

Editorial

The Journal of Pathology 2008 Jeremy Jass Prize for Research Excellence in Pathology

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Abstract

The first Jass Prize for Research Excellence has been awarded to a group from Hannover in Germany. These authors discovered the epigenetic inactivation of microRNA gene *hsa-mir-9-1* in human breast cancer and characterized its biological and clinical relevance. This frequent epigenetic silencing was found to occur early in the development of breast cancer, and illustrates another mechanism by which tumour development is influenced by genes that operate without expression as proteins.

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The first issue of *The Journal of Pathology* appeared in 1892 and contained articles by Metchnikoff and Virchow, among others. Since then, the Journal has had a major place in the pathological, clinical, and scientific literature, and many important papers have appeared in its pages [1]. The entire backfile archive can be accessed from the Journal website, including the articles by Metchnikoff and Virchow [2,3]. In the winter of 2009, the Editorial team of *The Journal of Pathology*, together with the Officers of the Pathological Society, announced the institution of an annual prize to be awarded to the authors of the research paper published in *The Journal of Pathology* adjudged by the Editorial team to be the best published in a calendar year. The judgement would be based on scientific excellence, novelty, and importance, with the award being made annually one year in arrears, with the first award being made in January 2010 for a paper that had been published in the calendar year 2008. The decision to name this award after the late Jeremy Jass was made with the permission of Johanna Jass (Jeremy's widow), Joanna and Simon (his children), Leon (his father), and other members of the family. It was anticipated that this new award would recognize and commemorate the enormous contribution that Jeremy Jass made to pathological research

[4]. The decision to work a year in arrears was predicated on the view that by looking at work published in the recent past (as opposed to the immediate past), one could perhaps have a more objective view of the impact and importance of work, and also employ surrogate measures such as downloads and citation to gauge material that is truly having some influence on the research community.

To undertake any such analysis of 'quality' is fraught with danger and potential pitfalls. By definition, all the material published has been through a full peer review process and is thus deemed to be of high quality. Judging 'worth' is dangerous since some observations and discoveries do not appear to be important in a broad context until long after their initial report. Take, for example, the near simultaneous reports in 1979 of what we now call p53 [5–10]. At the time, these reports appeared to be a relatively arcane set of observations and the enormous impact of this discovery was not apparent for more than a decade [11,12]. Considering the work of Jeremy Jass, his provocative thesis that metaplastic (hyperplastic) polyps were not as innocent as many (if not all) thought [13] was barely considered for nearly two decades, yet the idea of serrated pathways to colorectal cancer are now well accepted [14]. Other pieces of work are published



Figure 1. The authors of the Jass Prize award winning paper. From left to right, Hans Kreipe, Florian Länger, Ulrich Lehmann, Matthias Christgen, and Britta Hasemeier. Top inset: Mirco Müller; bottom inset: Daniel Römermann

with great fanfare, with press releases and claims of breakthrough, yet subsequently turn out to be of more modest import, or even of no significance in the greater scheme of scientific endeavour. Surely, then, to attempt to identify real quality from the highlights of a single year might be considered a risky business. Certainly there are measures one could use: citations and downloads are easily measured and one could use such simple devices to identify the best. But, of course, such metrics have many limitations. A paper might be downloaded frequently because it lies in an area with a large number of workers (the p53 field suffers this problem, for example). Other work may be of major importance but the field in which it lies might be relatively small: it could never compete! Citation indices suffer from similar problems. The views of a number of external reviewers could be used, but again personal foible and unwitting bias could come into play. Notwithstanding the many limitations of *any* such analysis of the nearly 200 papers published by the Journal in any calendar year, it was hoped that this award would help to recognize the many gifted and talented scientists who contribute to the pages of *The Journal of Pathology*.

The approach that the Editorial team finally settled on for identifying the annual winner of the Jass Award for Research Excellence in Pathology was a mixture of methods and included human factors. The Managing Editor and her colleagues produced a table listing all research articles published in a calendar year and annotated this with citations and downloads. The file was reviewed by all Editors and Associate Editors, and from this and their own review of the papers in the year in question, they drew up a short list of 'their personal highlight papers' in a ranked order using the criteria of 'scientific excellence, novelty, and importance' and the award is therefore influenced by what excited each of us individually. From the data of the Deputy

Editor and all Associate Editors, the Editor-in-Chief (who did not make an assessment at this stage) collated the information and identified papers that were recognized by more than one 'reviewer'. A simple scoring system then allowed the identification of the paper to be awarded the Jass Prize for Research Excellence. The role of the Editor-in-Chief was to manage the process and to cast a deciding vote in the case of a tie. In this first award cycle, the Deputy and Associate Editors identified and nominated ten papers whose diversity reflects the spectrum of research fields covered by pathological science [15–24], with one being nominated by a majority [24] and thus being declared the first winner of the Jass Prize for Research Excellence (Figure 1).

The winning paper looked at the regulation of expression of microRNAs in breast cancer. Until maybe 10 years ago, most studies of RNA would not have been able to recognize alterations in the expression of these short regulatory RNA species. They do not encode a protein, yet may influence the expression of large sets of genes and thereby affect tumour behaviour (reviewed in ref 25). Lehmann *et al* [24] demonstrated that certain microRNAs can have their expression suppressed not by mutation (as has been described for some microRNAs) but by an epigenetic mechanism — promoter methylation. Importantly, this was found to occur early and often in the development of breast cancer, and so this work illustrates another mechanism by which tumour development is influenced by genes that operate without expression as proteins.

The entire Editorial team congratulates the authors of this excellent work. The corresponding author will be presented with a medal and certificates for the co-authors at the Winter Meeting of the Pathological Society on 7 January 2010 at Imperial College, London.

References

- Herrington CS. *The Journal of Pathology*: past, present and future. In *Understanding Disease. A Centenary Celebration of the Pathological Society*, Hall PA, Wright NA (Eds). Wiley: London, 2006; 97–107. (A free PDF of this article this can be obtained from http://www3.interscience.wiley.com/homepages/1130/The_history_of_the_Journal_of_Pathology.pdf).
- Metchnikoff E. On aqueous humour, micro-organisms, and immunity. *J Pathol* 1892;**1**:13–20.
- Virchow R. Transformation and descent. *J Pathol* 1892;**1**:1–12.
- Shepherd NA, Morson BC. Professor Jeremy R Jass: an appreciation. *J Pathol* 2009;**217**:467–468.
- De Leo AB, Jay G, Appella E, Dubois GC, Law LW, Old LJ. Detection of a transformation-related antigen in chemically induced sarcomas and other transformed cells of the mouse. *Proc Natl Acad Sci U S A* 1979;**76**:2420–2424.
- Kress M, May E, Cassingena R, May P. Simian virus 40-transformed cells express new species of proteins precipitable by anti-simian virus 40 serum. *J Virol* 1979;**31**:472–483.
- Lane DP, Crawford LV. T antigen is bound to a host protein in SV40-transformed cells. *Nature* 1979;**278**:261–263.
- Linzer DIH, Levine AJ. Characterization of a 54 K Dalton cellular SV40 tumor antigen present in SV40-transformed cells and in infected embryonal carcinoma cells. *Cell* 1979;**1**:43–52.
- Melero JA, Stütt DT, Mangel WF, Carroll RB. Identification of new polypeptide species (48–55 K) immunoprecipitable by antiserum to purified large T antigen and present in simian virus 40-infected and transformed cells. *J Virol* 1979;**93**:466–480.
- Chang C, Simmons DT, Martin MA, Mora PT. Identification and partial characterization of new antigens from simian virus 40-transformed mouse cells. *J Virol* 1979;**31**:463–471.
- Baker SJ, Fearon ER, Nigro JM, Hamilton SR, Preisinger AC, Jessup JM, *et al.* Chromosome 17 deletions and p53 gene mutations in colorectal carcinomas. *Science* 1989;**244**:217–221.
- Nigro JM, Baker SJ, Preisinger AC, Jessup JM, Hostetter R, Cleary K, *et al.* Mutations in the p53 gene occur in diverse human tumour types. *Nature* 1989;**342**:705–708.
- Jass JR. Relation between metaplastic polyp and carcinoma of the colorectum. *Lancet* 1983;**1**:28–30.
- Goldstein NS. Serrated pathway and APC (conventional)-type colorectal polyps: molecular–morphologic correlations, genetic pathways, and implications for classification. *Am J Clin Pathol* 2006;**125**:146–153.
- Nakai N, Ishikawa T, Nishitani A, Liu N-N, Shincho M, Hao H, *et al.* A mouse model of a human multiple GIST family with KIT-Asp820Tyr mutation generated by a knock-in strategy. *J Pathol* 2008;**214**:302–311.
- Liegl B, Kepten I, Le C, Zhu M, Demetri GD, Heinrich MC, *et al.* Heterogeneity of kinase inhibitor resistance mechanisms in GIST. *J Pathol* 2008;**216**:64–74.
- Angulo B, Suarez-Gauthier A, Lopez-Rios F, Medina PP, Conde E, Tang M, *et al.* Expression signatures in lung cancer reveal a profile for EGFR-mutant tumours and identify selective PIK3CA overexpression by gene amplification. *J Pathol* 2008;**214**:347–356.
- Edward M, Quinn JA, Mukherjee S, Jensen M-BV, Jardine AG, Mark PB, *et al.* Gadodiamide contrast agent activates fibroblasts: a possible cause of nephrogenic systemic fibrosis. *J Pathol* 2008;**214**:584–593.
- Vockerodt M, Morgan SL, Kuo M, Wei W, Chukwuma MB, Arrand JR, *et al.* The Epstein–Barr virus oncoprotein, latent membrane protein-1, reprograms germinal centre B cells towards a Hodgkin's Reed–Sternberg-like phenotype. *J Pathol* 2008;**216**:83–92.
- Leucci E, Cocco M, Onnis A, De Falco G, van Cleef P, Bellan C, *et al.* MYC translocation-negative classical Burkitt lymphoma cases: an alternative pathogenetic mechanism involving miRNA deregulation. *J Pathol* 2008;**216**:440–450.
- Storci G, Sansone P, Trere D, Tavolari S, Taffurelli M, Ceccarelli C, *et al.* The basal-like breast carcinoma phenotype is regulated by SLUG gene expression. *J Pathol* 2008;**214**:25–37.
- Weigelt B, Horlings HM, Kreike B, Hayes MM, Hauptmann M, Wessels LFA, *et al.* Refinement of breast cancer classification by molecular characterization of histological special types. *J Pathol* 2008;**216**:141–150.
- Bergamaschi A, Tagliabue E, Sørli T, Naume B, Triulzi T, Orlandi R, *et al.* Extracellular matrix signature identifies breast cancer subgroups with different clinical outcome. *J Pathol* 2008;**214**:357–367.
- Lehmann U, Hasemeier B, Christgen M, Müller M, Römermann D, Länger F, *et al.* Epigenetic inactivation of microRNA gene *hsa-mir-9-1* in human breast cancer. *J Pathol* 2008;**214**:17–24.
- Taft RJ, Pang KC, Mercer TR, Dinger M, Mattick JS. Noncoding RNAs: regulators of disease. *J Pathol* 2010;DOI:10.1002/path.2638.