Hypertrophy versus Hyperplasia

How much organs can grow depends on whether their functional units increase in size or in number.

Richard J. Goss

If it were technically possible to exchange livers between mouse and elephant embryos, I suspect that each transplanted organ would grow to a size appropriate for its new host. Kidneys, on the other hand, probably would not. The explanation for these predictions lies in the ability of some organs to increase indefinitely the number of structural units upon which their functions depend (hyperplasia), whereas others must resort to the physiologically less rewarding process of increasing the size of those units (hypertrophy). These alternative modes of growth, both of which are stimulated by functional demands, may be expressed at several levels of organization. It is at the level of the cell, however, that the nexus between growth and function is especially instructive (*1*).

Differentiation and Mitosis

There are two kinds of cellular differentiation with respect to mitotic competence (*2*). Cells that retain within their cytoplasm the end products of their specific synthesis forfeit the ability to divide when they become fully differentiated. Those which secrete their products into the extracellular compartment, however, can proliferate even after completing differentiation. Examples of these two categories, together with the specific end products synthesized in each case, are itemized in Table 1.

In view of this correlation, the long-recognized classification of tissues and organs of the body according to their patterns of mitotic activity (*3*) is subject to revision. Nonmitotic cells are characteristic of static tissues and of the mature cells of renewing tissues, the two differing from each other in the size, life-span, and provisions for replacement of their cells. Expanding organs consist of cells capable of division despite their fully differentiated state. Yet the functional activity of these cells is apparently held in abeyance during mitosis, and there is now evidence that dedifferentiation, mediated by lysosomes, may occur when such cells prepare for division (*4*).

Cellular hyperplasia is clearly an important factor contributing to the growth and maintenance of organs, but even in the absence of mitosis some tissues are capable of considerable growth (as in hypertrophy of muscle fibers). Others (lung, kidney) are limited in their adult growth potential because they cannot increase the number of histological units of which they are composed no matter how many times their cells may divide. Thus hypertrophy at one level of organization is the result of hyperplasia at lower levels, and it is the relationship between the potential number and size of functional units upon which the growth potential of an organ depends.

Size and Work Load

The size of an organ is largely determined by the work it has to do, for the physiological mechanisms which regulate function also control growth (*5*). Responses to functional demands, however, vary from one organ to another. Some organs seem capable of limitless growth, whereas others have very circumscribed potentials for enlargement. Such discrepancies have traditionally been explained in terms of the varying mitotic capabilities in renewing, expanding, and static tissues (*3*). It is now apparent that any classification of tissues and organs based solely on *cellular* hyperplasia may be inappropriate. The capacity of an organ to grow to meet its physiological responsibilities depends mostly on its ability to multiply the *functional units* of which it is composed, regardless of whether these are represented by organelles, by cells, or by tissues.

A functional unit may be defined as the smallest irreducible structure still capable of carrying out organ-specific physiological activities. In some organs such units may be represented at the tissue level by such histological entities as nephrons, osteons, exocrine acini, pulmonary alveoli, intestinal villi, hepatic parenchymal cords, and thyroid and ovarian follicles. In other cases, functional units may be identified with cells, as exemplified by erythrocytes, leukocytes, and the cells of many endocrine glands. At the subcellular level of organization, the myofibrils, or rather the sarcomeres, are the functional units of striated muscle; and the terminal connections of neurons, or perhaps even the neurofilaments, may logically be regarded as the functional units of nervous tissue. Although the designation of these units is admittedly arbitrary, and even unrealistic in the case of some tissues (for example, cartilage and skin), such a classification must be attempted if the growth of organs is to be understood in terms of their function.

Determinate and Indeterminate Structures

There are in the vertebrate body certain structures which occur in fixed, species-specific numbers. Formed early in ontogeny, they grow only in size throughout the remaining maturation period of the organism. Their numbers are therefore determinate (Fig. 1). For example, the number of hairs, feathers, or scales on the body is constant regardless of how big the animal may grow. There is also a species-specific population of villi and glands in the gut (*6*). Similarly, the number of internodes along the myelin sheaths of nerve fibers is determinate (*7*). All of these structures can be replaced if lost, but their normal numbers cannot be augmented. In contrast, neurons and striated muscle fibers can be neither replaced nor multiplied (Fig. 2).

It is with these latter structures that the functional units of the lungs and kidneys are to be classified, for the numbers of alveoli and nephrons cannot be augmented beyond early stages of development, nor can they be replaced

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if lost. In the human (Fig. 1), new pulmonary alveoli gradually cease to develop during childhood years (*8*), and the last renal nephrons are formed several weeks before birth (*9*). Although these two organs remain mitotically competent, further growth can be achieved only by hypertrophy of their pre-established functional units. Hence the dimensions of alveoli (*10*) and nephrons (*11*) are larger in adults than in immature individuals, and if their numbers are reduced, or the physiological demands otherwise increased, even greater enlargement may take place by compensatory hypertrophy (Figs. 2 and 3). The lost ability to make new functional units does not preclude adaptive growth in the lung (*12*) and kidney (*13*), but the physiological disadvantages of hypertrophy, as compared with hyperplasia, constitute a serious handicap to functional efficiency.

Indeterminate organs never lose the ability to make new functional units (Fig. 1). Those which make new functional units at the cellular or histological level have unlimited potentials for growth (Fig. 4). That this potential may seldom if ever be fully expressed does not diminish the significance of the regenerative reserves in these organs. Indeed, it emphasizes the dependence of their growth on the regulating influences of functional demands.

Table 1. Correlations between mitotic behavior of fully differentiated mammalian cells and the intra- or extracellular locations of specific end products.

Cells	End products
Non-mitotic	Intracellular
Muscle fiber	Myofilaments
Neuron	Neurofilaments
Schwann cell	Myelin
Retinal rod	Rhodopsin
Lens fiber	Crystallin proteins
Epidermal cell	Keratin
Adipose cell	Fat globule
Erythrocyte	Hemoglobin
Sperm	Acrosome
Mitotic	Extracellular
Endocrine cells	Hormones
Plasma cell	Antibodies
Fibroblast	Collagen
Chondrocyte	Chondroitin
Odontoblast	Dentin
Liver cell	Plasma proteins
Acinar cells of:	
Pancreas	Digestive enzymes
Salivary gland	Saliva
Lachrymal gland	Tears
Mammary gland	Milk

This regulation is best illustrated in the various tissues of the body in which functional units are constantly or periodically being lost and replenished. Red blood cells in man are destroyed at the rate of about two million per second, yet the demand of the body for oxygen stimulates a commensurate rate of erythropoiesis in the marrow. (Investigations of comparable control mechanisms for leukocyte and platelet production are greatly to be desired.) The gradual renewal of cells in the adrenal cortex and the cyclical turnover of follicles in the ovary are regulated by trophic hormones upon which the functions of the organs depend. Less well understood are the slow erosion and reconstruction of Haversian systems that occur in bone, presumably in response to a combination of chemical and physical factors associated with skeletal mechanics.

Many organs in which there is normally no turnover of functional units can make new units when occasion demands. Liver regeneration (*14*) takes place by the production of additional parenchymal cords in the hepatic lobules. Such exocrine organs as the pancreas, salivary, and mammary glands can form new secretory acini under appropriate physiological stimuli, and the thyroid gland can always produce new follicles. Other endocrine glands with functional units at the cellular level of organization remain mitotically competent throughout life. The growth of all such organs theoretically has no limit, for in them, unlike the lung and kidney, the number of functional units to be produced is dictated by the physiological demands of the systems they serve.

Intermediate between the determinate and indeterminate types of organs are those having subcellular functional units. Nerve endings (*15*), neurofilaments (*16*), and myofibrils (*17*) can usually regenerate or increase in number under special circumstances. Yet their numbers can be augmented only within the confines of the neuron or muscle fiber. Therefore their hyperplasia is limited not so much by any innate lack of proliferative potential as by spatial restrictions in the cells of which they are a part.

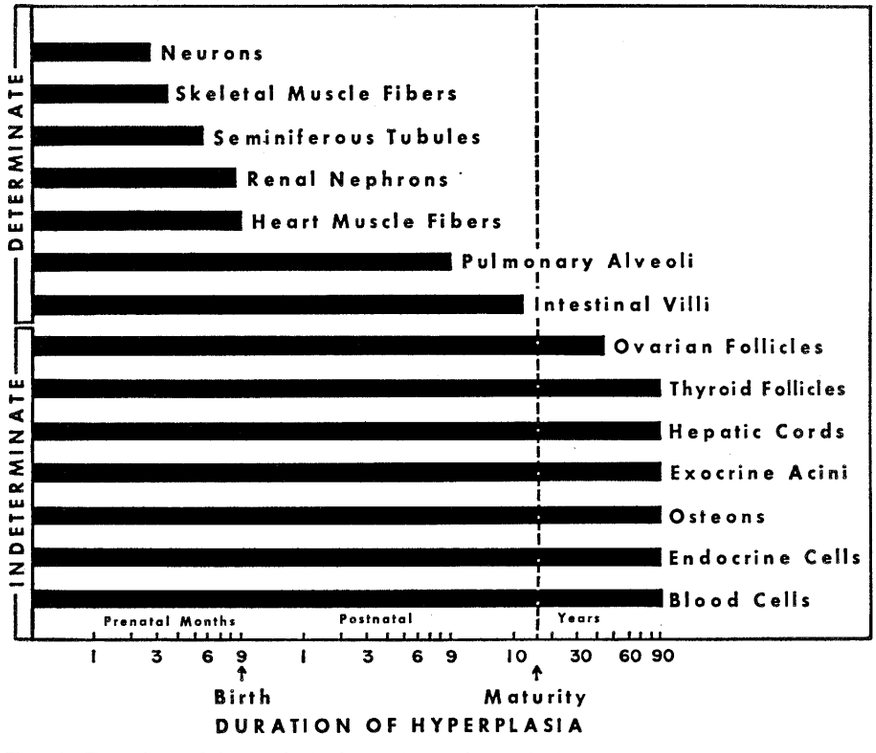


Fig. 1. Duration of hyperplasia in various histological structures in man. Organs in which substructures cease to multiply before maturity have determinate numbers of structural units and restricted capacities for growth beyond normal adult dimensions. Those which retain their hyperplastic abilities throughout life can grow potentially without limit, and therefore have indeterminate numbers of structural units.

Levels of Organization

Tissues and organs of the body may be classified according to the highest level of organization at which they are capable of hyperplasia. The effects of hyperplasia at three different levels (organelle, cell, tissue) are illustrated in Fig. 5. In each instance the organ as a whole may undergo hypertrophy, but the mechanism by which this is achieved differs. When only organelles can multiply, as in striated muscle,

then hypertrophy occurs at the cellular and tissue levels of organization. If hyperplasia is possible as high as the cellular level, histological structures hypertrophy as a result of the increased population of constituent cells. When proliferation can occur at the tissue level of organization, then everything multiplies except the organ itself, which becomes hypertrophic. In each of these three categories the number of organelles increases equally but their distribution among cells and tissues differs. Hyperplasia of most organelles, like that of molecules, is therefore a phenomenon characteristic of all cells irrespective of their mitotic capacities.

For maximum growth potential, an organ must first be able to multiply its functional units. Most adult organs can do this, with the exception of the lung and kidney. But even if hyperplasia of functional units is possible, the extent of their proliferation may eventually be limited by the lack of proliferative ability at the next higher level of organization. For example, the size to which striated muscle can enlarge is restricted not by any inherent inability of myofilaments, sarcomeres, or myofibrils to multiply, but by the failure of the muscle fibers to divide. Similarly, the lack of hyperplasia at the tissue level in the kidney puts a ceiling on mitotic activity among the cells of the nephrons.

Other things being equal, it is therefore to the advantage of the organ to have functional units at as high a level of organization as possible. It is equally important that organs do not lose the capacity to multiply their functional units, a process which becomes increasingly complex at higher levels of organization. Despite these conditions, most organs of the body are endowed with functional units at a level of organization not incompatible with their unrestricted hyperplasia.

Such structures as follicles and acini, being histological entities, are subject to little or no spatial limitation on their proliferation. If cells are not organized into specialized tissues, as in blood and many of the endocrine glands, then hyperplasia of functional units is achieved by unrestricted mitosis. When cells are arranged into histological structures that cannot proliferate, as in pulmonary alveoli and renal nephrons, then growth of the whole organ is limited even though the cells remain mitotically competent. Similarly, hyperplasia of organelles in nerves and muscles is of limited value when cellular proliferation is impossible. It is because of these differences, then, that organs must be classified according to whether or not the proliferation of their functional units, if it occurs at all, is restricted by the absence of hyperplasia at higher levels of organization.

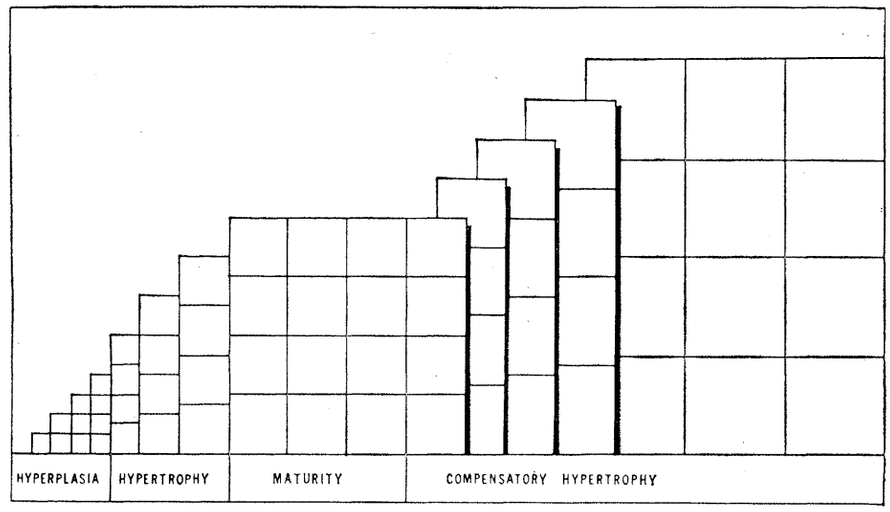


Fig. 2. Growth profile of determinate tissues such as nerve or muscle. Each square represents n neurons or muscle fibers. After an early period of hyperplasia, these units can grow to adult dimensions only by hypertrophy. With increased functional demands, however, further compensatory enlargement is possible.

Mechanisms of Hyperplasia

There are two ways to have your cake and eat it too. One is to divide the cake in half; the other is to make another cake. Both of these methods (division and replication) may be used to increase the numbers of units in the body.

Inasmuch as the failure of some structures to reproduce themselves is a major limiting factor for growth in certain organs, it is especially important to determine why hyperplasia is not a universal attribute at all levels of organization. To this end, let us examine how hyperplasia occurs among those units which do possess the ability to increase in number.

Molecules, of course, cannot multiply by division without becoming something different. At this level of organization, therefore, hyperplasia is achieved only by the synthesis of new units, even though this may sometimes depend upon "parent" molecules to serve as templates for replication.

Cells, on the other hand, cannot be created anew. Their only recourse is to proliferate by mitotic division. Not all cells, however, are capable of mitosis, and in at least one instance (blood platelets derived from megakaryocytes) reproduction may take place by cytoplasmic fragmentation.

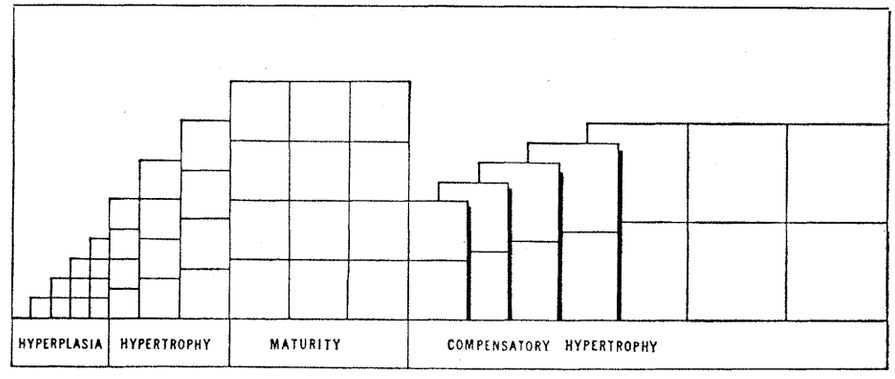


Fig. 3. Graphic representation of growth in the lung or kidney, in which the alveoli or nephrons (squares) lose the capacity for hyperplasia before maturity but continue to grow by hypertrophy. Following partial resection of these organs, compensatory hypertrophy of the remaining functional units occurs.

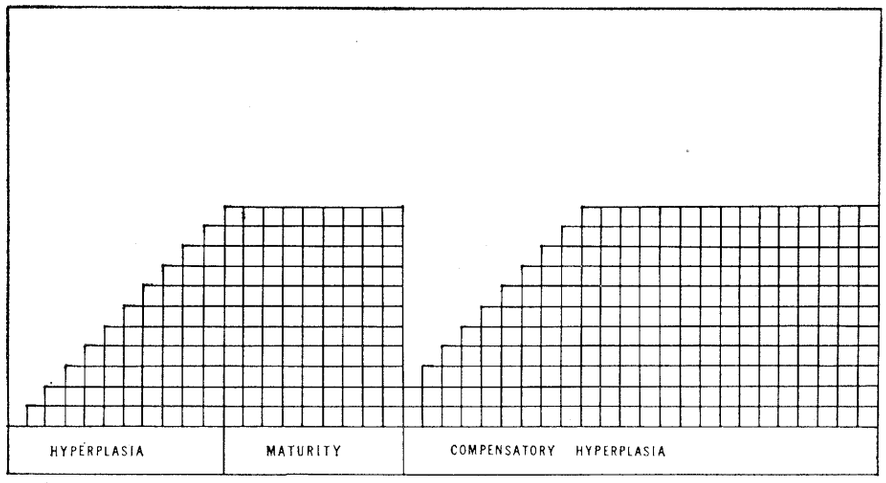


Fig. 4. Pattern of growth in organs with indeterminate numbers of functional units (for example, exocrine and endocrine glands). Hyperplasia of these units keeps pace with the overall growth of the body throughout life, and makes unlimited regeneration possible.

At the level of the organelle and tissue, however, both methods of hyperplasia are encountered. And in fact some structures are even capable of multiplying either by division or by neogenesis.

Organelles usually reproduce by a developmental process not involving division of pre-existing structures. Cilia, microvilli, and motor end plates, for example, are all formed *de novo*. Ribosomes, Golgi apparatus, spindle fibers, and pigment granules also have ontogenies independent of homologous parent structures. Myofibrils are normally synthesized autonomously, but the possibility that they can be formed by longitudinal splitting of those already made cannot be ruled out (*18*).

Although mitochondria and chloroplasts may develop from microbodies (*19*) or proplastids (*20*) of uncertain origin, they are likewise capable of reproducing by division (*20, 21*), an attribute they share with chromosomes and centrioles. These four intracellular structures, then, are the sites of their own synthesis and are also organelles known to contain DNA.

Hyperplasia among histological units of function in the adult recapitulates their mode of formation in the embryo. By virtue of the persistence of generative tissues in the mature organism, new structures may originate from incompletely differentiated cells. Buds of cells that sprout from the duct epithelium of exocrine glands develop into secretory acini, and it is from the germinal epithelium of the ovary that Graafian follicles are derived. Thyroid follicles may either take shape out of undifferentiated interstitial cells, or bud off mature follicles. In growing bone, new osteons are organized from the osteogenic layer, whereas in the mature skeleton they are reconstructed at the site of their resorbed predecessors. Thus, with the exception of thyroid follicles and Haversian systems, which may sometimes form at the expense of, or in place of, fully developed homologous structures, most units of function at the tissue level owe their existence in the adult to a reserve population of germinative cells.

The absence of such reserves may account for the inability of the kidney to form new nephrons and of the lung to augment its adult complement of pulmonary alveoli. In the immature animal, the histological units of function in these two organs are formed by recruitment of undifferentiated cells. When this supply has been consumed, no further units can be developed. Unable to proliferate by subdividing their preformed nephrons or alveoli, the kidney and lung must forever make do with their original endowment of functional units, a situation which may account for their high susceptibility to pathological disorders in old age.

In the development of these organs there is a race between the rate of proliferation of undifferentiated cells and the rate at which these cells become incorporated into differentiated structures. Hyperplasia of functional units ceases when the rate of incorporation exceeds the rate of proliferation. This is reminiscent of how the growth of long bones is arrested in most higher vertebrates. Osteogenesis in the cartilaginous plate eventually overtakes the process of chondrogenesis, thereby exhausting the resources for further development. If, as in the rat, this does not occur, then the growth of bones can continue throughout life.

A comparable situation is believed to exist in fish kidneys, which have no theoretical upper size limit. Here the number of nephrons parallels the growth of the body, presumably because of the lifelong retention of nephrogenic cells.

Ontogeny and Phylogeny

There seems to be a reciprocal relationship between hyperplasia and hypertrophy. Although there are some interesting exceptions to the rule, most structural units able to proliferate maintain a fairly constant size. Those which cannot proliferate must enlarge, and their dimensions are therefore more variable (Fig. 6). Hence the mode of growth can in many instances be inferred from size comparisons during ontogeny and phylogeny (*22*).

For example, many differentiated structures do not change appreciably in size despite the overall growth of the body. Thus if young and old, or small and large, animals are compared, the dimensions of some structures remain nearly constant (Fig. 6*A*). This means that the hyperplastic mode of growth is tissue- and organ-specific, regardless of the ultimate size attained by the adult organism. If a unit can multiply throughout ontogeny, it need not resort to hypertrophy during the phylogenetic enlargement of body size.

Structures which undergo hypertrophy during ontogeny, however, are usually larger in big species (Fig. 6*C*). In the course of development both hyperplasia and hypertrophy go on for longer periods of time in a large animal, giving rise to units that are not only more numerous but also greater in size than in smaller species.

Heart muscle is an anomaly. Although its fibers hypertrophy as an animal matures (Fig. 6*B*), they are remarkably constant in size in most adult mammals (*23*). Hence the total number of cardiac muscle fibers to differentiate in the embryo is proportional to the normal adult size of the heart, regardless of how big or little the animal is. It follows that myocardial hyperplasia must persist to later stages of development in larger species. Although cardiac muscle fibers may double their

size in hypertrophic hearts (*18*), the consistency of their normal adult dimensions clearly suggests a size limit commensurate with optimum physiological activity. Compared with skeletal muscle, this may be related to their low tolerance for lactic acid and their greater dependence upon adequate supplies of oxygen (*24*).

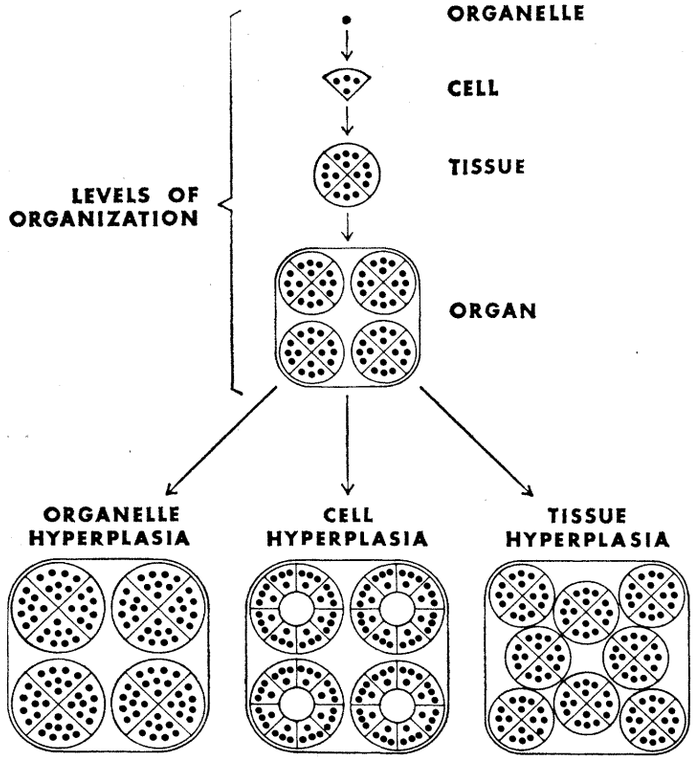
Skeletal muscle fibers, being multinucleate, are permitted wider latitudes in size during ontogeny and phylogeny (*25*). In general, the larger an animal is, the longer and thicker are its muscle fibers. Under the influence of functional overload they are capable of additional hypertrophy in width, but not in length. The upper limits of this compensatory growth are usually not dictated, as is often assumed, by the greater distances through which materials must diffuse between the innermost myofibrils and the capillaries outside the cell. A hypertrophic muscle fiber in a mouse is considerably smaller than its normal homologue in an elephant. The difference is that the skeletal muscle fibers in the elephant are longer than their counterparts in the mouse, and they have proportionally more nuclei. It is the latter attribute which appears to be most closely bound up with how big a skeletal muscle fiber can grow. Early in development, therefore, the ultimate size of the fiber is ordained by how many nuclei are included in it. Thus the adult size of an animal is anticipated, if not determined, well before birth.

Conclusions

Although all tissues and organs of the body are normally subject to the growth-regulating influences of functional demands, some are potentially capable of unlimited growth while others are not. This depends on whether hyperplasia of their functional units ceases prior to maturity or can continue throughout life. In the former case, further growth is limited by the extent to which hypertrophy can enhance physiological efficiency. Some of the body's most vitally essential organs (heart, brain, kidney, lung) lack the ability to make additional structural units in the adult and are therefore handicapped in compensating for the depreciations of advancing age. Theoretically, at least, other organs (glands, renewing tissues) possess unlimited powers of regeneration because they never lose the capacity (latent or expressed) for hyperplasia.

There is a strategy in the way growth mechanisms have evolved. It may be significant that the so-called "hypertrophic" organs lose the capability for hyperplasia, because not to do so might jeopardize their growth regulation. If size is determined by functional demands, then the latter must not operate continuously lest growth go on without interruption and lead to overproduction of functional units. Only renewing tissues can tolerate perpetual growth because they get rid of excess structures as fast as they are formed. Endocrine and exocrine glands are in most cases known to function discontinuously and are thus not in danger of being overstimulated. The heart, lungs, and kidneys (and brain?), however, must work incessantly. Were their functional units capable of hyperplasia and at the same time subject to control by functional demand, then overgrowth would seem to be inevitable. By giving up the potential for hyperplasia in favor of the necessity for constant function, these organs have adopted a strategy that enables them to become hypertrophic to a limited extent while doing their jobs efficiently.

It is a curious fact that the unrestricted proliferation of biological structures cannot occur at all levels of organization. The counterpart of cancer, which is a cellular phenomenon, does not exist among molecules or cytoplasmic organelles, nor is it



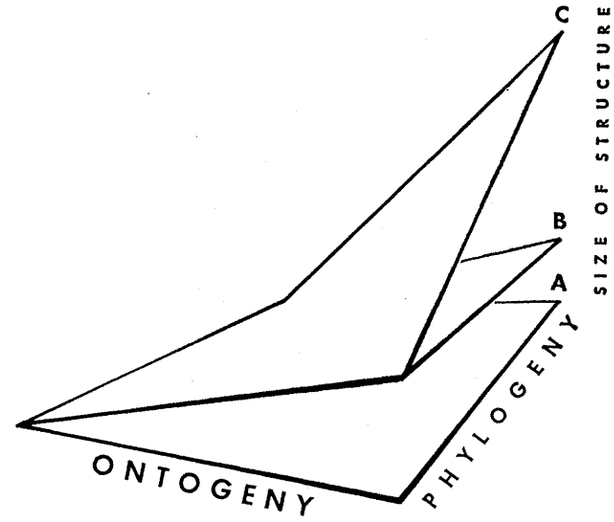


Fig. 5 (left). Diagram illustrating alternate ways in which organs can double their size according to the levels of organization at which hyperplasia takes place. Growth potential increases from left to right. Fig. 6 (right). Schematic comparison of dimensional changes in mammalian structures in relation to increasing body size during ontogeny and phylogeny. *A* represents units which are approximately the same size regardless of age or species, such as organelles, proliferative cells, exocrine acini, hepatic parenchymal cords, and thyroid and ovarian follicles. *B* is characteristic of cardiac muscle fibers which enlarge during ontogeny, but not with phylogenetic increases in body size. *C* is typical of hypertrophy from young to old, or small to large species, as in neurons, skeletal muscle fibers, Purkinje fibers, pulmonary alveoli, and renal nephrons.

known to occur at the histological level of organization. Even in organs made up of histological units of function and having the potential for unlimited hyperplasia (for example, liver, exocrine glands, thyroid, ovary), the population of functional units never exceeds the number needed to fulfill the physiological requirements of the body. Above and below the level of the cell, therefore, structures are not permitted to escape the constraints of functional demands which control their production. The fact that cells can occasionally do so when they become neoplastic may reveal as much as it conceals about the problem of growth regulation.

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NEWS AND COMMENT

The Coast Redwoods: Struggle over National Park Proposals

In the battle over the fate of northern California's coast redwoods, the saw has the current advantage. Approximately 85 percent of the nearly 2 million acres which originally stretched from the Oregon border to south of San Francisco have already been cut. Fewer than 300,000 acres of virgin redwoods remain; of this area, only about one-sixth is now publicly preserved. The Interior Department estimates that, at the present rate, the remaining virgin growth will be completely cut in 2 to 3 decades.

The desire to preserve a greater portion of the virgin redwoods while the trees remain standing in significant groves is the crux of the current struggle. For the conservationist, destruction of the redwoods is an irreversible tragedy. President Johnson emphasized the "now or never" need for redwood preservation when he told Congress in February, "It is possible to reclaim a river like the Potomac from the carelessness of man. But we cannot restore—once it is lost—the majesty of a forest whose trees soared upward 2000 years ago."

The coast redwood (*Sequoia sempervirens*) is one of the oldest living things, as well as the tallest living thing; the highest known tree exceeds 365 feet. (The much less extensive stands of the Sierra redwood—*Sequoia gigantea*—are already protected.) Conservationists often point out that some standing coast redwoods were alive at the time of Christ. Their great antiquity, as well as their size and beauty, leads some admirers to regard them as "holy trees."

The lumber companies argue that the coast redwood is an extremely fast-growing tree; some of their trucks carrying the giant logs to the mills flaunt the slogan "Redwoods Forever." While it is true that the coast redwood becomes commercially harvestable within a 40- to 80-year period, it requires several hundred years to reach its full stature. For the redwood conservationist, second-growth redwoods cannot be more than second-rate, at least in his experience or that of foreseeable generations. At a June Senate Interior Committee hearing, Ralph W. Chaney, professor emeritus of paleontology at the University of California, Berkeley, was even more pessimistic. He said that, because of climatic and other changes, "there are many of us who doubt that giant redwoods may ever grow extensively again."

The concern about the disappearance of the virgin coast redwood forests prompted President Johnson to request, on 23 February, that a Redwood National Park be created in northwestern California by combining the Jedediah Smith and Del Norte Redwoods state parks with land owned by the Miller Redwood Company in the Mill Creek watershed. The Redwood National Park proposal is unusual in that it is the most expensive national park ever requested by an administration (approximately $55 million), and that it is the first administration park proposal to concentrate specifically on preserving a single plant species.

The administration bill has acquired important backers, including Thomas H. Kuchel of California, Senate minority whip and ranking Re-