Universal Scaling Law in Human Behavioral Organization

Toru Nakamura,[1] Ken Kiyono,[2] Kazuhiro Yoshiuchi,[3] Rika Nakahara,[4] Zbigniew R. Struzik,[5] and Yoshiharu Yamamoto[5,*]

[1]*The Center for Advanced Medical Engineering and Informatics, Osaka University, 1–3 Machikaneyama, Toyonaka-shi, Osaka 560–8531, Japan*

[2]*College of Engineering, Nihon University, 1 Naka-gawara, Tokusada, Tamura-machi, Koriyama City, Fukushima, 963–8642, Japan*

[3]*Department of Psychosomatic Medicine, Graduate School of Medicine, The University of Tokyo, 7–3–1 Hongo, Bunkyo-ku, Tokyo 113–8655, Japan*

[4]*Department of Psychiatry, Teikyo University Mizonokuchi Hospital, 3–8–3 Mizonokuchi, Takatsu-ku, Kawasaki, Kanagawa 213–8507, Japan*

[5]*Educational Physiology Laboratory, Graduate School of Education, The University of Tokyo, 7–3–1 Hongo, Bunkyo-ku, Tokyo 113–0033, Japan*

(Received 29 April 2007; published 28 September 2007)

We describe the nature of human behavioral organization, specifically how resting and active periods are interwoven throughout daily life. Active period durations with physical activity count successively above a predefined threshold, when rescaled with individual means, follow a universal stretched exponential (gamma-type) cumulative distribution with characteristic time, both in healthy individuals and in patients with major depressive disorder. On the other hand, resting period durations below the threshold for both groups obey a scale-free power-law cumulative distribution over two decades, with significantly lower scaling exponents in the patients. We thus find universal distribution laws governing human behavioral organization, with a parameter altered in depression.

DOI: 10.1103/PhysRevLett.99.138103 PACS numbers: 87.19.St, 05.40.−a, 89.75.Da

Universality in waiting time distributions is abundant in a wide range of complex systems: e.g., earthquake occurrences [1,2], human communication [3,4], and in neuronal avalanches [5], where it has been associated with critical branching processes [6] and systems showing (on-off) intermittent dynamics [7].

In models of human dynamics at a macroscopic level of interhuman communication, a phenomenological priority concept has been suggested [3,4] to account for the non-Poisson statistics and universal scaling. Yet, the origins of the universality and the emergence of an apparent *physical law* of human communication dynamics are, to date, not evident. It is intriguing to observe that waiting time distributions at microscopic scales of neuronal avalanches in neurocortical circuits [5] exhibit universality of an analogical class to that of decision making based human communication dynamics, successfully modeled by a critical branching process [6]. However, a hierarchical distance in these multiscale human dynamics between the local neuronal circuits and social interactions is large.

In this Letter, we reveal an analogical universality class in human behavior at an intermediate scale. By studying quantitatively how periods of lower levels of activity are interwoven with those of higher levels of activity throughout daily life, we identify the underlying *criticality* signature of human behavioral organization governing the onset and termination of movement and its alterations in clinical depression [8].

We analyze so-called locomotor activity data in humans, capturing even slight bodily acceleration counts in a continuous fashion (Fig. 1) and report a robust statistical law governing the temporal organization of periods of lower levels of activity (*resting* periods) and higher levels of activity (*active* periods). We find that the cumulative probability distribution of resting periods with lower levels of activity takes a power-law form over two decades, while the cumulative distribution of active periods with higher activity levels takes a stretched exponential form. Surprisingly, these statistical laws, after being rescaled by the mean waiting times, are not affected by altering the threshold value to calculate the waiting times. They share the distribution form for individuals considerably different in their daily living, and regardless of whether healthy or suffering from major depression, therefore suggesting the presence of *universal* laws governing human behavioral organization.

Locomotor activity data, defined as counts of events in which an acceleration signal crosses zero-level within a predefined time, were acquired from 14 patients with major depressive disorder (age: 32.9 ± 5.9 years; mean $\pm$ S.D.) and 11 age-matched healthy control subjects (age: 32.7 ± 15.7 years) [11]. Zero-crossing counts were accumulated for every 1 min, both in the patients and in the healthy controls. The activity sensors have the capability of detecting small changes in wrist acceleration (up to $0.01g$) so that even slight movements of the subjects, ranging from, e.g., writing or working on a computer to physical exercise, are registered.

Examples of locomotor activity data for a control subject and a depression patient over 5 consecutive days (Fig. 1) show a clear circadian rest-activity cycle in the control subject, while in the patient, such a rhythmic pattern is much disrupted, reflecting the reported chronobiological abnormality in depression [15–17]. During the

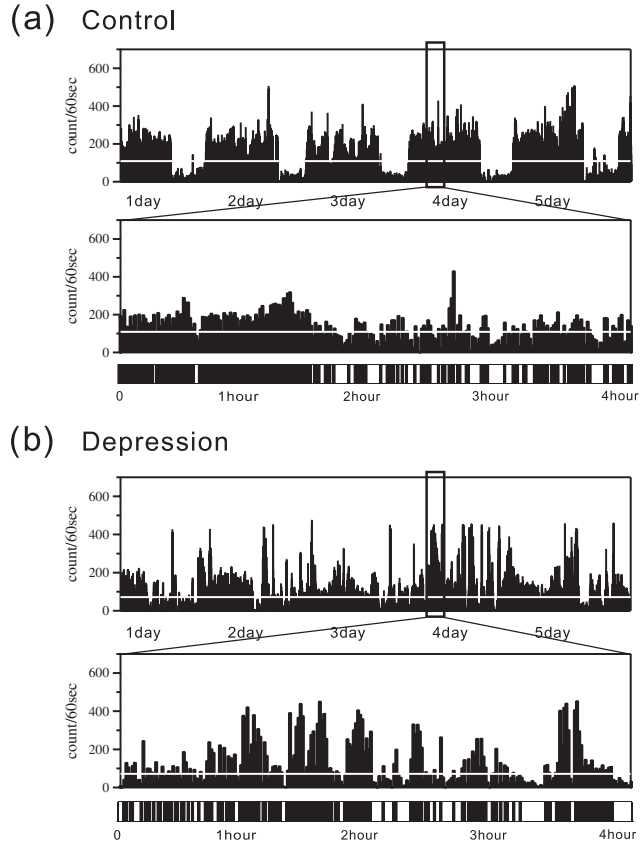

FIG. 1. Illustrative examples of locomotor activity data for a healthy control subject (a) and a depression patient (b) over five consecutive days (upper) and 4-hour periods during the 4th day (lower). The overall average of nonzero activity counts is used for a threshold, and the period in which the counts are successively below or above the threshold is coded as a resting (open bar in bottom panels) or active (closed bar) period, respectively.

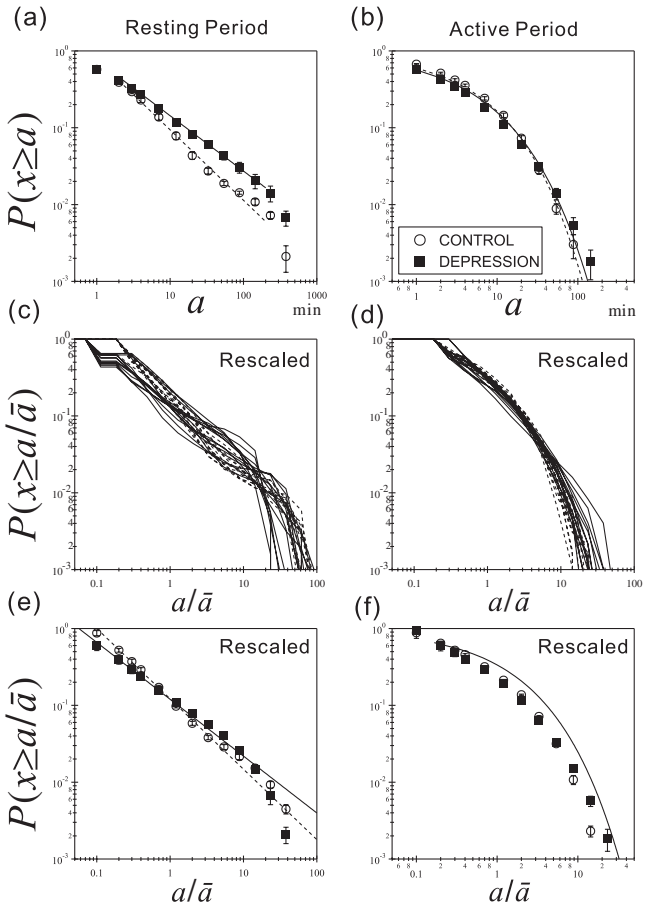

FIG. 2. Cumulative distributions of resting and active period durations in human locomotor activity. (a) Double logarithmic plots of cumulative distributions $P(x \geq a)$ of resting period durations a for control subjects (open circles) and for depression patients (filled squares). The error bars indicate the standard error of the mean. The straight lines are eye-guides with the mean scaling exponents from 2 min to 200 min for each group as the slopes. (b) The same as (a) but for active period durations. The curves are stretched exponential functions $P(x \geq a) = \exp(-\alpha a^{\beta})$ with the mean α and β values for each group. (c) Rescaled cumulative distributions of resting period durations with the rescaled time $a/\bar{a}$, where $\bar{a}$ is the individual mean of the resting period durations, for both healthy subjects (dotted lines) and patients with major depression (solid lines). (d) The same as (c) but for active period durations. (e) Group average rescaled cumulative distributions of resting period durations for controls (open circles) and depression (filled squares). The straight lines are eye-guides with the mean $\bar{\gamma}$ for each group as the slopes. (f) The same as (e) but for active period durations. The curve is a stretched exponential function with the overall mean $\bar{\alpha}$ and $\bar{\beta}$ values for both groups.

daytime, locomotor activity of the depression patient also shows intermittent bursts in the activity counts, while the data for the control subject are characterized by more sustained activity levels. To evaluate such an intermittency over the whole of the recording periods, we estimate the cumulative distribution $P(x \geq a)$ of durations a of both resting periods, where the activity counts are successively lower than a certain predefined threshold value, and of active periods, where the counts are successively higher than the threshold. We first describe the results when an overall average of nonzero activity counts is used as the threshold. An evaluation of the effects of the threshold on $P(x \geq a)$ follows.

The averaged cumulative distribution of resting period durations for depression patients is higher than that for control subjects [Fig. 2(a)], especially at longer times, implying more frequent episodes of longer resting periods in the patients; this suggests more episodes of slowing down or cessation of movement in the patients. The resting period distributions for healthy controls and depression patients take a power-law form $P(x \geq a) \sim a^{-\gamma}$ over more than two decades (from 2 min to 200 min) [18], with significantly ($p < 0.0001$) [19] different scaling exponents of $\gamma = 0.92 \pm 0.06$ for controls and $\gamma = 0.72 \pm 0.11$ for depression. This is associated with a significantly ($p < 0.001$) longer mean resting period duration in the patients (15.64 ± 6.19 min) than in the control subjects (7.72 ± 1.44 min). When the time a is rescaled with the individual mean of the resting period durations [Fig. 2(c)],

both the patients and the control subjects exhibit similar individual power-law distribution forms, except for the lower and higher ends of the waiting time, with the slope consistently greater in the control subjects, as reflected in the group-averaged, rescaled cumulative distributions [Fig. 2(e)]. This results in a significant ($p < 0.0001$) between-group difference in the scaling exponents for rescaled a ranging from 0.1 to 10.0: $\bar{\gamma} = 0.92 \pm 0.08$ for controls and $\bar{\gamma} = 0.74 \pm 0.12$ for depression.

On the other hand, active period durations for both controls and depression patients obey a stretched exponential functional form rather than a power-law distribution [Fig. 2(b), 2(d), and 2(f)]. The cumulative distribution well fits $P(x \geq a) = \exp(-\alpha a^{\beta})$ for a wide range of times, without significant ($p > 0.01$) between-group differences in their parameters (α; 0.41 ± 0.08 for controls and 0.53 ± 0.11 for depression, β; 0.61 ± 0.07 for controls and 0.53 ± 0.07 for depression). The rescaled cumulative distributions also collapse onto a stretched exponential function [Fig. 2(d) and 2(f)], with the overall $\bar{\alpha}$ and $\bar{\beta}$ values of 1.45 ± 0.11 and 0.54 ± 0.07, respectively. These parameters are not significantly ($p > 0.05$) correlated with the power-law exponent $\bar{\gamma}$ both for controls and depression, suggesting the independency of laws governing burst durations (active periods) and interburst intervals (resting periods).

The mean resting and active period durations are, by definition, influenced by the choice of threshold values; the higher the threshold, the longer the mean resting periods and the shorter the active periods. However, the shapes of the rescaled, cumulative distributions are not altered by choosing from a wide range of threshold values (Fig. 3). In Fig. 3, we test this for different threshold levels ranging from 0.6 to 1.6 times the overall nonzero activity counts used above.

Recently, the intermittent and/or heavy-tailed nature of human decision making activities has been attracting significant scientific interest. For instance, Barabási *et al.* [3,4] developed a model based on a queuing theory, successfully accounting for the heavy-tailed distribution of waiting time statistics observed in email communications, Web browsing, and trade transactions. In their model, humans are presented with multiple tasks and choose [with the probability of $\Pi(x) \sim x^{\lambda}$] between them based on some priority levels (x's) in order to decide the timing of the task execution. The waiting time probability distribution of this model, in the $\lambda \to \infty$ limit, predicts a universal class of $P(\tau) \sim \tau^{-1}$ observed for the various empirical data.

In our case of human locomotor activity, the exponent $\gamma = 0.92$ of the *cumulative* distribution of waiting times of the activity bursts (the resting period durations) for healthy subjects corresponds to $P(\tau) = \tau^{-1.92}$, a special case of Barabási's model [3] where the λ is close to unity and the task priorities are given randomly. Thus, it can be argued that the healthy subjects execute their (even slight) physical activity with the probability [$\Pi(x)$] proportional to a randomly given priority x, or a physiological demand, to initiate their actions. Further, the exponent $\gamma = 0.72$ for depression patients, compatible with much greater λ than unity [3], implies that the patients tend to react only to higher physiological demands [because of the $\Pi(x) \sim x^{\lambda}$ nature].

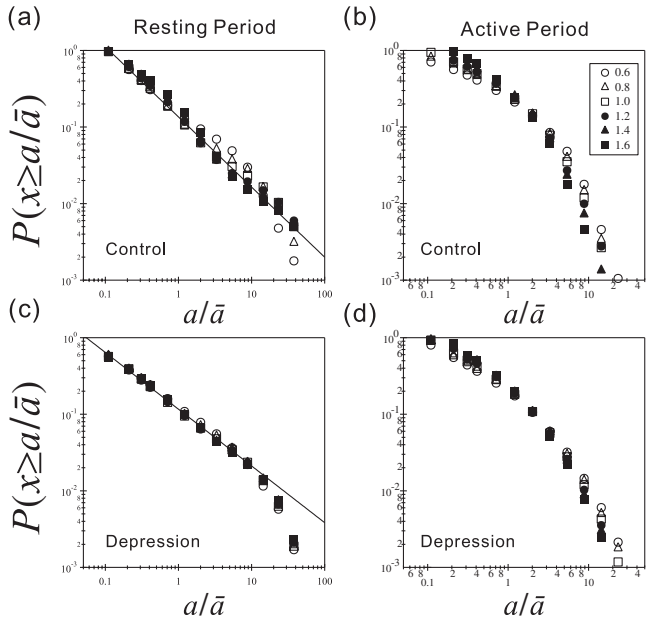

FIG. 3. Dependency of cumulative distribution of locomotor activity on the threshold to determine resting and active periods. (a) Double logarithmic plots of rescaled (by the mean resting duration $\bar{a}$) cumulative distributions $P(x \geq a/\bar{a})$ for control subjects with different threshold values of 0.6, 0.8, 1.0, 1.2, 1.4, and 1.6 times the overall average for nonzero activity counts. The straight line is an eye-guide with the mean $\bar{\gamma}$ for the control subjects as the slope. (b)–(d) The same as (a), but of active period durations (b), of resting periods for depression patients (c), and of active periods for depression patients.

An alternative hypothesis for the explanation of the above $P(\tau) \sim \tau^{-2}$ law of waiting time statistics in healthy subjects may involve the underlying avalanche type propagation of neural activity signaling onsets of activity by the subject. An analogical scaling law has been observed experimentally in living neural networks at criticality [5] and has been suggested theoretically in a critical branching process [6] as a model of neural avalanches. Indeed, we find that the *cumulative* distribution of burst size $P(x \geq S)$, where S is the total counts for active periods, for healthy controls takes a power-law form $P(x \geq S) \sim S^{-0.5}$ (Fig. 4) [20], which is compatible with the $P(S) \sim S^{-1.5}$ law in the neural avalanches and the critical branching model [5,6]. The time scale of human activity fluctuations is much longer than that of the neural bursts in the experiments and models above. Yet, the possibility of explaining the $P(\tau) \sim \tau^{-2}$ in distribution by the underlying neural criticality in healthy individuals and the apparent dissociation from this law in depression patients seems attractive and worth further attention.

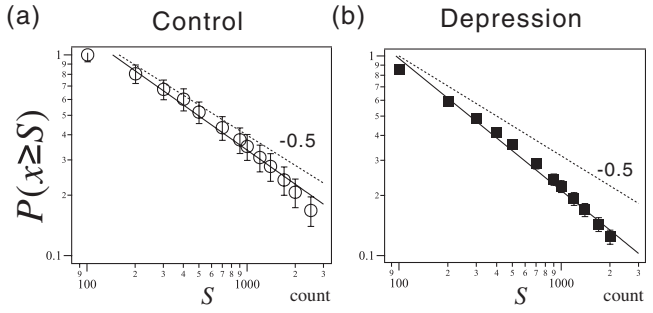

FIG. 4. Cumulative distributions of the burst size (S) for active periods. The threshold is the same as that used in Fig. 2. (a) Double logarithmic plots of cumulative distributions $P(x \geq S)$ for control subjects, with the solid regression line for [200, 2000] counts. The error bars indicate the standard error of the mean. The dotted line is an eye-guide with the slope of -0.5. (b) The same as (a), but for depression patients.

The active period durations of both healthy subjects and depression patients show the gamma-type ($\beta \sim 0.5$) stretched exponential distribution. It has been demonstrated that the products of a finite number of random variables lead to a stretched exponential distribution, $p(x) = \exp(-\alpha x^{\beta})$, where the exponent β is an inverse of the number of products in the multiplicative process [21]. Thus, it can be argued that the active periods are terminated based on a joint probability of *two* (i.e., about $1/\beta$) factors such as, e.g., less vigor and more fatigue.

In summary, we find an underlying robust law governing behavioral organization of humans. Specifically, the cumulative distribution of resting periods with lower levels of activity, or waiting times for active (burst) periods, shows the intermittent and/or heavy-tailed nature, of which the underlying mechanism is possibly shared by a wide range of human dynamics with heavy-tailed distributions of waiting time statistics. Importantly, we further demonstrate here that a parameter for the power-law distribution function of resting period durations, i.e., the scaling exponent γ, is significantly smaller in patients with major depression than in healthy control subjects. The finding of the alteration of a parameter of the robust law governing the organization of resting periods may form the basis for providing a quantitative index to characterize the severity of depressive disorders in clinical settings. We also believe that an understanding of the neurobiological basis for complex human behavior and social interactions will be facilitated by identifying the underlying multiscale mechanism of the heavy-tailed distribution of waiting times for active periods, which we show to be shared by that of both the local neuronal circuits and that of the social interactions.

*yamamoto@p.u-tokyo.ac.jp

[1] A. Corral, Phys. Rev. Lett. **92**, 108501 (2004).
[2] E. Lippiello, L. de Arcangelis, and C. Godano, Europhys. Lett. **76**, 979 (2006).
[3] A. L. Barabási, Nature (London) **435**, 207 (2005).
[4] A. Vázquez, J. G. Oliveira, Z. Dezsö, K. I. Goh, I. Kondor, and A. L. Barabási, Phys. Rev. E **73**, 036127 (2006).
[5] J. M. Beggs and D. Plenz, J. Neurosci. **23**, 11167 (2003).
[6] S. Zapperi, K. B. Lauritsen, and H. E. Stanley, Phys. Rev. Lett. **75**, 4071 (1995).
[7] J. F. Heagy, N. Platt, and S. M. Hammel, Phys. Rev. E **49**, 1140 (1994).
[8] Alteration in locomotor activity is one of the cardinal signs in psychiatric disorders, and many psychiatric disorders, including depression, indeed have diagnostic criteria which require an assessment of altered locomotor activity [9]. For instance, one of the known disease signs in depression is psychomotor retardation, involving a recognizable alteration in locomotor activity such as slowing down of movement, which is observable by others [10].
[9] M. H. Teicher, Harv. Rev. Psychiatr. **3**, 18 (1995).
[10] American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Press, Washington, D.C., 2000), 4th ed., Text Revision.
[11] All the patients were outpatients of the Teikyo University Mizonokuchi Hospital, gave their institutionally approved, informed, written consent, and were diagnosed according to DSM-IV [10]. The severity of depression of the patients was mild to moderate with an average Hamilton depression rating scale [12] of 13.3 ± 2.93 (range: 8.8–18.5). All the patients wore a small watch-type activity monitor equipped with a computer (Ruputer Pro, Seiko Instruments, Chiba) [13] on the wrist of their nondominant hand for at least 18 days (28.1 ± 7.4 days; range: 18 to 42 days). The activity monitoring device is analogous in performance [14] to the commercial Actigraph Mini-Motionlogger (Ambulatory Monitors Inc., Ardsley, NY) [9], which is widely used in clinical fields. All the healthy controls wore the Actigraph Mini-Motionlogger on the wrist of their nondominant hand for seven days.
[12] M. Hamilton, J. Neurol. Neurosurg Psychiatry **23**, 56 (1960).
[13] Y. Yamamoto, Z. R. Struzik, R. Soma, K. Ohashi, and S. Kwak, Ann. Neurol. **58**, 175 (2005).
[14] W. Pan, K. Ohashi, Y. Yamamoto, and S. Kwak, Movement disorders : official Journal of the Movement Disorder Society **22**, 1308 (2007).
[15] L. J. Siever and K. L. Davis, Am. J. Psychiatr. **142**, 1017 (1985).
[16] H. Schulz and R. Lund, Psychiatry Res. **16**, 65 (1985).
[17] M. H. Teicher, J. M. Lawrence, N. I. Barber, S. P. Finklestein, H. R. Lieberman, and R. J. Baldessarini, Arch. Gen. Psychiatry **45**, 913 (1988).
[18] A similar type of temporal, as well as spatial, power-law was reported for rats' motor activity [M. P. Paulus and M. A. Geyer, Psychopharmacology (Berl.) **104**, 6 (1991)].
[19] The Kolmogorov-Smirnov test is used throughout.
[20] The exponent for [200, 2000] counts is -0.56 ± 0.05 for healthy subjects and -0.66 ± 0.11 for depression patients. The upper limit of 2000 counts roughly corresponds to the product of mean active period durations (6.60 ± 0.97 min for controls and 6.49 ± 1.47 min for depression) and a typical upper limit of the monitoring devices (300 counts/min) [9].
[21] D. Sornette, *Critical Phenomena in Natural Sciences: Chaos, Fractals, Self-organization and Disorder: Concepts and Tools* (Springer, Berlin, 1994), 2nd ed.