Deadly strike mechanism of a mantis shrimp

This shrimp packs a punch powerful enough to smash its prey's shell underwater.

Stomatopods (mantis shrimp) are well known for the feeding appendages they use to smash shells and impale fish. Here we show that the peacock mantis shrimp (*Odontodactylus scyllarus*) generates an extremely fast strike that requires major energy storage and release, which we explain in terms of a saddle-shaped exoskeletal spring mechanism. High-speed images reveal the formation and collapse of vapour bubbles next to the prey due to swift movement of the appendage towards it, indicating that *O. scyllarus* may use destructive cavitation forces to damage its prey.

Stomatopod appendages were previously thought to be limited to a maximum speed of 10 m s^{-1} (ref. 1) but, by using new imaging technology and a faster species, we have measured the dactyl heel reaching peak speeds of 14–23 m s^{-1}, peak angular speeds of 670–990 rad s^{-1}, and peak acceleration of 65–104 km s^{-2} within an average period of 2.7 ms, making *O. scyllarus* perhaps the fastest appendicular striker in the animal kingdom.

To generate these extreme movements in water, a large amount of energy must be released over a short period. Stomatopods increase their power output through the use of a click mechanism, in which latches prevent appendage movement until muscle contraction is maximal[1,2]. When the latch is freed, stored energy is released over a shorter time than the duration of the original muscle contraction. Some extreme animal movements also require a specialized spring, because the muscle fibres and tendon store insufficient elastic strain energy[3–5]. We conservatively calculate that a minimum power requirement of 4.7×10^5 watts per kilogram of muscle is necessary for a typical strike, which is orders of magnitude higher than that available in the fastest-contracting muscles[3,6] known. Moreover, the lateral extensor muscle and apodeme (arthropod tendon) could store only a small fraction of the strain energy underlying the work requirements in an average strike (see supplementary information), which means that stomatopods need a specialized spring.

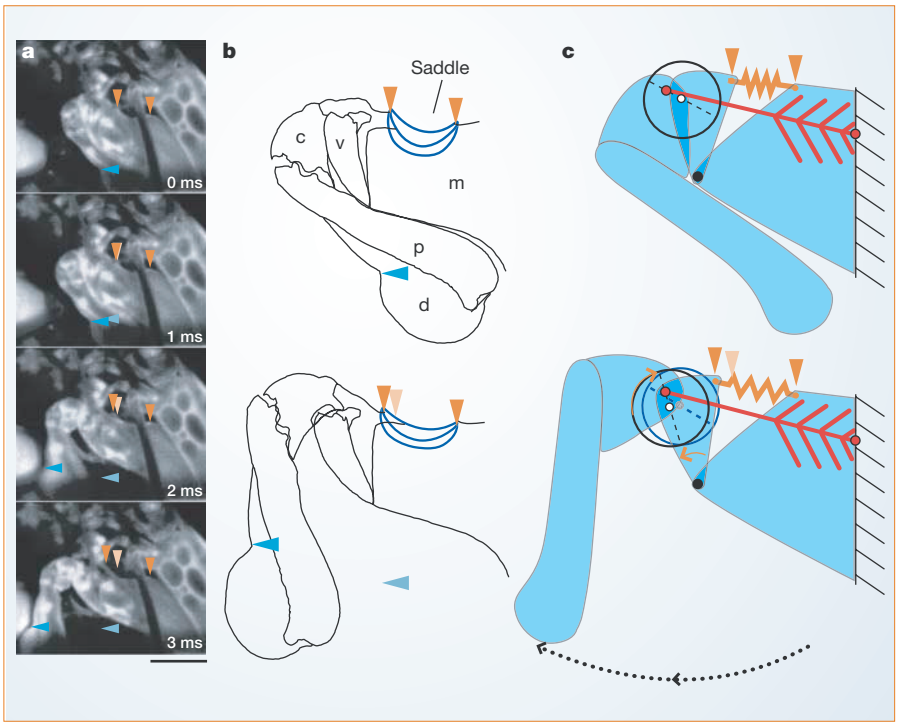

Figure 1 The mechanics of a stomatopod strike. **a,** A high-speed image sequence illustrates the distal extension of the saddle (orange triangles) occurring simultaneously with the extension of the smashing heel of the dactyl and propodus (blue triangles). Scale bar, 1 cm. Appendage kinematics were based on 6 individuals and 7–12 strikes per individual; measurement error, ±4%. Images were recorded at 5,000 frames s^{-1}. **b,** The compressed (top) and released (bottom) saddle on the raptorial appendage (raptorial segments: m, merus; v, meral–V; c, carpus; p, propodus; d, dactyl). **c,** The saddle modelled as a spring (orange) that stores elastic energy to drive the movement. Top, pre-strike phase. The lateral extensor muscle and apodeme (red) pull on the carpus to compress the saddle, while flexor muscles (not shown) engage a click mechanism to prevent extension of the appendage[1]. Bottom, the strike occurs when the latch is released[1], enabling the saddle to extend and two pivot points to rotate in opposite directions. The meral–V forms a pivot point (black circle) as it rotates distal–ventrally (anticlockwise here) and pushes the second pivot point, the carpal–meral fulcrum (white circle), distally. The isometrically contracted extensor muscle maintains a constant distance between its carpal and meral attachment points (red circles), forcing the carpus to rotate (clockwise here) and driving the dactyl heel towards the prey.

Figure 2 The mantis shrimp *Odontodactylus scyllarus* strikes a snail. Cavitation bubbles (arrow) form and collapse between the dactyl heel (red) and the snail (black). Scale bar, 1 cm.

In our model for the stomatopod strike, elastic energy is stored in a compressive, saddle-shaped spring (Fig. 1) that is a stiff exoskeletal structure located dorsally on the merus (the enlarged proximal segment) of all stomatopods. Hyperbolic–paraboloid (saddle-shaped or anticlastic) surfaces are used in engineering and architecture: their opposite and transverse curvatures reduce failure by distributing stresses across the three-dimensional surface. Likewise, the saddle shape of the stomatopod's spring minimizes the probability of local buckling while compressing and extending. To our knowledge, the stomatopod's saddle is the first biological hyperbolic–paraboloid spring to be described.

Stomatopod smashing produces a loud pop, and high-speed video images reveal evidence of cavitation (Fig. 2). Cavitation occurs in fluids when areas of low pressure form vapour bubbles that collapse and yield considerable energy (in the form of heat, light and sound); these can be sufficient to destroy boat propellers and other hard surfaces[7]. Cavitation has been noted in snapping shrimp, which appear to 'shoot' cavitation bubbles at prey items to stun them[8,9].

By contrast, cavitation in stomatopods occurs between the surface being struck and the dactyl heel. Although the heel is highly mineralized[10], the surface becomes pitted and damaged over time; stomatopods moult frequently and produce a new smashing surface every few months. In addition to their novel energy-storage mechanism and remarkable striking speed, stomatopods may be using cavitation forces to process their prey.

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Origin of AIDS

Contaminated polio vaccine theory refuted

Despite strong evidence to the contrary[1–5], speculation continues that the AIDS virus, human immunodeficiency virus type 1 (HIV-1), may have crossed into humans as a result of contamination of the oral polio vaccine (OPV)[6–8]. This 'OPV/AIDS theory' claims that chimpanzees from the vicinity of Stanleyville — now Kisangani in the Democratic Republic of Congo — were the source of a simian immunodeficiency virus (SIVcpz) that was transmitted to humans when chimpanzee tissues were allegedly used in the preparation of OPV[6,7]. Here we show that SIVcpz is indeed endemic in wild chimpanzees of this region but that the circulating virus is phylogenetically distinct from all strains of HIV-1, providing direct evidence that these chimpanzees were not the source of the human AIDS pandemic.

Detection and molecular characterization of SIVcpz in chimpanzee communities in the vicinity of Kisangani should directly test the OPV/AIDS theory. An earlier survey of chimpanzees at Wanie-Rukula near Kisangani (Fig. 1a; W. D. Hamilton, M.W. and J. B. J., January 2000, see ref. 9) failed to identify SIVcpz viral (v) RNA in any of 34 faecal samples collected. However, western immunoblot analysis of 10 chimpanzee urine samples collected at the same time identified two specimens that showed strong crossreactivity with the HIV-1 core protein p24 (Fig. 1b). Such indeterminate urine antibody profiles were found in chimpanzees from Tanzania, where SIVcpz infection was subsequently demonstrated after amplification by polymerase chain reaction (PCR) and sequencing of DNA[10].

To confirm the existence of SIVcpz in the Kisangani apes and to identify circulating strain(s) at a molecular level, we resumed field-work in February 2003, this time collecting 97 faecal samples from three different sites (for map, see supplementary information). From these, we identified one SIVcpz vRNA-positive specimen from the Parisi forest by PCR amplification of *gag* (422 base pairs) and gp41/*nef* (699 base pairs) sequences. This result confirmed that natural SIVcpz infection was present in chimpanzees in the Kisangani region.

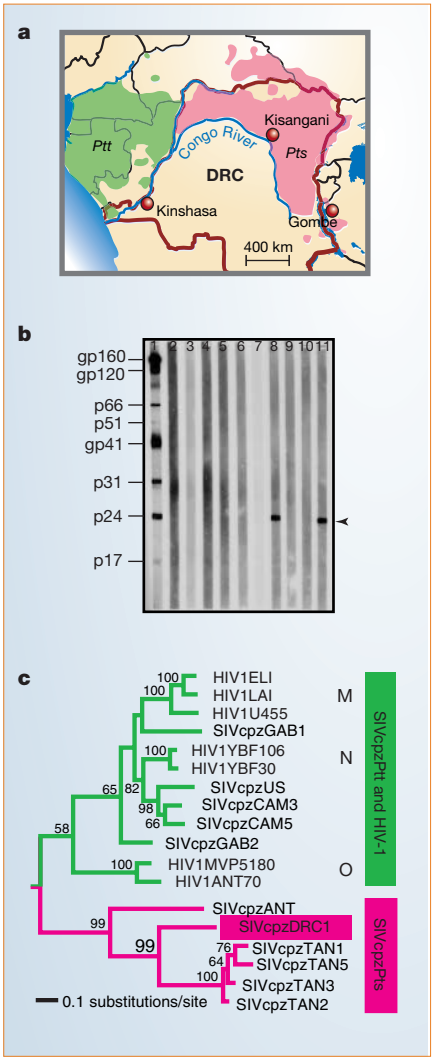

Figure 1 The Kisangani expeditions and refutation of the OPV/AIDS theory. **a,** Map of the Democratic Republic of Congo (DRC) and neighbouring countries showing the ranges of *Pan troglodytes troglodytes* (*Ptt*) and *Pan troglodytes schweinfurthii* (*Pts*). **b,** Western blot analysis of urine samples from 10 chimpanzees, collected during the 2000 expedition to rainforests near Kisangani; plasma from a positive HIV-1 control is shown in the left lane, with crossreactivity to the different viral proteins indicated. Two samples showed strong crossreactivity with HIV-1 p24 (arrowhead). **c,** Maximum-likelihood-estimated phylogenetic tree for gp41/*nef* sequence data, with bootstrap results. Bootstrap percentages are shown for all clades that were present in more than 50% of 1,000 maximum-likelihood-inferred bootstrap trees. Support for the SIVcpzDRC1/SIVcpzTAN clade was very strong (99%). Analysis of partial *gag* sequences supported the same phylogenetic position for SIVcpzDRC1 (data not shown). For further details of phylogenetic analyses, see supplementary information.

Phylogenetic analysis of the newly derived sequences revealed that the Kisangani virus clustered with high statistical support with SIVcpz strains that were infecting chimpanzees of the same subspecies (*Pan troglodytes schweinfurthii*) that lived about 800 km to the south-east in Gombe National Park in Tanzania[10,11]. The new virus, which we designate SIVcpzDRC1, represents a third lineage within the well circumscribed *P. t. schweinfurthii* SIVcpz radiation, and is clearly distinct from the *P. t. troglodytes* SIVcpz clade that includes all known strains of HIV-1 (Fig. 1c, and see supplementary information).

These results indicate that chimpanzees in the vicinity of Kisangani are endemically infected with SIVcpz that is highly divergent from HIV-1, thereby ruling out these apes as the source of HIV-1 and refuting the OPV/AIDS theory. Instead, each of the many circulating HIV-1 variants comprising groups M, N and O is linked to SIVcpz from *P. t. troglodytes* (Fig. 1c), the chimpanzee subspecies native to west-central Africa[1,12].

Given that fears about the safety of polio vaccines are currently threatening the global campaign to eradicate the disease[8], our clear-cut evidence against one of the key sources of concern is timely. The molecular epidemiological data presented here, together with data suggesting that HIV-1 group M originated 30 years before OPV trials were conducted[1,13] and the absence of detectable SIVcpz or chimpanzee DNA in archival stocks of OPV[2,3], should finally lay the OPV/AIDS theory to rest.

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