Non-Kolmogorov Turbulence and Inverse Energy Cascade in Intracranial Aneurysm: Near-Wall Scales Suggest Mechanobiological Relevance

Simon Tupin¹, Khalid M. Saqr¹‡, Sherif Rashad²,³, Kuniyasu Niizuma²,³,⁴, Makoto Ohta¹, Teiji Tominaga³

(1) Biomedical Flow Dynamics Laboratory, Institute of Fluid Science, Tohoku University, Sendai, 980-8577, Miyagi, JAPAN.
(2) Department of Neurosurgical Engineering and Translational Neuroscience, Tohoku University Graduate School of Medicine, Sendai, Miyagi, 980-8574, JAPAN.
(3) Department of Neurosurgery, Tohoku University Graduate School of Medicine, Sendai, Miyagi, 980-8574, JAPAN.
(4) Department of Neurosurgical Engineering and Translational Neuroscience, Graduate School of Biomedical Engineering, Tohoku University, Sendai, Japan.

*Corresponding author: Khalid M. Saqr, Ph.D.
Email: k.saqr@tohoku.ac.jp, kh.saqr@gmail.com
ABSTRACT

The genesis, growth and rupture of intracranial aneurysm (IA) are open questions in neurovascular medicine until the present moment. The complexity of aneurysm mechanobiology and pathobiology staggeringly combine intertwining biological and physical processes that are tightly connected. Recently, transition to turbulence in IA blood flow is thought to play a central role in IA growth and rupture as it can be directly linked to endothelial dysfunction. However, the problem of turbulence brings unprecedented complications to the topic. We found turbulence in IA to be of non-Kolmogorov type. For the first time, we detected inverse kinetic energy cascade in blood flow and in non-Kolmogorov turbulence. Here, we hypothesize that the near-wall turbulence undergoing inverse energy cascade have scales that could affect the mechano-signaling of endothelial cells. Our findings could be a paradigm shift in the contemporary theory of aneurysm hemodynamics.

Keywords: Aneurysm, Turbulence, non-Kolmogorov, Inverse energy cascade, Mechanobiology
BACKGROUND

Rupture of intracranial aneurysm (IA) is a major cause of nontraumatic subarachnoid hemorrhage (SAH). Aneurysm pathophysiology and mechanobiology are directly influenced by hemodynamics [1]. Experimental measurements[2] and numerical simulations[3-5] showed that blood flow in intracranial aneurysm (IA) exhibits transition to turbulence[6] and direct energy cascade[7]. Disturbed flow (i.e. transitional/turbulent) promotes proinflammatory and degenerative pathways in endothelial cells (ECs)[8]. Nevertheless, the exact flow parameters which influence different biological pathways and mechanisms remain unclear[9]. Computational Fluid Dynamics (CFD) contributed extensively to the hemodynamic theory of IA in the past three decades, however, resulted in numerous controversies and ambiguities. A complete account of the role of hemodynamics in IA pathobiology can be found in the recent review and meta-analysis by Saqr et al [10]. Some computational studies in the past few years pointed the importance of considering transition to turbulence in IA as a possible hemodynamic variable that could influence its growth and rupture [11-13]. However, there is no complete theory that correlates the features of IA transitional/turbulent flow to its pathophysiology and mechanobiology. In simple words, we know that the flow in IA is transitional/turbulent, however we do not know the characteristics of such flow and we do not know how such flow affects IA on cell and tissue levels. The present work sheds the light on these questions via experimental measurements.

Fluid turbulence is one of the everlasting mysteries of classical mechanics. Therefore, it is important to briefly revisit the theory of turbulence to appreciate the findings of the present work. Turbulence is chaotic oscillations of a flow field that result in vortex cascade phenomena. Richardson conceptualized the phenomena of vortex cascade in turbulent flows[14]. Kolomogorov formulated a statistical theory that describes the cascade of turbulence kinetic energy in homogenous isotropic turbulence[15]. Such theory dictates that kinetic energy transfers from large slow vortices to smaller and faster ones[16]. Then, two decade later, Kraichnan predicted the possibility of inverse energy cascade in two-dimensional Kolmogorov turbulence [17]. Kraichnan predictions were validated in later experimental [18, 19] and numerical [20] works and became an essential part of the statistical theory of turbulence. Then, inverse energy cascade, associated with isotropic homogenous turbulence, has been detected in geophysical flows [21-23] and most recently in Jupiter’s atmosphere[24]. However, until the present moment there is no evidence on the existence of inverse energy cascade in non-Kolmogorov turbulence nor in biological flows.
Despite the fact that transition to turbulence of simple monoharmonic pulsatile flow in straight pipes is not fully understood [25, 26], substantial evidence show that such flow could undergo transition to turbulence at $Re_m = \frac{\rho u_m D}{\mu} < 1000$ and $\alpha = R \sqrt{\frac{\omega \rho}{\mu}} < 3$ [27-29]. Blood flow is a multi-harmonic nonsynchronous oscillating flow with 95% of the frequency spectrum falling below 12 Hz[30]. In addition to blood’s waveform complexity, anatomical variations of arteries and aneurysm geometries dictates shear-driven vortex formation, shedding, precessing, and a multitude of complex flow physics that are yet to be unveiled[26, 31]. Direct numerical simulations of intracranial aneurysm hemodynamics showed that an intermittent direct energy cascade exists at $Re_m \sim 500$ and $\alpha \sim 4$ [7, 32]. The primary objective of this article is to prove the existence of non-Kolmogorov turbulence in pulsatile flow in an IA model at Reynolds number less than 400 and Womersley number less than 3. The secondary objectives are to characterize some of the features of such turbulence and propose a hypothesis to combine such features with the recent findings of endothelial cell response to local forces applied on the cell cytoskeleton. We have used well established methodology to conduct the Particle Image Velocimetry (PIV) measurements and analyze the results in frequency domain.

**MATERIALS METHODS**

An ideal ICA sidewall aneurysm geometry was fabricated as shown in figure 1. It was composed of a cylindrical artery of diameter $d = 4$ mm and a sphere of diameter $D = 10$ mm located at a distance of 6.25 mm [33, 34]. This geometry was used to mould $200 \times 50 \times 50$ mm³ box-type models made of silicone (R’Tech, Japan), considered as rigid, and 12 wt.% PVA-H (polyvinyl alcohol hydrogel), for greater compliance [35], following a manufacturing process previously described in detail [36, 37]. A pulsatile flow pump (Alpha Flow EC-1, Fuyo Corporation, Japan) was used to reproduce physiological flow conditions, as shown in figure 2, with a flow rate ranging between 200 and 300 mL/min [38], and pressure between 70 and 120 mmHg [39], with waveforms shown in figure (1-b). The waveform period was $T = 1$ s and the time to peak systole was 0.3 s. Coriolis flow-meters (FD-SS2A, Keyence, Japan) and pressure sensors (AP-12S, Keyence, Japan) were used to monitor instantaneous flow conditions at the inlet and outlet of the models. Fourier decomposition of the inlet flow rate waveform revealed the complex physiological flow reproduction composed of 9 harmonics (figure 1-c). A blood-mimicking fluid, made of a mixture of glycerin (42.8%), water (47.6%) and sodium iodide (9.6%), was used to approximate blood
kinematic viscosity \( \nu_f = 4.0 \times 10^{-6} \, \text{m}^2/\text{s} \) and density \( \rho_f = 1270 \, \text{Kg/m}^3 \), and match models refractive index \( n_f = 1.41 \). The oscillating mean Reynolds and Womersley numbers were calculated as following: 

\[
Re_{os} = \frac{\rho u d}{\mu}, \quad Re_m = \frac{\rho U m d}{\mu}, \quad \alpha = r \sqrt{\frac{\rho \omega d}{\mu}}.
\]

The inflow waveform used in our experiment was multiharmonic, constituted of nine harmonics of cardiac frequency spectra \( f_1 = 1 \, \text{Hz}, 2 \leq f_n \leq 9 \, \text{Hz} \) superimposed to the mean blood flow (figure 1-b and 1-c). The flow oscillating, mean Reynolds and Womerlsey numbers were \( 271.89 \leq Re_{os} \leq 397.88, Re_m = 313.45, \alpha = 2.51 \), respectivily. The experimental setup is schematically shown in figure 2. The Fourier coefficients of the Womerlsey flow are detailed in table 1.

**Figure 1. Aneurysm model geometry and inflow waveform with its Fourier decomposition.** (a) Schematic of the aneurysm model geometry and dimensions, (b) the inflow and pressure waveforms as measured at the inlet of the model, the phase difference between flow and pressure waves is a well-documented property of the Womersley solution of Navier-Stokes equation\(^1\) and is attributed to the flow inertia which is proportional to the inflow waveform frequency. (c) Fourier decomposition of the inflow harmonics showing the nine main oscillating components and the inset shows the complexity of the secondary harmonics \( n = 2:9 \) which results in the self-sustained oscillations.

**Figure 2. Schematic of the experimental and laser PIV measurement setup.** (a) the flow circuit used in the experiment consists of a closed loop comprising a pulsatile flow pump generates physiological flow, surge tank, upstream and downstream flowmeters and pressure transducers connected by fixed 4 mm inner-diameter tubes and the pressure is controlled by a check valve
downstream the aneurysm model. The tubes used were all rigid and the measurements were filtered to remove any system generated noise. (b) the laser PIV measurement setup showing the measurement plane and high-speed camera arrangement.

| n  | a       | phi     |
|----|---------|---------|
| q₀ | 0.236341|         |
| 1  | 0.043718| 2.804445|
| 2  | 0.016012| -1.11386|
| 3  | 0.002393| 1.02666 |
| 4  | 0.003461| -0.01789|
| 5  | 0.004199| 2.304288|
| 6  | 0.001759| -1.57197|
| 7  | 0.0008  | -1.98073|
| 8  | 0.000964| 0.447477|
| 9  | 0.000313| 2.747776|

Rhodamine B encapsulated microspheres (FLUOSTAR, EBM, Japan) of 15 µm diameter were selected as fluorescent particles along with a long-pass filter (cut-off wavelength at 550 nm) placed in front of the camera. The PIV acquisition system was composed of a high-speed camera (Fastcam Mini UX100, Photron, Japan), mounted with a lens (f = 105 mm), and a CW DPSS laser (Millennia eV, Spectra-Physics, USA) with a power of 2W and a wavelength of 532 nm. A light sheet optics module (80X91, Dantec Dynamics, Denmark) was used to generate a ≈ 1 mm thickness laser sheet to illuminate the center plane of the model, where out-of-plane velocity component is assumed negligible due to model symmetry [43]. A total of 12000 images (1024 × 1024 pixels, 19.7 µm by pixel) were captured at a frame rate of 2000 Hz and a shutter speed of 2×10⁻⁴ s. Velocity fields were computed by cross-correlation of consecutives images (DaVis 8.4.0, LaVision GmbH, Germany). An adaptive multi-pass scheme (interrogation windows refined from
96 × 96 px to 48 × 48 px) was adopted with 75% overlapping and adaptive PIV option for the last pass. This adaptive PIV approach, as previously described [44], calculates the optimal local interrogation window size and shape based on flow gradients and image quality. The interrogation window size is varied according to the flow gradient (standard deviation of the displacement field \( \sigma \) inside the interrogation window) and the correlation value, \( C \), using a local weighting factor, \( W \), defined as: \( W = 1/(C \cdot \sigma) \)

The shape of the window is set as an elongated Gaussian weighted ellipse with principal direction, \( \alpha \), and eccentricity, \( \varepsilon \), computed by minimizing the differences of displacements within the window \( dV_\alpha = \sum_{\alpha=0}^{180} |v_\alpha - v_\alpha| \) and \( \varepsilon = \frac{4}{1+3(dV_\alpha/dV_{\alpha+90})} \)

Due to the lower flow velocity in the aneurysm compared to the vessel, analysis of the aneurysmal flow was performed using 1 image over 4 (equivalent to 500 Hz). The local kinetic energy in frequency domain was calculated as [45] \( E_l(f) = \frac{|F(\bar{u}_i(t))|^2}{L_f} \) where \( f \) the frequency, \( F \) the fast Fourier transform and \( L \) the length of \( \bar{u}_i \) matrix. Enstrophy was calculated by integrating the vorticity \( (\omega(t)) \) across the aneurysm domain using cumulative trapezoidal numerical integration (CTNI) at every time step. Then, enstrophy cascade was obtained by Fourier transform of the resulting integration. The local dissipation length scales were calculated as \( \frac{1}{\lambda_i} = \frac{2\pi^2}{\bar{u}_i^2\sigma_{\bar{u}_i}^2} \int_0^\infty f^2E_l(f)\,df \) where \( \bar{u}_i \) and \( \sigma_{\bar{u}_i} \) are the mean and standard deviation of \( \bar{u}_i \), respectively. This parameter was evaluated for each point in the domain to plot \( E(f, \lambda_i) \). A two-dimensional local smoothing function [46] was applied to improve the readability of the 3D surface plots and better visualize the qualitative changes of energy or vorticity versus length scale and frequency.

RESULTS AND DISCUSSION

Using physiologically relevant in vitro intracranial aneurysm model, we investigated the kinetic energy cascade in frequency domain without using Taylor’s hypothesis. The inverse cascade was detected in multiharmonic pulsatile flow using ms/\( \mu \)m laser PIV in rigid (silicone) and elastic (PVA hydrogel) sidewall aneurysm models suggesting its independence from wall compliance. The vessel to aneurysm diameter ratio used in this study was \( \frac{2}{5} \) (see figure 1-a for model dimensions) which corresponds to moderate to large
intracranial aneurysm size. Random transitional/turbulent oscillations during six flow pulses are clearly shown in the aneurysm in comparison with the periodic pattern observed in the parent artery, shear layer and separation region, as shown in figure 3. Through a distance as small as 2 mm, between the vessel center and the shear layer (subfigures C and D in figure 3, respectively), the flow transits from periodic to quasi-periodic pattern, and the value $\frac{\bar{u}}{u_m}$ increases one fold. In non-homogenous anisotropic regimes, where $|\frac{\bar{u}}{u_m}| > 1$, the use of Taylor’s frozen turbulence hypothesis is inappropriate[47, 48] and the energy cascade is only available in frequency domain $E(f)$ rather than the wavenumber domain $E(k)$. Also, we found that Kolomogorov’s $-\frac{5}{3}$ cascade and corresponding scales do not apply (see figure 4 and table 2).

In table 2, the (+,-) signs denote inverse and direct energy cascades, respectively. Kolmogorov-Obukhov theory of 1941 and its substantiation of 1962 inherited the homogeneity and isotropy from Taylor’s 1937 theory, and the former dictates that the rate of decay of kinetic energy as function of frequency or wavenumber (since wavenumber is linearly proportional to frequency in Taylor’s hypothesis) should be homogenous in Cartesian and ensemble spaces and isotropic in directionality. This does not apply to the negative (direct) decay rate values shown in table 1. The Kraichnan inverse energy cascade theory, proposed in 1967, was based on the work of Kolmogorov, hence it only applies in homogenous isotropic turbulence. This also does not apply on the positive (inverse) energy cascade shown in table 1. It should be noted that the unavailability of wavenumber in this work only shows the inverse cascade as function of frequency which should intuitively have the same meaning of Kraichnan inverse cascade. The fact that energy cascade has different power values at different locations, where the flow exhibits random and high velocity/frequency oscillations, stipulates the non-Kolmogorov nature of the flow. In order to confirm our findings, we conducted similar measurements using PVA-H (polyvinyl alcohol hydrogel) aneurysm models. The inverse energy cascade was detected in both models which indicates that the existence of inverse energy cascade, as well as the non-Kolmogorov turbulence, are independent from wall compliance, as shown in figure 5.
Figure 3. Velocity history profiles at selected locations in the vessel and aneurysm showing local turbulence characteristics. Six-seconds time series of oscillating velocity magnitude normalized by local mean velocity magnitude ($|\bar{u}/U_{m}|$) at selected points near the aneurysm wall and in the vessel. The points are indicated on the aneurysm model by letters (A-J) in the lower left inset. The periodic behavior is conserved in the vessel (A,B) and in the entry jet region (E). In contrast, random velocity fluctuations are observed in the shear layer (C), in the separation region (D) and near the aneurysm wall (F-J). The random velocity fluctuations are obvious marking local (i.e. non-homogenous) transitional features in the aneurysm. The values of $|\bar{u}/U_{m}| > 1$ and its spatial variation within the aneurysm geometry (D=10 mm) indicates large (i.e. anisotropic) structures.
Figure 4. Inverse energy cascade is detected near the aneurysm model wall and not in the vessel. Various points taken at different near-wall locations are shown on the lower left inset, while the absolute time-averaged velocity and streamlines are shown on the lower right inset.
Table 2. Energy cascade power values show variation that indicate non-Kolmogorov cascade and scales. The cascade power values, approximated to 3 decimal figures, at the selected points shown in fig. 1.

| point       | A   | B   | C   | D   | E   | F   | G   | H   | I   | J   | V   |
|-------------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
| [1:end]     | -0.530 | -0.434 | -0.855 |     |     |     |     |     |     |     |     |
| [1:40]      |     | -0.539 | -1.440 | -1.253 | -0.977 | -0.436 | -0.133 | -1.599 | -0.684 |     |
| [40:154]    |     | -1.17 | -0.868 | 1.431 | 1.230 | 0.829 | 1.312 | 0.127 | 0.385 |     |
| [154:end]   |     | -0.383 | 0.029 | 1.047 | -1.149 | -0.847 | -1.381 | 0.549 | -0.377 |     |

Figure 5. Inverse energy cascade is independent from wall compliance as indicated by comparing rigid and elastic models. (a) Photographs of time-averaged traces of fluorescent particles in silicone (rigid) and PVA-H (elastic) models. The photograph of the elastic model is less clear due to the elastic model compliance. (b) Examples of energy cascades at the vortex core (V) and at the first point downstream the entry jet region (F) showing the inverse cascade behavior in both models.

In figure 4, we show $E(f)$ portraits at selected points in the parent vessel and near aneurysm wall, where the ECs would be subjected to pathologic flow predisposing to dysfunction and/or rupture. The energy cascade in the main vessel, where the flow is apparently periodic (figure 3, sub B) is shown to be direct with no major energy transfer events at frequencies higher than 10 Hz (see figure 4, sub A and B). Near the aneurysm wall (figure 3, sub E to J) and in the aneurysm vortex core (figure 4, sub V), the flow exhibits inverse energy cascade. To observe the inverse
cascade in space domain, one-dimensional spatiotemporal visualization of the reconstructed $E(f)$ field on representative lines (figure 6-a) depicted the inverse energy cascade around the vortex core (figure 6-b) and in the flow recovery region (figure 6-c) and not in the vessel (figure 6-d). Near the wall, where inverse energy cascade was observed, energy levels at $f \geq 200 \text{ Hz}$ were of similar values to the energy levels of the dominant harmonics $1 \leq f \leq 10 \text{ Hz}$. In the main vessel, however, the energy cascade was only direct as shown in fig. 2D. The values of energy in the inverse cascade regions had the order of $0.1 \mu J/kg$.

Figure 6. Inverse energy cascade occur at high frequency spectra around the vortex core and near the aneurysm wall. One dimensional spatiotemporal $E(f)$ maps on vertical and horizontal lines passing across the aneurysm vortex core as depicted in (a). The direction of direct cascade in terms of frequency spectrum is indicated by arrows on the vertical lines (L1) and (L2) passing across (b) aneurysm and (c) vessel, respectively. The locations where inverse energy cascade is observed are denoted by dotted white lines in (b) and on the horizontal line (L3) passing across the vortex core denoted by (V). Inverse cascade takes place around the vortex core (b) and in the vortex recovery region (d) for frequency range $f \geq 80 \text{ Hz}$ and $f \geq 50 \text{ Hz}$, respectively.

In order to visualize length scale $\ell_s$ without using Taylor’s hypothesis; time averaged field of $\ell_s$ was calculated locally at each measurement point and plotted as color map in figure 7-a (see methods section for details). We found that $\ell_s$ near the aneurysm wall had the order of $1\sim10 \mu m$ (figure 7-a). In generalized two-dimensional Cartesian space, the kinetic energy in the vessel and aneurysm were plotted against length scale and frequency in figure 7-b and 7-c, respectively. We identified inverse energy cascade events at $f > 100 \text{ Hz}$ corresponding with $\ell_s = 1 \sim 10 \mu m$ (fig.
The energy levels at such values of $\ell_s$ have the order of $10^{-3} \sim 10^{-4} \mu J/kg$. As we observed the inverse energy cascade, we noticed that at certain frequencies the inverse cascade spans all the aneurysm domain, therefore, we selected some of these frequencies to study the energy as function of the space domain, as in figure 8. At $f = 154.33$ Hz, as shown in figure 8-a, the aneurysm domain exhibits a spike of kinetic energy which brings the level of energy to similar levels of the dominant frequency ($f = 1$ Hz). This energy spike is thought to be a hydrodynamic resonance resulting from self-sustained oscillations arising from the multiharmonic flow. Similar self-sustained oscillations have been briefly documented in the literature[49], however, this is the first observation in multiharmonic pulsatile flow.

**Figure 7.** Near the aneurysm wall, dissipative length scales are $1 \sim 10 \mu m$ in size with kinetic energy level of $10^{-3} \sim 10^{-4} \mu J/kg$. Time-averaged dissipative length scales are calculated locally at each measurement point and plotted in color maps in (a). Two-dimensional spatiotemporal maps of $E(f)$ as a function of the time-averaged dissipative length scales in the vessel and aneurysm are shown in (b) and (c). The dominant (inflow) harmonics are depicted with the maximum energy shown as bright yellow lines in (b) vessel and (c) aneurysm at $1 \leq f \leq 12$. The inset (d) shows the details of the inverse cascade in the aneurysm at $40 \leq f \leq 250$ Hz.
Figure 8. Kinetic energy in the aneurysm (a) and vessel (b), as well as the local vorticity field (c) as function of location in mm at selected frequencies indicate resonance at $f = 154.33 \text{ Hz}$. The direct cascade is evident in the vessel from 1 Hz to 200 Hz. In the aneurysm, a critical frequency of 154.33 Hz was observed where the energy budget increases significantly in the flow domain which denotes transition between direct and inverse cascades, and shows the resonance mode arising from the self-sustained oscillations.

In an attempt to further investigate such phenomena, local vorticity in frequency domain was investigated, as depicted in figure 8-c, and a non-characteristic near-wall high vorticity ring was detected at the critical frequency. In correlation with the length scales, the sudden increase in vorticity magnitude at the critical frequency was correlated with length scales as low as 2 $\mu m$, see figure 9. Enstrophy ($\epsilon_\omega = \int_S \omega^2(t) \, ds$) was calculated and plotted in frequency domain, see figure 9-c. Enstrophy is a global indication for the energy cascade, and the depiction of power-change of its cascade at $f \geq 40 \text{ Hz}$ confirms the existence of inverse cascade. Dissipative length scales as small as $\ell_s = 1\sim10 \mu m$ exhibit inverse cascade at high frequency, and with energy content in the order of 0.1$\sim$1 nJ/kg ($10^{-9} \sim 10^{-10}$ J/kg). With the former length scale, such energy level corresponds to instantaneous force of the order of $10^{-3}$ pN.
Figure 9. Enstrophy cascade and vorticity correlation with length scale and frequency confirms the existence of inverse energy cascade. The vorticity field in frequency domain is correlated with dissipative length scales in (a) and the spectra where the critical frequency denoting self-sustained oscillations are shown in inset (b). Global enstrophy field ($\epsilon_\omega$) is traced in frequency domain (c) and the change of its cascade at ($f \geq 40$ Hz) marks the inverse cascade.

ECs sense flow via a variety of cell surface and intracellular mechano-receptors[50]. One of the main mechano-transducers is the cell membrane glycocalyx (GCX)[51]. Glycocalyx was shown to sense and transduce the flow induced forces into cellular response [52]. GCX also was shown to undergo structural organization in response to flow, and these structural changes can impact the endothelial cellular response [51]. When we analyzed the near-wall $\ell_s$ that demonstrated the inverse energy cascade phenomenon, they fell into the micrometer range.
Interestingly, it was recently shown that focal tension forces, in pN order of magnitude and at the micrometre length scale, applied via membrane tethers, are capable of activating the Piezo channels, inducing Ca$^{2+}$ entry and downstream cellular signaling cascade[53]. Thus theoretically, kinetic energy transfer at such scale may induce ECs’ signaling cascade and a corresponding aberration may induce pathogenesis. ECs within aneurysms are known to have a pathologic phenotype, however the actual aneurysmal flow is still scrutinized and under debate [54]. While this holds true for aneurysmal ECs, the ECs within the parent vessel are exposed to physiologic flow stress and thus exhibit a physiologic phenotype [51]. Thus, we argue that the exposure to an inverse energy cascade in the aneurysm may induce a pathologic phenotype, which may explain the mechanobiological response to the aberrant flow field. Therefore, the exact effect of the inverse energy cascade on ECs should be investigated, and the effect of different energy cascades should be characterized. This will eventually lead to substantial improvement of the current hypothesis [1] about the mechanobiological role of wall shear stress (WSS) by including the role of local energy cascade and dissipation.

CONCLUSION

Blood flow in intracranial aneurysm appears to be much more complex than previously thought. The observation of non-Kolmogorov turbulence and inverse energy cascade in such flow constitute staggering shift in the aneurysm hemodynamics paradigm. Near-wall turbulence possesses length and force scales that could be significant and influential in the ECs mechano-signaling. On the other hand, from the viewpoint of turbulence physics, it is indeed overwhelming to observe such complex phenomena at $Re < 400$ and $\alpha < 3$. The complex dynamics of multiharmonic flow as well as the inherited geometrical sophistication of aneurysms trigger series of physical phenomena that are yet to be discovered. The present work points to the fact that the classical differentiation between laminar and turbulent flow regimes, based on the Reynolds criteria for pipe flow, is inappropriate in the aneurysm problem.

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AUTHOR CONTRIBUTIONS

Conceptualization and supervision: Saqr
Software, data curation and formal analysis: Tupin
Methodology: Tupin and Ohta
Funding acquisition and Resources: Ohta and Tupin
Investigation: Tupin, Saqr and Rashad
Visualization: Tupin
Project administration: Rashad and Niizuma
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Writing: Saqr, Rashad and Tupin
Editing and revision: Niizuma and Ohta

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