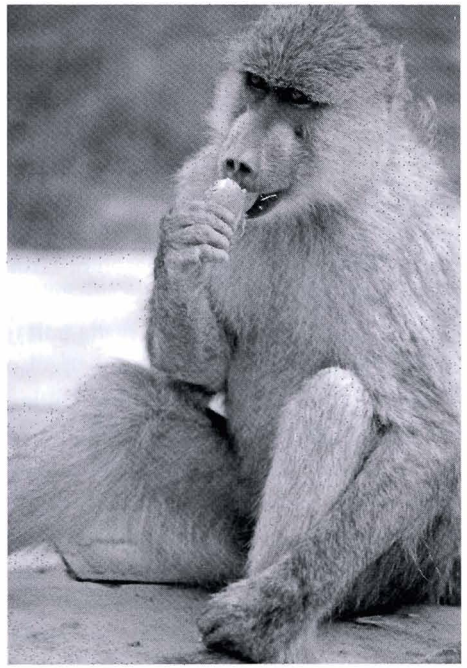
# 9 *Primate foraging adaptations: two research strategies*

STUART A. ALTMANN



## Introduction

In the course of evolution, probably every organ system of animals has been altered by the exigencies of obtaining food. An outcome of our rapidly

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expanding knowledge of primates in the wild is that numerous potential adaptations for foraging have been described. These proposed adaptations are at virtually every level of biological organization, including social and individual behavior, mental states, physiology, anatomy, and morphology (Rodman & Cant, 1984; Whiten & Widdowson, 1992; Miller, 2002). However, an adaptation proposed is not an adaptation confirmed.

Gould & Vrba (1982) pointed out the presence of two distinct adaptation concepts in the literature, one historical, emphasizing traits' origins and their past histories of selection, the other nonhistorical, emphasizing current functions of traits and their contributions to fitness. My discussion is limited to the latter.

From the standpoint of the current functions of phenotypic traits and their impact on selection, a trait variant is better adapted, relative to competing variants in other individuals of the same species, to the extent that it directly contributes to fitness. Competing variants of traits are those that potentially can become relatively more common at the expense of others. Trait variants are more likely to be competing the closer the subjects are in other respects, for example, in order of increasing proximity: members of the same species, same deme or local population, same group, cohort, season of birth, sex, and with mothers of nearly the same dominance rank.

Thus, in studies of living organisms, one can confirm that a trait is adaptive and measure its degree of adaptiveness by determining its effect on fitness. However, just identifying which traits are adaptive tells us little about the functional processes by which traits affect fitness or their relative contributions to it. For studying the adaptive significance of traits in extant species, several methods are available (Endler, 1986; Rose & Lauder, 1996; Altmann, 2005). The choice among them depends on what aspects of adaptation one wishes to study.

In what follows, I focus on two closely related strategies for studying foraging adaptations in extant, wild primates or other animals. Both are based on quantitative relationships between phenotypic traits, their proximate effects ("performances," "functions"), and biological fitness. This focus reflects an important distinction, that between phenotypic selection and the genetic response to selection.

> Natural selection acts on phenotypes, regardless of their genetic basis, and produces immediate phenotypic effects within a generation that can be measured without recourse to principles of heredity or evolution. In contrast, evolutionary response to selection, the genetic change that occurs from one generation to the next, does depend on genetic variation. *(Lande & Arnold, 1983)*



Of these two strategies for the study of adaptations in wild primates or other animals, one is based on a priori design specifications for optimal phenotypes and has been applied to primates in their natural habitats. The other is based on multivariate selection theory, which deals with the effects of selection acting simultaneously on multiple characters, and has been applied to various other animals. Emphasis here will be placed on what each strategy can reveal about adaptations, the types of data that each requires, and how one can get from one to the other. For details of field techniques, logistics, sampling methods, assumptions, data analysis, and so forth, the reader should turn to the primary literature.

## Measuring adaptiveness in extant species

### *First strategy*

This strategy utilizes methods developed by Russell Lande and Stevan Arnold for studying adaptations by measuring the impact of traits and their proximate effects on biological fitness (Arnold, 1983, 1988; Lande & Arnold, 1983). To make this approach concrete, consider a study of a fictitious primate.

> The study is carried out on a local population of arboreal monkeys for which long-term birth and death records are maintained. We suspect that the monkeys' fitness is limited primarily by their intakes of proteins and energy. Although all have access to and eat the same foods, some individuals eat more nuts than others, others more flower nectaries, and still others, more insects. Each day, we record the intake of each food that they consume. We also measure any other traits that are suspected of being correlated with intakes of nuts, flowers, and insects, our three prime candidates for limiting the monkeys' fitness. We collect and preserve samples of each food and have them analyzed for nutrients and any suspected toxins.
>
> Suppose that we want to measure the impact on biological fitness of individual differences in a given diet component, such as the amount of nut-meat consumed, independent of the quantities of other foods in the diet. How can we do this?

#### *Selection gradients*

To measure the potential impact of any given phenotypic trait on relative fitness, regress relative fitness $w$ on it (Lande & Arnold, 1983). However, because fitness-affecting traits may be correlated, use partial regression $\beta_{wz_i}$

(ordinary, not standardized) to measure the direct impact of the $i^{th}$ trait, $z_i$, on relative fitness w, with indirect effects from correlated traits thus held constant. Repeat for each of the other traits that may affect relative fitness. Then, to document any correlations among these traits, calculate their covariances (unstandardized correlations).

Relative fitness $w$ of an individual is defined with respect to the mean fitness in the population: $w = W/W^{-}$, where $W$ is the absolute fitness of an individual and $W^{-}$ is the mean absolute fitness in the population. The fitness of individuals can be estimated in several ways, particularly by using aspects of reproductive success, e.g., the number of surviving offspring. Individual fitness values can be determined at long-term study sites of populations for which birth and death data are consistently recorded. For data sets of shorter duration, it can be estimated from various components of fitness (Howard, 1979).

The partial regression of relative fitness on a given character is its *selection gradient*. It measures the change in relative fitness expected if that character were changed by a unit amount but none of the other characters varied. It can be thought of as an indication of the sensitivity of fitness to changes or differences in the character.

> So, from the birth and death records of our exemplar monkeys, we calculate the relative fitness of each subject. The partial regression of relative fitness on, e.g., nut consumption is the latter's selection gradient. It indicates the change in the monkeys' fitness per unit increase in nut consumption, with all other foods in the regression held constant at their mean value, and it thus provides a measure of the potential adaptiveness of nut consumption.

*Contributions of traits to fitness*

Of course, to understand the realized adaptiveness of traits, we want to know the *magnitude* of their influences on fitness, not just the latter's *sensitivity* to them. For this purpose, we use each trait's selection gradient to calculate that trait's average contribution to mean fitness.

> Suppose that, as suspected, the fitness gradients reveal three traits, nut-eating, flower-eating, and insect-eating, to which fitness is particularly sensitive. We would like to know how much each of these phenotypic traits contributes to mean fitness. Each such contribution is the product of that trait's average value and its selection gradient.



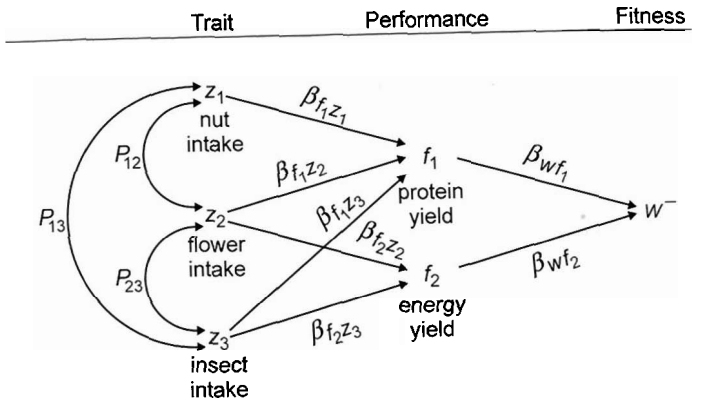


Figure 9.1. Path diagram representation of statistical relationships (beta terms, single-headed arrows) between any phenotypic trait $z_i$ (diet), each of its proximate effects $f_j$ (performance), and the latter's effects on fitness, $w^{-}$. The covariances among the traits are represented by double-headed arrows. For example, $P_{13}$ is the covariance between nut consumption and insect consumption. For simplicity, arrows indicating residual influences on performance and fitness are not shown.

$$
\begin{aligned}
w^{-} &= \text{average fitness} \\
&= \beta_{wz_1 \cdot \bar{z}_1} = \textit{average contribution of nut-eating} \\
&+ \beta_{wz_2 \cdot \bar{z}_2} = \textit{average contribution of flower-eating} \\
&+ \beta_{wz_3 \cdot \bar{z}_3} = \textit{average contribution of insect-eating} \\
&+ \ldots \quad = \textbf{\textit{contribution from other elements.}}
\end{aligned}
\tag{9.1}
$$

A remarkable result, due to Russell Lande, is that for a set of characters that affect fitness, their selection gradients, each calculated with effects of all correlated traits that directly affect fitness partialled out, include all the information about phenotypic selection (but not inheritance) that is needed to predict the directional response to selection.

*Partitioning selection gradients*

Although selection gradients evaluate causal links from traits to fitness, they do not tell us anything about the intervening functional effects of traits that augment fitness. Selection requires a mechanism. Arnold (1983) used Sewall Wright's method of path analysis to provide a convenient means of partitioning selection gradients (Figure 9.1). He showed that for any trait $z_i$ (such as nut consumption, $z_1$ in Figure 9.1) that affects only one fitness-related performance variable $f_j$ (e.g., protein yield, $f_1$), a selection gradient $\beta_{wz_i}$ can be partitioned into two parts, a *performance gradient* $\beta_{f_j z_i}$, representing the effect of the trait on that aspect of performance, and a *fitness gradient* $\beta_{wf_j}$, representing the effect of performance on fitness. That is,

$$\beta_{wz_i} = \beta_{fjz_i} \bullet \beta_{wf_j},$$
$$\begin{bmatrix}\text{selection} \\ \text{gradient}\end{bmatrix} = \begin{bmatrix}\text{performance} \\ \text{gradient}\end{bmatrix} \bullet \begin{bmatrix}\text{fitness} \\ \text{gradient}\end{bmatrix} \quad (9.2)$$

where $\beta_{wf_j}$ is the partial regression of relative fitness w on the $j^{th}$ performance variable, and $\beta_{fjz_i}$ is the partial regression of that performance variable on the $i^{th}$ trait variable.

A trait may affect more than one performance variable, resulting in branching paths. For example, the second trait $z_2$ in Figure 9.1 (flower-eating) affects two performance variables, $f_1$ and $f_2$ (protein and energy). In that case, the total path connecting character $z_2$ with relative fitness is the sum of the two paths, one through performance variable $f_1$ and one through performance variable $f_2$, as shown in Figure 9.1. The corresponding relationship in partial regression coefficients is $\beta_{wz_2} = \beta_{f_1z_2}\beta_{wf_1} + \beta_{f_2z_2}\beta_{wf_2}$. Thus, the total selection gradient can be partitioned into additive parts, corresponding to branching paths of influence on fitness, as well as factored along paths. These elementary results can readily be expanded for analysis of selection in situations considerably more complicated than that of the fictitious primate depicted in Figure 9.1.

*Contributions of traits to performance*

We can use each trait's performance gradient to calculate that trait's average contribution to the mean value of a given proximate effect. To illustrate, consider our exemplar primate.

> Suppose that, as we suspected, the fitness gradients reveal two performance variables, protein intake and energy intake, to which fitness is particularly sensitive. We would like to know how much, on average, each phenotypic trait contributes to the animals' protein intake (and similarly, to their energy intake). The protein intake of the average animal in the local population can be expressed as the sum of the contributions made by each forage-related trait. Each such contribution is the product of that trait's average value and its performance gradient for protein yield:

$$\begin{aligned} f_1 &= \textit{average protein yield} \\ &= \beta_{f_1z_1}\cdot\bar{z}_1 = \textit{average contribution of nut-eating} \\ &+ \beta_{f1z_2}\cdot\bar{z}_2 = \textit{average contribution of nectary-eating} \\ &+ \beta_{f1z_3}\cdot\bar{z}_3 = \textit{average contribution of insect-eating} \\ &+ \ldots \quad = \textit{contribution from other elements.} \end{aligned} \quad (9.3)$$



*Contributions of performance variables to fitness*

We can proceed similarly for the second causal link, evaluating the average contribution made by each performance variable to mean fitness.

> For example, the mean fitness $w^-$ of the monkeys can be partitioned into additive components:

$$w^- = \beta_{wf_1} \bullet \bar{f}_1 + \beta_{wf_2} \bullet \bar{f}_2 + \textit{contribution from other elements}, \quad (9.4)$$

> where the first two terms in the summation on the right are the contributions to mean fitness made by the average monkey's intake of protein and energy, respectively.

Such contributions of performance variables to fitness are excellent indicators of their relative adaptiveness.

In sum, the first strategy enables us to study the effects of correlated traits on fitness, by measuring both the sensitivity of relative fitness to variability in individual phenotypic traits, holding other traits constant, and the independent contribution that each trait makes to fitness. In so doing, it measures the adaptiveness of traits. In addition, the first strategy enables us to partition that sensitivity into two causal links, those from traits to performance variables, and those from performance variables to fitness. It enables us to estimate the mean contribution of each phenotypic trait to the average value of each performance variable, and—perhaps the best measure of a performance variable's adaptiveness—each performance variable's contribution to fitness.

### *Second strategy*

An optimality model of foraging behavior specifies how an individual of a given species should behave, under prevailing circumstances, in order to optimize (maximize or minimize, as appropriate) a performance variable. For example, what selection of foods would maximize energy intake? What hunting strategy would minimize hunting time? That performance variable, the "currency," is selected because it is expected to be a major contributor to mean fitness. The objective variable that is to be optimized is written as a function (the "objective function") of its causative traits.

> Consider a model for maximizing mean daily energy intake E applied to our paradigm monkeys, with their diet of nuts, flower nectaries, and insects. Suppose that the energy obtained from foods is 15 kilojoules per gram of nuts, 3 kJ per gram of nectaries, and 4 kJ

per gram of insects, and let $c_i$ represent respectively the amounts (grams consumed per day) of the $i^{th}$ food. The objective then is to find those values of $c_1$, $c_2$, *and* $c_3$ *that would maximize* $E$, *where* $E = 15c_1 + 3c_2 + 4c_3$.

Of course, there are no benefits without costs, "no such thing as a free lunch." The second component of an optimality model consists of *costs* (*constraints, limiting factors*), such as nutrient requirements at the lower end, toxins or other hazards at the upper, that represent the animals' limitations and that keep the currency from going to zero or infinity. They too are written as functions.

Some costs may vary continuously with the objective variable. If such costs can be expressed in the same units as the objective variable, then the objective can be to optimize the difference between them, the net benefit, or "trade-off."

> Suppose for simplicity that for our exemplar monkeys, flower nectar is their only source of energy and that as they begin to exhaust their local supply of flowers with filled nectaries, they range ever farther away from their home range center. As they do so, they encounter adjacent groups with increasing frequency, resulting in progressively more energy-consuming chases. They eventually reach a "point of diminishing returns" beyond which the extra energy gained from additional nectar is less than the loss from being chased. Going just that far to feed on nectaries is the optimal solution, unless doing so would not already have put them beyond some other upward-limiting constraint.

Other costs can each be approximated by a step function, a discrete boundary beyond which the animal cannot remain indefinitely without seriously impairing some vital function, perhaps fatally, but within which further increases have no significant effect. For example, nutrient intakes above the minimum required to prevent deficiency symptoms are claimed by nutritional scientists to have no further beneficial effect. The same may be true of many other constraints. So, for example, if our monkeys' gut sizes and food passage times limit them to 500 g of food per day, their consumption constraint, in grams/day, is $c_1 + c_2 + c_3 \leq 500$.

In short, such models consist of an equation for the objective function (a putative fitness-enhancing performance variable that is to be maximized or minimized, written as a function of contributing phenotypic traits), and various other functions representing constraints on the objective function (attributes of the organism or its relationship with the environment). For



example, in an optimal diet model whose objective is maximizing daily energy intake, the simultaneous solution to the equations of the model would indicate an amount of each available food, which, if consumed by the subjects, would maximize mean daily energy intake and give them an otherwise adequate diet without taking too much time, exceeding the animals' consumption capacity, and so forth. A simultaneous solution to these equations is required. The animals' actual diets and their effects can then be compared with their optimal diets. In 1978, Steve Wagner and I published a method for providing closed-form solutions to such sets of equations if linear, not knowing that an iterative procedure, developed by Dantzig, was already well established.

### *Data requirements*

Note that the first and second strategies include the same three empirical components. First, they require quantitative samples, taken in a local population of a species, of individual variants in a set of phenotypic traits. Typically, these are traits known or thought to affect a particular vital activity, such as getting food or obtaining mates. Next, they require quantitative samples of proximate effects (performances) of these variants, whether known a priori to be functional or otherwise. Third, they require estimates of the biological fitness of each subject.

In addition, the second strategy makes use of an optimality model that relates phenotypic traits to their fitness-enhancing proximate effects, and so, requires specific information, as follows. Quantitative data – presumably obtained in the performance samples described above – are required for each individual's success on that performance variable (or those variables) that are the basis of the model's objective function. Beyond that, information is required to establish the model's constraints: quantitative data on various traits of the animals and of their relationships with the environment, as needed to establish upper and lower constraints on the optimization.

### *An example*

The optimal diet model that I applied to the foraging of yearling baboons illustrates the feasibility of such models (Altmann, 1998, chapter 8). On the assumption that energy is the primary fitness-limiting component of the baboons' diets, I took optimal diets to be those that maximize the yearlings' daily energy intake while simultaneously keeping them above their minima for nutrients and below their maxima for various constraints such as toxin tolerances, time limits, gut processing capacities, and so forth. In addition to the linear objective function, the model had 72 constraint functions, each in the form of a linear equation! Yet, models much larger than this can now

quickly be solved by readily available computer programs. The model was adjusted and recalculated for data taken at 10-week intervals to take into account seasonal changes in available foods and age-related changes in the yearling's requirements and tolerances.

Obtaining data on the diets of 11 yearlings occupied a year, during which I recorded food intakes in 18 460 feeding bouts during 333 hours of in-sight sample time. For practical reasons, data analysis was limited to 52 core foods on which the yearlings spent the most time feeding and that collectively accounted for 93% of their feeding time. Data on the subsequent survivorship and reproduction of these subjects were obtained as part of routine long-term demographic monitoring in the Amboseli baboon project, and continued for the rest of the subjects' lives, which in the extreme case, female Dotty, took 27.7 years (Bronikowski *et al.*, 2002). However, well before that, strong patterns became apparent, and one need not wait until the last subject has died before examining available data. Eight components of fitness were evaluated as of a cut-off date that was, on average, 14.4 years after the yearlings were born. (By that date, all but two were dead.) These data on each individual's dietary intake, its costs and benefits, and the subject's fitness were then used to test the optimal diet model.

### *Confirmation*

Models are elaborate hypotheses, and are often regarded as attempts to describe some small aspect of the world. From this descriptive perspective and given an assumption that natural selection tends to eliminate suboptimal traits, a model of optimal foraging or diet would be considered confirmed to the extent that the observed foraging behavior or food consumption matches the model's specifications. A goodness-of-fit test usually would be appropriate and would tell us whether the traits of individuals or their population mean were significantly different from specified optimal trait values. Yet, if so, we would not be able to distinguish the shortcomings of the model from the shortcomings of the animals. For a host of reasons (reviewed by Maynard Smith, 1978; Emlen, 1987; Rose & Lauder, 1996; Altmann, 1998), organisms may not perform at or near their optima.

An alternative strategy, and the one that I adopted (Altmann, 1998, chapter 1), is to consider optimality models as normative, not descriptive. In a normative strategy, deviations of traits from values specified by an optimality model of adaptive traits are regarded not as tests of the model but as indications of potential differences in fitness. The model itself would be tested by showing that those individuals whose traits are closer to the putatitive optimum have higher fitness as a consequence and, conversely, that those that deviate sufficiently from the optimum have predictable functional



impairments, such as nutrient deficiencies or toxicity effects, which lead to a reduction in fitness. Confirming an optimality model in this way is, at the same time, a confirmation of adaptive differences in the specified traits and in the functional mechanisms by which these traits affect fitness.

Not surprisingly, the yearlings' dietary intakes were not optimal. Every baboon at every age at which I sampled it took in suboptimal quantities of virtually all macronutrients (water, minerals, proteins, fiber, other carbohydrates, energy and lipids), relative to quantities specified by their age-specific diet for maximizing energy. Indeed, there was but one exception out of 227 yearling-age-macronutrient comparisons: female Eno, like all the others, took in suboptimal quantities of all macronutrients, except that, at 30–40 weeks of age, she took in the optimal quantity of lipids, but just barely (Altmann, 1998 table 7.8).

All of these macronutrient shortfalls of the yearlings deviated less than 8% from the shortfall in their total dietary mass, but that total was, on average, just 52% of the mass of an energy-maximizing diet. That is, on average, the yearlings ate close to a balanced diet, in the sense that the macronutrients in their diets were in ratios moderately close to what would be needed to obtain an energy-maximizing diet, they just didn't eat enough of them. None of their diets, at any age in the 30–70 week age-interval in which they were sampled, were within two standard deviations of that optimal mass.

In short, many more of the yearlings' nutrient shortfalls were attributable to inadequate food consumption than to poor choices of foods – to the quantity of their diet, rather than its quality. I do not know the source of these food deficiencies, but suspect that in Amboseli the sparse distribution of foods, not their quality, abundance, or seasonal unavailability, is the primary factor limiting the baboons' intakes. For energy, this may have affected the lactation capacity of the yearlings' mothers: 45% of the yearlings' mean energy shortfall resulted from the discrepancy (1.31 MJ/day in milk energy) between the amount of nursing that they did and the amount prescribed by the energy-maximizing diet.

Clearly, the energy-maximizing diet model for Amboseli's yearlings would be rejected outright by anyone following the descriptive strategy. Neither the yearlings nor their mean were anywhere near optimal. However, with a normative strategy, the optimal diet model was confirmed to a remarkable degree (Altman, 1991, 1998). Several characteristics of the yearlings' diets and other traits, many of which are intercorrelated, provide good predictions of components of fitness. I here focus on two, *energy shortfall* (deviation of energy intake from optimal energy intake, as a percentage of the latter) and *protein surplus* (deviation of protein intake from amount in an energy-maximizing diet, as a percentage of the latter). *Reproductive success* – the

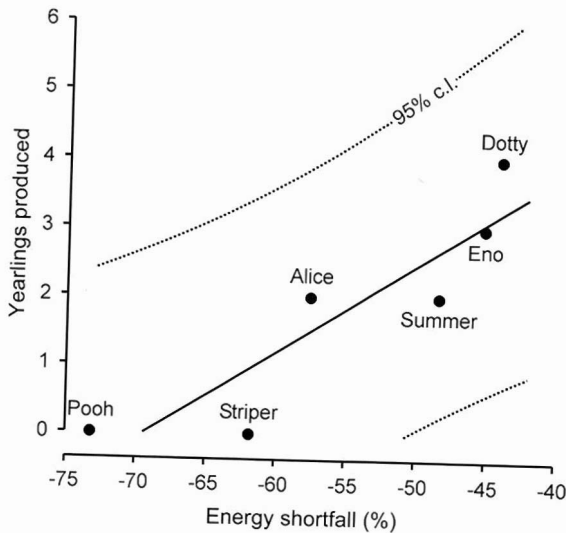


Figure 9.2. Reproductive success of females, on the y-axis (numbers of surviving yearlings they each produced in their lifetime) as a function of the females' energy shortfalls when they were yearlings, *x*-axis (percent deviation of their actual intakes from their respective optimal intakes). Regression: $n = 6$, adjusted $R^2 = 0.76$, $p \leq 0.05$, $y = 8.8 + 0.127x$. From Altmann (1998, Fig. 8.9).

number of yearlings that each female produced by the cutoff date (mean age 14.4 years, two females surviving) – was taken as the fitness value for the six females in my study. (No paternities for the male subjects were known at the time.) A linear regression of fitness on energy shortfall indicated that energy shortfall accounted for 76% of the variance in female fitness ($p \leq 0.05$). Those females who took in more energy, and so had smaller energy shortfalls, produced more surviving infants (Figure 9.2). On average, each eight percentage point difference in energy shortfall during a female's childhood translated into an additional surviving yearling over her lifetime.

I then calculated a multivariate linear regression of female fitness (reproductive success, as defined above) on a combination of energy shortfall and protein surplus (Figure 9.3). The resulting linear equation is:

$$\text{fitness} = 33.22 + 30.53(\text{energy shortfall}) - 21.57(\text{protein surplus}) \quad (9.5)$$

This linear combination of energy shortfall and protein surplus accounted for 94% of the variance among females in their fitness. In short, the fitness of these females *as adults* was highly predictable from what they ate when they were yearlings, just 30–70 weeks of age.



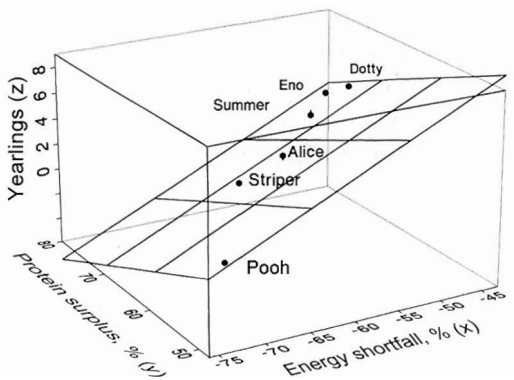


Figure 9.3. Reproductive success, on the *z*-axis – that is, numbers of yearlings produced by the females – is predicted as a linear combination (the tilted plane) of their energy shortfalls as yearlings, on the *x*-axis (percent deviation of their actual energy intakes from their respective optimal intakes) and their protein surpluses, on the *y*-axis (energy intakes above requirements), each calculated with the effect of the other predictor variable held constant at its mean value. Named dots show individual observed values; deviations are vertical lines (very short) from observed to predicted values. Regression equation is equation (9.5) in text, $n = 6$, adjusted $R^2 = 0.94$, $p \leq 0.01$. From Altmann (1998, Fig. 8.13).

Considering how highly correlated these two performance variables are (product moment correlation 0.95), adding protein as a second predictor variable might have seemed an unlikely way to improve the prediction of fitness. However, the coefficients of a multiple regression are, in fact, the beta values of partial regression, and each indicates the rate of change in the dependent variable (fitness) per unit in that predictor variable, with the others held constant at their mean value. Thus, the protein surplus coefficient in Equation 9.5 indicates the rate of change in fitness per unit change in daily protein intake in excess of requirement, independent of the effects of energy on fitness; and conversely, for the energy shortfall.

The resemblance of Equations 9.4 and 9.5 is no coincidence: the coefficients of a multivariate linear regression are the partial regression coefficients of the variables. The equations differ in two respects. In Equation 9.4, the dependent variable is relative fitness, whereas I used absolute fitness – a simple difference in units. Second, Equation 9.4 regresses fitness values on values of performance variables. In contrast, Equation 9.5 regresses fitness values on *deviations* in values of performance variables from values in an optimality model. It provides an answer to the central question: Do those individuals whose diets come closer to the putative optimum have higher fitness? Confirming an optimality model in this way is a confirmation of

adaptive differences in specified traits and in our identification of the functional mechanisms by which these traits affect fitness. At the same time, the model provides testable hypotheses for relationships between traits and their proximate effects, not just the null hypothesis that some effects occur.

In the process of applying standard methods for linear optimization to calculate the constituents of an optimal diet, numerous related questions are also answered (Altmann, 1984, 1998 chapter 5). Here are examples. What attributes of the animals or of their environment limit the amount of energy in the diet? How much could each attribute of the organisms or environment that does not limit energy intake change before it became a limiting factor? How sensitive is the energy content of the diet to the values of each of the limiting factors? How much could the energy density of a food change, or its estimated value be in error, without changing the composition of the optimal diet? Is the optimal diet unique? That is, is there more than one combination of foods that would maximize the objective function while satisfying all constraints?

The second strategy, like the first, is concerned with the effects of traits on performance and of each of these on fitness. Nothing in the second strategy would preclude making use of gradients of selection, performance, and fitness, described in the first strategy, to clarify these causal links. On the contrary, the two methods can fruitfully be combined (Arnold, 1988).

### *Nonlinearities*

In my model of energy-maximizing diets for yearling baboons, the various equations – for the objective function, for lower-bound constraints (nutrients) and for upper-bound constraints (toxin tolerances, gut capacity, and so forth)— were all linear. In that study, 94% of the yearlings' individual differences in fitness were accounted for by a linear combination of their deviations from the amounts of protein and energy in an energy-maximizing diet. However, in some situations, some of these relationships may be appreciably nonlinear, and their treatment should be considered.

#### *Some examples*

1. In optimal foraging studies, the most common examples of nonlinearity are ones in which the objective function is a cost–benefit ratio or any other ratio of two random variables, such as energy obtained per minute of foraging time. Michael Altmann (in Altmann, 1998, Appendices 7 and 8) has provided a method for maximizing or minimizing objective functions of this type. For methods of optimizing other types of nonlinear objective functions, an appreciable literature on nonlinear optimization is available.
2. Nutrient requirements, toxin tolerances, and probably many other constraints that are approximated by lines (in two dimensions, e.g., just two foods) or by hyperplanes (multiple dimensions) are actually probability distributions, e.g., the probability that any given dietary intake of ascorbic acid would result in scurvy. By drawing the lines at, say, the mean plus two standard deviations of tolerances for toxins (and conversely for nutrients), one would reduce the probability of advocating a diet that is high in energy but debilitating or even lethal.
3. Many nutrients interact with each other and with toxins in plants or other forage-related hazards. Some of these interactions result in nonlinear constraints. For example, because of the genetic variability of malarias, the chance and severity of malaria infection probably increases exponentially with time spent foraging in malaria-infested areas. However, many other such interactions may be nonlinear. For example, oxalic acid in some foods reacts with calcium ions to form insoluble calcium oxalate, thereby rendering that much calcium biologically unavailable. However, this reaction just requires representing intakes of available calcium as mols of calcium consumed minus mols of oxalate consumed.
4. The relationships described herein between phenotypic traits, their proximate effects, and fitness are based on directional selection. For stabilizing and other forms of nonlinear selection, see Arnold (2003).



### *Practicability*

Knowledge of mechanisms, and of requirements and limits for food components, is the great advantage that studies of foraging behavior have over studies of many other forms of behavior, in that we can say, at least to a first approximation, what a well-adapted primate should eat. If it takes in too little iron, it will become anemic, too much and it may suffer from siderosis. If it takes in too little vitamin D, rickets (infants) or osteomalacia (adults) results; too much, and demineralization of bone and mineralization of soft tissue result. If it eats too many seeds of certain legumes, it may suffer the toxic effects of trypsin inhibitor, too few and it may not get enough protein. For most nutrients and a few toxins in foods, quantitative upper and lower limits are moderately well established (Altmann, 2005). Yet, who could do the same

for, say, play behavior? What kinds and what amounts are better? Where is the research that could be used to advise a maturing male primate of when and how hard to hit or slash at his opponent in a fight and when to back off, or to tell a primate mother which adult females she should allow to hold her infant and for how long?

Whether the two types of study that I have described are practicable for a set of traits or their proximate effects depends on whether the requisite data can be obtained, namely, quantitative data on individual differences in traits, in proximate trait effects, and in components of fitness. To secure all three requires a combination of short- and long-term research plans.

Two major practical problems occur in implementing either of these research strategies for studying primate foraging adaptations. First, sampling large numbers of subjects is difficult. My sample size, 11 infants – or, for reproductive success, just six females – might not have revealed statistically significant differences were it not for the subjects' great variability in survivorship and reproductive success and strong individual differences in their dietary intakes. During a year of field work, I sampled the foraging of the entire cohort of infants in the main study group that were between 30 and 70 weeks of age at any time during my study (nine subjects); in a pilot study the previous year, I had sampled two others in the same age range. To enlarge the sample appreciably would have required additional observers and groups. In addition, the amount of sampling that I did per individual was less than ideal. Data that I obtained in a year of sampling the foraging of 11 yearling baboons and the chemical characteristics of their foods were sufficient to differentiate many but not all pairs of individual intakes on the basis of seven macronutrients during each of four, 10-week age classes.

The other major practical problem is the time required to obtain good estimates of fitness. However, basic demographic data – dates of births, deaths, emigrations of individuals from and immigrations into groups, and sightings of solitary individuals – are routinely obtained in almost all long-term studies. They provide information that is needed for a wide variety of projects. The advantage of studying infants, as I did, is that one can thereby capitalize on the high mortality that is characteristic of many mammals. As for reproductive success, my cutoff date for evaluating the fitness of my subjects was at an age that was half the life span of our oldest animal of known age.

Even with very long-lived primates such as chimpanzees, the study of adaptations in the wild is feasible. Of more than 40 chimp study sites in Africa, four have resulted in studies of chimp communities extending more than 15 years, and two – Kasakela at Gombe (Wilson & Wrangham, 2003) and the Boussou group in Guinea (Sugiyama, 2004) – have been studied for



43 years and 26 years, respectively, through 2003. Compare these numbers with some key demographic values. Mean age at first parturition averages 10.9 years to 14.6 years in chimps, depending on location. Survival to first parturition ranges from 22%–58% (Sugiyama, 2004). Thus, by a happy coincidence, these apes, with one of the longest life expectancies among primates, are also the subject of some of the most sustained programs of field research. With appropriate combinations of short- and long-term planning, the adaptive value of a wide variety of traits could be confirmed, particularly by taking advantage of high infant mortality.

### *Other options*

What can one do to study the adaptiveness of forage-related traits if data on lifetime fitness values of the subjects are not (yet) obtainable? Several opportunities are available (Altmann, 2005), including both of the strategies described above. The first strategy is based on Arnold's (1983) separation of fitness into two parts: a performance gradient representing the effect of the trait on some aspect of performance and a fitness gradient representing the effect of performance on fitness. "The point of this distinction," he wrote, "is that even when effects on fitness cannot be measured, it will often be possible to measure the effects on performance." We can take advantage of the ability of performance gradients to isolate the effect that each trait has on a given performance from effects of correlated traits, and we can quantitatively evaluate the contribution made by each trait to each performance variable. If we assume that, through their impact on vital processes, each of these performance variables affects fitness, they are indicative of the adaptiveness of the traits, even though in the absence of fitness data, we would be unable to test that assumption.

Similarly, if we have an optimality model (second strategy), we would already have hypothesized how to combine trait variables into quantitative predictions of each individual's level of performance on a major fitness-enhancing performance variable, and thus to predict its fitness relative to other members of its local population. Far more optimality models have been applied to humans than to any other species of primates, albeit without fitness correlates (Winterhalder & Smith, 1981, 2000; Smith, 1983; Smith *et al.*, 2001, and references therein).

## Coda

In long-term studies, estimates of fitness components, such as survivorship, become possible first, then eventually, lifetime fitness. As these estimates

become available, we can evaluate the impacts both of traits on functions and of functions on fitness with increasing accuracy. The benefits that we then reap go far beyond being able to say, yes, these traits are demonstrably adaptive. We obtain a far richer understanding of the mechanisms and processes by which they affect natural selection and so, in turn, are shaped by it.

I end with Arnold's (1983) ending. It is not enough to complain that adaptation is often invoked without critical evidence (Williams, 1966; Lewontin, 1979; Rowell, 1979; Baldwin & Baldwin, 1979; Gould & Lewontin, 1979). We also need an analytical approach that emphasizes what can be accomplished. The strategy outlined here is a step in the right direction.

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# 10 *The predictive power of socioecological models: a reconsideration of resource characteristics, agonism, and dominance hierarchies*

ANDREAS KOENIG AND CAROLA BORRIES

## Introduction

Beginning with Wrangham's work (1979, 1980), testing predictions regarding patterns of female agonistic behavior, social structure, and dispersal as a

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