

Is West, Brown and Enquist's model of allometric scaling mathematically correct and biologically relevant?

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Introduction

Geoffrey West and colleagues published a series of papers, mostly in *Nature*, *Science* and *Proceeding of the National Academy of Sciences of the USA*, on scaling of different aspects of physiology and life history in organisms as distinct from each other as plants and animals (West, Brown & Enquist 1997, 1999a,b; Gillooly *et al.* 2001). Recently they even extended their considerations down to cells, mitochondria and respiratory complexes (West, Woodruff & Brown 2002). All these papers invoke the $3/4$ scaling of metabolic rate claimed to be derived in their first and basic paper (West *et al.* 1997; WBE hereafter). It is therefore extremely important to check the legitimacy of their model.

First we show that to make WBE's model mathematically consistent either metabolic rate must be directly proportional to body mass (recall that the model is aimed to explain the $3/4$ exponent for metabolic rate) or one of the basic model assumptions, that is, the size-invariance of terminal supplying vessels, must be violated. Then we show that animals built according to WBE's model cannot represent a broad range of sizes, because for large animals the volume of blood vessels would exceed body volume. Later we demonstrate that many features of the plant vascular system, insect tracheal system, vertebrate lung or vertebrate cardiovascular system do not conform to WBE's model assumptions. Finally, we argue that $3/4$ scaling for metabolic rate is by no means universal, and therefore WBE's model was built to explain a non-existent pattern.

The anatomy of West *et al.*'s (1997) model

WBE write:

We develop a quantitative model that explains the origin and ubiquity of quarter-power scaling; it predicts the essential features of transport systems, such as mammalian blood vessels and bronchial trees, plant vascular systems, and insect tracheal tubes. It is based on three unifying principles or assumptions: First, in order for the network to supply the entire volume of the organism, a space-filling fractal-like

branching pattern is required. Second, the final branch of the network (such as the capillary in the circulatory system) is a size-invariant unit and third, the energy required to distribute resources is minimized.

Size-invariance here means that capillary number scales isometrically (with exponent 1) with size. Although WBE acknowledge that 'Biological networks vary in the properties of the tube (elastic to rigid), the fluid transported (liquid to gas), and the nature of the pump ...' they mainly use the terms of the cardiovascular system to describe both the structure and function of the network.

Their network consists of a hierarchy of branching vessels starting, in the case of the cardiovascular system, from the aorta and ending with capillaries. The assumption that the final branch is size-invariant causes the number of levels to be a function of body size (or vice versa): more levels are required to fill a larger volume with the same density of final vessels.

Further on, terms characteristic of the cardiovascular system will be given in italics if the system is more general or if it cannot represent the cardiovascular system because of some additional assumptions. The consecutive levels of branches of the network are indexed with the symbol k , where $k = 0$ for *aorta* and $k = c$ for *capillaries*. A branch k has length l_k and radius r_k . Flow velocity through a vessel of the level k equals \bar{u}_k , and the number of such vessels is N_k . The volume rate of flow in a single vessel at each level is \dot{Q}_k , and because the fluid (or gas) must be conserved throughout the system, the following relation must hold (eqn 2 in WBE):

$$\dot{Q}_0 = N_k \dot{Q}_k = N_k \pi r_k^2 \bar{u}_k. \quad \text{eqn 1}$$

Because equation 1 holds for all levels,

$$\dot{Q}_0 = N_c \dot{Q}_c = N_c \pi r_c^2 \bar{u}_c. \quad \text{eqn 2}$$

Now, denoting metabolic rate by B and body mass by M , a new assumption is formulated: 'Because the fluid transports oxygen and nutrients for metabolism, $\dot{Q}_0 \propto B$; thus, if $B \propto M^a$ (where a will later be determined to be $3/4$), then $\dot{Q}_0 \propto M^a$. Equation 2 therefore predicts that the total number of capillaries must scale as B , that is, $N_c \propto M^a$.' The implicit part of this assumption is that the entire group of organisms compared shares the same exponent for the relationship between metabolic

rate B and body mass M (although at this point of the considerations they may differ in their intercepts). It is not clear at this point whether the amount of oxygen or nutrients is proportional to the fluid volume, although it seems from further considerations that such an implicit assumption has also been made. It is likely that in animals the amount of oxygen per blood volume will be larger for small animals often having smaller erythrocytes and/or higher haemoglobin concentration (Kostelecka-Myrcha & Cholostiakow-Gromek 2001; Gregory 2002). At this point the origin of exponent a is not specified, and $a < 1$ may reflect transport ability per fluid volume decreasing with size.

Now WBE introduce scale factors β_k for vessel radius and γ_k for vessel length:

$$\beta_k \equiv r_{k+1}/r_k; \gamma_k \equiv l_{k+1}/l_k.$$

The next assumption they make is that the scaling factor for length does not depend on the vessel level k ($\gamma = \gamma_k$ for all k). The scaling factor for radius also does not depend on the level k in a non-pulsatile system ($\beta = \beta_k$ for all k), but depends on the level in a pulsatile system. WBE further assume that at each node of level k the vessel splits into n vessels of level $k + 1$, with n independent of the level. This is a very strong assumption, because this branching is completely independent of the geometry of the organism: full regularity is assumed. WBE need this assumption in order to replace the number of capillaries, N_c , with the number of branching levels N , that is, $N_c = n^N$. This assumption results from the self-similarity of the fractal, being a stronger version of one of the basic explicit assumptions cited at the beginning of this section.

WBE consider the network system in terms of a self-similar fractal this way: 'A space-filling fractal is a natural structure for ensuring that all cells are serviced by capillaries. The network must branch so that a group of cells, referred to here as a "service volume," is supplied by each capillary.' WBE rightly consider entire body volume W (proportional to body mass M) as the sum of spheres (service volumes) with diameters l_c (the length of capillaries). Thus the total volume equals $4/3\pi(l_c/2)^3 N_c$, and because of the assumption that l_c is size-invariant it follows that the number of capillaries N_c must be proportional to body mass M . This contradicts WBE's earlier assumption that $N_c \equiv M^a$, unless a equals 1. The only way to overcome this inconsistency for $a \neq 1$ is to assume that l_c changes with size (l_c increases with size for $a < 1$), but this violates the assumption of capillaries' size-invariance. Thus to keep the model mathematically consistent either capillary length must decrease with size or metabolic rate must be proportional to body mass ($a = 1$).

Although it is clear that WBE's model must be rejected because of its basic internal inconsistency, the error in deducing the $3/4$ exponent should be pinpointed in any event. Let us follow their reasoning. They write: 'Because $N_c = n^N$, the number of generations of branches scales only logarithmically with size':

$$N = \frac{a \ln(M/M_0)}{\ln n}. \quad \text{eqn 3}$$

In fact, the next important assumption is introduced here: the proportionality of capillary number to metabolic rate B directly and to M^a indirectly does not suffice to write equation 3. The entire group of compared organisms must share not only the exponent but also the intercept in the equation relating metabolic rate to body mass. WBE overcome this problem by introducing a poorly defined term, 'normalization scale' M_0 .

In the next step, WBE eliminate body mass M and the number of branching levels N from equation 3, and replace them with a combination of scaling factors β and γ . To achieve this goal they consider the volume of the fluid in the system V_b :

$$V_b = \sum_{k=0}^N N_k V_k = \sum_{k=0}^N \pi r_k^2 l_k n^k \quad \text{eqn 4}$$

where V_k represents the amount of fluid in one vessel of level k . If scaling factors β , γ , n are constant for all levels, the terms in equation 4 represent $N + 1$ consecutive terms in a geometric progression with the first term $\pi r_0^2 l_0 = V_0$ and the quotient $n\gamma\beta^2$, or alternatively the first term $\pi r_0^2 l_0 n^N$ and the quotient $1/n\gamma\beta^2$ (WBE apply calculations in both directions later on). Using the expression for the sum in the geometric progression we get

$$V_b = \frac{(n\gamma\beta^2)^{-(N+1)} - 1}{(n\gamma\beta^2)^{-1} - 1} n^N V_0. \quad \text{eqn 5}$$

WBE combine equations 5 and 4 into one expression (their eqn 4), ignoring the fact that equation 4 holds true for all networks, and equation 5 only for non-pulsatile networks (with β constant for all levels k). Thus the considerations following equation 5 cannot be applied to a cardiovascular system. Now WBE write: 'As shown below, one can also prove from the energy minimization principle that $V_b \propto M$.' They intensively exploit this relation to derive the exponent $3/4$ for a rigid-pipe model with area-preserving branching (with the sum of the cross-area of all vessels equal at all levels k) without proving it. They approximate equation 5 with

$$V_b = \frac{V_0}{1 - n\gamma\beta^2} = \frac{V_0(\gamma\beta^2)^{-N}}{1 - n\gamma\beta^2}, \quad \text{eqn 6}$$

taking into account that $n\gamma\beta^2 < 1$ (which is true for realistic parameters) and $N \gg 1$. It may be checked with numerical calculations that this approximation really is good for a broad range of parameters, especially in area-preserving branching. Because V_0 is size-invariant, and also $V_b \propto M$ is assumed, $(\gamma\beta^2)^{-N} \propto M$ according to WBE. Here the error in reasoning occurs. Recall that total body volume is the sum of 'service areas' containing one capillary each. Because of capillary size-invariance, fluid volume in capillaries ($V_c N_c$) must be proportional to body mass M , not total fluid volume

(V_b). V_b must increase with body mass faster than linearly, because more and more blood is contained in vessels not directly supplying tissues. In fact this is not obviously true for real organisms, but only for WBE's organisms with size-invariant capillaries.

The erroneous assumption that $(\gamma\beta^2)^{-N} \propto M$ allowed WBE to replace M/M_0 in equation 3 with $(\gamma\beta^2)^{-N}$, which results in WBE's equation 5:

$$a = -\frac{\ln n}{\ln(\gamma\beta^2)} \quad \text{eqn 7}$$

WBE write: 'To make further progress requires knowledge of γ and β . We shall show how the former follows from the space-filling fractal requirement, and the latter, from the energy minimization principle.' In fact the latter is simply derived geometrically from the assumption that $\beta = r_{k+1}/r_k$ for all k and from the cross-section area-preserving property $\pi r_k^2 = n\pi r_{k+1}^2$. Under these assumptions $\beta = n^{-1/2}$. Total body volume W not only can be expressed as the sum of service volumes of capillaries $W = 4/3\pi(l_c/2)^3 N_c$, but also can be approximated by the sum of service volumes at each vessel level $W = 4/3\pi(l_k/2)^3 N_k$. Thus $4/3\pi(l_{k+1}/2)^3 N_{k+1} \approx 4/3\pi(l_k/2)^3 N_k$, which means that $\gamma_k^3 \equiv (l_{k+1}/l_k)^3 \approx N_k/N_{k+1} = 1/n$. From this it follows that $\gamma = \gamma_k \approx n^{-1/3}$ for all vessel levels. Substituting $\beta = n^{-1/2}$ and $\gamma = n^{-1/3}$ into equation 7 gives $a = 3/4$. Recall that this derivation is unjustified because equation 7 is built on the false premise that $(\gamma\beta^2)^{-N} \propto M$.

About the relationship $\gamma = \gamma_k \approx n^{-1/3}$ WBE write: 'This result for γ_k is a general property of all space-filling fractal systems that we consider.' As shown earlier, this result contradicts the assumption of capillary size-invariance. Thus, complex considerations of pulsatile flows, leading according to WBE to the exponent a for metabolic rate equal to $3/4$, must also be rejected as unjustified. Interestingly, Dodds, Rothman & Weitz (2001), reanalysing WBE's model, found flaws in this part and concluded in Appendix B: 'So, in the case of Poiseuille and pulsatile flow a fractal network is not necessary for energy dissipation to be minimized. Additionally, in the case of a pulsatile flow network, $\alpha = 3/4$ cannot be derived from the optimization problem as stated.' The exponent $3/4$ is proved neither for a rigid-pipe model with area-preserving branching nor for a pulsatile cardiovascular system.

Table 1 gives a numerical example built on WBE's assumptions and showing that their model cannot work. Model 'organisms' have supply systems branching at each node to $n = 5$ small vessels. Capillary length is size-invariant, as assumed by WBE, with length $l_c = 0.03$ mm and radius $r_c = 0.0003$. According to WBE, the radius ratio for consecutive levels $\beta = n^{-1/2}$ equals 0.04472, and length ratio for consecutive levels $\gamma = n^{-1/3}$ equals 0.5848. Three sizes of 'organisms' are represented in the table, equivalent to the number of generations of branches 7, 9 and 11. These numbers of generations are equivalent to body volumes M_v 1.10, 27.61 and 690.29 mm³, and assuming specific mass 1 g cm⁻³, to body masses 1.10,

27.61 and 690.29 mg. Body volume was calculated as the sum of spheres representing the service volumes of all capillaries. Columns M_v of Table 1 (body volume, proportional to body mass) show that WBE correctly assumed that body volume can be well approximated by the sum of spheres at all levels.

It is clear from Table 1 that the amount of blood in capillaries $N_c V_c$ is proportional to body mass M , and total blood volume V_b increases with body mass (body volume) much faster. Using five body sizes (five numbers of vessel generations) we found that $V_b = 0.059M^{4/3}$ and the dependence is strictly linear on a log-log scale. This is because with the size increase more and more blood is contained in large vessels not directly supplying tissues. Interestingly, blood volume per body volume increases very rapidly with body size, from 6.1% at the size of 1.10 mg to 52.7% at the size of 690.29 mg Banavar *et al.* (2002) call attention to the fact that for all supplying systems with size-invariant demand of the final unit, blood volume increases so rapidly that it exceeds total volume for large organisms. They correctly argue that such systems cannot exist for broad size ranges, but can occur for narrow ranges. Thus we should expect specific metabolic rate to decrease with body mass, accompanied by a simultaneous increase of capillary length with body mass, but only for broad size ranges. By no means can this correct relation be deduced from WBE's internally contradictory model. Also $(\gamma\beta^2)^{-N}$, an expression important in WBE's derivation of the $3/4$ exponent, is proportional to $M^{4/3}$ and not to M as they assume.

Biological relevance of the model

WBE claim that the supplying networks they analyse, being space-filling self-similar fractals, are represented in nature by blood vessels of mammals as well as by plant vascular systems. Here we discuss the relevance of WBE's model to particular supply systems.

PLANT VASCULAR SYSTEM

Plant vascular systems consist of vessel bundles. A single vessel goes from the base of the stem (in fact from a root tip) to the leaves. Each vessel is independent of each other, because in WBE's idealized model it does not communicate with other vessels. In real plants there is some communication possible through lateral pits, which serve to equalize water pressure, especially when one of the vessels is blocked by an air bubble. If we accept WBE's neglect of these weak connections, their sentence 'These network systems must therefore be treated as a complete integrated unit; they cannot realistically be modelled by a single or a few representative vessels' becomes unjustified for plants. Moreover, can the vessel system of plants represent a space-filling self-similar fractal? First, vessels do not really branch; they diverge at the 'branching' points. In the model, a vessel of the higher order splits to n vessels of the lower order and disappears. In real plants such a mode of

Table 1. Numerical examples of WBE’s model. Three parts of the table represent organisms having the number of generations of branches N equal to 7, 9 and 11. Each vessel branches into $n = 5$ small vessels. Capillary length is size-invariant with length $l_c = 0.03$ mm and radius $r_c = 0.0003$. Radius ratio for consecutive levels $\beta = n^{-1/2}$ equals 0.04472, length ratio for consecutive levels $\gamma = n^{-1/3}$ equals 0.5848, k represents consecutive levels and c capillary level, l_k and r_k the length and the radius of the vessel at level k , N_k the number of vessels of level k , $N_k V_k$ the amount of blood in all vessels of level k , V_b total blood volume, and M_v body volume (proportional to body mass M) estimated at each level as the sum of N_k spheres with diameter l_k

k	$N = 7$					$N = 9$					$N = 11$				
	r_k	l_k	N_k	$N_k V_k$	M_v	r_k	l_k	N_k	$N_k V_k$	M_v	r_k	l_k	N_k	$N_k V_k$	M_v
0	0.0839	1.28	1	0.0283	1.1045	0.4193	3.75	1	2.0709	27.612	2.0963	10.97	1	151.38	690.29
1	0.0375	0.75	5	0.0166	1.1045	0.1875	2.19	5	1.2111	27.612	0.9375	6.41	5	88.53	690.29
2	0.0168	0.44	25	0.0097	1.1045	0.0839	1.28	25	0.7082	27.612	0.4193	3.75	25	51.77	690.29
3	0.0075	0.26	125	0.0057	1.1045	0.0375	0.75	125	0.4142	27.612	0.1875	2.19	125	30.28	690.29
4	0.0034	0.15	625	0.0033	1.1045	0.0168	0.44	625	0.2422	27.612	0.0839	1.28	625	17.71	690.29
5	0.0015	0.09	3 125	0.0019	1.1045	0.0075	0.26	3 125	0.1416	27.612	0.0375	0.75	3 125	10.35	690.29
6	0.0007	0.05	15 625	0.0011	1.1045	0.0034	0.15	15 625	0.0828	27.612	0.0168	0.44	15 625	6.06	690.29
7	0.0003	0.03	78 125	0.0007	1.1045	0.0015	0.09	78 125	0.0484	27.612	0.0075	0.26	78 125	3.54	690.29
						0.0007	0.05	390 625	0.0283	27.612	0.0034	0.15	390 625	2.07	690.29
						0.0003	0.03	1 953 125	0.0166	27.612	0.0015	0.09	1 953 125	1.21	690.29
											0.0007	0.05	9 765 625	0.71	690.29
											0.0003	0.03	4.9E + 07	0.41	690.29
V_b	0.0673					4.9644					364.0				
V_b/M	0.0609					0.1798					0.527				
$N_c V_c/M$	0.0006					0.0006					6E-04				

branching is never or almost never represented, and instead there appear either dichotomous branching with n always equal to 2, or more commonly herringbone branching with the vessel bundle of the higher order not disappearing at the branching but becoming thinner. Branching itself is completely dependent on plant geometry, and xylem containing vessel bundles also plays an extremely important mechanical role. Vessels are placed parallel (without interconnections) along the leaves (monocotyledons), or else they form a more or less dense mesh of interconnected vessels (dicotyledons). Thus neither arrangement resembles a fractal form with constant length of final vessels. In fact WBE state (page 124) that their basic eqn 2 does not hold for leaves, citing Canney (1993). What is the final unit and service volume in such a case?

INSECT TRACHEAL SYSTEM

According to WBE, insect tracheae are another representation of the fractal network. There are, however, some substantial deviations from the assumed pattern. The space-filling fractal arrangement of tracheae is repeated in each separate body segment in larval forms, and also in imago, especially in the abdomen (Chapman 1998). Thus, an increase of body size is partly realized through multiplication of similar-sized, separate fractal networks, rather than building up subsequent levels of a single network, as assumed by WBE. This clearly should affect the allometry of metabolic rate in their model.

Area-preservation of cross-sections is considered to be a rule, but with numerous exemptions, especially at the tracheole level (Buck 1962; Chapman 1998; Locke 2001). Furthermore, oxygen diffuses not only from terminal tracheoles, but also from tracheae (Buck 1962), which contradicts WBE's definition of a 'service volume'. In a small insect there is no mass flow of gas or fluid in such a system, but diffusion of particles according to partial pressure gradients. In larger and highly active insects there is active ventilation (compressible air sacs, spiral folding of the wall permitting accordion-like movements; Schmidt-Nielsen 1997; Chapman 1998). Can the same equations and the same argument be used for mass flow and diffusion? What represents the resistance of the network in a diffusion system? Most importantly, a tracheal system adjusts very well to oxygen demands. Tracheoles are most abundant in metabolically active tissues, as in flight muscles, or tissues with chemical synthetic activity, as in pheromone glands (Chapman 1998). Moreover, it is possible to change the density of the tracheal system in a growing insect by manipulating partial pressure of oxygen in air (Locke 2001), and a tracheal system grows easily into implanted tissues with demand for oxygen (Buck 1962). Chapman (1998) describes contractile cytoplasmic threads that can drag a tracheole to the oxygen-deficit region, even 150 μm apart. Spiracles are not open all the time, but open and close in a highly regulated way, according to oxygen demand or the need to remove carbon dioxide

(Chapman 1998). To restrict access of oxygen to tissues and thus to limit oxidative stress, tracheoles are partly filled with a liquid under low oxygen demand (Kestler 1985). All these facts indicate that the supply system does not limit metabolic rate, as WBE assume, but adjusts to metabolic needs.

LUNGS

The lungs of mammals probably most resemble a self-similar space-filling fractal. Reptiles and amphibians, however, have spongy lungs: there is no single way for air to come to a given blood capillary. Although a sponge can represent a fractal (Imre 1999), such a structure is completely different from a branching network, and cannot be described with the same equations.

VERTEBRATE VASCULAR SYSTEM

The geometry of the vertebrate vascular system depends partly on body shape, and herringbone splitting is often present. Furthermore, capillaries are organized in a mesh connecting arterioles with veins, with blood flow through capillaries strictly regulated according to tissue needs. Such anatomy does not match WBE's hierarchical branching system. There is not enough blood in the organism to fill all the capillaries entirely at the same time (Schmidt-Nielsen 1997). Such circulatory system construction means that it is more likely that the supplying system adjusts to cover the demands of tissues, and not that the supplying system limits the basal metabolic rate, as assumed by WBE. Nor are capillary radius and capillary length size-invariant, as WBE assume (Dawson 2001). Exercise training elicits a considerable, 20%, increase in capillary density both in humans (e.g. Geiser *et al.* 2001) and animals (e.g. White *et al.* 1998). A sizable, 40%, increase in capillary density can also result from acclimation to cold (Wickler 1981).

RESTING VS ACTIVE METABOLIC RATE

From the functional point of view, the basic but hidden assumption of WBE's model is that fractal-like supplying systems constitute a rate-limiting step in the O_2 delivery cascade, and thus impose the $3/4$ scaling of the whole-body basal metabolic rate. However, endothermic animals usually operate at metabolic rates four or five times higher than BMR (e.g. Speakman 2000). Flying insects increase their metabolic rate 15–100-fold in relation to the resting metabolic rate (Chapman 1998), and even at walking the metabolic rate can increase roughly 10-fold (Rogowitz & Chappell 2000). Obviously this would be impossible with an O_2 delivery rate limited already at the resting level. As Darveau *et al.* (2002) rightly pointed out, a several-fold difference between resting and active metabolism implies that at the resting level considered in WBE's model the supplying systems must operate well below their maximum capacity. This clearly contradicts the logic of the model.

To summarize, all or almost all real supplying systems and their functions are far removed from the idealized cases considered by WBE. The differences are not in the details but in the basic structure. These differences are unlikely to be quantitatively neutral to the scaling exponents under consideration.

Does $3/4$ scaling really exist?

The ubiquity of $3/4$ scaling claimed by WBE has been re-examined by many authors recently and has repeatedly failed close scrutiny. Re-examination of the allometries from earlier classic studies has led Dodds *et al.* (2001) to reject the null hypotheses of the $3/4$ power scaling for data on mammalian and avian metabolic rates compiled by Brody (1945), Hemmingsen (1960), Kleiber (1961), Bartels 1982), Bennett & Harvey (1987) and Heusner (1987). In their recent analysis of data featuring 619 species and encompassing five orders of magnitude of body mass variation, White & Seymour (2003) found that the BMR of mammals scales to the $2/3$ power. Lovegrove (2000) analysed data on the BMR of 487 species of mammals and found significantly different slopes for large and small mammals. Our analysis of his data set revealed that in three out of six mammalian orders represented by at least 20 species the slopes significantly differ from $3/4$ (Kozłowski, Konarzewski & Gawelczyk 2003). Our analysis of the distribution of allometric slopes reported by Peters (1983), and cited by WBE in support of their model, revealed systematic deviations from $3/4$ scaling in various taxa, which cannot be explained by measurement errors. Thus, the ubiquity of $3/4$ scaling claimed by WBE must be rejected.

Conclusions

WBE's model is mathematically incorrect. The authors did not discover their error apparently because they did not play with numerical examples. Therefore, $3/4$ power scaling for metabolic rate cannot be accepted as derived in their original paper. Most importantly, their model is based on assumptions that are biologically unjustified. It was built to explain a non-existent pattern, that is, universal $3/4$ scaling of the metabolic rate. The positive influence of their work lies in reviving interest in metabolic rate scaling. Future models must incorporate more biological realism. In particular, they should take into account different mechanisms inherent in the scaling of the supply and demand systems underlying maximum and basal metabolic rates. They should also explain why the scaling exponents of metabolic rate systematically differ between taxonomic groups while their values fall between $2/3$ and $3/4$ for broad taxa.

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