Are fast-moving elephants really running?

Despite their unseemly bulk, elephants can hit high speeds — but use an unusual style.

It is generally thought that elephants do not run[1–5], but there is confusion about how fast they can move across open terrain and what gait they use at top speed. Here we use video analysis to show that Asian elephants (*Elephas maximus* L.) can move at surprisingly high speeds of up to 6.8 m s^{-1} (25 km h^{-1}) and that, although their gait might seem to be a walk even at this speed, some features of their locomotion conform to definitions of running.

Elephants moving rapidly have been estimated[2–4] to reach speeds of about 4 m s^{-1} (15 km h^{-1}), although anecdotal evidence[1] claims that they can reach 11 m s^{-1} (40 km h^{-1}). To investigate the gait used by elephants at top speeds, we used video analysis to study 42 healthy, active Asian elephants throughout Thailand (for details, see supplementary information). The skin was marked with non-toxic tempera paint dots over the limb-joint centres of the elephants (right fore- and hindlimbs; Fig. 1), estimated by palpation and manipulation of the joints; these dots were used for later video digitizing. Mahouts guided the elephants along a 30-metre course, parallel to the field of view of the video camera (60 Hz). Elephants had at least 10 m to accelerate or decelerate before and after this 30-m course. A total of 188 trials were carried out; trials with sudden accelerations or decelerations in the 10-m videotaped stretch of the course were omitted.

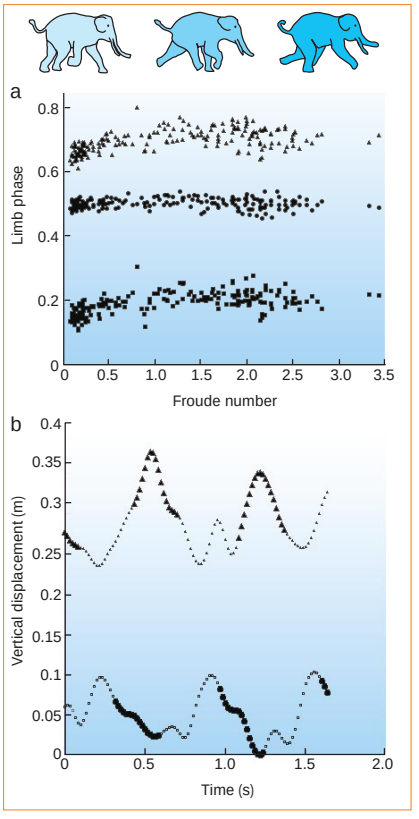

Figure 2 Biomechanics of rapid movement by Asian elephants (*Elephas maximus* L.). **a,** Relative limb phase (fraction of stride time for which each foot comes into contact with the ground after the left hind foot) plotted against dimensionless speed (Froude number). At all speeds, the left hind foot, then the left front (squares), then the right hind (circles), then the right front foot (triangles) contact the ground in the same sequence. Images of elephants moving at 6.6 m s^{-1} (Froude number, 3.1; see movie in supplementary information) illustrate these footfall patterns, which are typical of quadrupedal walking[5]. **b,** Vertical displacements of the hip (circles) and shoulder (triangles) joints plotted against time for one individual moving at 6.8 m s^{-1} (Froude number, 2.8). Large symbols indicate stance phase; small symbols indicate swing phase. This downward hip movement was typical of all fast-moving elephants; many showed a downward-then-upward pattern during stance. Roughly 2.5 strides are shown, beginning just before and ending just after the 10-m section of the observation course.

Our digitization of the hip-joint markers (using Peak Motus, Peak Performance, Colorado) measured the average velocity along the central 10 m of the track. We used the length of the thigh segment (between the centres of hip- and knee-joint markers) to scale the digitized video coordinates to real dimensions. Photocell timers at either end of the track gave an average velocity across the entire 30 m, which was used as a preliminary gauge of which elephants were the fastest, as well as for comparison with the 10-m velocity to monitor speed changes. Speeds over the 10-m and 30-m courses were generally similar, indicating that the elephants did not suddenly speed up or slow down.

Of the elephants, 32 reached top speeds of over 4.0 m s^{-1}, 20 exceeded 5.0 m s^{-1}, and three attained speeds greater than 6.0 m s^{-1}. The fastest gait used by elephants has been variously described as a walk, amble, trot, pace, rack or a running walk[1–5], but — given that these speeds are relatively fast — how well does this gait of the fastest elephants fit the definitions of running?

Several kinematic factors distinguish quadrupedal walking from running. First, trotting and galloping are running gaits with footfall patterns that are distinct from walking[5]. Second, an aerial phase (a period during which no foot touches the ground) often marks the transition from walking to running[5]. Third, a run has been defined as any gait with a duty factor (the fraction of a complete sequence of footfalls for which a given foot is in contact with the ground) of less than 0.50 (ref. 5). Our elephants maintained the same walking footfall pattern (Fig. 2a) and always kept at least one foot in contact with the ground, although they used duty factors as low as 0.37.

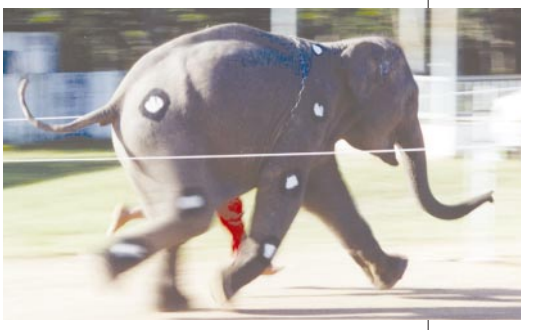

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Figure 1 An Asian elephant marked with dots for gait analysis.

Walking and running can also be distinguished by the forces involved. The Froude number (Fr) is a dimensionless speed[6] calculated as velocity^2/(acceleration of gravity × hip height). At speeds beyond Fr 1.0, the force needed to keep the body mass on a circular arc during stance exceeds the force of gravity. Theoretically, this requires a walking animal to leave the ground and run. Most animals usually switch from a walk to a run at Fr ≈ 0.50, presumably because of an energetic or mechanical trigger[7], and quadrupeds switch from a trot to a gallop at Fr ≈ 2.5 (ref. 6). The elephants routinely exceeded Fr 1.0, reaching Fr values as high as 3.4 — speeds that are inconsistent with a quadrupedal walking gait. Other animals, such as running birds[8], also have non-aerial gaits at high Froude numbers.

It is the exchange of gravitational potential and kinetic energy that fundamentally distinguishes walking and running[9–11]. The centre of mass is highest at mid-stance in walking, but lowest at mid-stance in running[12]. Our analysis shows that at low speeds, as expected for walking, elephants' shoulder and hip joints rise and then fall (indicating vertical motion of the centre of mass) during the stance phase. At the highest speeds, the vertical movements of the shoulder indicate walking, but hip motion indicates running (Fig. 2b). During the stance phase of the forelimb, shoulder motion resembles walking, moving upwards and then downwards while the front foot is on the ground, whereas the hip's motion during the stance phase of the hindlimb is characteristic of running, moving downwards and then upwards.

Usually, the various criteria for walking and running are consistent, making it relatively easy to distinguish walking from running, but this is not true in the case of elephants. Our observations suggest that, at greater speeds, elephants do more than merely walk. Ground-reaction-force data are needed to demonstrate conclusively whether the elephants' gait involves spring-

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like running kinetics[9–12] and to understand why elephants avoid using an aerial phase.

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Cell biology

Developmental predisposition to cancer

Many human cancers occur in renewing epithelial tissues, in which cellular lineages typically go through two distinct phases: early in life, cell populations expand exponentially to form the tissue, and for the remainder of life, the tissue is renewed by stem cells dividing to create an almost linear cellular history[1]. Here we use a simple mathematical model to show that mutations that arise during the exponential phase probably seed tissues with stem cells carrying mutations that may predispose to cancer. Susceptibility to late-life cancers, such as those of the skin and colon, may therefore be influenced by somatic mutations that occur during early development.

Mutations accumulate during exponential cellular growth according to the Luria–Delbrück distribution[2]. Let u_e be the mutation probability per cell division during exponential growth, and N the number of stem cells produced during development to seed the tissue. Starting with one cell, the number of cell divisions required to produce N cells is $N-1$, and the total number of mutation events during the cellular history is $M = u_e(N-1) \approx u_e N$. (The phases of cellular growth are shown in Fig. 1a.)

Figure 1b shows the probability distribution for the number of stem cells that carry mutations. These are the mutations that arise during exponential growth, before further division of the stem lineages to renew the local tissue. The average frequency of stem cells with mutations is $\overline{x} \approx u_e \ln(N)$ (ref. 2).

Although the frequency of stem cells that initially contain a mutation may be small, those mutations can contribute substantially to the total risk, R_T, of cancer. Suppose that k rate-limiting mutations are needed for cancer to develop[3,4], then the total risk is $R_T = N(1-x)R_k + NxR_{k-1}$, where x is the frequency of stem cells that start with one mutation, and R_k is the risk that a particular stem-cell lineage acquires k mutations during the phase of linear division and tissue renewal. The risk, R_k, is given roughly by the gamma distribution, which describes the probability of the occurrence of the kth event over a particular time interval. From the gamma distribution, $R_k \approx (u_s\tau)^k/k!$, where u_s is the mutation rate per stem-cell division, and τ is the total number of stem-cell divisions.

The expected fractional increase in cancer risk arising from stem cells that begin with one mutation can be calculated as $F = \overline{x}\, R_{k-1}/R_k \approx u_e \ln(N) k/u_s\tau$. The next step is to assign approximate magnitudes to these quantities. We can take $k \approx 5$ for the number of rate-limiting mutations required to cause epithelial cancer in humans[3,4], $\ln(N) \approx 20$, and τ to range from 100–1,000. This gives the fractional increase in cancer risk, F, ranging from $u_e/10u_s$ to u_e/u_s.

If mutations accumulate with the same probability per cell division during exponential growth and linear stem-cell division ($u_e = u_s$), then the increase in risk from mutations arising in development ranges from 10–100%. If, as has been claimed[5], mutation rates in stem cells are much lower than those during exponential growth, ($u_s \ll u_e$), then almost all cancer arises from predisposed stem-cell lineages that were mutated during development.

The risk of cancer from somatic mutations during development could be quantified by studying inbred rodents. The number of predisposing mutations per individual will vary according to the Luria–Delbrück distribution (Fig. 1b). Individuals with many predisposing mutations are likely to develop multiple independent tumours relatively early in life, whereas those with few predisposing mutations should develop few tumours relatively late in life. Controlled experiments have shown variability in cancer susceptibility among inbred rodents[6], but the causes of this variability have not been determined.

Mutations during development seed a young individual with a small fraction of mutated stem cells. Those relatively few mutated lineages may therefore be responsible for a substantial proportion of the cancers that develop later in life.

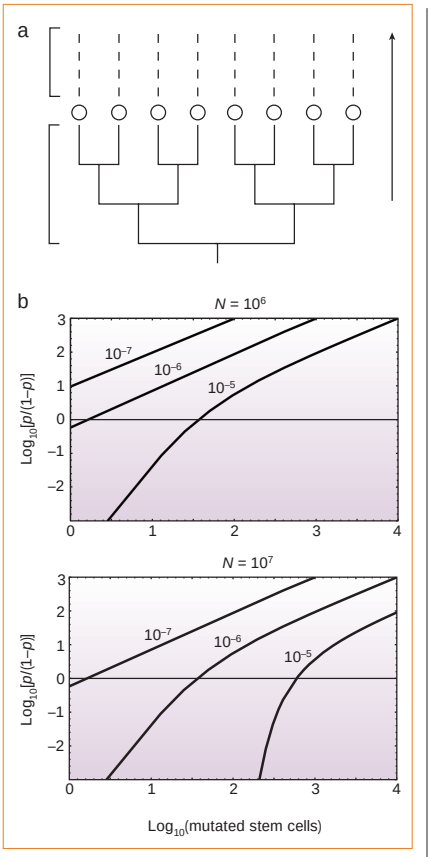

Figure 1 The role of tissue architecture in the accumulation of mutations during development. **a,** The phases of cellular growth in epithelial tissues. Cell populations increase exponentially during development, shown by a branching phase of division; at the end of development, stem cells differentiate in each tissue compartment (circles); stem cells renew each compartment by dividing to form a nearly linear cellular history (dashed lines) — each stem-cell division gives rise to one daughter stem cell that continues to renew the tissue and to one daughter transit cell that divides rapidly to produce a short-lived lineage that fills the tissue. **b,** The cumulative probability, p, for the number of stem cells mutated during development. By plotting $\log_{10}[p/(1-p)]$, the zero line gives the median of the distribution. N, number of stem cells produced during exponential growth. The number above each line is u_e, the mutation probability per cell added to the population during exponential growth. For a single gene, u_e is probably of the order of 10^{-7}; thus, if there are 100 genes for which initial mutations can influence the progression to cancer, then u_e is roughly of the order of 10^{-5}. The Luria–Delbrück distribution plotted here was calculated using equation (8) of ref. 7.

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