

Transport-healing scaling for Omori exponents: A minimal model and its limits

Giuseppe Petrillo ^{*}

CNRS, ENS de Lyon, LPENSL, UMR5672, 69342 Lyon Cedex 07, France



(Received 28 May 2026; revised 10 August 2026; accepted 15 September 2026;
published 24 September 2026)

The temporal decay of triggered seismicity is commonly described by the Omori law $r(t) \sim (t + c)^{-p}$. I introduce a minimal phenomenological framework that relates this temporal decay to two concurrent processes: the spatial spreading of a triggering perturbation and the progressive depletion of elements that remain susceptible to failure. The model yields the scaling relation $p = 1 - \alpha d/2 + \gamma$, where α characterizes spreading, d is an effective spatial dimension, and γ describes depletion or healing. This relation provides a joint consistency test rather than a unique inversion of the Omori exponent for material healing. Numerical simulations recover the predicted scaling. A comparison with published earthquake-pair statistics illustrates that slow spatial spreading can be identified, but also that spatial and temporal exponents cannot be combined unless their underlying observables and event selections are compatible. The main implication is that the Omori exponent alone cannot generally be interpreted as a direct measure of fault or reservoir healing; testing the proposed relation requires compatible estimates of spatial spreading, event-rate decay, effective geometry, and, where possible, an independent recovery observable.

DOI: [10.1103/fgw2-2sxn](https://doi.org/10.1103/fgw2-2sxn)

I. INTRODUCTION

The temporal decay of seismic activity following a stress perturbation is one of the most robust empirical laws in earthquake physics. Since the original formulation of Omori law [1] and its generalization by Utsu *et al.* [2], aftershock rates have commonly been described by a power-law decay

$$r(t) \sim (t + c)^{-p},$$

with typical exponents close to $p \simeq 1$. This empirical regularity has motivated interpretations based on critical relaxation [3,4], nonequilibrium dynamics [5], branching processes [6–8], and epidemic models of seismic triggering [9–11].

In statistical seismology, the epidemic-type aftershock sequence (ETAS) model [9,10,12] provides a natural branching-process interpretation of Omori decay. In ETAS, each earthquake can trigger secondary events according to a direct Omori-type kernel, usually written as $(t + c)^{-1-\theta}$. The observed aftershock sequence results from the superposition of many generations. Helmstetter and Sornette [13] showed that this cascade can renormalize the bare kernel. Importantly for the

^{*}Contact author: giuseppe.petrillo@ens-lyon.fr

present work, anomalous spatial spreading and continuous-time random-walk (CTRW) descriptions of earthquake triggering predate our model: Huc and Main [14] measured a slowly increasing correlation length in stacked earthquake-pair data and discussed anomalous stress diffusion, while later studies applied fractional-kinetic CTRW models to earthquake migration [15,16].

Physical models provide complementary explanations. In the rate-and-state formulation of Dieterich [5], the seismicity rate following a positive Coulomb stress step relaxes approximately as $1/t$, providing a mechanical route to Omori-like decay. Related approaches based on constitutive heterogeneity, viscoelastic relaxation, and ductile-controlled stress redistribution also tend to generate Omori exponents of order unity [3,17,18]. In addition, rate-and-state asperity models have shown that creep, strengthening, and the state of the seismic cycle can modulate Omori decay and modify the apparent aftershock-rate exponent [19].

Fluid-driven seismicity introduces an additional level of complexity, but it is not a single phenomenological class. Pressure diffusion, poroelastic stress transfer, correlated permeability, aseismic slip, and preexisting permeable fracture networks can produce distinct spatial footprints and time histories [20–26]. Classical hydraulic diffusion predicts $\ell(t) \sim t^{1/2}$; anomalous migration is an empirical possibility rather than a generally accepted description. CTRW is used below only as one effective coarse graining of heterogeneous transport, not as a microscopic model of hydraulic fracturing or as an alternative established over poroelastic descriptions.

The temporal reference time also depends on the problem. For a tectonic aftershock sequence, it is normally the mainshock time; for postinjection decay, it may be the shut-in time. It is generally not the start of injection. Poststimulation behavior varies strongly among sites, and event-event aftershock triggering may be absent, obscured by the injection rate, or comparable to tectonic triggering [26–30]. For event-event triggering in fluid-induced sequences, measured Omori exponents can likewise be of order unity. Aggregate seismicity decay after shut-in is a different observable and can be steeper and strongly site and protocol dependent [31,32]. Moreover, a steep power law can be difficult to distinguish from exponential or stretched-exponential relaxation over a limited time range.

I introduce a minimal coarse-grained closure designed to isolate transport and depletion. Internal relaxation is described by a time-dependent susceptible fraction,

$$\phi(t) \sim t^{-\gamma}, \quad (1)$$

which represents the progressive depletion of elements that remain sufficiently close to failure to be triggered. Within this activation-volume framework, the aftershock rate is controlled by the growth of the active region and by the decay of the susceptible population. This yields the scaling relation

$$p = 1 - \frac{\alpha d}{2} + \gamma, \quad (2)$$

where α is the anomalous-diffusion exponent, d is the effective spatial dimension, and γ is an effective healing exponent. Within this conditional closure, the common observation $p \simeq 1$ for tectonic aftershock sequences implies $\gamma \simeq \alpha d/2$, corresponding to an approximate balance between the spreading and depletion contributions to the observed decay exponent.

The equation is a conditional result: It follows only if the rate factorizes as the rate of newly explored volume times a spatially uniform susceptible fraction. Power-law recovery from broad relaxation spectra was derived previously for rocks and other disordered media [33,34]; that ingredient is not new. The combined relation instead provides a consistency condition. If independently estimated p , α , and d imply $\gamma < 0$, or if direct recovery measurements disagree with the inferred γ , the separable closure is rejected. The observables must, however, have definitions compatible with the closure; an exponent fitted to the amplitude of a cumulative spatial distribution is not automatically an Omori exponent. Coda-wave interferometry supplies one possible independent proxy for material recovery [35]. If the exponent characterizing velocity recovery is identified with the effective healing exponent γ , the scaling relation gives $p - \gamma = 1 - \alpha d/2$. Thus, $p = \gamma$ only in the special case $\alpha d = 2$; otherwise, the spreading contribution causes the seismicity-rate and

recovery exponents to differ. This interpretation remains conditional because velocity recovery is not necessarily identical to the recovery of the susceptible fraction represented by $\phi(t)$.

II. CONTINUOUS-TIME RANDOM WALK AND FRACTIONAL DIFFUSION EQUATION

I model the propagation of an effective activation signal through a heterogeneous fault or reservoir by a continuous-time random walk. A walker moves on a d -dimensional lattice with spacing a and performs nearest-neighbor jumps separated by independent waiting times τ drawn from the heavy-tailed density

$$\psi_\alpha(\tau) = \frac{\alpha \tau_0^\alpha}{(\tau + \tau_0)^{1+\alpha}}, \quad 0 < \alpha < 1, \quad \tau_0 > 0. \quad (3)$$

At long times, $\psi_\alpha(\tau) \sim \tau^{-(1+\alpha)}$, so that the mean waiting time diverges and the resulting transport is subdiffusive.

This choice is intended as a minimal coarse-grained representation of transport in fractured and fluid-saturated media. In such systems, the propagation of an activation signal associated with stress transfer or pore-pressure redistribution may be delayed by a broad spectrum of hydraulic, frictional, and structural relaxation times. Examples include fluid drainage through multiscale crack networks, trapping in poorly connected pores, transient permeability changes, and local mechanical relaxation across asperities or contact junctions. Broad distributions of trapping or response times are precisely the setting in which CTRW models lead, in the hydrodynamic limit, to fractional diffusion equations [36–38].

The use of Eq. (3) should therefore not be interpreted as a microscopic model of a specific hydraulic pathway. Rather, it encodes the effective consequence of unresolved heterogeneity: The activation signal spreads anomalously, with a characteristic length growing more slowly than in ordinary diffusion.

The origin $t = 0$ denotes the impulsive perturbation, whose subsequent relaxation is being modeled. It is the mainshock time for a tectonic sequence and may be the shut-in time for a postinjection sequence. The present Green's function problem does not describe the finite-duration injection stage. In particular, it is not applicable while hydraulic fracturing is actively changing the medium, because the stationarity and fixed-propagator assumptions entering Eq. (3) are then violated.

Let $P_i(t)$ denote the probability that the walker occupies lattice site i at time t . Since the walker performs instantaneous jumps separated by random waiting times drawn from $\psi_\alpha(\tau)$, the evolution of $P_i(t)$ is governed by a renewal master equation, commonly known as the Montroll-Weiss equation [36]:

$$P_i(t) = \Psi_\alpha(t) P_i(0) + \int_0^t \psi_\alpha(t - t') \frac{1}{2d} \sum_{j \in \text{NN}(i)} P_j(t') dt'. \quad (4)$$

The meaning of each term is the following.

The factor $\Psi_\alpha(t)$ is the probability that no jump has occurred between $t = 0$ and the current time t . Explicitly,

$$\Psi_\alpha(t) = 1 - \int_0^t \psi_\alpha(\tau) d\tau, \quad (5)$$

which is the tail (survival function) of the waiting-time distribution. The contribution $\Psi_\alpha(t) P_i(0)$ is therefore the probability that the walker started at site i and has remained there without making any jump up to time t .

The second term accounts for the possibility that the last jump occurred at some earlier time t' , with waiting time $\tau = t - t'$. The probability density for this event is $\psi_\alpha(t - t')$. If the last jump occurred at time t' , the walker must have been at one of the nearest neighbors of i at that time.

Because jumps are assumed to be instantaneous and symmetric, the walker chooses each nearest neighbor with probability $1/(2d)$. The factor

$$\frac{1}{2d} \sum_{j \in \text{NN}(i)} P_j(t') \quad (6)$$

therefore represents the conditional probability that the walker was at time t' at a site from which it can jump to i . Integrating over all possible renewal times t' yields the full gain term.

For exponential waiting times, $\psi(\tau) = \lambda e^{-\lambda\tau}$, Eq. (4) reduces to the standard Markovian master equation. For heavy-tailed $\psi_\alpha(\tau)$, instead, the dynamics becomes non-Markovian, with long memory encoded by the survival function $\Psi_\alpha(t)$.

Introducing the spatial Fourier transform $\hat{P}(\mathbf{k}, t) = \sum_i e^{-i\mathbf{k} \cdot \mathbf{r}_i} P_i(t)$ and temporal Laplace transform $\tilde{P}(\mathbf{k}, s) = \int_0^\infty e^{-st} \hat{P}(\mathbf{k}, t) dt$, Eq. (4) can be solved explicitly. For small wave vectors $|\mathbf{k}| \ll 1$ and small Laplace parameter $s \rightarrow 0$, one obtains, to leading order [37],

$$\tilde{P}(\mathbf{k}, s) = \frac{s^{\alpha-1}}{s^\alpha + D_\alpha |\mathbf{k}|^2}, \quad D_\alpha = \frac{a^2}{2d \tau_0^\alpha}. \quad (7)$$

Equation (7) coincides with the propagator of the time-fractional diffusion equation

$$\partial_t P(\mathbf{r}, t) = D_\alpha {}_0D_t^{1-\alpha} \nabla^2 P(\mathbf{r}, t), \quad (8)$$

where ${}_0D_t^{1-\alpha}$ is the Riemann-Liouville fractional derivative of order $1 - \alpha$ [39]. Thus, in the long-wavelength and long-time hydrodynamic limit, the CTRW with heavy-tailed waiting times is described by the fractional diffusion equation (8).

III. SCALING FORM OF THE GREEN'S FUNCTION

I now consider the continuum probability density $P(\mathbf{r}, t)$ solving Eq. (8) with a point-source initial condition at the origin, $P(\mathbf{r}, 0) = \delta(\mathbf{r})$. The corresponding Green's function is radially symmetric, $P(\mathbf{r}, t) = P(r, t)$ with $r = |\mathbf{r}|$, and obeys the self-similar scaling form [40]

$$P(r, t) = t^{-\alpha d/2} \mathcal{F}\left(\frac{r}{t^{\alpha/2}}\right), \quad (9)$$

where $\mathcal{F}$ is a dimensionless function decaying rapidly for large arguments. Physically, Eq. (9) encodes the subdiffusive growth of the characteristic length,

$$\ell(t) \sim t^{\alpha/2}, \quad (10)$$

and the concomitant decay of the peak amplitude $P(0, t) \sim t^{-\alpha d/2}$.

For later use, I introduce an effective coarse-grained activation field $A(\mathbf{r}, t)$ proportional to the local density of walkers,

$$A(\mathbf{r}, t) = A_0 P(\mathbf{r}, t), \quad A_0 > 0. \quad (11)$$

This field should be interpreted as a scalar measure of the probability that a stress or pore-pressure perturbation has reached a given region, rather than as a literal elastostatic stress tensor. Equation (9) then implies

$$A(r, t) = A_0 t^{-\alpha d/2} \mathcal{F}\left(\frac{r}{t^{\alpha/2}}\right). \quad (12)$$

A. Active front and Omori exponent

I now link the fractional-diffusion activation field to the spatial extent of the region explored by the stress or pore-pressure perturbation. This region is interpreted as the portion of the fault or reservoir reached by the propagating activation signal.

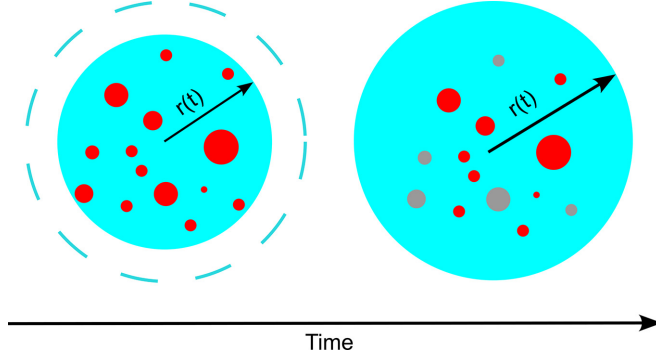


FIG. 1. Schematic view of the active region and internal healing. The cyan disk represents the region explored by the stress or pore-pressure perturbation, whose characteristic radius increases in time due to anomalous diffusion. Red circles denote defects or asperities that are still susceptible to failure, whereas gray circles indicate defects that have healed or become mechanically inactive. The aftershock rate reflects the competition between the geometric growth of the explored volume and the progressive depletion of failure-ready defects.

The underlying picture is sketched in Fig. 1: A perturbation spreads through a heterogeneous medium over an increasing characteristic length scale, while the population of defects that remain close enough to failure is progressively depleted by internal healing.

For a CTRW governed by Eq. (8), the characteristic spreading length is

$$\ell(t) \sim t^{\alpha/2}. \quad (13)$$

This length should be understood as the typical spatial extent reached by the perturbation, for example, as measured by the scaling variable of the Green's function or by the mean-square displacement $\langle r^2(t) \rangle \sim t^\alpha$. Possible logarithmic or model-dependent corrections associated with a specific definition of the activation front do not affect the leading scaling argument used below.

The corresponding explored volume scales as

$$V_{\text{act}}(t) \sim \ell(t)^d \sim t^{\alpha d/2}, \quad (14)$$

where d is an effective spatial dimension. In this minimal coarse-grained description, $V_{\text{act}}(t)$ represents the volume within which failures are mechanically admissible because the perturbation has reached the local population of defects.

Equation (14) neglects fault-network geometry and treats candidate defects as countable elements of equal statistical weight. Earthquake rupture areas and seismic moments are broadly distributed, including in injection-induced sequences [41]. This does not change an event-count rate if size is independent of encounter and survival, but it does change moment release and can change the count scaling when size, susceptibility, and healing are correlated. Such correlations are outside the present closure and cannot in general be absorbed into d .

The growth of the explored active volume, Eq. (14), determines the rate at which new regions of the fault or reservoir become mechanically accessible to triggering. However, the event rate at time t depends not only on the size of this region, but also on the fraction of its elements that remain susceptible to failure.

A variety of internal relaxation processes, including frictional healing, sealing and compaction of microcracks, pore-pressure dissipation, and the logarithmic aging of rate-and-state friction, reduce the number of critically stressed asperities as time elapses. In heterogeneous fractured media, these relaxation processes are expected to occur over a broad range of timescales. Broad distributions of relaxation times are a generic feature of disordered systems and naturally emerge in trap-model and CTRW descriptions of anomalous relaxation [37,42]. Postseismic seismic-velocity recovery

provides direct evidence for evolving elastic properties of damaged rock [35]. Broad recovery spectra and their power-law or approximately logarithmic transients have been developed previously in rock-physics models [33,34].

The emergence of broad healing times can be understood from a disordered energy-landscape picture. Following the standard trap-model description of glassy relaxation [42], I assume that each defect or asperity is characterized by an activation barrier E controlling the healing dynamics through an Arrhenius relation,

$$\tau_h = \tau_0 e^{E/E_T}, \quad (15)$$

where E_T is an effective activation-energy scale.

If the distribution of barrier heights is exponential,

$$P(E) = \frac{1}{E_0} e^{-E/E_0}, \quad (16)$$

the corresponding distribution of healing times follows from the change of variables,

$$\rho(\tau_h) d\tau_h = P(E) dE. \quad (17)$$

Using Eq. (15), one obtains

$$\rho(\tau_h) \propto \tau_h^{-1-\gamma}, \quad \gamma = \frac{E_T}{E_0}. \quad (18)$$

This standard change-of-variables argument is included to define the parametrization, not as a new derivation; closely related distributed-relaxation constructions were given by Kawada *et al.* [33] and Snieder *et al.* [34]. In this interpretation, small γ corresponds to highly disordered systems with broad barrier distributions, whereas larger γ reflects narrower distributions and faster effective healing.

To represent this effect, I assume that each defect or asperity is associated with a characteristic healing time τ_h , after which it is no longer available for triggering. Motivated by the barrier-distribution argument above, I therefore introduce the heavy-tailed healing-time distribution

$$\rho(\tau_h) = \frac{\gamma \tau_0^\gamma}{(\tau_h + \tau_0)^{1+\gamma}}, \quad \gamma > 0, \quad (19)$$

where τ_0 is a microscopic healing timescale.

The fraction of defects that remain susceptible at time t is then the survival probability of the healing-time distribution,

$$\phi(t) = \int_t^\infty \rho(\tau_h) d\tau_h = \left(\frac{\tau_0}{t + \tau_0} \right)^\gamma. \quad (20)$$

Thus, at long times,

$$\phi(t) \sim t^{-\gamma}. \quad (21)$$

In this interpretation, γ quantifies the breadth of healing times: Small γ corresponds to highly heterogeneous and slowly relaxing systems, whereas larger γ corresponds to a faster depletion of the population of failure-ready defects.

The underlying assumption is that seismic events are triggered when the propagating perturbation encounters previously unactivated defects that are still sufficiently close to failure. In this coarse-grained picture, the seismicity rate is therefore controlled by the rate at which new susceptible regions are explored by the activation front. Thus, in the simplest activation-volume approximation, I write

$$r(t) \propto \frac{d}{dt} V_{\text{act}}(t) \phi(t) \sim t^{\alpha d/2-1} t^{-\gamma} \sim t^{-p}, \quad (22)$$

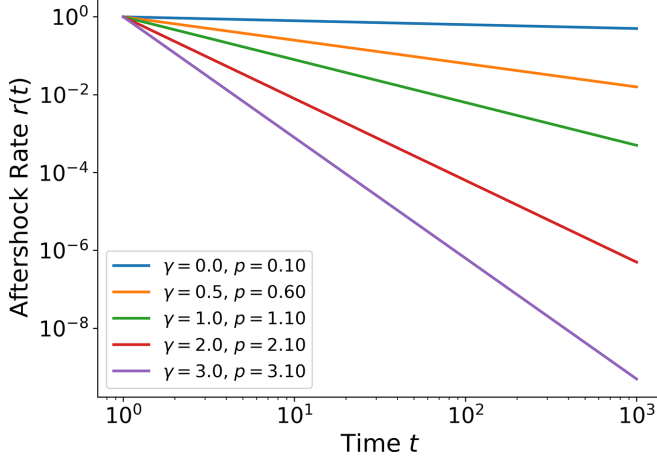


FIG. 2. Theoretical effect of internal healing on the Omori decay. Aftershock rate $r(t) \propto t^{-p}$ for several values of the healing exponent γ at fixed anomalous-diffusion exponent $\alpha = 0.6$ and effective spatial dimension $d = 3$. Within the present activation-volume approximation, the predicted Omori exponent is $p = 1 + \gamma - \alpha d/2$.

where

$$p = 1 - \frac{\alpha d}{2} + \gamma. \quad (23)$$

Thus, the Omori exponent receives a geometric contribution ($\alpha d/2$) from the anomalous spreading of the activation field and an independent contribution (γ) from internal healing or strengthening mechanisms. The combined influence of subdiffusive spreading and internal healing on the Omori decay is illustrated in Fig. 2. As γ increases, the depleted fraction of failure-ready defects grows faster and the Omori exponent becomes correspondingly larger.

To verify that the scaling relation is recovered beyond the purely analytical argument, I performed a minimal numerical simulation of the activation-volume model. The explored volume was prescribed as $V_{\text{act}}(t) \sim t^{\alpha d/2}$, and the number of newly explored defects in each logarithmic time bin was taken proportional to dV_{act} . Each newly encountered defect was assigned a healing time τ_h drawn from Eq. (19); the defect was counted as susceptible only if $\tau_h > t$. The simulated rate was then obtained as the number of susceptible newly explored defects per unit time.

Figure 3 shows the result for fixed $\alpha = 0.6$ and $d = 3$, varying the healing exponent γ . The rates display clear power-law decays, and the fitted Omori exponents agree with the theoretical prediction $p_{\text{th}} = 1 - \alpha d/2 + \gamma$ within the fluctuations across independent realizations. This numerical check confirms that the additive decomposition of the Omori exponent follows directly from the combination of anomalous spreading and distributed healing times.

B. What can, and cannot, be compared with Huc and Main [14]

The separable model makes a joint prediction that can be tested without identifying a microscopic transport mechanism. If a measured spatial scale is demonstrably the counterpart of the model length $\ell(t)$ and follows $L(t) \propto t^H$, comparison with Eq. (10) gives

$$\alpha = 2H. \quad (24)$$

If, independently, the corresponding incremental event rate decays as t^{-p} over the same regime, the activation-volume closure requires

$$\gamma = p - 1 + Hd. \quad (25)$$

Thus, $p \geq 1 - Hd$ is a necessary condition for a nonincreasing susceptible fraction ($\gamma \geq 0$).

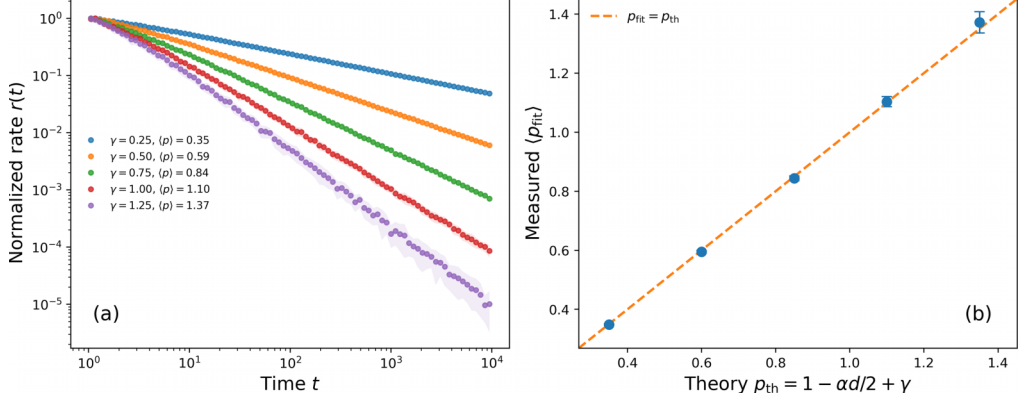


FIG. 3. Numerical validation of the activation-volume scaling. (a) Simulated aftershock rates for different healing exponents γ , obtained by sampling healing times from the distribution $\rho(\tau_h) \propto (\tau_h + \tau_0)^{-1-\gamma}$ and counting only newly explored defects that remain susceptible at time t . Rates are normalized by their maximum value for clarity. (b) Measured Omori exponent $\langle p_{\text{fit}} \rangle$ vs the theoretical prediction $p_{\text{th}} = 1 - \alpha d/2 + \gamma$ for fixed $\alpha = 0.6$ and $d = 3$. Error bars indicate the standard deviation over 30 independent realizations. The dashed line denotes $p_{\text{fit}} = p_{\text{th}}$.

Huc and Main [14] analyzed 13 798 shallow ($M_w \geq 5$, depth < 100 km) earthquakes using cumulative time windows and fitted the background-corrected spatial conditional probability as

$$P(r; t) = A(t)r^{-a(t)} \exp[-r/L(t)]. \quad (26)$$

Their text reports $L(t) = 13.6t^{0.06}$ km and $a(t) = 0.18t^{-0.06}$, while the caption of their Fig. 5 gives fitted slopes 0.058 for L and 0.067 for the mean separation. They also fit

$$A(t) = 406 \int_0^t x^{-0.60} \exp(-x/T_c) dx, \quad (27)$$

with $T_c = 16$ days in the main text (15 days in the Fig. 5 caption). Figure 4 reproduces the Table I values and these published parametrizations.

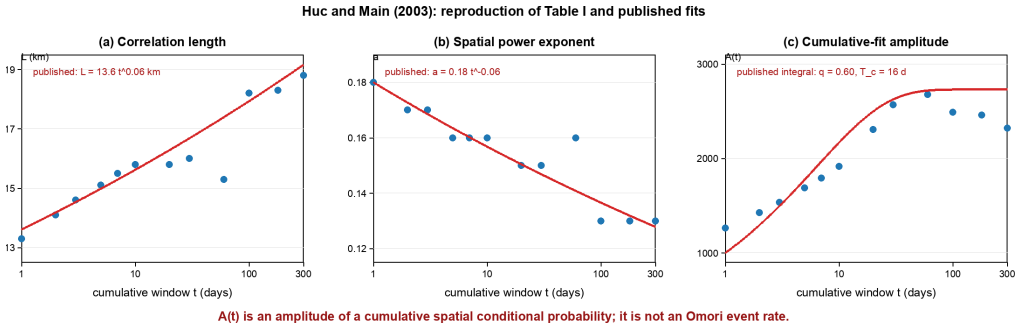


FIG. 4. Reproduction of quantities fitted by Huc and Main [14] from stacked, background-corrected earthquake pairs. Points are their Table I values. Curves use the parametrizations reported in their text: (a) $L(t) = 13.6t^{0.06}$ km; (b) $a(t) = 0.18t^{-0.06}$; and (c) $A(t) = 406 \int_0^t x^{-0.60} \exp(-x/T_c) dx$ with $T_c = 16$ days. Their Fig. 5 caption instead states $T_c = 15$ days and gives slopes 0.058 for L and 0.067 for mean distance; these are minor reporting or rounding differences in the source. Crucially, $A(t)$ is the amplitude of a cumulative spatial conditional probability, not an Omori event rate, so its exponent is not inserted into Eq. (25).

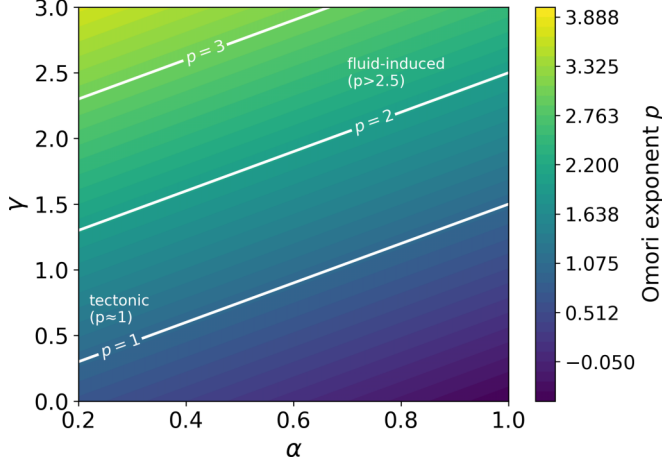


FIG. 5. Model phase diagram of the Omori exponent. Contour plot of $p = 1 + \gamma - \alpha d/2$ in the (α, γ) plane for $d = 3$. The white curves correspond to $p = 1$, $p = 2$, and $p = 3$. The plot displays the algebraic behavior of the separable activation-volume closure only; it is not an empirical assignment of tectonic or induced-seismicity classes.

These definitions do not permit a quantitative inversion within the present framework. In Eq. (26), $A(t)$ is the amplitude of a cumulative spatial distribution, not the total event count and not the incremental Omori rate. Even if the fitted expression were integrated over $0 < r < \infty$, its one-dimensional normalization would be

$$C(t) = A(t)L(t)^{1-a(t)}\Gamma[1 - a(t)], \quad (28)$$

so its derivative depends on A , L , and a , not on dA/dt alone. Moreover, identifying their pair-correlation length L with the activation-front scale ℓ is an additional modeling assumption. Consequently, Huc and Main's exponent 0.60 is not an Omori exponent in the present closure, and their data do not imply either a positive or a negative value of γ . At most, if L is provisionally identified with ℓ , the reported $H \simeq 0.06$ corresponds to $\alpha \simeq 0.12$ and provides a qualitative benchmark for very slow spreading. A valid joint test requires an independently estimated event rate and compatible spatial and recovery observables from the same selection of events.

IV. DISCUSSION

The analysis above shows that the Omori exponent emerging from our minimal model can be written in the compact form

$$p(\alpha, \gamma) = 1 - \frac{\alpha d}{2} + \gamma. \quad (29)$$

Here, α controls the assumed spreading law and γ the assumed depletion law. The algebraic additivity is exact only within the factorization $r(t) \propto \dot{V}_{\text{act}}(t)\phi(t)$. It does not show that healing is the physical cause of an observed large p ; exponential postinjection decay, a changing forcing, catalog incompleteness, triggering cascades, permeability evolution, or nonseparable space-time dynamics can yield different interpretations. In particular, coda-wave velocity recovery is a more direct observable of evolving elastic properties than the Omori exponent alone [35].

The phase diagram in Fig. 5 is consequently a map of model behavior, not an empirical classification of tectonic and fluid-induced sequences. Within the model, increasing γ steepens the rate and decreasing α reduces the geometric expansion term. Outside the model, neither trend is

unique. The Huc-Main audit in Fig. 4 demonstrates why similarly named fitted exponents cannot be combined unless their observables and conditioning are compatible with the closure.

If the assumptions are independently supported, the equation may be inverted to define a conditional effective parameter

$$\gamma_{\text{eff}} = p_{\text{obs}} - 1 + \frac{\alpha d}{2}, \quad (30)$$

but γ_{eff} is a diagnostic residual rather than a measurement of healing. A negative value falsifies the assumed nonincreasing susceptible fraction. A positive value becomes a healing estimate only after comparison with an independent recovery observable.

Further limitations are material. The model ignores rupture-size and fault-geometry distributions, treats the propagator as stationary, neglects poroelastic coupling and failure-permeability feedback, and assumes that transport and susceptibility are statistically independent. These conditions are not satisfied during active hydraulic fracturing. More realistic extensions should derive susceptibility from a state or damage variable, include distributed rupture sizes, and compare predicted space-time rates with catalogs and coda-wave recovery measured on the same sequence.

V. CONCLUSIONS

I have derived a conditional scaling relation for a minimal activation-volume model. Its separate ingredients, anomalous transport and distributed power-law recovery, have clear precedents; the useful output is the joint constraint $p = 1 - \alpha d/2 + \gamma$. Reexamination of Huc and Main’s stacked earthquake-pair statistics shows that their slowly growing correlation length can serve as a qualitative spatial benchmark, but their cumulative spatial amplitude is not an Omori rate and cannot be used to infer γ . The relation is therefore best viewed as a falsifiable diagnostic and a baseline for richer models, not as a universal proxy relating an Omori exponent to fault or reservoir healing.

ACKNOWLEDGMENT

The author acknowledges support from the DyDéCo project and from the CPJ Chair at ENS de Lyon and CNRS.

DATA AVAILABILITY

There are no publicly available research data or software supporting this manuscript. Requests for further information or data should be sent to the authors.

-
- [1] F. Omori, On the after-shocks of earthquakes, *J. Coll. Sci. Imp. Univ. Tokyo* **7**, 111 (1894).
 - [2] T. Utsu, Y. Ogata, and R. S. Matsu’ura, The centenary of the Omori formula for a decay law of aftershock activity, *J. Phys. Earth* **43**, 1 (1995).
 - [3] G. Petrillo, F. P. Landes, E. Lippiello, and A. Rosso, The influence of the brittle-ductile transition zone on aftershock and foreshock occurrence, *Nat. Commun.* **11**, 3010 (2020).
 - [4] E. A. Jagla, F. P. Landes, and A. Rosso, Viscoelastic effects in avalanche dynamics: A key to earthquake statistics, *Phys. Rev. Lett.* **112**, 174301 (2014).
 - [5] J. Dieterich, A constitutive law for rate of earthquake production and its application to earthquake clustering, *J. Geophys. Res.: Solid Earth* **99**, 2601 (1994).
 - [6] A. Saichev and D. Sornette, Superlinear scaling of offspring at criticality in branching processes, *Phys. Rev. E* **89**, 012104 (2014).
 - [7] I. Spassiani, G. Petrillo, and J. Zhuang, Distribution related to all samples and extreme events in the ETAS cluster, *Seismol. Res. Lett.* **95**, 3234 (2024).

- [8] G. Petrillo, I. Spassiani, and J. Zhuang, A full space–time–magnitude extreme-event distribution for an ETAS cluster, *Seismol. Res. Lett.* **97**, 2925 (2026).
- [9] Y. Ogata, Statistical models for earthquake occurrences and residual analysis for point processes, *J. Am. Stat. Assoc.* **83**, 9 (1988).
- [10] Y. Ogata, Space-time point-process models for earthquake occurrences, *Ann. Inst. Stat. Math.* **50**, 379 (1998).
- [11] G. Petrillo and E. Lippiello, Testing of the foreshock hypothesis within an epidemic like description of seismicity, *Geophys. J. Int.* **225**, 1236 (2021).
- [12] G. Petrillo and M. Taroni, Adding strain rate information into a short-term seismicity model improves forecasting performances: The case of Campi Flegrei, Italy, *Seismica* **4** (2025).
- [13] A. Helmstetter and D. Sornette, Diffusion of epicenters of earthquake aftershocks, Omori’s law, and generalized continuous-time random walk models, *Phys. Rev. E* **66**, 061104 (2002).
- [14] M. Huc and I. G. Main, Anomalous stress diffusion in earthquake triggering: Correlation length, time dependence, and directionality, *J. Geophys. Res.: Solid Earth* **108**, 2001JB001645 (2003).
- [15] G. Michas and F. Vallianatos, Modelling earthquake diffusion as a continuous-time random walk with fractional kinetics: The case of the 2001 Agios Ioannis earthquake swarm (Corinth Rift), *Geophys. J. Int.* **215**, 333 (2018).
- [16] G. Michas and F. Vallianatos, Stochastic modeling of injection induced seismicity based on the continuous time random walk model, *Sci. Rep.* **14**, 4951 (2024).
- [17] E. Lippiello, G. Petrillo, F. Landes, and A. Rosso, Fault heterogeneity and the connection between aftershocks and afterslip, *Bull. Seismol. Soc. Am.* **109**, 1156 (2019).
- [18] G. Petrillo, The role of disorder in foreshock activity, *Geosciences* **15**, 226 (2025).
- [19] P. Dublanchet, P. Bernard, and P. Favreau, Creep modulation of Omori law generated by a Coulomb stress perturbation in a 3-D rate-and-state asperity model, *J. Geophys. Res.: Solid Earth* **118**, 4774 (2013).
- [20] X. Li, I. G. Main, and A. Jupe, Induced seismicity at the UK “hot dry rock” test site for geothermal energy production, *Geophys. J. Int.* **214**, 331 (2018).
- [21] T. H. W. Goebel and E. E. Brodsky, The spatial footprint of injection wells in a global compilation of induced earthquake sequences, *Science* **361**, 899 (2018).
- [22] B. Maillot, S. Nielsen, and I. G. Main, Numerical simulation of seismicity due to fluid injection in a brittle poro-elastic medium, *Geophys. J. Int.* **139**, 263 (1999).
- [23] S. A. Miller, Aftershocks are fluid-driven and decay rates controlled by permeability dynamics, *Nat. Commun.* **11**, 5787 (2020).
- [24] O. Khajehdehi, K. Karimi, and J. Davidsen, The effect of correlated permeability on fluid-induced seismicity, *Geophys. Res. Lett.* **49**, e2021GL095199 (2022).
- [25] N. Igonin, J. P. Verdon, J.-M. Kendall, and D. W. Eaton, Large-scale fracture systems are permeable pathways for fault activation during hydraulic fracturing, *J. Geophys. Res.: Solid Earth* **126**, e2020JB020311 (2021).
- [26] R. Schultz, R. J. Skoumal, M. R. Brudzinski, D. Eaton, B. Baptie, and W. Ellsworth, Hydraulic fracturing-induced seismicity, *Rev. Geophys.* **58**, e2019RG000695 (2020).
- [27] X. Bao and D. W. Eaton, Fault activation by hydraulic fracturing in western Canada, *Science* **354**, 1406 (2016).
- [28] K. Karimi and J. Davidsen, Aftershock triggering and spatial aftershock zones in fluid-driven settings: Discriminating induced seismicity from natural swarms, *Geophys. Res. Lett.* **48**, e2020GL092267 (2021).
- [29] O. Khajehdehi and J. Davidsen, On the potential role of viscoelasticity in fluid-induced seismicity, *J. Geophys. Res.: Solid Earth* **128**, e2023JB027349 (2023).
- [30] G. Rodríguez-Pradilla, D. W. Eaton, and J. P. Verdon, Basin-scale multi-decadal analysis of hydraulic fracturing and seismicity in western Canada shows non-recurrence of induced runaway fault rupture, *Sci. Rep.* **12**, 14463 (2022).
- [31] A. M. Farahbod and H. Kao, Aftershock decay rate of large injection-induced earthquakes in northeast British Columbia: A case study for two sequences in the Montney Play, *CSEG Rec.* **44**, 1 (2019).
- [32] C. Langenbruch and S. A. Shapiro, Decay rate of fluid-induced seismicity after termination of reservoir stimulations, *Geophysics* **75**, MA53 (2010).

- [33] Y. Kawada, H. Nagahama, and H. Hara, Irreversible thermodynamic and viscoelastic model for power-law relaxation and attenuation of rocks, [Tectonophysics](#) **427**, 255 (2006).
- [34] R. Snieder, C. Sens-Schönfelder, and R. Wu, The time dependence of rock healing as a universal relaxation process, a tutorial, [Geophys. J. Int.](#) **208**, 1 (2017).
- [35] F. Brenguier, M. Campillo, C. Hadziioannou, N. M. Shapiro, R. M. Nadeau, and E. Larose, Postseismic relaxation along the San Andreas fault at Parkfield from continuous seismological observations, [Science](#) **321**, 1478 (2008).
- [36] E. W. Montroll and G. H. Weiss, Random walks on lattices. II, [J. Math. Phys.](#) **6**, 167 (1965).
- [37] R. Metzler and J. Klafter, The random walk's guide to anomalous diffusion: A fractional dynamics approach, [Phys. Rep.](#) **339**, 1 (2000).
- [38] B. Berkowitz, Characterizing flow and transport in fractured geological media: A review, [Adv. Water Resour.](#) **25**, 861 (2002).
- [39] For $f \in C^1$, ${}_0D_t^{1-\alpha} f(t) = \frac{1}{\Gamma(\alpha)} \frac{d}{dt} \int_0^t (t-t')^{\alpha-1} f(t') dt'$.
- [40] F. Mainardi, *Fractional Calculus and Waves in Linear Viscoelasticity: An Introduction to Mathematical Models*, 2nd ed. (World Scientific, 2022).
- [41] S. A. Shapiro, C. Dinske, and J. Kummerow, Probability of a given-magnitude earthquake induced by a fluid injection, [Geophys. Res. Lett.](#) **34**, 2007GL031615 (2007).
- [42] J.-P. Bouchaud, Weak ergodicity breaking and aging in disordered systems, [J. Phys. I](#) **2**, 1705 (1992).