如何提升科技期刊国际影响力

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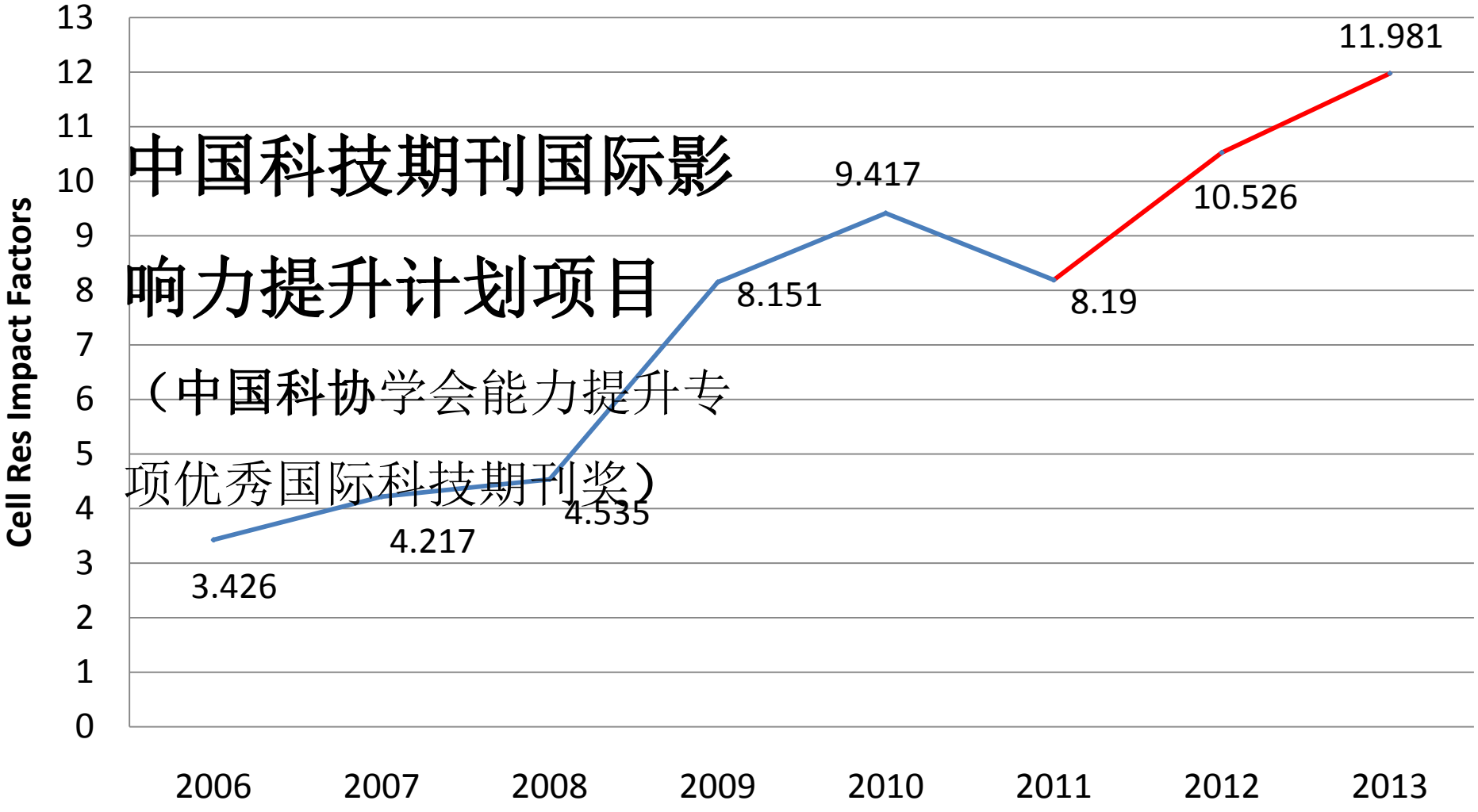
Cell Research　编辑部主任

南京，25 Oct 2014

国际分子细胞生物学领域一线期刊



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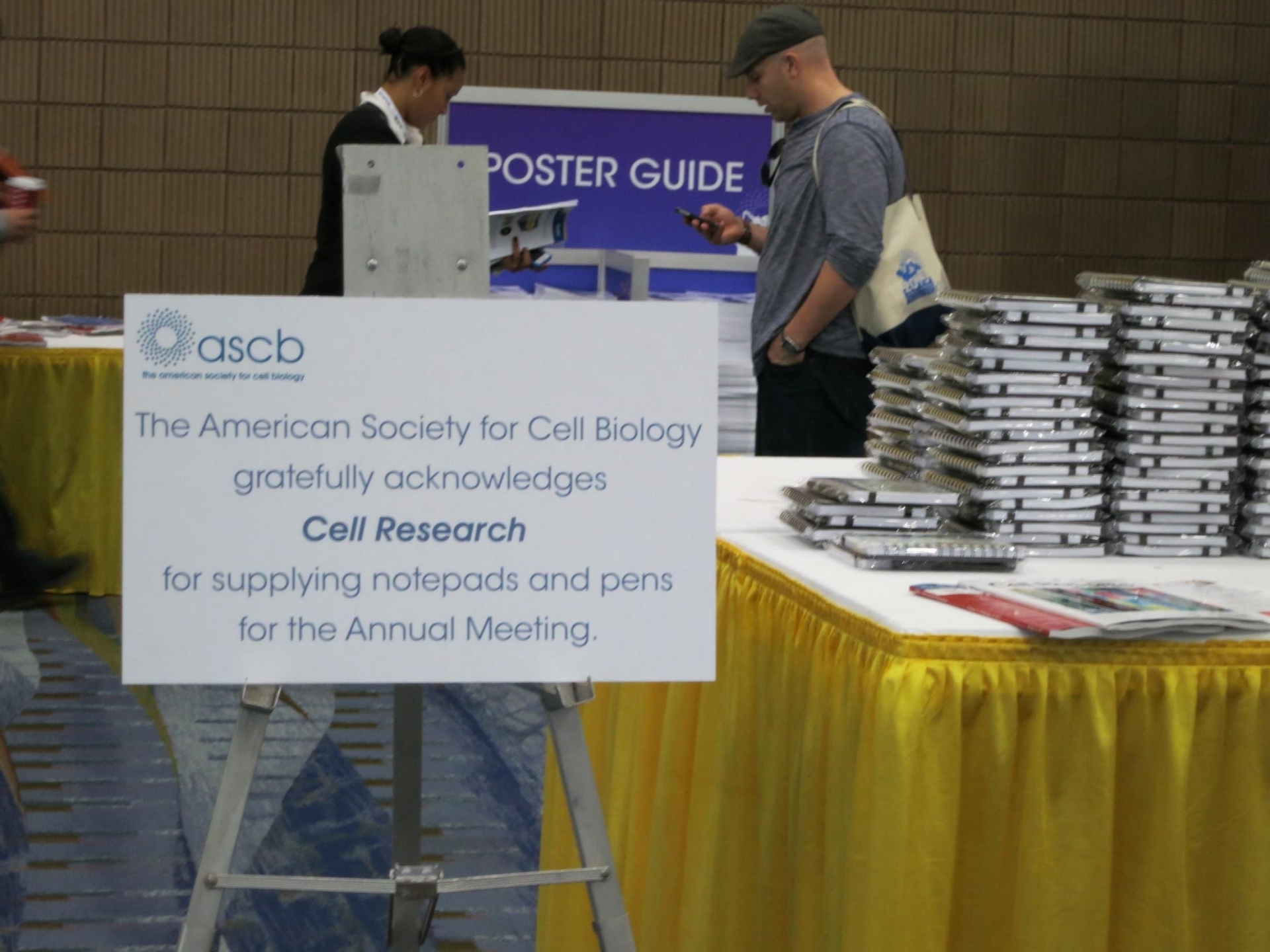
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gratefully acknowledges
Cell Research
for supplying notepads and pens
for the Annual Meeting.

Jena Johnson

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发件人：Jena Johnson<JJohnson@isscr.org>
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Dear Lei,

I hope you are well. The annual meeting was a fantastic success! Your journal was a hot item at our publication depot – I stopped to refill your supply a number of times through the program.

As promised, please find attached an updated web banner that is a static image, rather than the rotating banner we are currently running. Below is the link we would like this banner to direct to. Can you please update this when you have the chance?

http://www.isscr.org/home/regionalforums/singapore-2014?utm_source=cellresearch&utm_medium=banner&utm_campaign=singapore2014

Thanks so much!
Jena

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Autophagy in Stress, Development & Disease

Autophagy at the Intersection of Health and Disease

March 16-21, 2014
Renaissance Tuscany Il Ciocco Resort
Lucca (Barga), Italy

Chairs:
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Related Meeting Information

The Autophagy in Stress, Development & Disease Gordon Research Conference was held in conjunction with the **Autophagy in Stress, Development & Disease Gordon Research Seminar**. Please refer to the Autophagy in Stress, Development & Disease GRS web page for more information.

Autophagy is a cell biological process used to recycle cytoplasmic organelles and other constituents by delivering them to the lysosome for degradation. The past decade has seen an exponential expansion of our understanding of the molecular and cellular mechanisms of autophagy. Autophagy is tied to stress responses through a number of key signaling pathways, and is orchestrated by a series of core autophagy proteins (ATG proteins) that are evolutionarily conserved. The ATG proteins and genes constitute an integrated system that controls the internal milieu of the cell to support the multiple specialized functions that free-

Contributors

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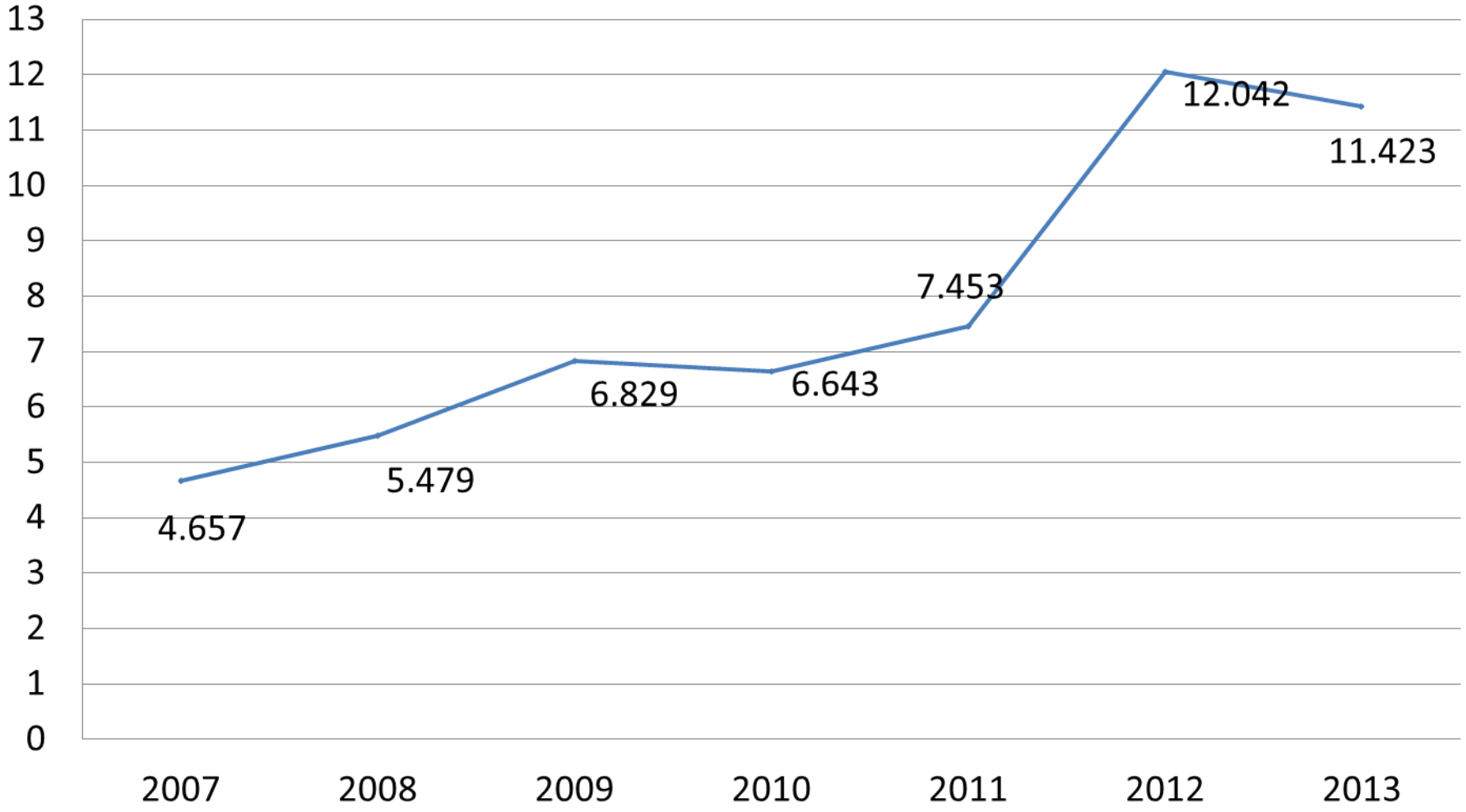

2013年发表热点专题CRISPR/Cas9研究论文

Original Article

Cell Research (2013) **23**:1163–1171. doi:10.1038/cr.2013.122; published online 27 Aug 2013

Multiplexed activation of endogenous genes by CRISPR-on, an RNA-guided transcriptional activator system

OPEN

Albert W Cheng[1,2,*], Haoyi Wang[1,*], Hui Yang[1], Linyu Shi[1], Yarden Katz[1,3], Thorold W Theunissen[1], Sudharshan Rangarajan[1], Chikdu S Shivalila[1,4], Daniel B Dadon[1,4] and Rudolf Jaenisch[1,4]

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Received 1 August 2013; Revised 10 August 2013; Accepted 13 August 2013
Advance online publication 27 August 2013

Abstract

Top

Technologies allowing for specific regulation of endogenous genes are valuable for the study of gene functions and have great potential in therapeutics. We created the CRISPR-on system, a two-component transcriptional activator consisting of a nuclease-dead Cas9 (dCas9) protein fused with a transcriptional activation domain and single guide RNAs (sgRNAs) with complementary sequence to gene promoters. We demonstrate that CRISPR-on can efficiently activate exogenous reporter genes in both human and mouse cells in a tunable manner. In addition, we show that robust reporter gene activation *in vivo* can be achieved by injecting the system components into mouse zygotes. Furthermore, we show that CRISPR-on can activate the endogenous *IL1RN*, *SOX2*, and *OCT4* genes. The most efficient gene activation was achieved by clusters of 3-4

Top 20 PDF

(*Period 1/1/13 -12/31/13)

Year	Title	Issue	Visits
2013	Multiplexed activation of endogenous genes by CRISPR-on, an RNA-guided transcriptional activator system	10	5508
2013	Generation of gene-modified mice via Cas9/RNA-mediated gene targeting	5	5035
2013	Genome editing with RNA-guided Cas9 nuclease in Zebrafish embryos	4	4370
2013	Efficient genome editing in plants using a CRISPR/Cas system	10	2042
2013	Targeted mutagenesis in rice using CRISPR-Cas system	10	1649

国际顶级科学家研究论文

Original Article

Cell Research (2013) **23**:1172–1186. doi:10.1038/cr.2013.112; published online 10 September 2013

Driving vascular endothelial cell fate of human multipotent Isl1$^+$ heart progenitors with VEGF modified mRNA

OPEN

Kathy O Lui[1,2,3,*], Lior Zangi[1,2,4,*], Eduardo A Silva[5,6], Lei Bu[1,2,3], Makoto Sahara[1,2], Ronald A Li[3,7], David J Mooney[5] and Kenneth R Chien[1,2,8]

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Received 7 November 2012; Revised 12 March 2013; Accepted 30 April 2013
Advance online publication 10 September 2013

Abstract

Distinct families of multipotent heart progenitors play a central role in the generation of diverse cardiac, smooth muscle and endothelial cell lineages during mammalian cardiogenesis. The identification of precise paracrine signals that drive the cell-fate decision of these multipotent progenitors, and the development of novel approaches to deliver these signals *in vivo*, are critical steps towards unlocking their regenerative therapeutic potential. Herein, we have identified a family of human cardiac endothelial intermediates located in outflow tract of the early human fetal hearts (OFT-ECs), characterized by coexpression of Isl1 and CD144/vWF. By comparing angiocrine factors expressed by the human OFT-ECs and non-cardiac ECs, vascular endothelial growth factor (VEGF)-A was identified as the most abundantly expressed factor, and clonal assays documented its ability to drive endothelial specification of human embryonic stem cell (ESC)-derived Isl1$^+$ progenitors in a VEGF receptor-dependent manner. Human Isl1-ECs (endothelial cells differentiated from hESC-derived ISL1$^+$ progenitors) resemble OFT-ECs in terms of expression of the cardiac endothelial progenitor- and endocardial cell-specific genes, confirming their organ specificity. To determine whether VEGF-A might serve as an *in vivo* cell-fate switch for human ESC-derived Isl1-ECs, we established a

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nature biotechnology

Receptive to replication

Do replication studies belong in top-tier journals?

Like many other scientific journals, *Nature Biotechnology* places a strong emphasis on novelty when selecting research for publication. As a result, studies describing replications or confirmations of previously published reports are less likely to be chosen. And studies detailing null or negative findings may not meet stringent editorial requirements for significance and relevance to our broad readership. Why then are we publishing on p. 965 a replication of a report published elsewhere in the literature?

One reason is that the new report, resulting from a collaboration between miRagen Therapeutics and Monsanto, clarifies what were controversial findings in a paper previously published in *Cell Research* (**22**, 107–126, 2012). The latter study, led by Chen-Yu Zhang of Nanjing University, China, required a corrigendum (*Cell Res.* **22**, 273–274, 2012) and sparked vigorous debate because it reported the presence of plant microRNA (miRNA) in human blood plasma and suggested that one in particular, miRNA168a, from ingested rice could traverse into the circulation of mice resulting in the modulation of miRNA target genes in the animal.

In contrast to these findings, the report on p. 965 finds no evidence for uptake of plant miRNA168a in the plasma and liver of mice fed a rice diet. Enzyme-linked immunosorbent assay data from the current study also contradict western blots from the Zhang paper that suggested miR168a directly suppressed levels of low-density lipoprotein receptor adapter pro-

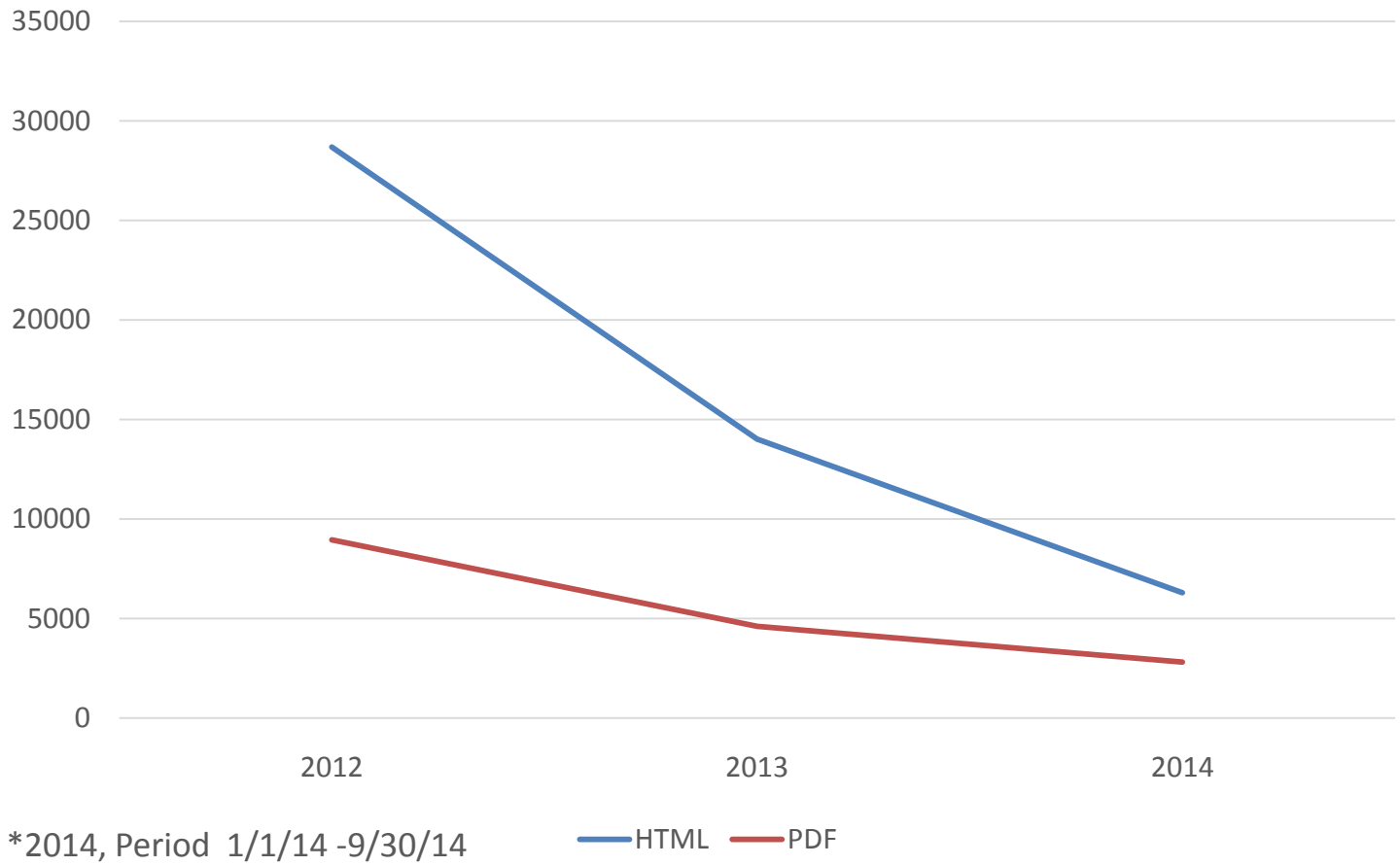
When an initial report prompts this level of concern and involves a considerable investment of time, effort and resources from both researchers and regulators in evaluating its findings and understanding its implications, then a carefully controlled and executed replication study clearly warrants publication. It is unfortunate it was not published in *Cell Research* where it could have been bi-directionally linked to the original paper.

Providing space for publishing a replication study is one way in which *Nature Biotechnology* and other top-tier journals can facilitate the process of self-correction of the scientific literature. Journals could also actively solicit papers that seek to replicate research studies where corroboration by independent laboratories would be of particular interest (e.g., to corroborate a controversial finding).

The Reproducibility Initiative (*Nat. Biotechnol.* **30**, 806, 2012) represents another way of replicating research. A collaboration between the Science Exchange and *PLOS ONE*, the initiative offers to broker independent validation of a researcher's work in return for a fee, with subsequent publication in the journal. In October, the Laura and John Arnold Foundation provided $1.3 million to the initiative to authenticate 50 high-profile cancer papers from the past two years (only ~$20,000 per study).

But the faction with the greatest motivation to replicate academic findings must surely be industry. Companies have the deepest financial resources, and they have the most to gain. And it was groups at Amgen

Downloads



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Original Article

Cell Research advance online publication 24 October 2014; doi: 10.1038/cr.2014.135

Discovery of the migrasome, an organelle mediates release of cytoplasmic contents during cell migration

OPEN

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Received 28 August 2014; Revised 6 September 2014; Accepted 25 September 2014
Advance online publication 24 October 2014

Abstract

Top

Cells communicate with each other through secreting and releasing proteins and vesicles. Many cells can migrate. In this study, we report the discovery of migracytosis, a cell migration-dependent mechanism for releasing cellular contents, and migrasomes, the vesicular structures that mediate migracytosis. As migrating cells move, they leave long tubular strands, called retraction fibers, behind them. Large vesicles, which contain numerous smaller vesicles, grow on the tips and intersections of retraction fibers. These fibers, which connect the vesicles with the main cell body, eventually break, and the vesicles are released into the extracellular space or directly taken up by surrounding cells. Since the

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