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Matalon S. A critical review of the American Journal of Physiology-Lung Cellular and Molecular Physiology: 2012–2015. Am J Physiol Lung Cell Mol Physiol 307: L911–L916, 2014; First published November 7, 2014; doi:10.1152/ajplung.00330.2014.—I have had the privilege of serving as Editor-in-Chief of the American Journal of Physiology: Lung Cellular and Molecular Physiology from 1/1/2012 to 1/1/2015 and have been reappointed for another 3-year term. When I took over as editor, I published an editorial in AJP-Lung in which I highlighted my vision and outlined the tasks to be accomplished to transform AJP-Lung into “The best place to publish basic, translational, and hypothesis-driven clinical lung research.” Herein I review our accomplishments during the first term. As promised, we review each article submitted to this journal and our reviews always help the quality and impact of every paper. We recognized the contributions of junior authors by establishing a number of awards and increased the visibility of AJP-Lung by establishing Facebook and Blog electronic pages and sponsoring symposia in scientific meetings. Our impact factor increased from 3.523 in 2011 to 4.041 in 2012 and, thanks to our calls for papers, we are receiving large numbers of high-quality papers in all aspects of pulmonary cell biology and lung diseases. The best is yet to come.

Major Accomplishments

1. We assembled and posted a list of the most highly cited papers published in AJP-Lung during the last 20 years (http://ajplung.physiology.org/). We recognize important contributions by young investigators by highlighting their papers in our electronic media (www.facebook.com/AJPLung) and blog page (http://ajplung.blogspot.com). In addition, we have established a number of monetary awards for best papers by young investigators.

2. We solicited and published two retrospective reviews (edited by Dr. Lester Kobzik) by the senior authors of two of the most highly cited papers (53, 67).

3. We published four Physiology in Medicine Reviews by eminent scientists in the fields of autophagy (33), sepsis (8),...
influenza (34), and lung ischemia-reperfusion injury (7). These articles are authoritative bench-to-bedside reviews, highly relevant to basic and translational scientists and clinicians alike.

4. Two Associate Editors (Drs. Morty and Prakash) and two highly valued Editorial Board members (S. Herold and I. Vadasz) spent an enormous amount of time writing three state-of-the-art Perspectives highlighting and integrating findings published in AJP-Lung with what has been published in other major journals. The areas covered were bronchopulmonary dysplasia (27), airway reactivity and remodeling (41), and acute lung injury and barrier dysfunction (18). These highly informative Perspectives show the significant impact that we have on three timely and clinical relevant topics.

5. We published two reviews by Drs. Ochs and Muhlfeld, intended as primers for those interested in performing quantitative lung morphology (32, 36). These two papers contain a wealth of information and specific examples of how to conduct artifact-free morphological measurements in both adult and newborn animals. Dr. Weibel also wrote an editorial on the developments of lung morphometry and how he became interested on this topic (54).

6. Our three best-cited reviews were published by Dr. Collawn et al. (“The ENaC-CFTR debate,” Ref. 10); Dr. Seeley et al. (“Inflection points in sepsis biology,” Ref. 48); and dos Santos et al. (“The inflammaosome in lung diseases,” Ref. 12). Reviews published in 2012 received 25.3 ± 3.2 citations (mean ± SE; n = 6).

7. We started a new feature on the History of Physiology. Dr. John B. West, who first described the distribution of ventilation and perfusion in the lungs and is the author of the most widely used book on respiration physiology by medical students, wrote eleven articles (56–66) tracing the history of pulmonary physiology and highlighting the contributions of the giants in our field starting with Drs. Fenn and Rahn. These articles are highly recommended to those who are interested in the history and teaching of respiratory physiology.

8. We recognized the outstanding contributions of our younger colleagues (graduate students, postdoctoral or clinical fellows, and assistant professors) by establishing “The Outstanding Paper by a Junior Investigator” Awards. We solicited nominations from the pulmonary community at large asking nominators to describe the contribution of each nominee to the paper, its importance to the field, and his/her potential of becoming an independent and productive scientist. Three awards were given in 2012 and five awards in 2013. The honor roll includes Drs. Christina Alvira (19), Lyubov Brueggemann (4), Adrian R. West (55), Julia E. Rager (43), James Londino (25), Ezra Roan (44), and Tracy Schmidt (46). Congratulations to these outstanding young scientists. We are all very proud of them and look forward to receiving more papers from their graduate students and postdoctoral fellows! We feel that this initiative has contributed significantly to increased submissions of original manuscripts and insightful reviews.

9. In an effort to promote the very best original research papers, The American Physiological Society established a virtual journal (APSselect, http://apsselect.physiology.org). The Editor and Associate Editors nominate papers that provide significant new insights into unanswered questions, stimulate new venues for research, and were highly rated by the reviewers. The APS Publications Committee screens all nominations and chooses the best for inclusion in APSselect. I am delighted to tell you that six AJP-Lung articles were included in APSselect in 2014. Even a casual perusal of these articles reveals the breadth of our coverage of all aspects of basic, translational, and hypothesis-driven lung research:

• Lim et al. (24) showed that maternal stress increases susceptibility of newborns to allergens.
• McAuley et al. (30) presented evidence that human mesenchymal stem cells restored sodium-dependent alveolar fluid clearance in human lungs resected for transplantation. There is considerable excitement in the use of stem cells in the treatment to acute lung injury. I refer interested readers to the excellent review by Gotts and Matthay (16).
• Mannam et al. (28) showed that MKK3 deficiency reduced the lethality of sepsis in mice by lowering levels of lung and mitochondrial injury as well as reactive oxygen species. Furthermore, MKK3 deficiency appeared to simultaneously increase mitochondrial biogenesis and mitophagy. This is a critical area of research because therapies for sepsis, a major cause for the development of acute lung injury, are limited. Please see the outstanding Physiology in Medicine article by Christiaans et al. (8) discussing the clinical management of sepsis and the saga of activated protein C as a potential therapeutic agent as well as the synopsis of a symposium organized by Drs. Choi and Gillespie on mitochondria in lung biology and pathology (47).
• Lee et al. (22) showed that MJ33 (an inhibitor of phospholipase A2) decreases LPS-induced injury in mice by preventing the activation of NADPH oxidase type 2 (NOX2) and thus decreasing reactive oxygen species generation. Again I refer the readers to the Physiology in Medicine review by Dr. Chattjee (7) as well as a comprehensive review on the role of NAPDH in the regulation of lung amiloride-sensitive epithelial sodium channels (13). On this topic, Sharma et al. (49) showed that activation of ATII cell NADPH during ischemia-reperfusion injury leads to the production of CXCL1, which contributes to the recruitment and activation of neutrophils and lung injury. Also, in a fascinating paper, Noel et al. (35) presented evidence indicating that PECAM-caveolae mechanosensing machinery on the endothelium is able to sense the changes in membrane tension during stoppage of flow that triggers a signaling cascade that leads to reactive oxygen species production.
• Chronic obstructive pulmonary disease is the third leading cause of death in the USA and accounted for ~135,000 deaths in 2010 (6). Chronic exposure to cigarette smoke is the main factor contributing to the development of pulmonary emphysema and chronic bronchitis (42) although genetic factors play an important role as well (3). Sauer et al. (45) showed that macrophage migration inhibitory factor (MIF) and its receptor CD74 are required for maintenance of normal alveolar structure in mice and that decreases in MIF are associated with COPD in human subjects. MIF is a pleiotropic factor that antagonizes both apoptosis and premature senescence which contribute to the pathogenesis of COPD. As mentioned above, Yao and Rahman (67) published an important review on the role of histone deacetylase 2 (HDAC2), which regulates a number of cellular processes including senescence and apoptosis in the development of COPD. Also Harra et al. (17)
Table 1. Impact factors of respiratory journals for the indicated years

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<th>Journal</th>
<th>Impact Factor</th>
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<td></td>
<td>2010</td>
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<tr>
<td>ARRCCM*</td>
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<tr>
<td>THORAX*</td>
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<td>ERJ*</td>
<td>5.922</td>
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<tr>
<td>AJRCMB</td>
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<td>3.127</td>
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*Published mainly clinical articles (basic science article should contain data on human subjects). Impact factors are posted on each journal’s website.

showed that exposure of human bronchial epithelial cell extract to cigarette smoke extract caused mitochondrial fragmentation which leads to increased senescence that was prevented by Mito-TEMPO, a mitochondrial targeted antioxidant.

- As pointed out in the Physiology in Medicine review on influenza by Drs. Noah and Noah (34), “influenza type A and B virus infections in humans result in an estimated 150,000–200,000 hospitalizations and 30,000–50,000 deaths in the United States annually and many thousands more globally.” Influenza H5N1 viruses are highly pathogenic and result in over 60% mortality in humans. Hypercytoxia is a key mechanism responsible for organ failure in H5N1-infected patients. In a recent article, Pan et al. (37) showed that blocking autophagy significantly attenuated H5N1-induced cytokine and chemokine production in vitro as well as in C57BL/6 mice. The authors then demonstrated that autophagy-mediated inflammatory responses involved the activation of NF-κB and p38 MAPK signaling pathways. As pointed out in the outstanding Physiology in Medicine review by Dr. Choi (33), induction of autophagy may either exacerbate the course of a number of pulmonary diseases or promote healing. To name just two examples, impairment of autophagy decreases ventilation-induced injury by blocking NF-κB (26) and insufficient autophagy contributes to the pathogenesis of pulmonary fibrosis by failing to modulate epithelial cell senescence (2).

10. We request Editorial Focus articles by prominent members of the pulmonary community to highlight research papers that were deemed by the reviewers to be in the top 5–10% of all manuscripts. I am very pleased and frankly awed by the high quality and diversity of articles we have published in AJP-Lung during the last three years. To name just a few: Dr. Janssen wrote a highly informative editorial highlighting the paper by Gallos et al. (14, 20) on the role of airway smooth muscle GABA receptors in controlling airway reactivity; Dr. Gotts and Matthay’s editorial focus highlighted the paper by Tropea et al. on the role of stem cells in bronchopulmonary dysplasia (16, 51); Dr. Collawn discussed the paper of Li et al. which showed that CFTR is necessary for the maintenance of liquid layer in the airways of pig lungs (10, 23); Drs. Clifford and Knox discussed the findings of An et al. who showed that TAS2R activation promotes airway smooth muscle relaxation (1, 9); Drs. Vohwinkel et al. highlighted the findings of Londino et al. showing that one of the influenza proteins (M2) inhibits the activity of CFTR (25, 52); Dr. Creighton’s editorial focus on vascular permeability highlighted the findings of Melton et al. showing that induction of phospholipase A2 caused lung inflammation and injury (11, 31). We make a special plea to members of the pulmonary community to send us suggestions for editorial focus and to volunteer to write these highly informative articles.

11. We have issued seven Calls for Papers to stimulate research and increase submissions of original manuscripts in the following areas: 1) Sex Differences in the Respiratory System; 2) Nanoparticles and the Lung: Friend or Foe?; 3) Biomarkers in Lung Diseases: from Pathogenesis to Prediction to New Therapies; 4) Real-time Visualization of Lung Function: from Micro to Macro; 5) Bioengineering the Lung: Molecules, Materials, Matrix, Morphology, and Mechanics; 6) Translational Research in Acute Lung Injury and Pulmonary Fibrosis; and 7) Household Air Pollution (inactive). Papers in response to these calls are published under the appropriate heading and in general are cited more often. Again we welcome ideas from the pulmonary community for additional topics.

12. In an effort to increase the visibility of AJP-Lung and attract more papers, we sponsored three symposia in international meetings. Invited speakers were encouraged (but not required) to submit manuscripts on the topic of their talk to AJP-Lung. (see https://www.facebook.com/AJPLung). They were as follows:

1) “The Enigma Variations: The Many Faces of the Myofibroblast in Fibrotic Disease.” Organized by: Rachel C. Chambers (Associate Editor) and Paul F. Mercer (Editorial Board member) for the 2014 Experimental Biology meeting (San Diego California). Please see Refs. 2, 5, 21, 40 published in AJP-Lung on this topic.

2) “Bioengineering the Cardiopulmonary System: From Molecules to Mechanics.” Organized by: YS Prakash (Deputy Editor) and Kurt R. Stenmark (Associate Editor) for the 2014 Experimental Biology meeting (San Diego, CA). We have an

Table 2. American Journal of Physiology: Lung Cellular and Molecular Physiology (APS Reports)

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<th>2010</th>
<th>2011</th>
<th>2012</th>
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<td>No. research manuscripts</td>
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<td>376</td>
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<td>266*</td>
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<td>24.3*</td>
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<td>Total manuscripts/month</td>
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<td>31.3</td>
<td>27.5</td>
<td>29.5*</td>
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<tr>
<td>Acceptance rate for research manuscripts</td>
<td>45%</td>
<td>51%</td>
<td>51%</td>
<td>61%</td>
<td>40%</td>
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<tr>
<td>No. days to first decision for research manuscripts</td>
<td>32</td>
<td>29</td>
<td>23</td>
<td>23</td>
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*As of 1/1/2014-9/30/2014
active call for papers on Bioengineering the Lung, which has resulted in the publication of some outstanding papers during the last three years. Please see Refs. 38, 39, 50, 55 as examples.


13. The Associate Editors restructured the Editorial Board to ensure balance among senior and junior scientists. Editorial Board members are expected to review about 12 manuscripts per year, submit original papers to AJP-Lung, and contribute ideas for reviews and call for papers. We also appointed five prominent members of the community as Consulting Editors to provide sage advice on all issues (http://ajplung.physiology.org/edboard).

Have We Been Successful in Increasing the Impact and Visibility of AJP-Lung?

How does one gauge success? I am listing below a number of subjective and objective criteria showing that we are in the right path.

The pulmonary community feels that their manuscripts receive positive and constructive reviews. The Associate Editors and I spent a lot of time summarizing the comments of the reviewers and communicate to the authors what needs to be done. Research papers are sent to two reviewers, or more if there is a clear difference of opinion that cannot be resolved by the Associate Editor.

Our impact factor increased. There is great debate about the pros and cons of the impact factor as a measure of the quality of a journal. Unfortunately, it is the most important determinant used by junior faculty when they submit manuscripts. Please see Table 1 for the Impact Factors of journals publishing mainly basic and clinical research. As can be seen, our impact factor increased from 3.523 to 4.041 and we are in a virtual tie with the American Journal of Respiratory Cell and Molecular Biology. Our 5-year impact factor is 4.338 and our 2013 Cited Half Life is 7.7 years (http://www.the-aps.org/mm/Publications/Journals/Impact-Factors.html). Actually, according to the H-index (“an index that attempts to measure both the productivity and impact of the published work of a scientist or scholar or journal”), we are currently the top-ranked journal publishing nonclinical pulmonary research articles (source: http://www.scimagojr.com/journalrank.php?category=2740).

We have shortened the time to first decision and reversed the decline in submissions. This information is shown in Table 2. Most journals experienced a decline in the number of submissions due to the decrease of NIH funding and the keen competition by the ever increasing number of online journals. As can be seen in Table 2, we have reversed the decline in submissions and we are hopeful that our submissions will be higher in 2014 than in 2013. Most important, we have decreased the number of days of first decision from 31.6 days in 2010 to 21.74 days in 2014. In addition, we make every effort to reach a decision on revised manuscripts within 14 days from submission. Our sincere thanks go to our current editorial assistant (Amy McEver) and to the two previous assistants (Gloria Y. Son and Emily Sher) who send gentle but firm reminders to all of us if we do not return our reviews within two weeks.

In summary, it has been a real privilege to serve as Editor in Chief of this highly prestigious journal. I was delighted when Dr. Raff, Chairman of the Publications Committee of the American Physiological Society, informed me that I was reappointed for another three-year term. I promise you that the “best is yet to come.” We will spare no effort to elevate AJP-Lung to new heights so it is perceived as “The best journal to publish basic, translational, and hypothesis-driven clinical lung research.”

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

S.M. researched and wrote this article.

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46. Tropea KA, Leder E, Aslam M, Lau AN, Raiser DM, Lee JH, Balasubramaniam V, Fenidrenburg LE, Alex MS, Kourenmanas S, Kim CF. Bronchialveolar stem cells increase after mesenchymal stromal


