ELSEVIER

Editorial

Contents lists available at ScienceDirect

Redox Biology



journal homepage: www.elsevier.com/locate/redox

Off to a good start and a promising future in communicating cutting edge developments in redox biology

As we approach the peak of the season (summer in the Northern hemisphere, winter in the Southern) we can gladly state that we have reached the figure of 50 accepted contributions since the journal's launch. Since our last editorial we feel significant progress has been made on the journal's profile and public awareness of it, and we are proud to say that it is rapidly being accepted into the major bibliographic databases such as Pubmed Central, Scopus and Google Scholar. We believe that Redox Biology is growing rapidly and steadily as it begins to realize its potential to serve the scientific community.

During the past few months the journal has published a series of highly interesting articles in the diverse domains of the redox field. Starting with method fine-tuning, the group of Garry Buettner describe the methods to evaluate the removal of extracellular hydrogen peroxide in cell cultures and improve experimental design for exposure of cells to hydrogen peroxide [1]. In a similar vein, Romanowicz et al. described the use and potential utility of a promising device to tightly control NO release at cell surfaces [2]. The journal has continued to attract significant interest in the signaling arena, gathering an outstanding series of articles, including articles on the relationship of heme oxygenase-1 and beta-catenin/hnRNPK in lung repair [3], activation of MAPKs by the cigarette smoke aldehyde Acrolein [4] and the redox-dependent expression of Interleukin-1 α [5]. These are just paradigmatic examples of the novel research that Redox Biology has been privileged to handle.

Clearly, a niche in which the Editors feel that progress has to be made regarding the journal's impact on the whole redox field is in bringing redox chemistry akin to pathophysiological and pathological contexts. In an effort to fill this gap, Balaraman Kalyanaraman has done a splendid job of providing the conceptual framework for transmitting the basis of redox biochemistry to a general reader [6] in an extensive graphical review. This series of detailed slides will allow many graduate students and seasoned researchers alike to put into perspective an important number of articles related to pathophysiology and to the pathological settings in which redox mechanisms play a role. Among these, our journal has hosted several contributions of diverse origin including the role of cullin 3 in various pathologies [7,8], the importance of nitrite conversion to NO in the gastrointestinal system [8] and the usefulness of peroxidation-resistant fatty acids for the evaluation of Friedreich's ataxia [14]. The crucial transcriptional regulator, Nrf2, has also been the object of intense attention, especially in relation to diabetes. The work of Heiss et al. [9] shows the importance of this molecule in the regulation of glucose metabolism. In addition, Kumar and cols [10] review graphically how Nrf2 and NF-kB are key players in diabetic neuropathy and how their targeting may subserve a therapeutic role.

Mitochondrial biology continues to be center stage in regard to redox-regulated cellular functions. This is reflected by articles covering the role of heme oxygenase in mitochondrial function in macrophages [11], the constant debate on the mitochondrial sites involved in ROS generation and its dependence on substrates [11] and the adaptive upregulation of mitochondrial LON protease in acute oxidative stress [12]. The antioxidant facet of the last series of accepted articles is represented by the interesting observation about the capacity of quercetin to prevent left ventricular hypertrophy in a model of atherosclerosis (Apo E KO mouse) and by another article detailing how sex hormones affect circulating antioxidants [13].

Overall, the readers of Redox Biology, a community we expect to grow exponentially in the next few years, have ample material for delectation in many different directions and at this stage the journal continues to offer a wide selection of high quality articles in every category. We are off to a good start and we are confident the future will be even brighter.

References

- [1] B.A. Wagner, et al., An assay for the rate of removal of extracellular hydrogen peroxide by cells, Redox Biology 1(1) (2013) 210–217, http://dx.doi.org/10. 1016/j.redox.2013.01.011.
- [2] Genevieve E. Romanowicz, et al., Novel device for continuous spatial control and temporal delivery of nitric oxide for in vitro cell culture, Redox Biology 1 (1) (2013) 332–339, http://dx.doi.org/10.1016/j.redox.2013.06.002.
- [3] Guang Yang, et al., Heme oxygenase-1 regulates postnatal lung repair after hyperoxia: role of β-catenin/hnRNPK signaling, Redox Biology 1 (1) (2013) 234–243, http://dx.doi.org/10.1016/j.redox.2013.01.013.
- [4] Matthew J. Randall, et al., Acrolein-induced activation of mitogen-activated protein kinase signaling is mediated by alkylation of thioredoxin reductase and thioredoxin 1, Redox Biology 1 (1) (2013) 265–275, http://dx.doi.org/ 10.1016/j.redox.2013.02.001.
- [5] Donald A. McCarthy, et al., Redox-control of the alarmin, Interleukin-1α, Redox Biology 1 (1) (2013) 218–225, http://dx.doi.org/10.1016/j.redox.2013.03.001.
- [6] B. Kalyanaraman, Teaching the basics of redox biology to medical and graduate students: oxidants, antioxidants and disease mechanisms, Redox Biology 1 (1) (2013) 244–257, http://dx.doi.org/10.1016/j.redox.2013.01.014.
- [7] AC Andérica-Romero, et al., Cullin 3 as a novel target in diverse pathologies, Redox Biology 1 (1) (2013) 366–372, http://dx.doi.org/10.1016/j.redox.2013. 07.003.
- [8] C. Pereira, et al., The redox interplay between nitrite and nitric oxide: from the gut to the brain, Redox Biology 1 (1) (2013) 276–284, http://dx.doi.org/ 10.1016/j.redox.2013.04.004.
- [9] E.H. Heiss, et al., Glucose availability is a decisive factor for Nrf2-mediated gene expression, Redox Biology 1 (1) (2013) 359–365, http://dx.doi.org/ 10.1016/j.redox.2013.06.001.
- [10] Yerra V. Ganesh, et al., Potential therapeutic effects of the simultaneous targeting of the Nrf2and NF-κB pathways in diabetic neuropathy, Redox Biology (2013), http://dx.doi.org/10.1016/j.redox.2013.07.005.
- [11] C.L. Quinlana, et al., Sites of reactive oxygen species generation by mitochondria oxidizing different substrates, Redox Biology 1 (1) (2013) 304–312. (10.1016/j.redox.2013.04.005).
- [12] Jenny K. Ngo, L.C.D. Pomatto, K.J.A. Davies, Upregulation of the mitochondrial Lon protease allows adaptation to acute oxidative stress but dysregulation is associated with chronic stress, disease, and aging, Redox Biology 1 (1) (2013) 258–264, http://dx.doi.org/10.1016/j.redox.2013.01.015.

Associate Editors Tak Yee Aw LSU Health Sciences Center, Molecular and Cellular Physiology, Shreveport, LA, USAAssociate Editor Santiago Lamas Centro de Biología Molecular "Severo Ochoa"(CSIC-UAM), Cell Biology, Madrid, Spain

- [13] Francesco Bellantia, et al., Sex hormones modulate circulating antioxidant enzymes: impact of estrogen therapy, Redox Biology, 1, 340–346, http://dx. doi.org/10.1016/j.redox.2013.05.003.
- [14] M. Grazia Cotticelli, et al., Insights into the role of oxidative stress in the pathology of Friedreich ataxia using peroxidation resistant polyunsaturated fatty acids, Redox Biology 1 (1) (2013) 398–404, http://dx.doi.org/10.1016/j. redox.2013.06.004.

Co-Editor-in-Chief Tilman Grune Institute of Nutrition, Jena, Germany

Victor Darley-Usmar* University of Alabama, Department of Pathology, Birmingham, AL, USA E-mail address: darley@uab.edu

^{*} Corresponding author. Tel.: +1 205 975 9686; fax: +1 205 934 7447.