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The matches, achieved by natural selection, between biological capacities and their natural loads

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Abstract. Natural selection tends to eliminate unutilized capacities because of their costs. Hence we ask how large are the reserve capacities by which biological capacities exceed natural loads, and how closely are related biological capacities matched to each other. Measured capacities (V_{max} values) of small intestinal brush-border nutrient transporters are typically around twice their natural loads (dietary intakes of their substrates); the ratio is higher for a transporter of a hyperessential nutrient. Preliminary evidence suggests matching of capacities between different steps in carbohydrate metabolism, and between the intestine, liver, kidneys, and spleen. Symmorphosis – the postulated matching of capacities to each other and to loads – is a testable hypothesis of economic design, useful in detecting and explaining cases of apparently uneconomic design.
Key words. Small intestine; brush border; nutrient absorption; sugar absorption; lactation; cold exposure; symmorphosis; safety margin; reserve capacity.

Introduction

This paper examines the quantitative match between biological capacities and the natural loads upon those capacities, and also the match between related capacities. As examples of capacities, we have measured the maximal reaction velocities (V_{max} values) of intestinal nutrient transporters and hydrolases; and as examples of corresponding loads, the daily dietary intakes of the substrates of those transporters and hydrolases. However, one could pose the same question for the strengths of bones in relation to the natural stresses on bones, or for the lung's oxygen diffusing capacity in relation to actual oxygen consumption [18], or for any other biochemical, physiological, or anatomical capacity. For each such comparison we ask whether the biological system is designed with some reserve capacity, such that actual capacity exceeds natural loads by some margin of safety. In effect, Darwin showed that biological capacities are qualitatively matched to their loads; we now ask about the quantitative match.

At the outset, one might wonder why natural selection or God did not endow animals with large reserve capacities in all systems, such that the animal's body could never limit the animal's behavior. The answer surely is that any biological capacity incurs costs (such as allocations of biosynthetic energy and space), but that animals have access to only finite biosynthetic energy, and that the space within an animal's body, cell membranes, and cytoplasm is utilized almost to the limit of standing-room-only. An animal squandering resources on one system thus draws down the resources available to other systems. Hence such an uneconomical animal would tend to be replaced by economical ones.

Many familiar examples illustrate the evolutionary outcome that disused capacities tend to become eliminated by natural selection. Cave animals tend to lose functional eyes, while some volant birds (especially rails) colonizing remote and predator-free islands tend rapidly to lose the ability to fly. The large and energetically costly flight

muscles are a necessity for birds on continents with native weasels and cats, but become an expensive liability in the absence of those predators.

Can one really be sure, though, that these evolutionary losses of disused capacities are due to natural selection – i.e., that the animal actually gains an advantage by eliminating a costly capacity that no longer serves a function? Might not the loss instead just be due to mutations that inactivate the capacity, that would have been screened out by natural selection as long as the capacity had a function, but that become neutral and accumulate once the structure becomes non-functional? In the following instructive example [9], the former interpretation is clearly the correct one. The wild type of the bacterium *Escherichia coli* contains enzymes that synthesize the essential amino acid tryptophan. Various mutants lack functional tryptophan-synthesizing enzymes. Naturally, if one tries to grow both the wild type and the mutants on a medium lacking tryptophan, the mutants die, while the wild type survives by synthesizing its own tryptophan. What happens if one grows them on a medium containing tryptophan, so that the mutants as well can now survive on exogenous tryptophan? If the now-unnecessary tryptophan-synthesizing enzymes were a negligible burden for the wild type, it would be at no competitive disadvantage compared to the mutants. In fact, the mutants outgrow the wild type, demonstrating that the latter's now-unnecessary waste of energy and space on tryptophan-synthesizing enzymes constitutes a significant disadvantage.

Some previous estimates of safety margins

These anecdotes of cave animals, island birds, and auxotrophic bacteria convince us that biological capacities incur selectively significant costs that make the maintenance of huge unutilized reserve capacities uneconomical. They do not tell us, however, how large a reserve capacity should be considered 'economical'. Do actual capacities tend to be 1.1, 10, or 100 times their natural loads? This quantitative question pervades all of biology.

Some indications of answers can be gleaned from clinical experience and the experimental literature. For example, healthy people can safely donate one kidney to a sibling or child with no functional kidney. If the donated kidney is not rejected, both the host and the donor can live relatively normal lives with just one kidney each. This suggests that our natural quota of two kidneys provides us with at least twice the capacity that we actually need. Again, patients with a damaged pancreas do not suffer malabsorption until pancreatic enzyme output drops below 10% of normal levels [7]. Conversely, patients with no functioning pancreas escape malabsorption only if pancreatic enzymes are supplied exogenously at at least 10% of their normal secretion rate. These observations suggest that the pancreas's enzyme secretory capacity exceeds natural loads by about a factor of 10. Finally, bones studied experimentally typically exhibit irreversible deformation under applied stresses double the peak stresses occurring under natural conditions [1, 17].

In these three examples, then, ratios of biological capacities to their natural loads involve factors of ≥ 2, about 10, and about 2, respectively. Kidney, pancreas, and bones all appear to be designed with some, but not too much, reserve capacity.

Safety margins of intestinal nutrient transporters

We have been comparing capacities with natural loads for brush-border nutrient transporters of the small intestine [5]. The advantage of these transporters for capacity/load studies is that capacities are readily defined and measured as the transporters' V_{max} values, while natural loads are defined and measured as the daily dietary intakes of those transporters' substrates.

First, a few words of background about the transporters. Recall that much of our dietary nutrient intake is in the form of proteins and complex carbohydrates, which are polymers of amino acids and of simple sugars (monosaccharides) respectively. Pancreatic enzymes (proteases and amylase, respectively) hydrolyze these polymers into small oligomers, which are then hydrolyzed by peptidases and disaccharidases bound to the small intestinal brush-border into amino acids and monosaccharides. These monomers are taken up out of the intestinal lumen into the intestinal mucosal cells by brush-border transport proteins specific for particular nutrients or nutrient classes. For example, there is one transporter for the aldohexose glucose, another for the ketohexose fructose, others for acidic or basic or neutral amino acids, and so on. Finally, the taken-up nutrients are exported from the mucosal cells into the bloodstream or lymph by a separate set of specific transport proteins located in the basolateral membranes of the intestinal mucosal cells.

To measure V_{max} values of the brush-border transporters, we excise the small intestine, evert a cylindrical sleeve of intestine such that mucosal cells now face outwards, and incubate the sleeve in a well-stirred solution containing a radiolabeled nutrient at a concentration far exceeding its transporter's Michaelis-Menten constant (K_m). After appropriate corrections for radiolabel in adherent fluid and for passively absorbed radiolabel have been applied, the rate of label uptake yields a measure of the transporter's V_{max} value, normalized per mg mass or per cm length of intestinal tissue [14]. The measurement is carried out on sleeves sampled from along the intestine's length. This method has the advantage that the tissue is intact rather than homogenized, so that fewer assumptions are involved in calculating the transport capacity of the whole length of the small intestine under physiological conditions [6].

Safety margins in cold-exposed mice

We have used this everted-sleeve preparation to measure brush-border transporter capacities for comparison with

the daily nutrient intakes that represent the loads upon these transporters. Our goal is to determine by how much, if at all, the measured capacities exceed the measured loads. However, one might still wonder how well our capacity measurements in vitro agree with physiological values prevailing in vivo. We therefore designed a quick-and-dirty test for existence of reserve capacity, involving only observations on living animals. The experimental principle was to induce a mouse suddenly to increase its food intake rate severalfold, and to monitor the mouse's digestive efficiency (fraction of ingested food absorbed) as a function of time. If mouse intestine normally possessed some reserve transporter capacity, the mouse's transporters would be able to cope with some increased food intake, still absorb the extra substrates, and not suffer any reduced digestive efficiency. If however the transporters were already operating near the limit of their capacity, the extra substrates would pass out the feces unabsorbed and be reflected in decreased digestive efficiency.

To induce mice to increase their food intake suddenly, we transferred mice from room temperature (22 °C) to a cold room at 6 °C, causing them immediately to begin losing heat and to increase their metabolic rate in order to maintain body temperature[21]. As figure 1 A illustrates, the mice fuelled this new raised metabolic rate by increasing their food intake within 12 h. Within 24 h food intake reached a new plateau value 2.5 times its previous value. However, fecal output (fig. 1 B) similarly jumped up within 12 h and reached a new plateau about 2.5 times the previous value within 24 h. Since fecal output and food intake increased by virtually the same factor, apparent dry-matter digestive efficiency (defined as food intake minus fecal output, divided by food intake) remained unchanged at 80% (fig. 1 C). (The 20% of food apparently unabsorbed presumably represents dietary indigestible fiber plus fecal output of bacterial matter, endogenous secretions, and shed enterocytes, rather than malabsorbed nutrients.) Thus, this quick-and-dirty experiment tells us that mouse intestine is normally capable of digesting at least 2.5 times the normal quantity of ingested food.

However, a further observation indicates that the intestine's capacity is probably not greatly in excess of 2.5 times the normal load. Within 4 days, the mice exposed to 6 °C temperatures experienced a hypertrophy of intestinal mass by 18%, resulting in correspondingly increased transporter capacities. This hypertrophy makes functional sense only if the mouse's body somehow detected (probably by changes in intestinal luminal nutrient concentrations) that intestinal transporter reserve capacity was becoming close to used up, and if the mouse somehow signalled the intestine (probably by releasing trophic hormones) to grow and thereby generate more transporter capacity. Hence these quick-and-dirty observations suggest that capacity is at least 2.5 times load, but not greatly in excess of 2.5 times load.

Figure 2 gives our actual values of intestinal brush-border glucose transporter capacity measured in everted sleeves, as compared with dietary glucose intake. Glucose

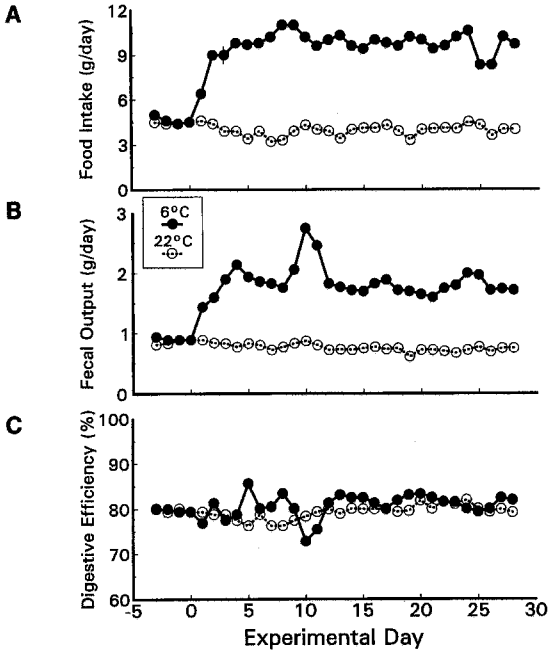

Figure 1. Food intake (1 A), fecal output (1 B), and apparent dry-matter digestive efficiency (1 C) of Swiss-Webster white mice suddenly transferred at t = 0 to an ambient temperature of 6 °C (•), compared to control mice maintained at 22 °C (o). Digestive efficiency was calculated as food intake minus fecal output, divided by food intake. A synthetic diet containing 55% sucrose was used in this and all subsequent figures except figures 3 and 5 (see Diamond and Karasov[6] for composition). Note that food intake and fecal output both increase 2.5-fold to new plateau values within 24 h of exposure to low temperature, but that digestive efficiency remains unchanged, implying the existence of some reserve capacity. Based on Toloza et al.[21].

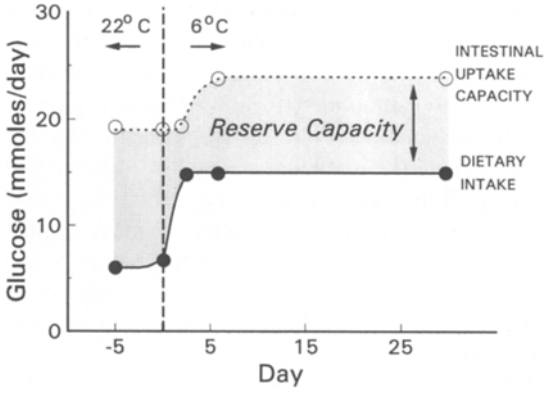

Figure 2. Dietary glucose intake per day (below), compared with the daily uptake capacity of the brush-border glucose transporter summed over the whole length of the small intestine (above), in mice. On day 0 ambient temperature was lowered from 22 to 6 °C, resulting in increased body heat loss, hence increased metabolic rate, hence increased food consumption and glucose intake. The excess of uptake capacity over dietary intake constitutes reserve capacity or safety margin. Note that uptake capacity exceeds dietary intake severalfold at 22 °C, that the sudden increase in intake at 6 °C nearly wipes out this safety margin, and that a subsequent small increase in uptake capacity due to intestinal hypertrophy reexpands the safety margin. Based on Toloza et al.[21].

transporter capacity in mice at 22 °C is 19 mmole/day, comfortably exceeding the actual glucose intake of 6 mmole/day by a factor of 3.2. At 6 °C glucose intake jumps to 15 mmole/day. This would use up most of the reserve capacity; in reality, it might at times exhaust it completely, because nutrient arrival at the small intestine is distributed unevenly over the 24-h cycle. However, within a few days the intestine grows, and glucose transporter capacity increases to 24 mmole/day, accounting for the mouse's ability to process 15 mmole/day glucose intake with undiminished efficiency. Thus, the function of intestinal hypertrophy is to restore some of the intestine's safety margin. Our measurement that the capacity/load ratio at 22 °C is about 3.2 compares well with our conclusion, based on the quick-and-dirty reasoning of the preceding two paragraphs, that the ratio is at least, but not greatly in excess of, 2.5.

Interestingly, the mice exposed to 6 °C temperatures experienced within a few days not only a 18% hypertrophy of the small intestine but also 16–20% hypertrophy of the liver, spleen, and kidney. This suggests that all four organs at 22 °C are similarly close to exhausting their reserve capacities. That is, not only does the small intestine have a modest reserve capacity for absorbing nutrients: the liver has a similarly modest reserve capacity for processing the absorbed nutrients, the kidney for excreting the resulting metabolic wastes, and the spleen for eliminating bacteria ingested with the nutrients.

Safety margins in lactating mice

In the preceding section we used the sudden 2.5-fold increase in food intake associated with low ambient temperatures to test for the existence of reserve capacity. As a much more challenging test, let us now consider the much larger increase in food intake associated with lactation, which for most mammals is the energetically most demanding part of the life cycle. Every mother discovers that mother love does not come cheaply. For example, a mother mouse weans her normal litter of 8 pups when each pup weighs nearly half of the mother's own weight. At the end of the suckling period the mother is thus supplying all the nutrients for 4 times her body mass: her own mass, plus 8 pups with a collective mass 3 times her mass. That is as if a 60-kg human mother were to give birth to sextuplets and to nourish them solely by breast feeding until each 'baby' attained a weight of 30 kg at around age 8.

We did not content ourselves, however, with this already massive increase in food intake that Nature normally imposes on a mother mouse. To push our test of reserve capacity to its limits, we cross-fostered newborn pups from other litters onto a mother mouse that had just given birth, so as to increase litter size experimentally up to 26 pups[12]. No mother was able to process enough food and produce enough milk to wean all 26 pups, but most mothers tested could wean 14 pups – nearly double

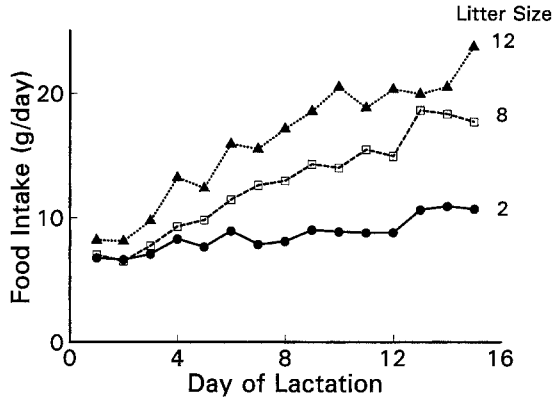

Figure 3. Food intake of chow-fed lactating mother mice, as a function of days after giving birth. Milk output reaches its peak on day 15, after which the pups begin to meet their food requirements by nibbling solid food. Litter size was experimentally adjusted to 2, 8, or 12 pups; 8 pups is the natural litter size. Note that the mother's food intake increases with number of pups, and also increased with day of lactation (as the pups grow larger), paralleling the demands that pups make on maternal milk production.

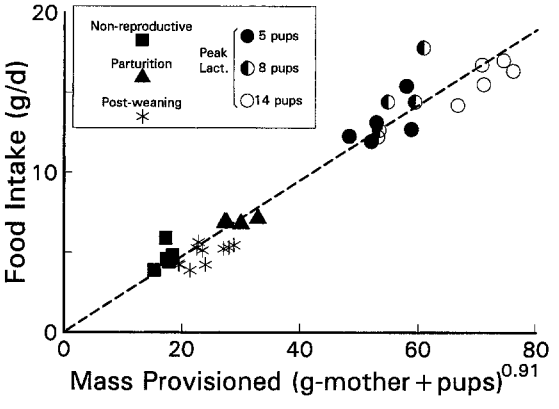

Figure 4. Food intake of female mice, as a function of reproductive state (different symbols) and of mass provisioned (the body mass of the mother herself and of the pups, if any, that she is nursing). 'Parturition' = the day on which the female gives birth; 'peak lact.' = the 15th day after giving birth, the peak day of lactation; 'post-weaning' = 14 days after the pups have been weaned. Litter size was experimentally adjusted to 5, 8, or 14 pups. The abscissa is taken as mass provisioned to the 0.91 power, the exponent of best fit for nonlinear regression of extra food consumed (beyond consumption in the nonreproductive state) against mass provisioned; but 0.91 does not differ significantly from either 1.0 or 0.75 when standard errors are considered. Note that food intake is directly proportional to (mass provisioned)$^{0.91}$, meaning that female mice in effect calculate accurately their food requirements. Based on Hammond and Diamond[12].

the usual litter size. Not surprisingly, the mother's food intake increases with her experimentally manipulated number of pups, and also increases (for a given pup number) with day of lactation, since each pup requires more milk (hence more maternal food intake) as it gets bigger (fig. 3). Figure 4 illustrates that the mother adjusts her food intake accurately so that it increases in direct proportion to the mass that she is nourishing (her own mass plus the mass of the pups). The mother's food intake at the peak of lactation (15 days after birth, just before the pups start to nibble solid food) is 3 times greater than her food intake in the nonreproductive state.

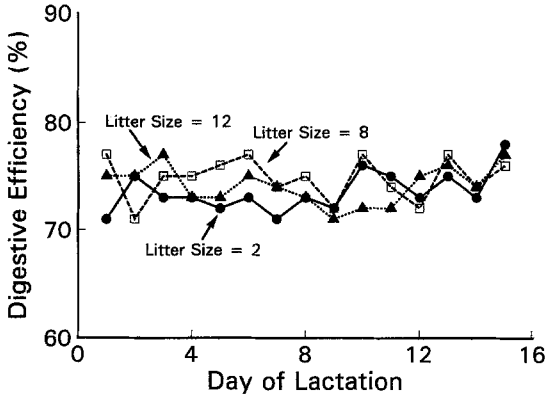

Figure 5. Apparent dry-matter digestive efficiency (calculated as in fig. 1 C) of chow-fed lactating mother mice, as a function of days after giving birth. Litter size was experimentally adjusted to 2, 8, or 12 pups. Note that digestive efficiency is independent of litter size and also independent of day of lactation – partly because mouse intestine already has considerable reserve capacity in the nonreproductive state (fig. 7), partly because the intestine hypertrophies.

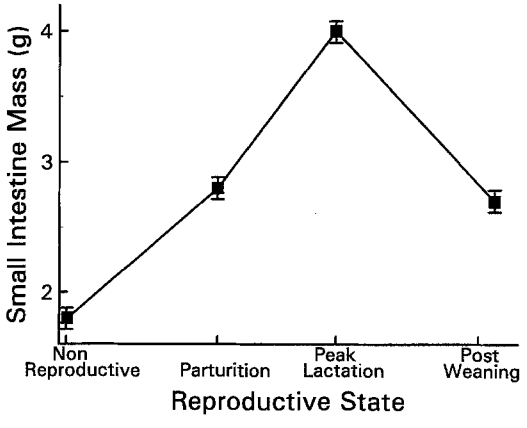

Figure 6. Small intestinal mass of female mice, as a function of reproductive state. Note that the intestine hypertrophies slightly during gestation and dramatically during lactation, then atrophies again after weaning, in parallel with changes in the females' food consumption.

Despite this large and rapid increase in food intake over a period of 15 days, digestive efficiency (fig. 5) does not vary over the course of lactation and does not vary with litter size (despite concomitant variation in food intake as shown in fig. 3). This implies either that the nonreproductive intestine already possessed enough reserve capacity to cope with additional food intake, or that the intestine grows sufficiently rapidly during lactation that growth in capacity keeps pace with growth in food intake. Both factors prove to contribute. The latter factor is documented by figure 6, which shows that gut mass hypertrophies nearly 2.5-fold from the nonreproductive state to peak lactation, then atrophies again when the young are weaned and the mother's food intake has declined back towards normal. The reversible hypertrophy of the intestine during lactation is analogous to the reversible hypertrophy of skeletal muscle in athletes who exercise and then cease training.

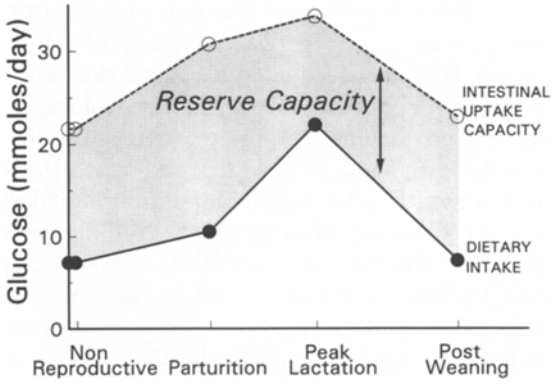

Figure 7. As figure 2, but for female mice as a function of reproductive state: dietary glucose intake per day (below), compared with the daily uptake capacity of the brush-border glucose transporter summed over the whole length of the small intestine (above). Note that uptake capacity exceeds dietary intake severalfold at 22 °C; that hyperphagia at peak lactation would nearly wipe out this safety margin, if it were not for an increase in uptake capacity due to intestinal hypertrophy (fig. 6); and that, as dietary intake declines again after weaning, so does uptake capacity due to intestinal atrophy. Based on Hammond and Diamond[12].

The last figure (fig. 7) shows the combined contributions of preexisting reserve capacity and intestinal growth to the intestine's ability to deal with lactational hyperphagia. In the nonreproductive state the intestine's glucose uptake capacity of 22 mmole/day exceeds glucose intake of 7 mmole/day by a comfortable safety margin. However, that reserve capacity would be used up by the glucose intake of 22 mmole/day at peak lactation – if it were not for the fact that intestinal hypertrophy increased uptake capacity to 34 mmole/day and regenerated some reserve capacity. Intestinal atrophy after the peak of lactation eliminates this added capacity again. The functional significance of the atrophy is surely similar to the significance of loss of wing muscles in island birds: the intestine's high rate of cell turnover makes it energetically one of the most costly tissues in the body, to be reduced when it becomes unnecessary.

Range of intestinal safety margins

Our laboratory has compared capacities with loads for the brush-border glucose transporter and several amino acid transporters, in six vertebrate species (rat, mouse, rabbit, cat, chicken, bullfrog), at various ages from birth or hatching until reproductive maturity[2–4,10,16,19,20]. We find that uptake capacities typically exceed loads (substrate intakes) by about a factor of 2, as we have discussed for the glucose transporter of mouse intestine. The highest factor is for cat intestine's arginine transporter, whose capacity exceeds its substrate intake by 10–30-fold depending on the cat's age[4].

Cat-lovers will recognize the ecological significance of this apparent anomaly. For cats, arginine is not merely an essential amino acid; it is a hyperessential nutrient[15]. Since wild cats are strict carnivores, their diet presents

them with large quantities of nitrogenous wastes to be excreted as urea, for whose synthesis exogenous arginine is required. If a cat consumes even a single meal without arginine, it is likely to die of nitrogen intoxication. As a result, cats go to great lengths to scavenge as much arginine as possible out of their diet, and it pays them to maintain a large reserve capacity for arginine uptake. In practice, since nutrient absorption proceeds sequentially along the length of the small intestine, the physiologically relevant variable to the cat is not its arginine transporter's capacity/load ratio. Instead, many transporter copies mean to the cat that it can absorb a high percentage (say, 99.5% instead of 90%) of ingested arginine as a meal traverses the length of the small intestine.

This example suggests that, with further information, differences among transporters in their capacity/load ratios may prove amenable to ecological interpretation. We anticipate that animals living in fluctuating environments, and transporters of critical nutrients, will tend to be characterized by high capacity/load ratios.

A testable hypothesis of animal design

Taylor and Weibel[18] have discussed an idealized model of economical design of an animal's body that they term symmorphosis. In effect, according to this model, natural selection will mold the body according to three principles. First, any given capacity will tend to be matched to its natural load. Second, within a given pathway of sequential steps, such as a metabolic reaction chain, capacities of steps should tend to be matched to each other; one may term this principle 'microsymmorphosis'. Third, capacities of different organs of the body (e.g., of energy-producing and energy-consuming organs, or of absorptive and excretory organs) will tend to be matched to each other; one may term this principle 'grand symmorphosis'.

If animals were really designed thus economically, no single step in a pathway would be rate-limiting. That would be uneconomic, since all other steps would be wasting energy and space on unutilizable reserve capacity, and since the animal could purchase an increase in flux through the whole pathway merely by adding capacity to a single step. This concept of economic design violates the belief of most biochemists that metabolic pathways are in fact governed by single dominant rate-limiting steps that serve as control points for regulation. In reality, evidence for this belief is highly controversial, and much (equally controversial) evidence supports belief instead in rate limitation distributed over a pathway's steps[13].

To broaden the issue from biochemical regulation in particular to symmorphosis in general, critics object to symmorphosis by pointing out many reasons why it seems to them unlikely to apply in practice[8, 11]. Still more reasons can be added to those adduced by the critics. Among the objections are the following:

1. Capacities should actually exceed peak loads by some reserve capacity; for capacities merely to be matched to peak loads would be too dangerous. We have seen that this reasoning does apply to intestinal brush-border transporters.
2. Reserve capacities should vary according to cost/benefit considerations: e.g., cheap capacities providing critical benefits should be present in large excess. The example of arginine transport in cat intestine supports this reasoning as well.
3. Reserve capacities should be appropriate to pathophysiological as well as physiological conditions. A reserve capacity that seems excessive in a healthy animal may actually be essential for a wild animal sapped by parasites and microbes. Perhaps the apparent 10-fold surfeit in pancreatic enzyme output is to be understood in this light.
4. Many capacities serve multiple functions. A capacity may have to exceed its load by far in one pathway, in order to be matched to load in another pathway.
5. Capacities may be mismatched to current loads because of developmental constraints, i.e. in order to arrive at a value appropriate to a load expected at a later stage of ontogenetic development.
6. Capacities may be mismatched to loads because of design constraints: it may simply be impossible for natural selection to achieve the most economic match.

We agree with the critics that examples of capacity/load mismatches for these and other reasons are likely to abound: we have already cited such examples. We disagree with the critics, however, that these mismatches invalidate the utility of the concept of symmorphosis. The concept is worth posing not because we believe it to be literally true, but because only by posing it as a testable hypothesis of economic design can one hope to detect where it breaks down, and to identify the interesting reasons for its breakdown.

As mentioned at the outset of this section, symmorphosis actually consists of three sets of related principles, of which capacity/load matching is only the first. For the other two of these principles – capacity/capacity matching within and between organs (microsymmorphosis and grand symmorphosis) – preliminary evidence with respect to nutrient processing yields surprising support. Activities of all major steps in carbohydrate digestion – complex carbohydrate splitting by pancreatic amylase, disaccharidase splitting by brush-border sucrase, monosaccharide uptake by the brush-border glucose transporter, and monosaccharidase export by the basolateral glucose transporter – are known to be upregulated by dietary carbohydrate levels, suggesting distributed rate control and matched capacities. Preliminary measurements in our laboratory (E. A. Lee, unpublished observations) reveal close match between capacities of sucrase and of the brush-border glucose transporter. Observations of comparable degrees of hypertrophy in

intestines, kidneys, livers, and spleens of cold-exposed hyperphagic mice suggest matches between capacities of these organs. Thus, symmorphosis – if regarded as an essential testable hypothesis rather than as a literal truth – may provide a unifying set of three principles for quantitative evolutionary understanding of biological design.

Acknowledgments. It is a pleasure to thank our many colleagues for stimulating discussion. This work was supported by National Institutes of Health grants GM 14772 and DK 42973.

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Pathways for oxidative fuel provision to working muscles: Ecological consequences of maximal supply limitations

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Abstract. The study of metabolic fuel provision and its regulation has reached an exciting stage where specific molecular events can be correlated with parameters of the organism's ecology. This paper examines substrate supply pathways from storage sites to locomotory muscle mitochondria and discusses ecological implications of the limits for maximal flux through these pathways. The relative importance of the different oxidative fuels is shown to depend on aerobic capacity. Very aerobic, endurance-adapted animals such as long distance migrants favor the use of lipids and intramuscular fuels over carbohydrates and circulatory fuels. The hypothesis of functional co-adaptation between oxygen and metabolic fuel supply systems allows us to predict that the capacity of several biochemical processes should be scaled with maximal oxygen consumption. Key enzymes, transmembrane transporter proteins, glucose precursor supply and soluble fatty acid transport proteins must all be geared to support higher maximal glucose and fatty acid fluxes in aerobic than in sedentary species.
Key words. Metabolic substrates; aerobic capacity; regulation; glucose; lactate; fatty acid; migration.

Introduction

Animals show an amazingly wide variety of locomotory adaptations allowing them to occupy otherwise inaccessible ecological niches. These specializations provide unique opportunities to live in particular environments or to support specific lifestyles. Habitat size, food quality and distribution, predator-prey interaction, and reproductive behavior are major ecological parameters that depend very strongly upon a species' aptitude for movement[2, 50]. Both endurance capacity and maximal speed are important determinants of these life-history characteristics. Here, I will focus on sustainable locomotion, a correlate of oxidative metabolism, rather than on maxi-