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From Sub-Rayleigh to Supershear Ruptures During Stick-Slip Experiments on Crustal Rocks

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Supershear earthquake ruptures propagate faster than the shear wave velocity. Although there is evidence that this occurs in nature, it has not been experimentally demonstrated with the use of crustal rocks. We performed stick-slip experiments with Westerly granite under controlled upper-crustal stress conditions. Supershear ruptures systematically occur when the normal stress exceeds 43 megapascals (MPa) with resulting stress drops on the order of 3 to 25 MPa, comparable to the stress drops inferred by seismology for crustal earthquakes. In our experiments, the sub-Rayleigh–supershear transition length is a few centimeters at most, suggesting that the rupture of asperities along a fault may propagate locally at supershear velocities. In turn, these sudden accelerations and decelerations could play an important role in the generation of high-frequency radiation and the overall rupture-energy budget.

Earthquake damage depends, in part, on the velocity of the rupture front (1). In 1973, Burridge demonstrated theoretically that in-plane shear ruptures (mode II) could propagate at velocities higher than the shear wave velocity (C_s) and up to the compressional wave velocity (C_p) (2). Since then, so-called supershear ruptures (V_r > C_s, where V_r is rupture velocity) have been observed during large strike-slip earthquakes (3–8). The stress and geometric conditions leading to the transition between sub-Rayleigh and supershear ruptures have been investigated with photoelasticity, both theoretically (9, 10) and experimentally, on brittle polymers (11–15). Although these experiments successfully illustrate supershear ruptures, the lack of experiments on rock samples limits the ability to understand these rare events observed in nature. In a recent experimental study, a photoelastic setup was coupled with an acoustic high-frequency–recording multistation array during stick-slip experiments on polycarbonate sheets. This allowed Schubnel et al. to use high-frequency acoustics to identify unequivocally the signature of both sub-Rayleigh and supershear ruptures (15). This advance has opened the possibility of revisiting experimental work performed on rocks (16–19).

Here, we report results from stick-slip experiments conducted on saw-cut Westerly granite samples (fig. S1), which serve as proxies for crustal rocks, during triaxial loading (where the tensile rupture of experiments conducted on saw-cut Westerly granite samples (fig. S1), which serve as proxies for crustal rocks, during triaxial loading (where the
principal stresses $\sigma_1 > \sigma_2 = \sigma_3$). Stick-slip experiments and earthquake mechanisms are analogous in nature because they both result from rapid frictional sliding along preexisting faults, leading to partial or total stress drop (16). In our experiments, the stress conditions were typical of the upper crust, ranging from 10 to 150 MPa in normal stress acting on the preexisting fault. In total, we recorded more than 200 stick-slip events (Fig. 1). For each event, we inverted the rupture velocity directly from our experimental records, using high-frequency acoustics as a tracking tool (fig. S2).

We used recorded accelerograms to track the Mach waveform arrival. Theoretical arrival time of the Mach waveform radiated away from the rupture tip was predicted using (i) the position of the rupture front determined from the inverted rupture velocity (fig. S2) and (ii) the shear wave velocity and the distance between the Mach front antenna (MFA) sensors to the fault, as defined in Fig. 2A (15). Our calculation assumes that the rupture velocity is constant. Importantly, we looked for ruptures with $\nu_s$ greater than the shear wave speed but different that $\sqrt{2c_s}$, for in this case, no Mach cone is expected (20).

We compared our calculations with waveforms recorded by the MFA array for a stick-slip event during which a supershear rupture velocity was predicted by the inversion (Fig. 2B). In agreement with theory (21), we first observed a weak $P$-wave arrival, which corresponds to the continuous emission of $P$ waves by the rupture tip as it propagates. However, the signal is dominated by the arrival of a large-amplitude, coherent wavefront just after the diffuse $P$-wave arrival. The relative amplitude of this waveform, when compared to the first $P$-wave amplitude, increases with distance to the fault. This is expected because the geometric attenuation of a conic waveform is smaller than that of spherical one. At each station, the arrival time of this waveform is consistent with the predicted arrival time of the Mach waveform.

To confirm our estimations of the rupture velocity, we used two-dimensional (2D) steady-state rupture model to conduct simulations (21). We observe an excellent fit, both in relative amplitude and for the general waveform shape, when comparing the experimental waveforms recorded on the MFA sensors during a subshear event and the synthetics obtained by our numerical simulation (Fig. 3B). We observed similar good correspondence between experimental waveforms and simulation of a subshear rupture (Fig. 3C). In both cases, we obtained the best fit between analytical and experimental records by using the rupture velocity estimated experimentally, confirming that our experimental estimate of the rupture velocity is accurate. Furthermore, we show that dynamic rupture models that can accurately simulate strong ground motions on the kilometric scale can also simulate accelerations in the kilohertz range on centimeter sized samples. In other words, dynamic rupture propagation is truly a self-similar mechanism.

Our experimental results demonstrate that the ruptures were dominantly mode II (fig. S3). For this mode, the transition between sub-Rayleigh and supershear rupture has been extensively discussed in theoretical and experimental studies (9, 12–15). Following 2D numerical studies, this transition is generally explained in terms of the seismic ratio $S = (\tau_p - \tau_{co})/(\tau_{p, co})$, where $\tau_p, \tau_{co}$, and $\tau_{co}$ are the peak frictional strength, the initial shear stress, and the residual frictional strength, respectively. The ratio $\tau_p/\sigma_n$ (where $\sigma_n$ is the normal stress), employed by Ben-David et al. (14), is equivalent to $S$, and both quantities are simply related by $\tau_p/\sigma_n = (f_d - f_s)/(1 + S) + f_d$, where $f_d$ and $f_s$ are the static and dynamic friction coefficients, respectively (Fig. 4). In our experiments, $\tau_p$ was continuously measured (Fig. 1). Taking $f_s = 0.85$ and $f_d = 0.1$, $S$ could be estimated for each individual stick-slip. Supershear propagation may happen under both of the following conditions: (i) $S < S_c$ (where $S_c$, the critical value of $S$ allowing supershear transition, is equal to 1.77 or 1.119 in 2D and 3D, respectively), which was always the case in our experiments (Fig. 4A), and (ii) when the rupture length exceeds the transition length $L_t$, estimated following the semi-empirical relation (9)

$$L_t = \frac{39.2}{\pi(1 + \nu)} \frac{1}{(S_c - S)^\frac{3}{2}} \left(\frac{\mu G}{1 + S}\right)^{\frac{1}{2}}$$

where $\nu, \mu, G$ are, respectively, the Poisson ratio, the shear modulus, and the fracture energy. In our case, condition (ii) can be met only if $L < L_t$, where $L_t$ is the finite length of the experimental fault. In our experiments, $G$ may range from
the lower bound 10 J/m², as given by single-crystal fracture energy values and stick-slip experiments performed at low normal stress (19), to the upper bound 10⁴ J/m², measured for intact Westerly granite samples at high confining pressures (22). From Eq. 1, L was calculated as a function of normal stress in two cases: (i) S = 1 and G = 10 J/m² and (ii) S = 0 and G = 1000 J/m² (Fig. 4A). Experiments performed at the lowest normal stresses are compatible with G = 10 J/m². However, experiments performed at intermediate normal stresses can be explained only by using larger fracture energy. This is consistent with our observation of an intense production of fine gouge particles at intermediate and high normal stresses. Indeed, for spherical particles, the ratio between G and surface energy of single crystals (τ) is $G/\gamma \approx 3w/d$, where w is the fault thickness and d is the average particle size. Our observation of gouge particles of 1-μm diameter and smaller is consistent with a fault roughness of less than 30 μm.

The match between the measured rupture velocities and the prediction that the minimum transition length L drops to a few centimeters (comparable to our sample size) at high normal stress (Fig. 4A) explains why, in our experiments, supershear rupture becomes a “normal” phenomena for $\sigma_n > 60$ MPa. It also explains why supershear ruptures were not clearly observed in previous experimental studies on rocks conducted at low normal stress (most often in biaxial conditions) (18, 19).

Finally, we observe a double correlation between the rupture velocity, the initial stress ratio $\tau_n/\sigma_n$, and the final stress drop (Fig. 4B). Sub-shear ruptures occurred for stress ratios $\tau_n/\sigma_n < 0.6$ and resulted in stress drops generally lower than 1.5 MPa. Conversely, supershear ruptures occurred for stress ratios $\tau_n/\sigma_n > 0.7$ and resulted in stress drops generally larger than 3 MPa. These results not only make sense physically but are also compatible with values previously observed on brittle polymers (14) and with field observations for the Kunlunshan earthquake (23). Importantly, our findings are comparable to the average stress drops inferred by seismologists for most large crustal earthquakes. Note that our direct measurement of the stress drop is comparable to what a seismological estimate would be using the final slip $u$ (fig. S4).

Based on our experimental results, why is there a paucity of supershear ruptures observed in nature? A first straightforward explanation is related to the difference in fault geometry between our experiments and seismogenic faults. Our experiments consisted of a perfectly planar fault geometry with very low initial roughness at high normal stress, leading to uniform and large stresses on the fault plane. Seismogenic faults, on the other hand, are most often nonplanar and exhibit self-affine roughness (24). The occurrence of kinks and dilatational jogs could slow down or even arrest locally the propagation of seismic ruptures (25). The few documented examples of supershear earthquakes are on very smooth, planar fault sections (3–8). In addition, the presence of a gouge layer along the fault interface may slow the propagation of the rupture, as well as thermohydro-mechanical coseismic processes within the breakdown zone [such as thermal pressurization (26), frictional melting (27), mineral reactions (28), and off fault damage, including pulverization (29)], which dissipate part of the released strain energy available, resulting in a deceleration of the rupture front. Alternatively, the paucity of supershear rupture observation in nature might also be due to limitations in instrumentation and/or spatial coverage. Nevertheless, the experimental values of L and stress drops reported here for a classical crustal lithology (Westerly granite) under upper-crustal conditions (<150 MPa) demonstrate that rupture velocity may exhibit important variations at the scale of small (centimetric) asperities, so that the seismological estimate of rupture velocities over long fault segments is an average that could well have little importance at the scale of an asperity. Our experimental results strongly suggest that, despite the scarcity of compelling measurements on natural earthquakes, supershear ruptures may frequently occur at the local scale of asperities, for which the stress drop generally inferred is quite large. In turn, these sudden accelerations and decelerations of the rupture front should play an important role in generating high-frequency radiation, which will influence the total rupture-energy budget.

References and Notes
Stepwise Evolution of Essential Centromere Function in a Drosophila Gene

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Evolutionarily young genes that serve essential functions represent a paradox; they must perform a function that either was not required until after their birth or was redundant with another gene. How young genes rapidly acquire essential function is largely unknown. We traced the evolutionary steps by which the Drosophila gene Umbrea acquired an essential role in chromosome segregation in D. melanogaster since the gene’s origin less than 15 million years ago. Umbrea neofunctionalization occurred via loss of an ancestral heterochromatin-localizing domain, followed by alterations that rewired its protein interaction network and led to species-specific centromere localization. Our evolutionary cell biology approach provides temporal and mechanistic detail about how young genes gain essential function. Such innovations may constantly alter the repertoire of centromeric proteins in eukaryotes.

To gain insight into the birth and evolution of essential function, we focused on one newly evolved gene in Drosophila. Umbrea (also known as HP6 and CG15636) arose via duplication of the intronless Heterochromatin Protein 1B (HP1B) gene into an intron of the dumphy gene (Fig. 1A) (7). HP1B is a chromosomal protein that predominantly localizes to heterochromatin in D. melanogaster cells and regulates gene expression (8). HP1B is dispensable for viability (8), yet RNA interference (RNAi) knockdown phenotypes show Umbrea to be essential in D. melanogaster (1, 9). The 100% late larval-pupal lethality upon Umbrea knockdown could be rescued by an Umbrea–green fluorescent protein (GFP) fusion (fig. S1). Genetic knockout experiments (fig. S1) further confirmed that Umbrea is essential in D. melanogaster.

We traced Umbrea’s evolutionary path after duplication from HP1B to understand when and how essential function was gained by comparing the localization of HP1B and Umbrea proteins in D. melanogaster Kc cells. GFP-tagged HP1B proteins from both D. melanogaster and D. ananassae (whose divergence predates the birth of Umbrea (7)) localized to pericentric heterochromatin and euchromatin (Fig. 1B and fig. S2). In contrast, Umbrea-GFP predominantly localized to interphase centromeres, but not telomeres (Fig. 1C and fig. S3, A and B). Specific antibodies raised against Umbrea (fig. S4A) confirmed its centromere localization in developing spermatocytes and larval imaginal discs (Fig. 1D and fig. S4, B and C).

On the basis of its essentiality and centromere localization, we hypothesized that Umbrea was required for chromosome segregation. Upon depletion of Umbrea by RNAi knockdown (fig. SSA), relative to control cells, D. melanogaster S2 cells displayed increased mitotic errors, including delayed chromosome alignment, early anaphase onset, lagging anaphase chromosomes, and multipolar configurations (P < 0.05) (Fig. 1, F and G, fig. S5B, and movies S1 to S3). These data suggest that Umbrea promotes proper chromosome segregation, but it is not required for the localization of the centromeric histone Cid (Fig. 1F).

To date the origin of Umbrea and subsequent changes, we sequenced the Umbrea locus from 32 Drosophila species (fig. S6A). Whereas HP1B was preserved (7), we found Umbrea in only 20 of 32 species, dating its monophyletic origin to 12 to 15 million years ago (Fig. 2A and fig. S6B). Using maximum likelihood methods, we observed evidence of both episodic and recurrent positive selection acting on Umbrea (fig. S7, A to D). These findings, together with the altered localization, lead us to conclude that neofunctionalization, not subfunctionalization, drove the divergence of Umbrea (10). Although Umbrea is essential in D. melanogaster, it was lost at least three independent times—in D. fuyamai, D. eugracilis, and in the suzukii clade (Fig. 2A)—which suggests that Umbrea was not essential at or immediately after its birth.

Four lineages retained full-length Umbrea genes, two of which encode an intact chromodomain (CD) and ancestral residues essential for binding histone H3 trimethyl Lys9 (H3K9me) (fig. S8) (11). However, most extant Umbrea genes have lost their CDs, and encode only the chromoshadow domain (CSD), which mediates protein–protein interactions (12) (Fig. 2A). We first tested how CD loss affected HP1B function. We found that an HP1B-GFP fusion lacking the CD lost heterochromatin localization (Fig. 2B), consistent with the requirement of HP1 CD for H3K9me binding (13). Furthermore, fusion of the HP1B CD and hinge to Umbrea-GFP reverted localization from centromeres to heterochromatin (Fig. 2C), which suggests that loss of the ancestral CD