EDITOR'S CHOICE

How did A V Hill understand the VO_{2max} and the "plateau phenomenon"? Still no clarity?

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ABSTRACT

Objectives: A recent editorial in *Medicine and Science in Sports and Exercise* concludes that the study of Hawkins and colleagues confirms "beyond any doubt the proposition of Hill and Lupton". It is not clear which of Hill and Lupton's propositions have been proved "beyond any doubt".

Methods: A review of all the relevant publications of A V Hill and his colleagues.

Results: Hill and Lupton believed (i) that myocardial ischaemia limits maximal exercise performance by inducing circulatory failure; (ii) that a "governor" protects the ischaemic heart by causing a "slowing of the circulation" during maximal exercise; (iii) that the oxygen cost of running increases exponentially at speeds above 16 km per hour; and (iv) that humans reach their highest measurable oxygen consumption of about 4 litres per minute at that running speed. Hill and Lupton neither invented the concept of the "plateau phenomenon" nor utilised this concept to establish that a "true" maximum oxygen consumption (VO_{2max}) had been achieved. Nor did they measure cardiac output during exercise.

Conclusion: Accepting uncritically this modern interpretation of Hill and Lupton's theory that the cardiac output limits maximal exercise performance whether or not the plateau phenomenon is present fails to answer the question first posed more than 20 years ago: What causes the termination of exercise when the "true" VO_{2max} is achieved without the "plateau phenomenon"? According to the Hill and Lupton model, this cannot be because a limiting cardiac output has been reached. Since a majority of maximal exercise tests terminate in the absence of the "plateau phenomenon", this is seemingly an important question.

A recent editorial in *Medicine and Science in Sports and Exercise*[1] poses the question: Is it necessary to establish a "plateau phenomenon" in oxygen consumption (VO_2) in order to prove that the highest possible maximum value for VO_2 (a true VO_{2max}) has been measured in any particular test? The author concludes that the paper of Hawkins *et al*[2] proves that the VO_{2max} measured during incremental exercise testing will always produce the highest VO_{2max} value regardless of whether or not a "plateau phenomenon" has been identified. He includes information from two other studies[3 4] which apparently support this conclusion.

Hawkins *et al*[2] analysed group data to draw conclusions about individual responses, a risky approach if not all individuals respond identically. Indeed, some subjects in that study developed lower VO_{2peak} values during "supramaximal" than during incremental exercise testing. Since there is no plausible biological explanation for this finding, the physiological relevance of those specific results must be questioned. Exclusion of those data would leave some subjects whose VO_{2max} values were higher during supramaximal than during incremental exercise testing, disproving the conclusion of Dr Howley and Dr Hawkins and his colleagues.

For if even one subject achieved a higher VO_{2max} value during supramaximal than during incremental exercise, then the conclusion cannot be drawn that incremental exercise testing will *always* produce the highest VO_{2max} value even when the "plateau phenomenon" is not present. Unfortunately absolutist interpretations can be disproved by a single contrary finding unless there is an alternative explanation for that finding.

The study of Hawkins *et al*[2] supports two clear conclusions. First, since some subjects reach higher VO_{2max} values during "supramaximal" than during incremental exercise testing, it cannot be assumed that incremental exercise testing will always produce the highest VO_{2max} value in any *individual*. Second, the mean VO_{2max} value measured with either incremental or supramaximal exercise in a group of subjects will not likely be different, as also shown by others.[3–5]

This indicates that each individual does indeed have a ceiling VO_{2max} value that can be determined in a number of ways, not just by conventional incremental exercise testing, regardless of whether or not there is a "plateau phenomenon". This point no longer requires debate. However, this finding does not really advance our understanding of the nature of the factors "limiting" maximal exercise performance since it is unable to differentiate between the predictions of two currently popular models.[2 3 5–15]

In this context, the key statement by Dr Howley is that the findings of Hawkins *et al* "confirm beyond any doubt the proposition of Hill and Lupton". But since Dr Howley does not state exactly what in his mind Hill and Lupton proposed, his editorial leaves unanswered some important questions. For example, did Hill and Lupton ever conceive the word "plateau phenomenon" in their writings? Or did Hill and Lupton ever establish that the "plateau phenomenon" actually developed in their own experiments? Or did Hill and Lupton invent the concept of the "plateau phenomenon" to prove that they had measured a "true" VO_{2max}? Or did Hill and Lupton utilise this concept as the biological foundation for their (cardiovascular/anaerobic/catastrophic or ischaemic heart) model of factors limiting performance during maximum exercise[6] with which, as Dr Howley writes,

they developed the "concept of the VO_2 max and its dependence on cardiac output"?

Did Hill and Lupton ever conceive the term "plateau phenomenon" in their writings?

Perhaps the key statement of Hill and Lupton is: "In running the oxygen requirement increases continuously as the speed increases attaining enormous values at the highest speeds: the actual oxygen intake, however, reaches a maximum beyond which no effort can drive it …. The oxygen intake may attain its maximum and remain constant merely because it cannot go any higher owing to the limitations of the circulatory and respiratory system".[16] Whilst this description includes features of the "plateau phenomenon", neither here, nor anywhere else in their writings, could I find those exact words. My conclusion, therefore, is that Hill and Lupton did not ever conceive the term "plateau phenomenon" in their "proposition". Rather it appears that Taylor *et al*[17] may have been the first to create this concept when they wrote that: "Each day the speed was increased until the oxygen intake during the standard collection time had reached a plateau" (p.74).

Interestingly Taylor *et al*[17] did not in fact describe a "plateau", which is defined in the Oxford English Dictionary as "(to) reach a level or stable state after an increase" and is derived from the French word *platel*, which is "a small *flat* surface". Rather these authors defined their "plateau" as an *increase* in VO_2 of less than 150 ml/min between two consecutive workloads. It would appear that Taylor and his colleagues bent the meaning of the English language to describe what they believed *should* happen according to the Hill model (i.e., an abrupt levelling off in VO_2 at the onset of myocardial ischaemia and the attainment of a truly maximal cardiac output) but which they were unable to show.

Did Hill and Lupton establish the presence of the "plateau phenomenon" in their own experiments?

If Hill and Lupton did not conceive the term, did they ever look for the presence of what Taylor *et al*[17] would recognise as a "plateau" in their own studies? I have repeatedly presented evidence in three different articles published in *Medicine and Science in Sports and Exercise*[18–20] as well as in this journal and elsewhere[6 21] showing that Hill and Lupton did not look for the presence of the "plateau" in any of the *individual* data they reported. The key evidence can be found in figs 2 and 3 in reference [18], fig 3 in reference [19] and fig 1 in reference [20] and in the accompanying text. Rather the authors persistently expressed the view, described later, that all humans had a maximum VO_2 of ~4 l/min. This suggests that they failed to recognise that individuals could have different VO_{2max} values higher than 4 l/min. The errors that led them to this incorrect conclusion have been presented in detail previously (fig 2 in reference [20] and accompanying text) and do not need to be repeated here since the evidence is clear.

Did Hill and Lupton invent the concept of the "plateau phenomenon" to prove that they had measured a "true" VO_{2max} in different individuals?

This is the thrust of Dr Howley's editorial.

I argue that on this point the evidence is clear.[20] Whereas Hill and Lupton did recognise the presence in the literature of different individual VO_{2max} values ranging from 2.1 to 3.8 l/min (table IV in reference [22]), the maximum values they measured were generally higher (3.5–4.2 l/min)[22] (p.154), leading them to conclude that there was an absolute VO_{2max} value in *all* humans of ~4.0 l/min. Thus Hill and Lupton wrote: "The oxygen intake attains its maximum value, which is strikingly constant (in the case of running) at about 4 L per minute"[23] (p.157); "It is obvious, therefore, that up to about 4,175 cc of oxygen per min. can be taken in during running by a man of 73-kilo body-weight"[22] (p.154); "The amount of work which the heart has to do is enormous, and it seems to reach its limit, in the case of athletic people, when about 4 L of oxygen are taken in per minute"[24] (pp. 230–231) and "The form, however, of the oxygen intake curve of Figure 1 approaching a constant level of 4 litres per minute, makes it obvious that no useful purpose would be served by investigating higher speeds in this way"[23] (p.157).

On the basis of that interpretation, Hill and Lupton produced a figure (fig 3 in reference [22]) reproduced here (fig 1) which shows that, according to Hill and Lupton, the VO_2 approaches a maximum of 4 l/min at a running speed of 260 m/min (15.6 km/h) (lower line in fig 1) but that this VO_2 was much less than the oxygen requirement which they incorrectly believed began to rise exponentially at running speeds greater than 200 m/sec (12 km/h) (upper line in fig 1). Thus they would have seen no reason systematically to test athletes at progressively faster running speeds beyond those that produced a VO_2 of 4 l/min since such exercise would merely have produced progressively higher "oxygen debts" which did not contribute to the VO_{2max} measurement.

In an earlier attempt to "prove" that Hill and his colleagues actually measured the "plateau" phenomenon in individual subjects and did not rely solely on the logic of their fig 1 to show that testing subjects at high running speeds could never produce VO_2max values higher than ~4.0 l/min, Bassett and Howley[8] have written that: "After 2.5 min of running at 282 $m.min^{-1}$, his VO_2 reached a value of 4.080 $L.min^{-1}$ (or 3.730 $L.min^{-1}$ above that measured at standing rest). Since the VO_2 at speeds of 259, 267, 271 and 282 $m.min^{-1}$ did not increase beyond that measured at 243 $m.min^{-1}$, this confirmed that at high speeds his

Table 1 Oxygen consumption (VO_2) of AV Hill when running at different speeds in two different experiments

Running speed		
(m/min)	(km/h)	Oxygen consumption (l/min)
172*	10.3	3.08
181*	10.9	2.66
181†	10.9	2.66
197*	11.8	3.49
200*	12.0	3.30
202*	12.1	3.01
202*	12.1	3.32
203†	12.2	3.21
203†	12.2	3.01
205*	12.3	3.21
205*	12.3	3.14
235*	14.1	3.49
243*	14.6	4.18
259*	15.5	3.87
267*	16.0	3.95
267†	16.0	4.06
271*	16.3	4.06
282*	16.9	4.08

*Refers to data (table 1; p156)[23]. †refers to data (table III; p150)[22].

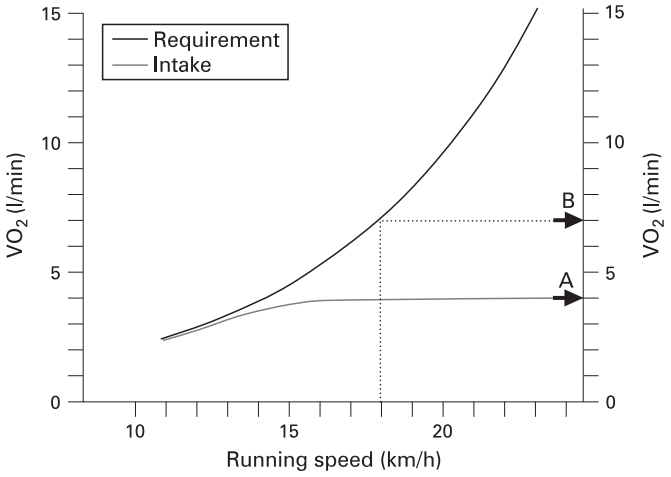

Figure 1 Hill and colleagues believed that the cost of running increased exponentially especially at running speeds above about 14 km/h (upper line). This was because they overestimated the "anaerobic" contribution to the energy cost of running. Since they also believed that the highest VO_{2max} value of which humans are capable is approximately 4 l/min (Arrow A), they did not develop nor did they ever describe a method to establish that an individual had reached a VO_{2max} value, for example by showing a "plateau phenomenon". Arrow B shows that Hill and his colleagues predicted that the oxygen consumption was approximately 7l/min in subjects running at 18 km/h or nearly twice the VO_2 that they considered to be the maximal of which humans were capable (Arrow A). It was for this reason that Hill and his colleagues did not believe it necessary to test athletes at running speeds faster than about 17 km/h (table 1) since, according to their understanding, such speeds would produce only increasing levels of "anaerobic" metabolism (based on fig 3 in reference [22], p.157).

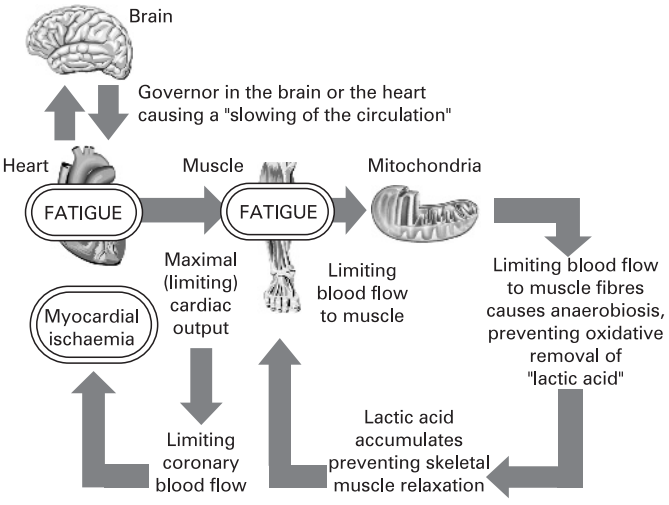

Figure 2 Hill and his colleagues described a model of human exercise physiology in which the onset of ischaemia of the heart (at running speeds in excess of about 16 km/h) caused an abrupt reduction in the blood flow to muscle leading to skeletal muscle anaerobiosis and a failure of the oxidative removal of lactate produced in skeletal muscle. Since Hill believed that the anaerobic production of lactic acid was necessary to initiate skeletal muscle contraction,[44] any failure of lactic acid removal would, according to their explanation, have caused skeletal muscle rigor to develop. But, more importantly, Hill and Lupton appreciated that myocardial ischaemia would lead to heart damage if unregulated. Thus they proposed the presence of a "governor", the function of which was to reduce myocardial contractile function as soon as myocardial ischaemia developed. A more effective model would be one in which the brain regulates the demands for skeletal muscle blood flow by regulating central (brain) motor output and hence the number of motor units that are active in the exercising limbs at any time. This model has been termed the Central Governor Model out of respect for this highly original idea of Hill and his colleagues.

VO_2 reached a maximum beyond which no effort can drive it"[8] (p.71). But these data can be interpreted differently.

Table 1 lists all the values for oxygen consumption measured on Hill in the two studies[22 23] quoted by Bassett and Howley. They clearly establish that Hill's VO_2 increased progressively with increasing running speed but that the value at 243 m/min appears spuriously high since an increase in running speed of 8 m/sec (from 235 to 243 m/min) produced an 0.69 l/min increase in VO_2. In contrast a 20 m/sec increase in running speed from 205 to 235 m/min produced a substantially lower increase in VO_2 of 0.35 l/min. Furthermore, if the value of 4.18 l/min measured at 243 m/sec were indeed a true value then, according to the "plateau phenomenon", all subsequent values at running speeds of 259, 267, 271 and 282 m/min should also have been 4.18 l/min, which they were not (table 1).

In addition, if Hill had really believed that he reached his VO_{2max} of ~4.0 l/min at a running speed of 243 m/min (14.6 km/h), then he would have drawn fig 1 accordingly. But he did not. Rather he drew the graph to show that the VO_{2max} of 4 l/min had not been reached even at a running speed of 300 m/min (18 km/h) (fig 1) although the "levelling off" of the VO_2 clearly occurred at 260 m/min (15.5 km/h). Thus there is good evidence to believe that the value of 4.18 l/min for the running speed of 243 m/min was not interpreted by Hill as evidence that he personally had reached his "plateau" VO_{2max} value at a speed below 282–300 m/sec (table 1).

But, more to the point, Bassett and Howley are guilty of retrospectively looking for something in these data for which the primary authors did not themselves search. As argued in detail previously[19] Hill used a circular logic ((fig 2) in reference [19]) to conclude that he had personally reached his maximum oxygen consumption. Nowhere do Hill, Long and Lupton ever use the argument now advanced 77 years later by Bassett and Howley[8] to prove that they had definitely measured an absolute VO_{2max} in an individual subject, in this case A V Hill himself. The issue is not whether the "plateau phenomenon" exists; it is whether or not Hill and his colleagues described the phenomenon as we currently understand it. The answer is that they did not.

Rather, since Hill and Lupton believed that no human could have a VO_{2max} greater than ~4 l/min, they could have seen no need to use a "plateau phenomenon" to differentiate between different individual VO_{2max} values. Indeed, because they greatly overestimated the "anaerobic" component of running at low speeds (fig 1), they could only conclude that running at even modest speeds (18 km/h; 300 m/min) required almost twice the universal human VO_{2max} of 4 l/min (arrow A in fig 1). Thus they could not justify studying athletes at running speeds greater than 17–18 km/h, the highest speed at which their subjects ever ran (fig 1 in reference [20]) (table 1).

Therefore I conclude that, since Hill and Lupton did not either look for or report a single instance of the "plateau phenomenon" in any individual test, they could not ever have invented the concept of the "plateau phenomenon" as proof that a "true VO_{2max}" had been measured in their tested subjects.

Did Hill and Lupton utilise this concept of the "plateau phenomenon" as the biological foundation for their (ischaemic heart) model of factors limiting performance during maximum exercise?

As now argued for the fourth time, I contend that Hill and Lupton utilised a concept that they themselves failed to prove, namely that "however much the speed be increased beyond this limit, no further increase in oxygen intake can occur"[22] (p.156) as proof that "the heart, lungs, circulation and the diffusion of oxygen to the active muscle-fibres have attained their maximum activity … the requirement of the body for oxygen … cannot be satisfied"[22] (p.156) so that "the oxygen intake fails to exceed this value (of about 4 litres per minute), not because more oxygen is not required, but because the limiting capacity of the circulatory-respiratory system has been attained"[23] (p.157).

For surely the point is that if Hill and Lupton did not attempt to analyse their data in a way that could prove the interpretation that, in retrospect, others[2] now wish to apply, then their interpretation of the biological basis for a phenomenon they did not identify cannot be accepted as uncritically as Dr Howley again[1] wishes we must. For surely, if we are to believe that the VO_{2max} is indeed dependent on the cardiac output, then evidence other than the "proof" provided by Hill and Lupton must be presented.

Hill and Lupton did not ever measure the cardiac output in any of their experiments

For not only did Hill and Lupton not ever search for a "true VO_{2max}" or a "plateau phenomenon" in any of their subjects, but also, much more importantly, they did not ever measure the cardiac output. Surely to prove the "dependency" of the VO_{2max} on the cardiac output requires that the cardiac output, and not some unproven surrogate such as the "plateau phenomenon", must be measured under appropriate experimental conditions? Perhaps we need to remember the cautionary statement in 1958 of Mitchell and colleagues, whose work on the "plateau phenomenon"[10 14 25] is widely acknowledged, that: "The view that cardiac capacity is the determinant of maximal oxygen intake is surmise, not established fact".[14] Even P O Astrand, another exponent of the A V Hill model,[12] once wrote that: "The working capacity of the heart should determine that of the muscles"[26] (p.118). Yet he also wrote that: "… during maximal running and cycling the heart probably works "submaximally"[26] (p.120). It is difficult to understand how a heart working submaximally can "limit" either the VO_{2max} or the athletic performance.

For if the cardiac output "limits" the VO_{2max} then the cardiac output must always reach a limiting "plateau" value coincident with the development of the "plateau phenomenon" in VO_2. But as reviewed in detail elsewhere[6] and as subsequently debated in full,[27] with two notable exceptions from the same authors,[13 15] five other studies show that the cardiac output increases linearly with increasing work rate up to the VO_{2max} with no evidence for any developing "plateau phenomenon". If anything, cardiac function appears to be enhanced at the work rate that elicits the VO_{2max}, especially in elite athletes, who should be the most likely to develop a "plateau" in cardiac output. Furthermore, coronary blood flow is increased during maximal exercise in hypoxia,[28 29] proving that the heart functions with coronary flow (and hence metabolic) reserve when the VO_{2max} is measured in normoxia. Indeed, the recent study of Brink-Elfegoun *et al*[9] confirms that the heart works submaximally at VO_{2max}, although the authors have interpreted their finding somewhat differently.

The ischaemic heart model of Hill and Lupton

In fact Hill and Lupton used their (unproven in 1923) concept of a limiting maximum oxygen consumption to produce a biological model of the factors limiting the VO_{2max} that is currently so unpalatable that no one other than ourselves[6 20 30] ever wishes to acknowledge its existence. Rather some[2 8 9] have attempted to burden our model with the fatal weaknesses of the original model. For Hill and his colleagues concluded that when "the heart, lungs, circulation and the diffusion of oxygen to the active muscle-fibres" reaches a maximum, ischaemia of the heart must develop and this ischaemia would then explain why a (falling) cardiac output would limit the maximum oxygen consumption: "When the oxygen supply (to the heart) becomes inadequate, it is probable that the heart rapidly begins to diminish its output so avoiding exhaustion".[31] As a result: "It would seem possible that a deciding factor in the capacity of a man for severe prolonged exercise may often be the efficiency of the coronary circulation"[16] and: "A heart, adequate in every other way, might fail to allow its owner to undertake severe continued effort, simply because of the imperfect arrangement of its own blood supply" [23] (p.166). This interpretation is now known to be incorrect; coronary flow reserve is present during maximal exercise in healthy humans.[28 29] Left unanswered by the advocates of the Hill model is: how does the cardiac output limit the VO_{2max} without the heart first becoming ischaemic?

Recently Levine[32] has proposed on the basis of studies in four-legged animals (pigs and dogs) that the pericardium restricts ventricular diastolic filling, thereby setting the upper limit for the cardiac output in humans. But humans exercise in the upright position on two legs and achieve lower end-diastolic volumes in the upright position than when they exercise in the supine position.[33] Thus the end-diastolic volume is not maximal in humans during upright exercise. As a result "pericardial restraint" cannot be the factor "limiting" the maximal cardiac output in humans during maximal exercise.

So the question remains: What "limits" the cardiac output during maximal exercise in humans? And what prevents the development of myocardial ischaemia when the cardiac output reaches this supposedly maximum, limiting value?

Hill, Long and Lupton propose the presence of a "governor" to limit myocardial damage in their ischaemic heart model

As fully argued elsewhere,[20] Hill's solution to this inconvenient problem was to propose the existence of a "governor" to reduce the work of the heart the moment ischaemia developed: "… it would clearly be useless for the heart to make an excessive effort if by so doing it merely produced a far lower degree of saturation of the arterial blood; and we suggest that, in the body (either in the heart muscle itself or in the nervous system), there is some mechanism which causes a slowing of the circulation as soon as a serious degree of unsaturation occurs, and vice versa. This mechanism would tend to act as a 'governor' maintaining a high degree of saturation of the blood" [23] (pp.161–2). Hill, Long and Lupton[23] therefore proposed that "it is very probable indeed, that the heart is able to regulate its output, to some extent, in accordance with the degree of saturation of the arterial blood, either of that which reaches it through the coronary vessels or by some reflex in other organs produced by a deficient oxygen supply" (p.161). Thus the inconveniently complete Hill model is that depicted in fig 2. This model was accepted in the United States by Bock and Dill,[34] who wrote that: "The maximum oxygen consumption of 4 litres per minute, found by Hill and Lupton, can very likely only be reached during running…" (p.68) and that "a temporary lowering of the functional

capacity of the heart, induced by fatigue of its muscular fibres, might gradually bring about during exercise an insufficient blood-supply to the skeletal muscles and the brain. The lassitude and disinclination for exertion, often experienced on the day after a strenuous bout of exercise, has also been ascribed to fatigue of the heart as its primary cause'' (p.229).

Interestingly, those generations of exercise physiologists[10] who have followed Hill and Lupton in England and Dill and Bock in the United States have, for some inexplicable reason, forgotten that these iconic exercise scientists taught that failure of the heart, not of the muscles, was the initiating factor that limits maximum exercise performance (fig 2).

Rather, most modern exercise physiologists have euphemised Hill and Lupton's conclusion, as again does Dr Howley, to state that their findings developed ''the concept of VO_2max and its dependency on cardiac output''. But Hill and Lupton, and Bock and Dill, were absolutely clear. In their model it was the development of myocardial ischaemia, partially regulated by the actions of a governor in the heart or brain or other organ, that limited maximal exercise performance. They did not need a ''plateau phenomenon'' to support their conclusion, merely their twin beliefs that (i) all humans have a ceiling VO_{2max} of 4 l/min (lower line in fig 1) and (ii) that the oxygen cost of running rises, as an exponential function of running speed (upper line in fig 1), reaching very high values at quite modest running speeds. It was also necessary that Hill believed that an oxygen deficiency limits maximal exercise performance (fig 2 in reference [19]).

The subtle danger of Dr Howley's editorial is that it again[8] tries to establish as fact that which Hill and Lupton did not look for and could not therefore have found. But the real issue is that, if we continue to overlook this error, we become increasingly vulnerable to an even greater falsehood, that the factors that Hill and his colleagues believed to limit the VO_{2max}, specifically the development of a limiting cardiac output (leading in their minds to skeletal muscle anaerobiosis and a ''poisonous'' lactic acidosis (fig 2)) must be the same whether or not the ''plateau phenomenon'' is present. This is a jump of (il)logic that cannot be allowed to fester unchallenged.

Low prevalence of the plateau phenomenon during VO_{2max} testing

During the past decade it has become increasingly obvious that the detection of the ''plateau phenomenon'' is the exception rather than the rule during incremental exercise testing.[6 35] Importantly, the evidence supporting this conclusion has often been provided by some of the most ardent protagonists of Hill's model[3 4] and not just its opponents.[6 36] This finding invites the rather obvious question: What causes maximum exercise to terminate in subjects who do not show the ''plateau phenomenon''? This is the critical question first posed in 1988[18] and restated exactly a decade later.[20] Another decade on and the supporters of the Hill model continue to avoid this challenge since their theories can provide no reasonable explanations. For example, the finding that only 12 of 71 subjects tested by Day *et al*[4] showed a ''plateau phenomenon'' does not seem to disturb either Dr Howley or the article's authors since their interest is solely in whether or not these values were ''truly maximal''. They therefore draw great comfort from the finding that subsequent testing at work rates higher than those used to measure the original VO_{2max} does not cause higher mean VO_{2max} values to be achieved.

But the point of logic that continues to be ignored is simply this. If, in whatever guise, the Hill ischaemic heart model is indeed the absolute truth, then the absence of the ''plateau phenomenon'' in a majority of VO_{2max} tests can logically be interpreted in only one way: that factors other than a limiting cardiac output and the development of skeletal muscle anaerobiosis must cause the termination of exercise in the majority of VO_{2max} tests (fig 3 in reference [20]). According to a normal logic as I understand it, there can be no other defendable conclusion. Indeed, this interpretation has been applied by Lucia *et al*,[35] who reported a ''plateau phenomenon'' in only 47% of 38 professional road cyclists. They concluded that: ''In a good number of highly trained humans, the main factor limiting maximal endurance might not necessarily be oxygen-dependent''. We have previously shown an even lower incidence of the ''plateau phenomenon'' in a group of Olympic-class runners.[36]

What causes the termination of exercise in subjects who do not show a ''plateau phenomenon''?

Thus the inconvenient question demanding a logical answer remains unanswered: What causes these athletes to terminate exercise if they show no evidence for a limiting oxygen supply to their heart and muscles according to the traditional Hill model? Perhaps now after 20 years of avoidance it is finally time for the defenders of Hill's ischaemic heart model to engage intellectually with this uncomfortable paradox. For the clear problem is that, if maximal aerobic exercise always terminates before there is a catastrophic biological failure, such as the development of myocardial ischaemia or skeletal muscle rigor as logically predicted by the Hill model[6] (fig 2), then the absence of any such ''catastrophe'' suggests the presence of an anticipatory, complex, regulatory control system.[7 37]

Thus the inevitable intellectual danger of Dr Howley's editorial is that the finding of a singular VO_{2max}, whether or not a ''plateau phenomenon'' is also present, will now be used as definitive proof that Hill's cardiovascular/anaerobic/catastrophic model is correct ''beyond any doubt'' so that Hill's original speculations on the factors limiting maximal exercise performance must remain inviolate. But this intellectual leap, already taken by Hawkins *et al*[2] and Brink-Elfegoun *et al*,[9] is unjustified.

In the first place, there is still no definitive proof that the cardiac output is the primary determinant of the VO_{2max}. In particular, it has not been disproven that the VO_{2max} and the cardiac output are not both codependent on a third factor, specifically the number of motor units (number of muscle fibres; mass of muscle) recruited by the central motor output of the brain (central command) during such exercise.[11] Second, the finding of a singular VO_{2max}, regardless of whether or not there is a ''plateau phenomenon'', is not predicted solely by the cardiovascular/anaerobic/catastrophic model of exercise. Rather it is equally compatible with the predictions of a complex model of regulation such as the Central Governor Model (CGM).[7] For the CGM predicts that exercise always terminates whilst homeostasis is retained, so that any number of biological signals, other than simply the development of skeletal muscle anaerobiosis and ''lactic acidosis'', could cause the termination of maximal exercise at the same or a similar VO_{2max} and before the loss of homeostasis.

For example, the achievement of a peak cardiac output might regulate exercise performance not as a result of a limiting delivery of oxygen to the exercising muscles but as a consequence of the pressure generated in the pulmonary circulation. For it is logical that there must be a peak pulmonary blood flow and hence a capillary pressure at which pulmonary oedema will develop. Since pulmonary oedema is

What is already known on this topic

Work undertaken in the 1920s by Nobel Laureate A V Hill and his colleagues laid the foundation for the concept that skeletal muscles become "anaerobic" during maximal exercise and that the biochemical derangements resulting from such anaerobiosis directly cause the termination of maximal exercise. Subsequent generations of exercise scientists have presumed that the basis for this conclusion was the identification and description by Hill and his colleagues of the "plateau phenomenon" in all their subjects, confirming that maximal exercise is limited exclusively by the achievement of a maximal cardiac output with development of skeletal muscle anaerobiosis. Recently it has been concluded that the finding that all humans reach a singular VO_{2max} value, even in the absence of the "plateau phenomenon" and regardless of the testing protocol, proves that the cardiac output alone determines the VO_{2max} as originally proposed by Hill and colleagues.

What this study adds

This review again shows that Hill and his colleagues (i) neither conceived nor used the term "plateau phenomenon"; (ii) nor did they ever search for the "plateau" in their subjects as do modern exercise physiologists to prove that a "true" VO_2max has been measured; (iii) nor did they ever measure cardiac output during any of their studies. Rather these iconic scientists proposed that exercise is limited by the development of myocardial ischaemia, which causes the cardiac output to fall, inducing skeletal muscle anaerobiosis, "poisonous" lactic acidosis and the termination of exercise. Since they understood that myocardial ischaemia would lead to heart damage, they proposed the existence of a governor in the heart or brain that would reduce myocardial contractility, thereby limiting or preventing damage once myocardial ischaemia develops. It is time that Hill's theories were presented accurately. Certainly they should not be repeatedly advanced as "proof" that the cardiac output limits the VO_2max and determines the maximal exercise performance, as again proposed in a recent editorial in *Medicine and Science and Sports and Exercise* to which this review serves as a rebuttal.

not a usual consequence of maximal exercise even in those with pulmonary hypertension,[38] there must also be a regulatory control, the aim of which is to prevent pulmonary capillary pressure reaching dangerously high levels during exercise. It is now established that arterial-to-venous shunting of pulmonary blood flow occurs during exercise in humans.[39 40] This mechanism would allow a higher pulmonary blood flow (cardiac output) to be achieved (to sustain skeletal, cardiac and respiratory muscle function during maximal exercise) whilst maintaining pulmonary capillary pressure within safe limits. Indeed, this shunting appears to be greatest in those with higher VO_{2max} values[41 42] or higher cardiac outputs,[43] in keeping with this hypothesis.[39]

However, in any individual, exercise would always terminate at the same pulmonary artery pressure and hence the same cardiac output and similar VO_{2max}. Alternatively, the attainment of a limiting rate of heat production could regulate maximum exercise performance according to the anticipatory mechanisms identified during exercise in the heat.[37]

Summary

In summary, the original studies of Hill and his colleagues have encouraged the adoption of a reductionist model of exercise physiology[7] in which a single variable – cardiac output, for example – is considered to limit maximal exercise performance. This model requires that exercise is regulated in a feed-forward manner by the cardiac output and excludes any role for afferent sensory feedback to the brain which alone has the capacity to terminate the exercise before truly "limiting" conditions are reached in either the heart or the skeletal muscles. Yet, with the exception of two studies from the same laboratory,[13 15] there is no other direct evidence proving that the cardiac output "plateaus" during maximal exercise and therefore probably directly determines the VO_{2max} in humans.[27] Since Hill and his colleagues did not ever measure the cardiac output during maximal exercise, their studies cannot be cited as the definitive proof of this relationship as Dr Howley wishes we should. However, it is now established beyond doubt that the "plateau phenomenon" is not a prerequisite for the identification of the "true VO_2max" in a majority of (but not all) subjects.[2–4 9] But since the majority of subjects terminate maximal exercise without developing the "plateau phenomenon",[3 4 6 35 36] we must now conclude that, according to the Hill model, the achievement of a "limiting" cardiac output causing skeletal muscle anaerobiosis cannot be the exclusive reason why all subjects terminate maximal exercise. The predictions of the Hill model (fig 2) allow no other conclusion.

Perhaps now is the time to repeat the three questions continually avoided by Dr Howley and the other supporters[2–4 9 10 32] of the Hill model:

1. How does the Hill model explain similar VO_{2max} values whether or not the "plateau phenomenon" is present?
2. Which biological factors cause the termination of exercise in subjects who do not show a "plateau phenomenon"?
3. Are these factors the same whether or not the "plateau phenomenon" is present?

Hopefully it will not take another 20 years[18] before these fundamental questions are finally addressed by the supporters of A V Hill's model of exercise physiology.

Peer review: fair review articles

The rationale for "peer review: fair review" articles is to ensure that research is not buried simply because it is too challenging and too controversial. There are many examples of papers that were not accepted the first time they were submitted, but were accepted elsewhere and have made a great difference to clinical practice (Khan KM, Stovitz SD, Pluim B, *et al*. Addressing conflicts of interest and clouding of objectivity: *BJSM*'s "Peer review: fair review" section. *Br J Sports Med* 2008;**42**:79). *BJSM* is committed to encouraging debate and providing a "safe place" for ideas that are supported by evidence, but considered "too radical" elsewhere.

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