Control of human activity is complex, being influenced by many factors both extrinsic (work, recreation, reactions to unforeseen random events) and intrinsic (the circadian pacemaker that influences our sleep/wake cycle [1] and ultradian oscillators with shorter time scales [2]). The extrinsic factors may account for the apparently random fluctuations in human motion observed over short time scales while the intrinsic rhythms may account for the underlying regularity in average activity level over longer periods of up to 24 h. Further, human activity correlates with important physiological functions including whole body oxygen consumption and heart rate.

Actiwatch devices [3] are traditionally used to demarcate sleep versus wakefulness based on average activity levels, or to observe the mean pattern of activity as it changes across the day and night according to disease state (Fig. 1). Traditionally activity fluctuations are considered as random noise and have been ignored. We hypothesize that there are systematic patterns in the activity fluctuations that may be independent of known extrinsic and intrinsic factors.

To test our hypotheses, we evaluate the structure of human activity during wakefulness, using: (i) probability distribution analysis; (ii) power spectrum analysis, and (iii) fractal scaling and nonlinear analysis. To elucidate the presence of an intrinsic activity control center independent of known circadian, ultradian, scheduled and random factors, we apply 3 complementary protocols.

- (A) Daily routine protocol: We record activity data throughout two consecutive weeks in 16 healthy ambulatory domiciliary subjects (8 males, 8 females, 19-44 years, mean 27 years) performing their routine daily activities. The only imposed constraints are that subjects go to bed and arise at the same time each day (8 h sleep opportunity) and that they are not permitted to have daytime naps (Fig. 1).
- (B) Constant routine protocol: To assess intrinsic activity controllers (i.e. circadian or other neural centers) independent of scheduled and random external influences activity recordings are made in the laboratory throughout 38 h of constant posture (semi-recumbent), wakefulness, environment (21°C, dim light [< 8 lux]), dietary intake and scheduled events [4].

This protocol is performed in a subset of subjects (7 males, 4 females) that participated in the daily routine protocol. These highly controlled and constant experimental conditions result in reduced average and variance of activity levels.

- (C) Forced desynchrony protocol: To test for the presence of heretofore unidentified intrinsic activity control centers, independent of known activity regulators (circadian pacemaker), whilst accounting for scheduled and random external influences, we employ the validated Forced desynchrony (FD) protocol [1]. Six (4 male, 2 female) of the 16 subjects that participated in the daily routine protocol completed the FD limb of the study. For eight days subjects remain in constant dim light (to avoid “resetting” the body clock). Sleep periods are delayed by 4 h every day, such that subjects live on recurring 28 h “days”, while all scheduled activities become desynchronized from the endogenous circadian pacemaker. Thus, as measurements occur across all phases of the circadian clock, the effect of intrinsic circadian influences can be removed [1]. Average activity level and activity variance are also significantly reduced due to laboratory-imposed restrictions on the subjects activity (Fig. 1).

When the same subject is studied in different protocols, we find large differences in the probability distributions (Fig. 2). For example, during wakefulness greater values of activity occur most frequently during the daily routine, intermediate activity values occur during the forced desynchrony, and the highest frequency of low activity values is seen during the constant routine (Fig. 2a). Indeed, the largest activity values encountered during the constant routine protocol are approximately two orders of magnitude less frequent than similar activity values encountered in the daily routine protocol. We also find major differences between individuals in the distribution of activity values during the daily routine protocol (Fig. 2b). Such differences are expected given their different daily schedules, environments, and reactions to random events. However, by appropriately rescaling the distributions of activity values on both axes to account for differences in
FIG. 1: Independent contributors to the complex dynamics of human activity, depicted at the top of the figure, include: 1) reaction to extrinsic random events, 2) scheduled events, notably the endogenous circadian pacemaker which influences the sleep/wake cycle. Our findings of scale-invariant activity patterns (Figs. 2, 3) indicate a heretofore-unidentified intrinsic multi-scale control of human activity 4), which is independent of other extrinsic and intrinsic factors such as 1, 2, and 3. The second panel illustrates an actual one-week recording of human activity [3] during the daily routine protocol. Data structure highlights a 24-h sleep/wake periodic change in the mean activity — lowest during sleep (filled bars). The third panel, expanding a 16-h section of wakefulness, also shows patches of high and low average activity levels with apparent erratic fluctuations at various time scales. The bottom left panel is an activity recording from the same subject during the constant routine protocol with much lower average activity values compared to daily routine. The clear 2-h cycle is a result of scheduled laboratory events. The bottom right panel shows activity levels in the same subject during the forced desynchrony protocol. Of note here is the 28-h sleep/wake cycle as opposed to the 24-h rhythm in activity data during the daily routine.

average activity level and standard deviation [5], we find a remarkable similarity in the shapes of the probability distributions for all three protocols (Figs. 2c, 2e), and for all individuals when in the same protocol (Fig. 2d). The existence of a universal form of the probability distribution, independent of activity level in all individuals and in all protocols, suggests that a common underlying mechanism may account for the overall distribution of activity.

This probability distribution when plotted on a log-log scale reveals different characteristics above and below a distinct crossover point (Fig. 2e). At scales above the crossover activity level there is pronounced non-Gaussian tail (Fig. 2e). This tail on the log-log plot represents a power-law form, indicating an intrinsic self-similar structure for a range of activity values. Moreover, we find that the observed shape of the rescaled probability distribution remains unchanged when the data series are reanalyzed using a variety of observation windows ranging from 15 s to 6 min (Fig. 2f). This stability of the probability distribution over a range of time scales indicates that the underlying dynamic mechanisms controlling the activity have similar statistical properties on different time scales. Statistical self-similarity is a defining characteristic of fractal objects and is reminiscent of a wide class of physical systems with universal scaling properties. Our finding of a universal form of the probability distribution raises the possibility of an intrinsic mechanism that influences activity values in a self-similar “fractal” manner, that is unrelated to the individual’s daily and weekly schedules, reactions to the environment, the average level of activity, the phase of the circadian pacemaker, and the time scale of observation.

We next perform power spectral analyses for all three protocols to determine whether there exist any systematic intrinsic ultradian rhythms of activity with periods of less than 24 h duration [2, 6]. The data for each individual exhibit occasional peaks in the daily routine protocol for periods ranging from 30
FIG. 3: Power spectrum and correlation analyses of activity data during wakefulness. (a) Group average power spectral densities for all three protocols. For better clarity and to avoid overlap, curves are vertically offset. To present graphs on a common x-axis, power spectra are shown with decreasing frequency from left to right. The activity values for each scheduled behavior imposed throughout two separate weeks of daily routine, 38 h of constant routine, and 8 days of forced desynchrony protocols. (b) DFA scaling of activity for an individual during wakefulness throughout two separate weeks of daily routine, 38 h of constant routine, and 8 days of forced desynchrony protocols. (c) DFA scaling of the magnitude series of activity increments for the same signals as in (a). A scaling exponent \( \alpha_{mag} \approx 0.8 \) of similar value is observed for all three protocols. (d) Scaling exponents \( \alpha \) and \( \alpha_{mag} \) (left scale), and average activity levels (right scale) for all 16 subjects obtained from a 14-day daily routine protocol. Although the average activity level between subjects changes considerably (from 0.2 to 0.5), both scaling exponents are consistent for all subjects, exhibiting a group average of \( \alpha = 0.92 \pm 0.05 \) and \( \alpha_{mag} = 0.77 \pm 0.05 \). min to 4 h. However, we find no systematic ultradian rhythms within individuals from week to week, and no systematic ultradian rhythms in the group average for the daily routine protocol (Fig. 3a). The only systematic rhythms that are ostensibly in the ultradian range which emerge in the group data are at 4 h during the forced desynchrony protocol (with harmonics at 2 h and 80 min) and at 2 h during the constant routine protocol (with harmonics at 1 h and 30 min) (Fig. 1 and Fig. 3a). These peaks are caused by the controlled scheduled activities in the laboratory and are extrinsic to the body as they also occur in simulated scheduled activity data that assumes specific activity values for each scheduled behavior imposed throughout the laboratory protocols (Fig. 3a). Thus, we find no evidence of systematic intrinsic ultradian rhythms in our data.

To provide further insight into the dynamic control of activity, we next examine the temporal organization in the fluctuations in activity values. We perform detrended fluctuation analysis (DFA) which quantifies correlations in the activity fluctuations after accounting for nonstationarity in the data by subtracting underlying polynomial trends. The DFA method quantifies the root mean square fluctuations, \( F(n) \), of a signal at different time scales \( n \). Power-law functional form, \( F(n) \sim n^\alpha \), indicates self-similarity (fractal scaling). The parameter \( \alpha \), called the scaling exponent, quantifies the correlation properties in the signal: if \( \alpha = 0.5 \), there is no correlation (random noise); if \( \alpha < 0.5 \), the signal is anticoordinated, where large activity values are more likely to be followed by small activity values; if \( \alpha > 0.5 \), there are positive correlations, where large activity values are more likely to be followed by large activity values (and vice versa for small activity values).

Figure 3b shows that \( F(n) \) for a typical subject during wakefulness exhibits a power-law form over time scales from \( \approx 1 \) min to \( \approx 4 \) h. We find that the scaling exponent \( \alpha \) is virtually identical for records obtained during the first week of daily routine (\( \alpha = 0.92 \pm 0.04 \), mean \( \pm \) standard deviation among subjects), the second week (\( \alpha = 0.92 \pm 0.06 \)) of the daily routine, the constant routine protocol (\( \alpha = 0.88 \pm 0.05 \)), and the forced desynchrony protocol (\( \alpha = 0.92 \pm 0.03 \)). The value of \( \alpha \approx 0.9 \) for all protocols and all individuals indicates that activity fluctuations are characterized by strong long-range positive correlations. Furthermore, we find that this scaling behavior is not caused by the scheduled activities because simulated scheduled activity data that are generated by assigning a specific activity value for each scheduled event throughout the laboratory protocols yields an exponent of \( \alpha = 1.5 \) (Fig. 3b), which represents random-walk type behavior. These results suggest that the activity fluctuations are not a consequence of random events (in which case \( \alpha \) would be 0.5) or scheduled events, but rather relate to an underlying mechanism of activity control with stable fractal-like features over a wide range of time scales from minutes to hours. Since mean activity levels and the amplitude of the fluctuations are greatly reduced in the laboratory during the constant routine and forced desynchrony protocols (Fig. 1), we obtain smaller values of \( F(n) \) (downward shift of the lines in Fig. 3b). However, there is no change in the scaling exponent \( \alpha \). Similarly,
the scaling exponents for the daily routine protocol are independent of the average activity levels of the different subjects (Fig. 3d), and of the mean activity level on different days of the week, indicating that this newly-found scaling pattern of activity appears to be an intrinsic feature.

To test for the presence of nonlinear properties of the data, we analyze the “magnitude series” formed by taking the absolute values of the increments between consecutive activity values [7]. Again, from detrended fluctuation analysis of this series, we find practically identical scaling exponents, $\alpha_{\text{mag}}$, for all three protocols, despite large differences in mean activity levels between protocols (Fig 3c). Moreover, all individuals have very similar values of the scaling exponent $\alpha_{\text{mag}}$ (Fig. 3d), which are not systematically changed by the protocol. For the group, during the first week of daily routine, we find $\alpha_{\text{mag}} = 0.78 \pm 0.06$ (mean ± standard deviation among subjects), during the second week $\alpha_{\text{mag}} = 0.76 \pm 0.05$, during the constant routine protocol $\alpha_{\text{mag}} = 0.82 \pm 0.05$, and during the forced desynchrony protocol $\alpha_{\text{mag}} = 0.80 \pm 0.04$. Since $\alpha_{\text{mag}} \approx 0.8 (> 0.5)$, there are positive long-range correlations in the magnitude series of activity increments, indicating the existence of nonlinear properties related to Fourier phase interactions (Fig. 3c) [7, 8]. To confirm that the observed positive correlations in the magnitude series indeed represent nonlinear features in the activity data, we do the following test: we generate a surrogate time series by performing a Fourier transform on the activity recording from the same subject during daily routine as in Fig.3b, preserving the amplitudes of the Fourier transform but randomizing the phases, and then performing an inverse Fourier transform. This procedure eliminates nonlinearities, preserving only the linear features of the original activity recording such as the power spectrum and correlations. Thus, the new surrogate signal has the same scaling behavior with $\alpha = 0.93$ (Fig. 3b) as the original activity recording; however, it exhibits uncorrelated behavior for the magnitude series ($\alpha_{\text{mag}} = 0.5$) (Fig. 3c). Our results show that the human activity data contains important phase correlations which are canceled in the surrogate signal by the randomization of the Fourier phases, and that these correlations do not exist in the simulated scheduled activity. Further, our tests indicate that these nonlinear features are related to Fourier phase interactions, suggesting an intrinsic nonlinear mechanism [8]. The similar value of $\alpha_{\text{mag}}$ for all three protocols and all individuals, which is different from $\alpha_{\text{mag}} = 0.5$ obtained for the simulated scheduled activity and for the phase randomized data, confirms that the intrinsic dynamics possess nonlinear features that are independent of the daily and weekly schedules, reaction to the environment, the average level of activity, and the phase of the circadian pacemaker.

The consistency of our results among individuals, and for different protocols, suggests that there exist previously unrecognized complex dynamic patterns of human activity that are unrelated to extrinsic factors or to the average level of activity. We also showed these patterns to be independent of known intrinsic factors related to the circadian and to any ultradian rhythms. Notably, (i) these patterns are unchanged when obtained at different phases of the circadian pacemaker; (ii) we do not observe systematic intrinsic ultradian rhythms in activity among subjects in the daily routine experiment; (iii) imposing strong extrinsic ultradian rhythms at 4 h and 2 h in the laboratory protocols did not change the fractal scaling exponents $\alpha$ or $\alpha_{\text{mag}}$ or the form of the probability distribution; and (iv) we find consistent results over a wide range of time scales. Together, these findings strongly suggest that our results are unlikely to be a reflection of the basic rest activity cycles or ultradian rhythms. We attribute these novel scale-invariant patterns to a robust intrinsic multi-scale mechanism of regulation (Fig. 1). This regulatory mechanism presents a new challenge to understand nonlinear control of human motor activity and pathways of interaction with other physiologic dynamics such as heart rate, gait [9], finger tapping [10], human sway and muscle fluctuations [11].

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