurements on day 3 (see text).

this hypothesis is that sucrose-intolerant birds should suffer from net water loss when feeding on concentrated sucrose solutions. Indeed, American Robins (*Turdus migratorius*) fed on sucrose dramatically increased water consumption (Brugger and Nelms 1991). In addition, fecal sugar concentration in sucrose-fed European Starlings (*Sturnus vulgaris*) is approximately the same irrespective of the sucrose concentration in food (ca. 11% BRIX; Brugger et al. 1993).

Plasma glucose tests also supported the hypothesis that *D. carolinensis* and *L. purpureiceps* are sucrose intolerant. Although fasting PGL was variable, the negative correlation between fasting PGL and the increase in PGL after a glucose : fructose challenge indicates that in *D. carolinensis* a maximal PGL of about 536 mg/100 ml is defended (Fig. 2). We have no adequate explanation for why PGL showed a significant decrease relative to fasting levels after a sucrose challenge.

In vitro measurements indicated that sucrose intolerance in *D. carolinensis* seems to be caused by lack of intestinal sucrase activity. The magnitude of intestinal sucrase activity detected in *D. carolinensis in vitro* was too low to be of physiological significance and similar to that reported by Martínez del Rio (1990) for two species of thrushes (Muscicapidae). Intestinal maltase and isomaltase were present, albeit at low levels. Mean maltase activity in *D. carolinensis* was within the limits predicted for a bird lacking sucrase activity, supporting the hypothesis that birds unable to digest sucrose also have reduced maltose hydrolyzing abilities. The available data on isomaltase activity in birds is still too scanty to allow comparative conclusions.

To evaluate the possible consequences of reduced maltose hydrolysis for birds lacking intestinal sucrase activity, we calculated the maximal possible contribution of maltose to the energy intake of *D. carolinensis.* In our calculation we assumed that luminal digestion of carbohydrates into oligosaccharides by pancreatic amylases was not limiting. If maltose concentration is at saturating concentration in the gut (K_m for intestinal maltose hydrolysis in *D. carolinensis* is about 3 mM; Martínez del Rio unpubl. data) then *D. carolinensis* individuals can hydrolyze 0.6 ± 0.1 g maltose/h, which provides 9.6 ± 1.6 kJ/h (assuming 16 kJ/g of maltose; Weast and Selby 1967). The predicted field metabolic rate of a *D. carolinensis* individual weighing 36.3 g is 131 kJ/day (Nagy 1987). During a 12-h day, catbirds can hydrolyze a maximum of about 115 kJ maltose/day, or 88% of their metabolic needs. *Dumetella carolinensis* individuals apparently cannot meet their energetic demands from maltose alone. Their capacity to hydrolyze maltose is less than that required to fuel metabolism. Measurements on other vertebrate species that possess intestinal sucrase activity indicate maltase hydrolytic abilities that are several times higher than those needed to fuel metabolism on a maltose (or starch) diet (Hernández and Martínez del Rio 1992).

Our results also provide support for the hypothesis that sucrose intolerance is a shared-derived character of the sturnid-muscicapid lineage (Martínez del Rio 1990). Although the reasons why sucrase activity was lost and why sucrase has not been regained in the ancestor of starlings and thrushes are unclear, the consequences of sucrase absence in this lineage seem to be important. Lack of sucrase activity seems to limit the dietary choices of a large number of species (Martínez del Rio and Stevens 1989) and also probably plays a significant role in the interaction of birds with the plants whose seeds they disperse. Sucrose intolerance in starlings, mim-

ids and thrushes appears to be a strong selective force that contributes to the maintenance of low sucrose concentrations in fruit pulp and to the prevalence of glucose and fructose in present-day bird-dispersed plants (Martínez del Rio et al. 1992). Sucrose intolerance in birds may be a good example of the implications that single and seemingly trivial evolutionary events, such as the loss of activity in a single multifunctional enzyme, can have on the interaction between animals and plants.

ACKNOWLEDGMENTS

This manuscript is the result of a collective effort. We are grateful to those that participated in its completion. H. Suthers and E. Stiles helped in the capture of catbirds and in conditioning them for release after experiments. Catbirds were captured under permit PRT-761796 (U.S. Fish and Wildlife Service). C. Sheppard and A. Lyles generously provided the Purple-headed Glossy-Starlings and commented on the manuscript. L. Eguiarte, K. Brugger, D. Levey, A. Masters, and D. O'Brien read and criticized drafts of the manuscript. D. W. White, K. G. Murray, and G. D. Schnell constructively reviewed the manuscript. Our work was funded by an NSF grant to C.M.R. (BSR-9020909).

LITERATURE CITED

ALPERS, D. H. 1987. Digestion and absorption of carbohydrates and proteins. Pages 1469–1486 *in* Physiology of the gastrointestinal tract, vol. 2 (L. R. Johnson, Ed.). Raven Press, New York.

AURICCHIO, S., A. DAHLQVIST, G. MÜRSET, AND A. PRADER. 1963. Isomaltose intolerance causing decreased ability to utilize dietary starch. J. Pediatr. 62:165–173.

AURICCHIO, S., F. CICCIMARRA, L. MOAURO, F. REY, J. JOS, AND J. REY. 1972. Intraluminal and mucosal starch digestion in congenital defficiency of intestinal sucrase and isomaltase activities. Pediatr. Res. 6:832–840.

BAKER, H. G., AND I. BAKER. 1983. Floral nectar sugar constituents in relation to pollinator type. Pages 131–171 *in* Handbook of experimental pollination ecology (C. E. Jones and R. J. Little, Eds.). Scientific and Academic Editions, New York.

BAKER, I., H. G. BAKER, AND S. A. HODGES. 1993. Patterns in the sugar composition of nectar and fruit juices taken by Microchiroptera and Megachiroptera. Biotropica. In press.

BEAL, F. E. L. 1897. Some common birds in their relation to agriculture. U. S. Dep. Agric. Farmers Bull. 54.

BEECHER, W. J. 1978. Feeding adaptations and evolution in the starlings. Bull. Chicago Acad. Sci. 11:269–298.

BENT, A. C. 1948. Life histories of north American nuthatches, wrens, thrashers, and their allies. Bull. U. S. Natl. Mus. 195.

BLAKE, J. G., AND B. A. LOISELLE. 1992. Fruits in the diets of Neotropical migrants in Costa Rica. Biotropica 24:200–210.

BOLTEN, A. B., P. FEINSINGER, H. G. BAKER, AND I. BAKER. 1979. On the calculation of sugar concentration in flower nectar. Oecologia 41:301–304.

BRUGGER, K. E., AND C. O. NELMS. 1991. Sucrose avoidance by American Robins (*Turdus migratorius*): Implications for control of bird damage in fruit crops. Crop Protection 10:455–460.

BRUGGER, K. E., P. NOL, AND C. I. PHILLIPS. 1993. Repellency of sucrose to European Starlings: Will high-sucrose cultivars deter bird damage in fruit crops? Ecol. Appl. 3:256–261.

COHEN, R. R. 1966. Anticoagulation, centrifugation time and sample replicate number in the microhematocrit method for avian blood. Poult. Sci. 46:214–218.

DAHLQVIST, A. 1984. Assay of intestinal disaccharidases. Scand. J. Clin. Lab. Invest. 44:69–172.

DENSLOW, J. S., D. J. LEVEY, T. C. MOERMOND, AND B. C. WENTWORTH. 1987. A synthetic diet for fruit-eating birds. Wilson Bull. 99:131–134.

GROSCOLAS, R., AND A. RODRIGUEZ. 1981. Glucose metabolism in fed and fasted Emperor Penguins (*Aptedonytes forsteri*). Comp. Biochem. Physiol. A Comp. Physiol. 70:191–198.

GUDMAN-HØYER, E., P. A. KRASILNIKOFF, AND J. SKOVBERJ. 1984. Sucrose-isomaltose malabsorption. Pages 35–62 *in* Advances in nutritional research, vol. 6 (H. H. Draper, Ed.). Plenum Press, New York.

HAZELWOOD, R. L. 1984. Pancreatic hormones, insulin/glucagon molar ratios, and somatostatin as determinants of avian carbohydrate metabolism. J. Exp. Zool. 232:647–652.

HERNANDEZ, A., AND C. MARTINEZ DEL RIO. 1992. Intestinal disaccharidases in five species of phyllostomoid bats. Comp. Biochem. Physiol. B Comp. Biochem. 10:105–111.

HOYSAK, D. J., AND P. J. WEATHERHEAD. 1991. Sampling blood from birds: A technique and an assessment of its effects. Condor 93:746–752.

ISOKOSI, M., J. JUSSILA, AND S. SARNA. 1972. A simple screening method for lactose malabsorption. Gastroenterology 62:28–32.

KRASILNIKOFF, P. A., E. GUDMAN-HØYER, AND H. H. MOLTKE. 1975. Diagnostic value of disaccharidase tolerance tests in children. Acta Paedtr. Scand. 64:693–702.

MACKWORTH-PRAED, C. W., AND C. H. B. GRANT. 1955. Birds of eastern and northern Africa. Longmans, Green and Co., New York.

MARSH, R. L., C. CAREY, AND W. R. DAWSON. 1984. Substrate concentrations and turnover of plasma glucose during cold exposure in seasonally acclimatized House Finches, *Carpodacus mexicanus*. J. Comp. Physiol. B Comp. Biochem. 154:469-476.

MARTIN, A. C., H. C. ZIM, AND A. L. NELSON. 1951. American wildlife and plants: A guide to wildlife food habits. McGraw-Hill, New York.

MARTÍNEZ DEL RIO, C. 1990. Dietary and phylogenetic correlates of intestinal sucrase and maltase activity in birds. Physiol. Zool. 63:987-1011.

MARTÍNEZ DEL RIO, C., AND B. R. STEVENS. 1989. Physiological constraint on feeding behavior: Intestinal membrane disaccharidases of the starling. Science 243:794-796.

MARTÍNEZ DEL RIO, C., B. R. STEVENS, D. DANEKE, AND P. T. ANDREADIS. 1988. Physiological correlates of preference and aversion for sugars in three species of birds. Physiol. Zool. 61:222-229.

MARTÍNEZ DEL RIO, C., D. J. LEVEY, AND W. H. KARASOV. 1989. Physiological basis and ecological consequences of sugar preferences in Cedar Waxwings. Auk 106:64-71.

MARTÍNEZ DEL RIO, C., H. G. BAKER, AND I. BAKER. 1992. Ecological and evolutionary implications of digestive processes: Bird preferences and the sugar constituents of floral nectar and fruit pulp. Experientia 48:544-550.

MOSTELLER, F., AND J. W. TUKEY. 1977. Data analysis and regression. Addison Wesley, New York.

NAGY, K. A. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecol. Monogr. 57:11-128.

PRINZINGER, R., A. PRESMAR, AND E. SCHLEUCER. 1991. Body temperature in birds. Comp. Biochem. Physiol. A Comp. Biochem. 99:499-506.

PROP, J., AND T. VULINK. 1992. Digestion by Barnacle Geese in the annual cycle: The interplay between retention time and food quality. Funct. Ecol. 6:180-189.

SEMENZA, G., AND S. AURICCHIO. 1989. Small intestinal disaccharidases. Pages 2975-2997 *in* The metabolic basis of inherited disease, vol. 2 (C. R. Scriver, A. L. Beaudet, W. S. Sly, and D. Valle, Eds.). McGraw-Hill, New York.

SERLE, W., G. J. MOREL, AND W. HARTWIG. 1977. A field guide to the birds of West Africa. William Collins Sons and Co., Toronto.

SIBLEY, C. G., AND J. E. AHLQUIST. 1984. The relationships of the starlings (Sturnidae: Sturnini) and the mockingbirds (Sturnidae: Mimini). Auk 101:230-243.

SIBLEY, C. G., AND J. E. AHLQUIST. 1990. Phylogeny and classification of birds. Yale Univ. Press, New Haven, Connecticut.

SIBLEY, C. G., AND B. L. MONROE. 1990. Distribution and taxonomy of birds of the world. Yale Univ. Press, New Haven, Connecticut.

SKADHAUGE, E. 1981. Osmoregulation in birds. Springer-Verlag, Berlin.

STEVENS, C. E. 1988. Comparative physiology of the vertebrate digestive system. Cambridge Univ. Press, Cambridge.

SUTHERS, H. B., AND D. D. SUTHERS. 1990. Aging and sexing Gray Catbirds by external characteristics. N. Am. Bird Bander 15:45-52.

VONK, H. J., AND J. R. H. WESTERN. 1984. Comparative biochemistry of enzymatic digestion. Academic Press, London.

WEAST, R. C., AND S. M. SELBY. 1967. Handbook of chemistry and physics, 48th ed. Chemical Rubber Co., Cleveland, Ohio.

WHISTLER, R. L., AND J. R. DANIEL. 1985. Carbohydrates. Pages 69-138 *in* Food chemistry (O. R. Fennema, Ed.). Marcel Decker, Inc., New York.

WHITE, D. W., AND E. W. STILES. 1990. Co-occurrences of food in stomachs and feces of fruit-eating birds. Condor 92:291-303.