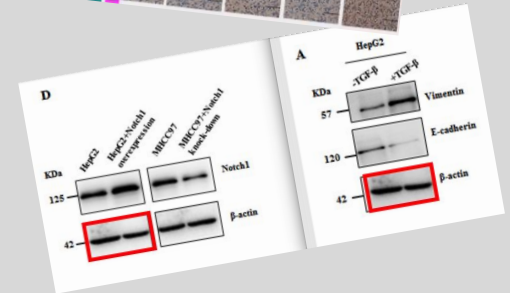
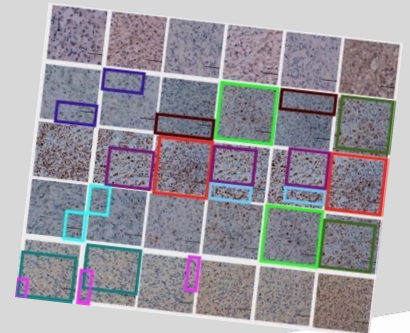
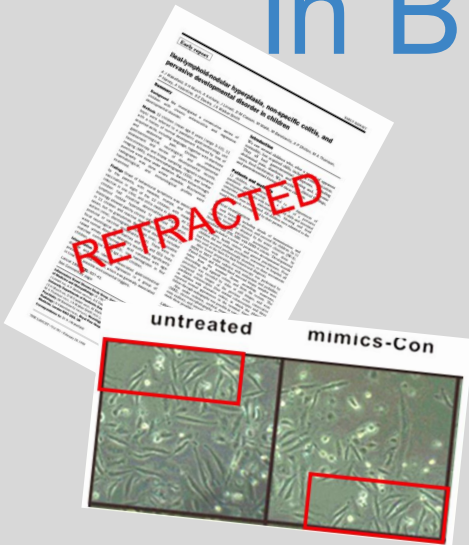


Double Trouble: Inappropriate Image Duplications in Biomedical Publications

Elisabeth Bik

www.ScienceIntegrityDigest.com

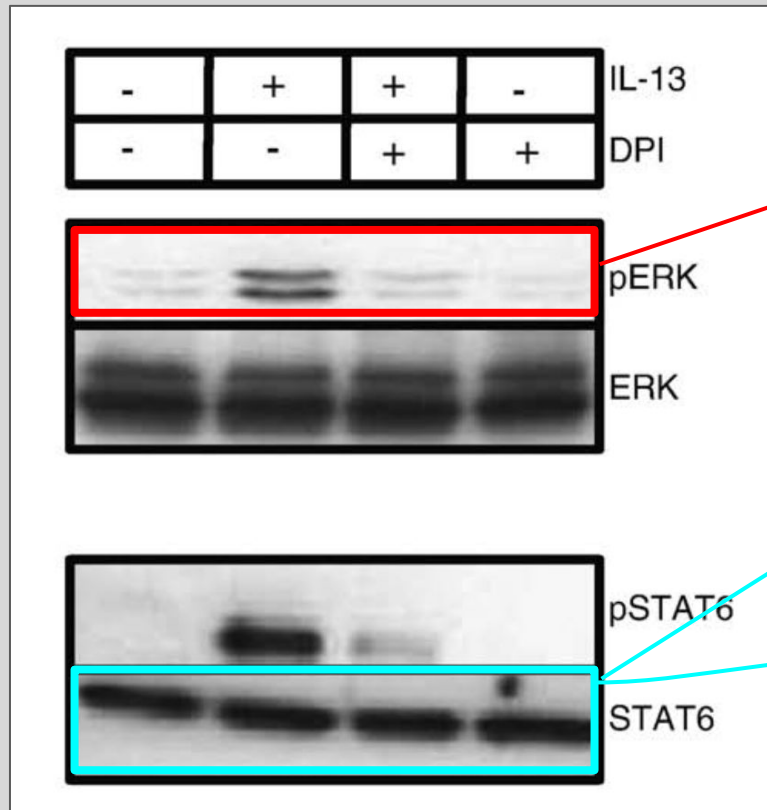
Twitter: @MicrobiomDigest



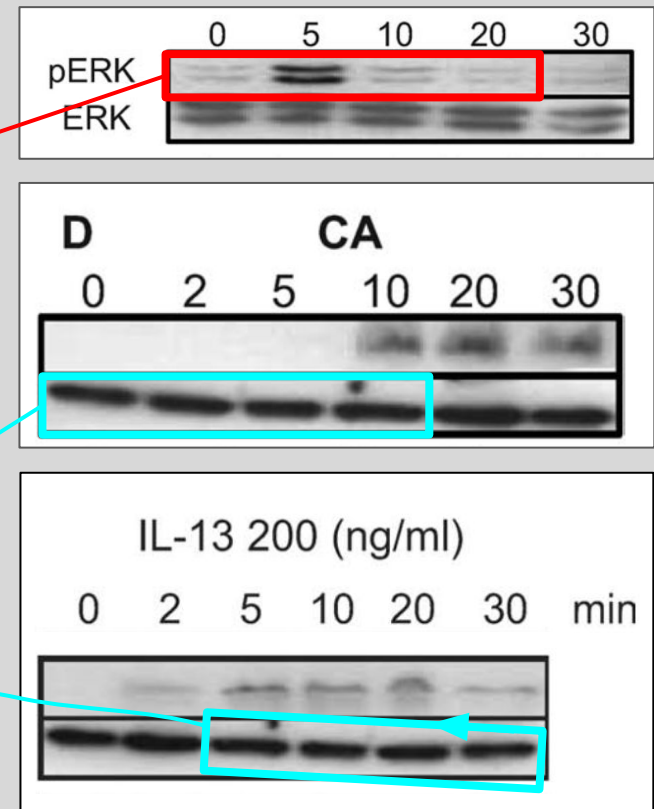
Financial disclosures:

- I receive consulting and speaker fees
- I receive donations through Patreon.com
- I am listed on 4 uBiome patents
- *uBiome founders charged with insurance fraud*

My first image duplication finding, 2014



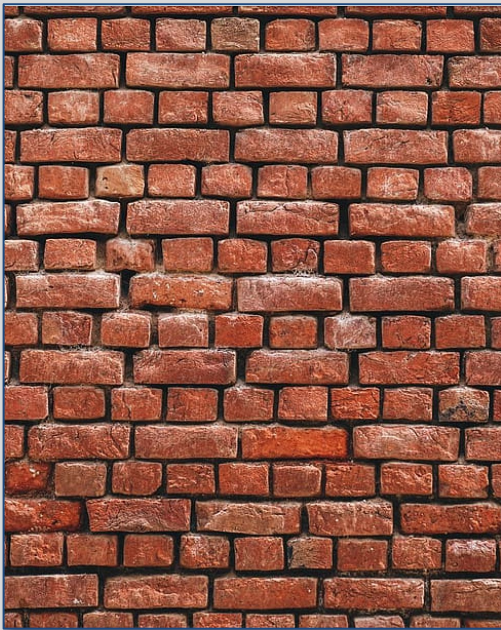
Cellular Signalling 22 (2010) 1485–1494
 DOI: 10.1016/j.cellsig.2010.05.017
 Reported Jan 2014, Retracted Sept 2016
 Cited by 44



Inflamm Bowel Dis 2010;16:753–764
 DOI: 10.1002/ibd.21133
 Reported Jan 2014, Retracted July 2015
 Cited by 43

Publications are the foundation of science

- Science is about finding the truth
- Science builds upon science: Publications as building blocks
- Built on trust, but science is not immune to fraud



www.piqsels.com



Ivan Radic, www.Flickr.com

Behind each misconduct case is a sad story

Cellular Signalling 22 (2010) 1485–1494

Contents lists available at ScienceDirect

Cellular Signalling

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



REDOX regulation of IL-13 signaling in intestinal epithelial cells: Usage of alternate pathways mediates distinct gene expression patterns



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ARTICLE INFO

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MAPK
TFF3
Bcl-xl

ABSTRACT

In the classic view interleukin-13 (IL-13) binds to a heterodimer protein complex of the IL-13Rα1 and IL-4Rα chains and signals through Janus kinase 1 (JAK1)-signal transducer and activator of transcription 6 (STAT6) mechanism. We recently reported that IL-13 also signals through the IL-13Rα2 chain initiating all three mitogen activated protein kinase (MAPK) pathways, and the relative expression of IL-13Rα1 and IL-13Rα2 modulates one another's transduction pathway. Therefore we investigated whether generation of reactive oxygen species (ROS) as second messengers may serve as a common nexus between these two pathways emanating from the individual receptor chains in intestinal epithelial cells (IEC). IL-13 stimulates intracellular reactive oxygen species synthesis within 5 min via IL-13Rα1-JAK1-STAT6- and IL-13Rα2-MEK1/2-ERK1/2-dependent activation of NADPH oxidase (NADPH oxidase-1 (NOX-1)). IL-13-induced ROS also constitutively regulates phosphorylation of ERK1/2 and STAT6, yielding a feed forward amplification loop. IL-13 also stimulates the stable, long-term gene expression of two other NADPH oxidase, NOX-2 and DUOX-2, which along with constitutive NOX-1, might facilitate elevated, continuous production of ROS in IL-13-activated IEC. The contribution of each signal transduction pathway mediated by ROS to such biological functions as wound healing, inflammation, and apoptosis varied by pathway and dependent on the biological context. Distinct usage patterns were observed, demonstrating not only that IL-13 signal transduction through STAT6, MAPK, and ROS is regulated in both an antagonistic and cyclic fashion, but also that each pathway plays a specific role in modulating the wound healing and anti-apoptotic capabilities of the intestinal epithelium.

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1. Introduction

Interleukin (IL)-13 is a proinflammatory cytokine secreted predominantly by activated T helper type 2 (Th₂) cells, NKT cells, human natural killer (NK) cells, renal cell carcinomas, and Hodgkin's lymphoma-derived tumor cells [1]. It is involved in allergic inflammation, emphysema, goblet cell hyperplasia, tumor cell growth,

helminth expulsion, and suppression of tumor immunosurveillance [1]. IL-13's diverse function is mediated by a complex receptor system including sharing the IL-4Rα chain and two other cognate cell surface proteins, IL-13Rα1 and IL-13Rα2 [2]. Consensus opinion maintains that IL-13 binds to IL-13Rα1, which then forms a heterodimer with IL-4Rα to craft a functional signaling complex that activates STAT6 [2]. In expanding this narrow view we recently reported that IL-13Rα2, previously considered a decoy receptor, uses the MAPK pathways to transduce a signal [3]. In addition, the relative expression of IL-13Rα1 and IL-13Rα2 modulates one another's transduction pathway, thus potentially biasing the pattern of gene activation and hence cell function [3]. Therefore we sought to identify a common nexus between these two pathways emanating from the individual IL-13 receptor chains. Reactive oxygen species (ROS) are widely recognized as important mediators of cell growth, adhesion, differentiation, senescence, and apoptosis [4]. Proteins with low-pKa cysteine residues, which are susceptible to oxidation by ROS, include the transcription factors nuclear factor-κB [5] and activator protein-1

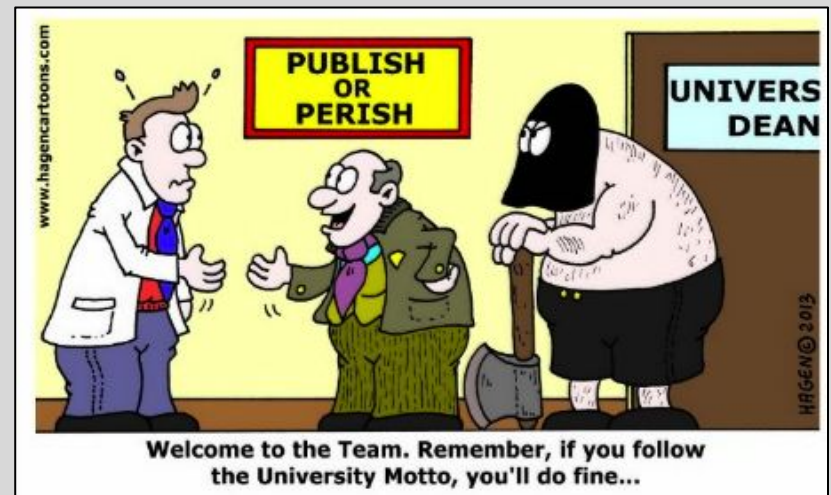
Abbreviations: COX, cytochrome c oxidase; DPI, diphenylene iodonium; DUOX, dual oxidase; H₂O₂, hydrogen peroxide; HBSS, Hank's balanced salt solution; IEC, intestinal epithelial cells; IL, interleukin; JAK, Janus kinase; MAPK, mitogen activated protein kinase; MFI, mean fluorescence intensity; NADPH, nicotinamide adenine dinucleotide phosphate; NOX, NADPH oxidase; Rac1, Ras-related C3 botulinum toxin substrate 1; ROS, reactive oxygen species; SEM, standard error of the mean; STAT, signal transducer and activator of transcription; TFF3, intestinal trefoil factor 3.

* Corresponding author. Department of Medicine, Case Western Reserve University School of Medicine 10900 Euclid Avenue, Cleveland, OH 44106-4952, United States. Tel.: +1 216 368 0342; fax: +1 216 368 0647.

E-mail address: alan.levine@case.edu (A.D. Levine).

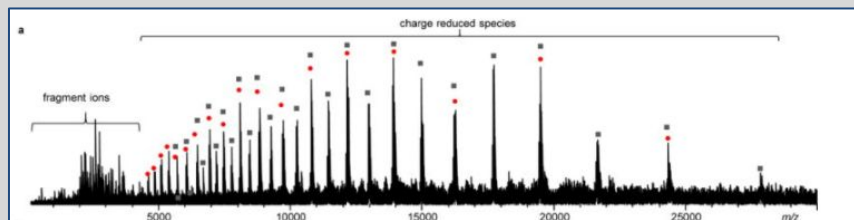
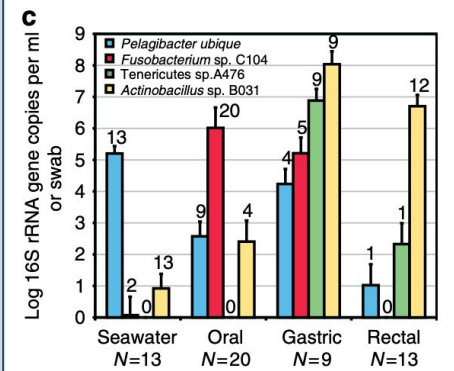
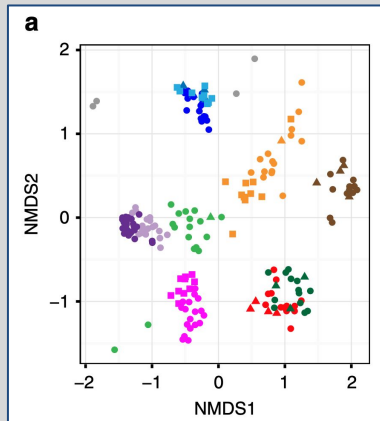
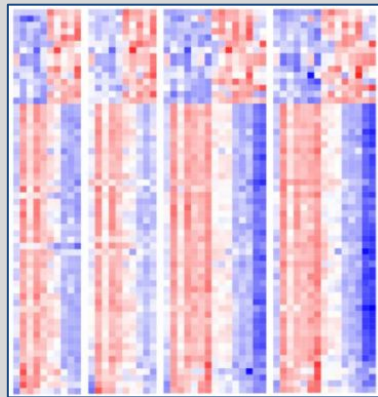
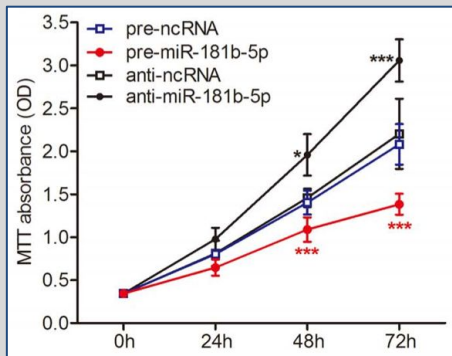
0898-6568/\$ – see front matter © 2010 Elsevier Inc. All rights reserved.
doi:10.1016/j.cellsig.2010.05.017

- Why do scientists commit fraud?
- Which author is responsible?
- All authors will be damaged
- Paper concerns vs. who did it

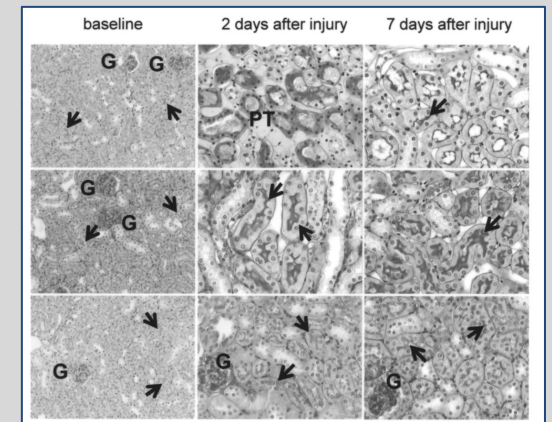
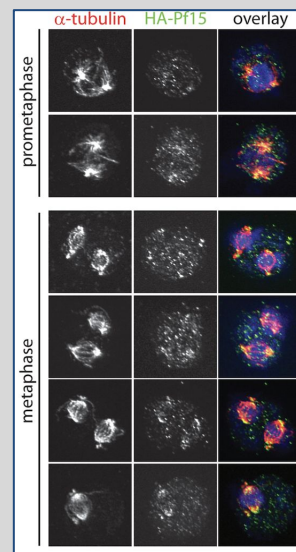
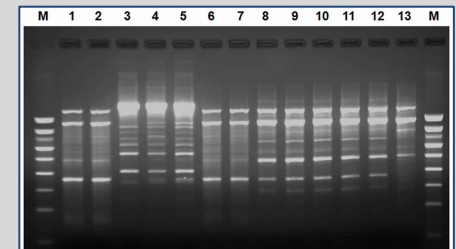
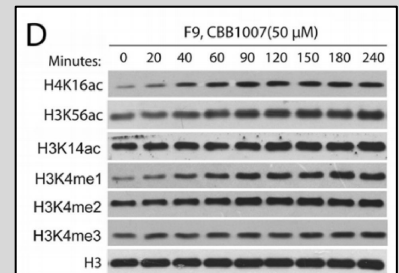
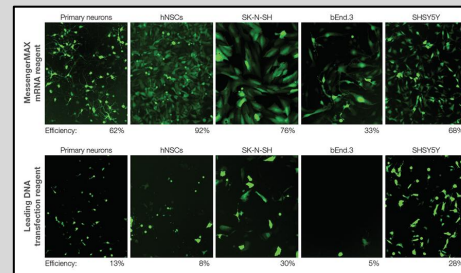


Figures found in scientific papers

Line graphs



Photos



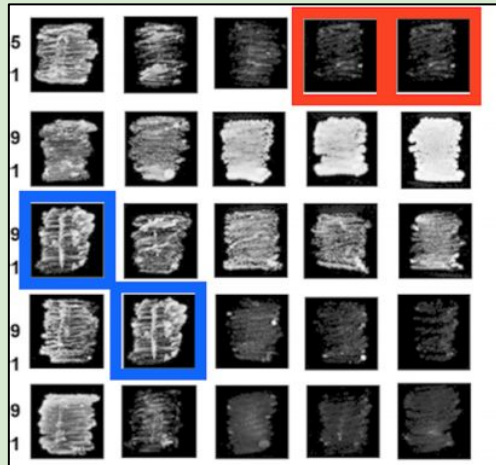
The temptation of image manipulation



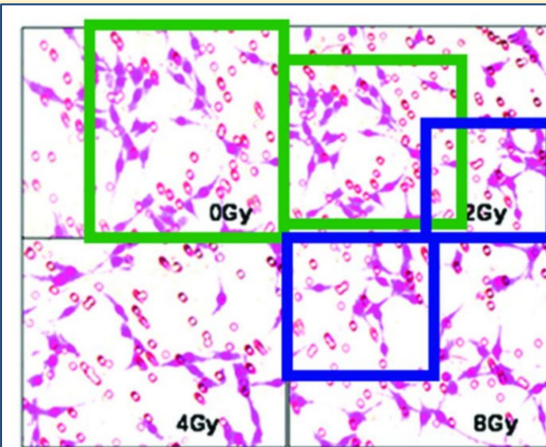
Source: Redbook Magazine, Anna Holmes, Jezebel / Washington Post

Inappropriate image duplication

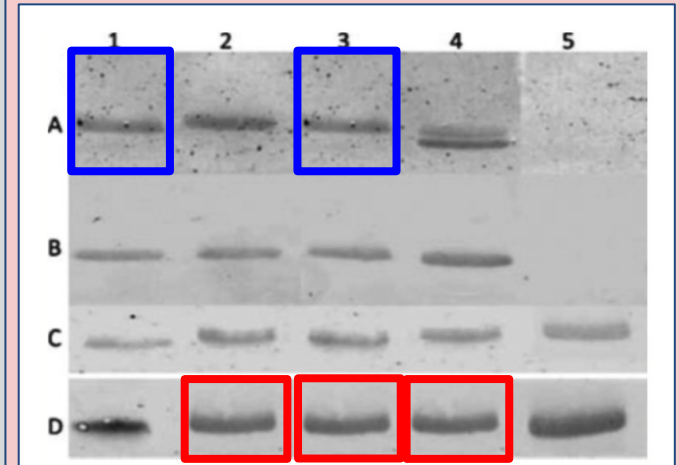
I: Simple



II: Repositioned



III: Alteration

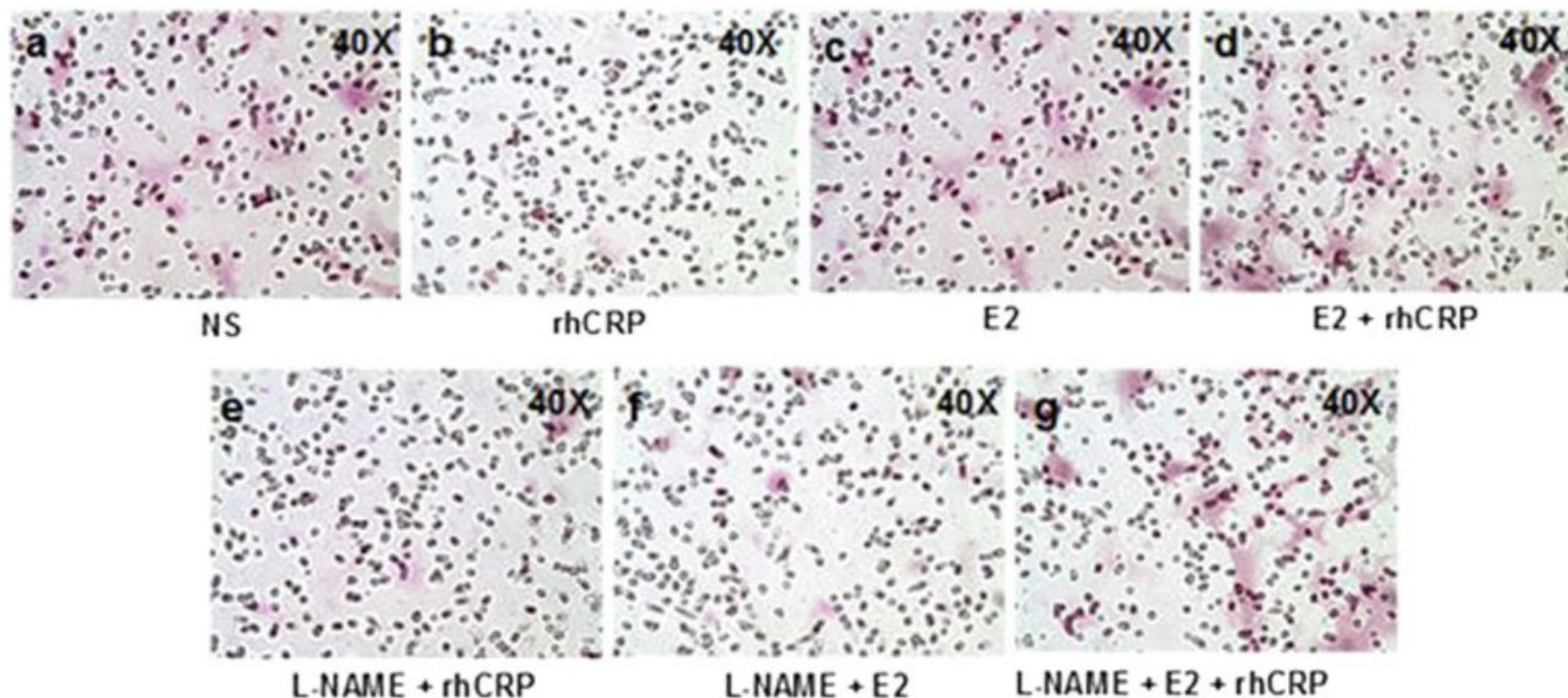


Honest error



Deliberately

Type I: Simple Duplication



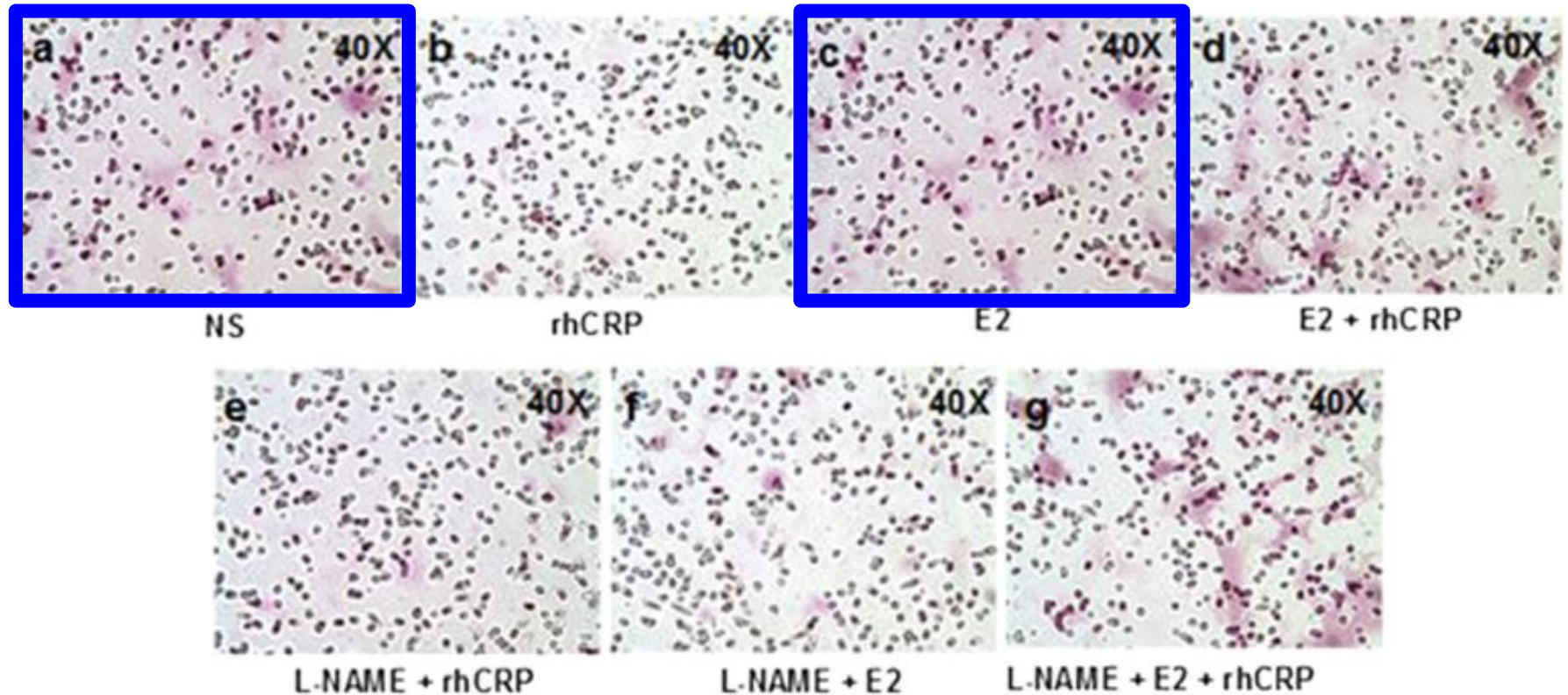
Estradiol inhibits vascular endothelial cells pro-inflammatory activation

Montreal Heart Institute, Canada

Molecular and Cellular Biochemistry (2013) , DOI: 10.1007/s11010-012-1482-9

Reported to journal: October 2015. No action yet.

Type I: Simple Duplication



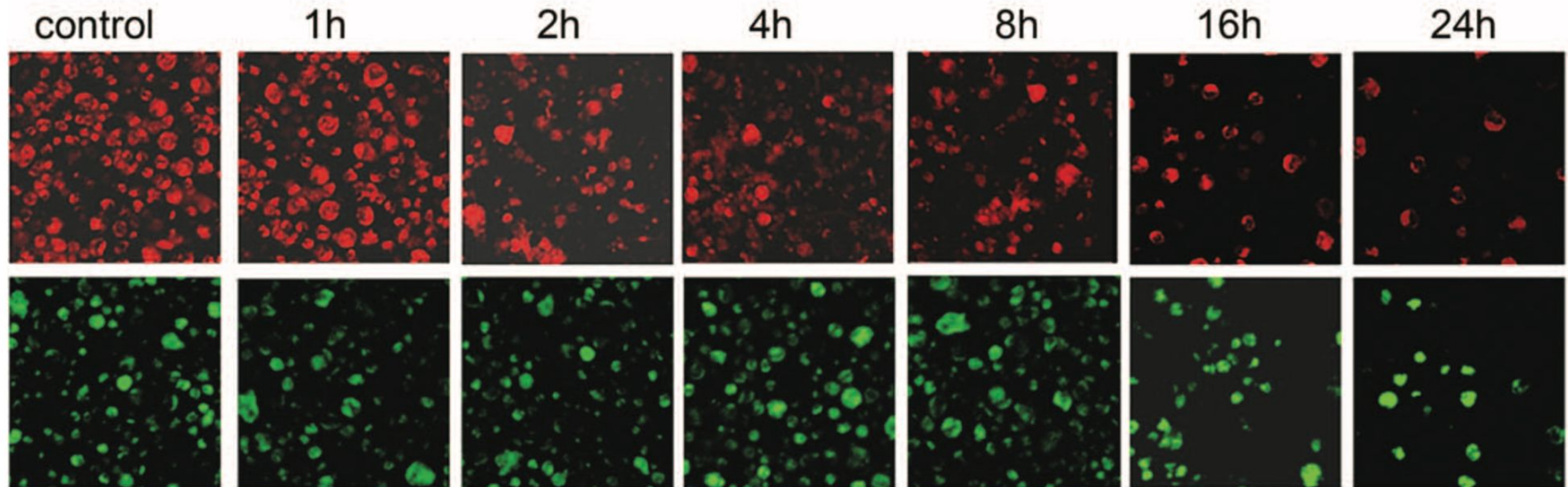
Estradiol inhibits vascular endothelial cells pro-inflammatory activation

Montreal Heart Institute, Canada

Molecular and Cellular Biochemistry (2013) , DOI: 10.1007/s11010-012-1482-9

Reported to journal: October 2015. No action yet.

Type II: Duplication with repositioning



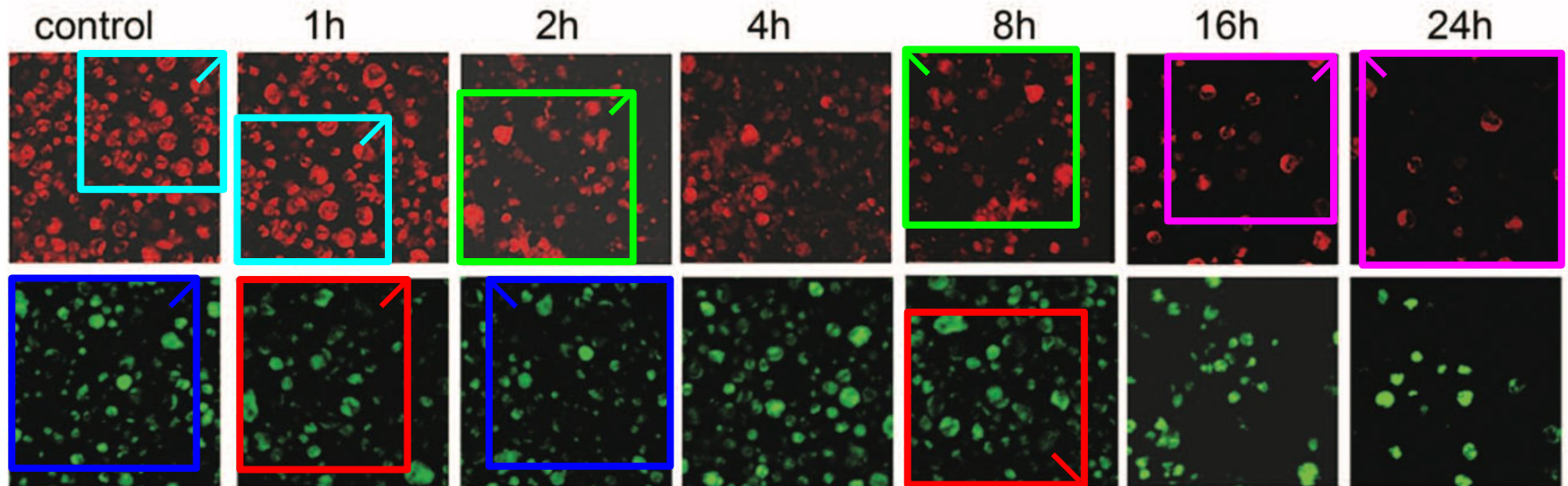
Apoptosis of myelodysplastic syndrome cell line by plant alkaloids

Department of Hematology, Zhejiang University School of Medicine, Hangzhou, China

Leukemia & Lymphoma (2007), DOI: 10.1080/10428190701216360

Reported Oct 2015, retracted March 2016, cited 19 times

Type II: Duplication with repositioning



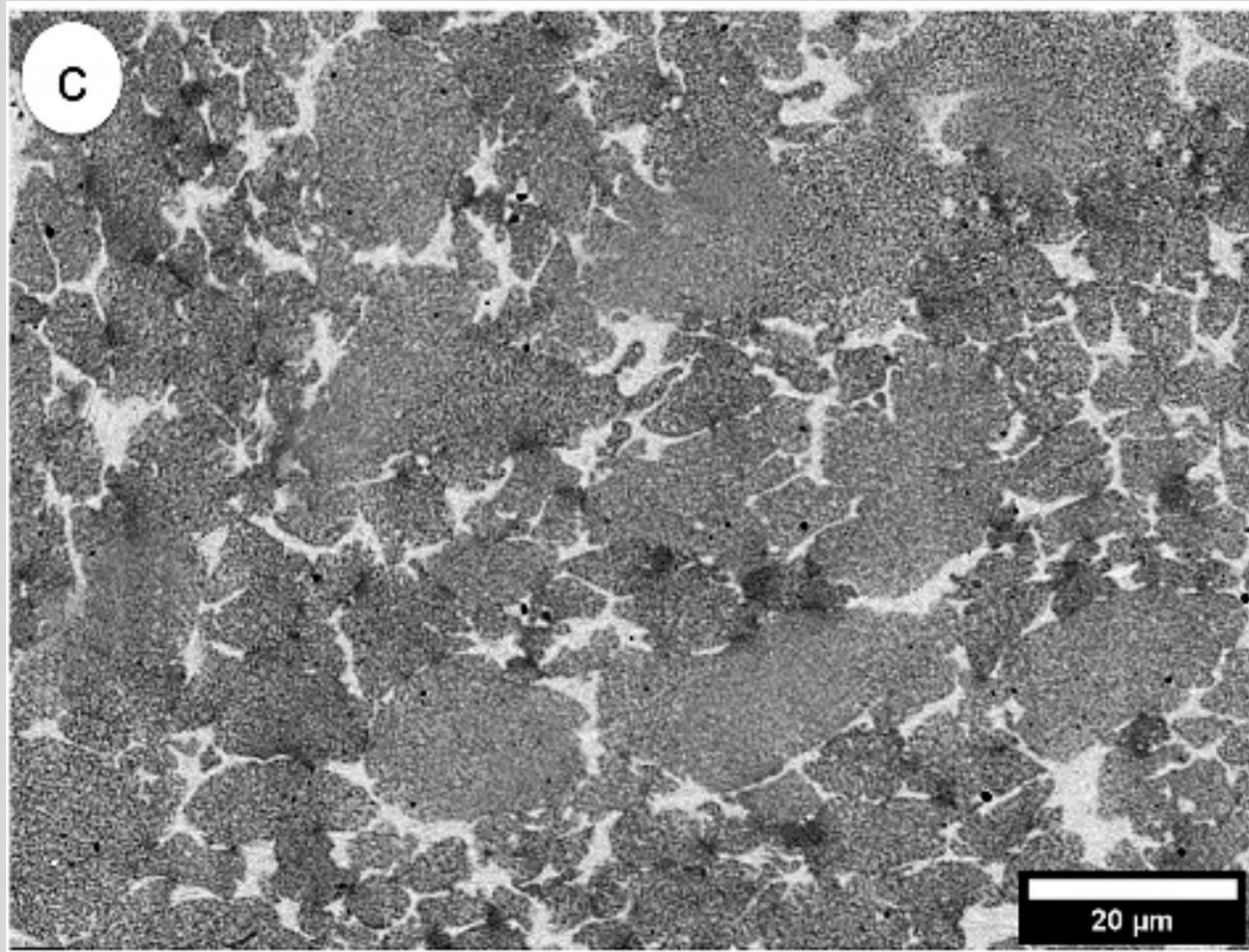
Apoptosis of myelodysplastic syndrome cell line by plant alkaloids

Department of Hematology, Zhejiang University School of Medicine, Hangzhou, China

Leukemia & Lymphoma (2007), DOI: 10.1080/10428190701216360

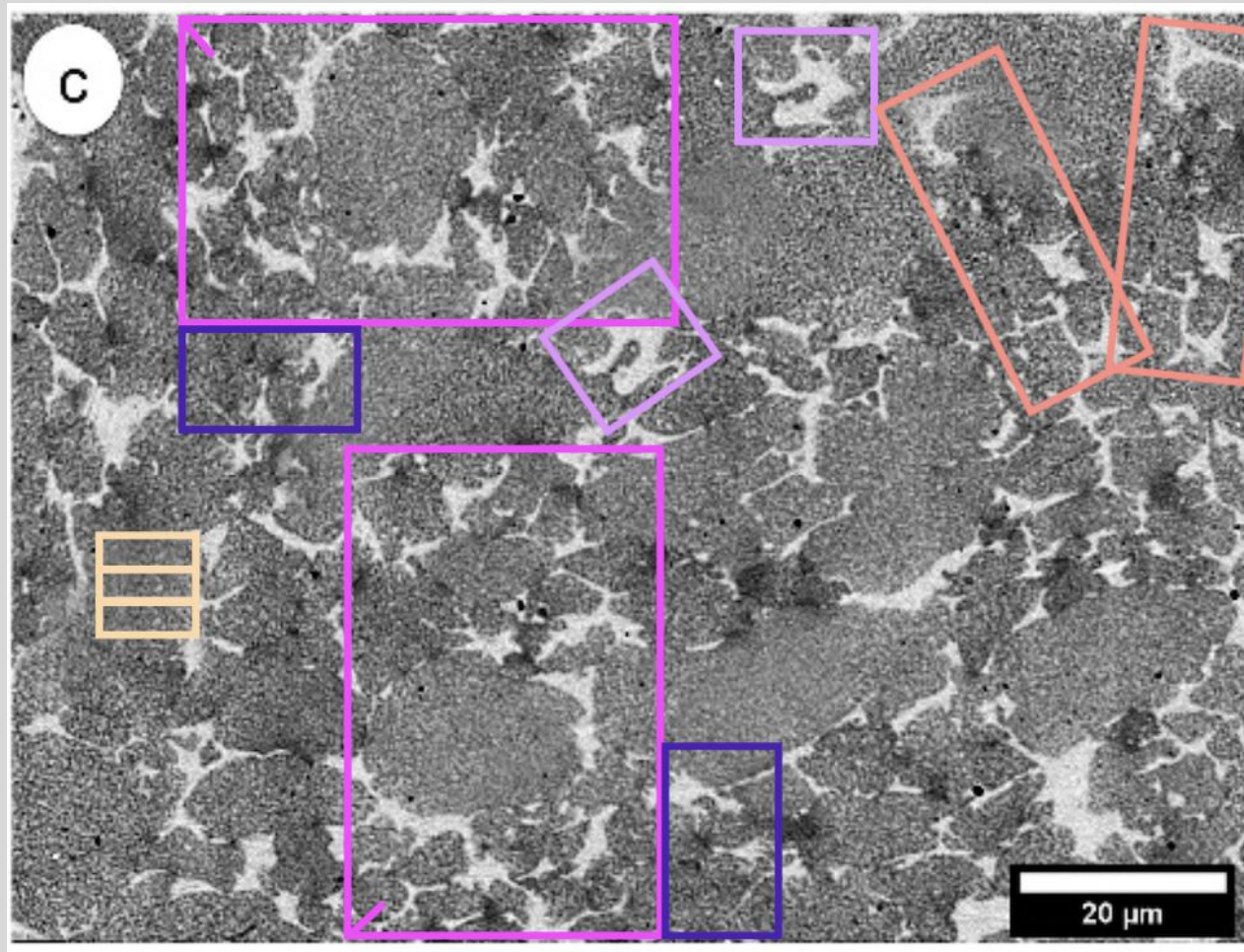
Reported October 2015, retracted March 2016, cited 19 times

Type III: Duplication with Alteration



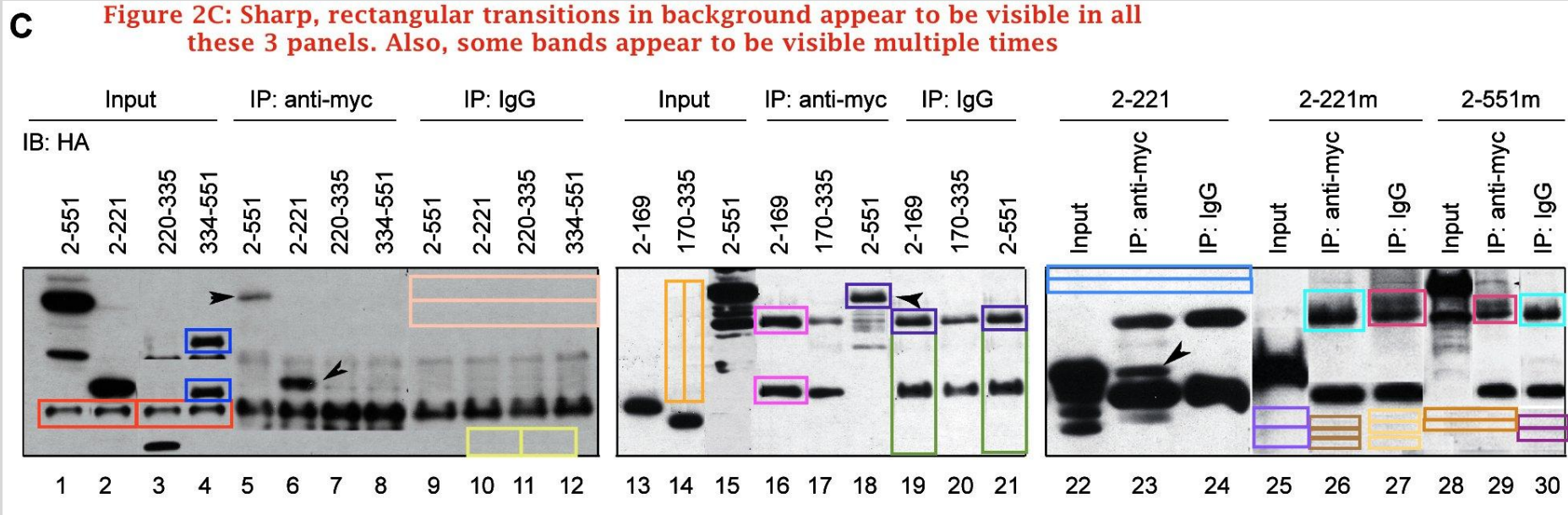
*National University of Science and Technology, Moscow, Russia
Materials (2022), DOI: 10.3390/ma15207366
Reported online September 2023 (first by Alexander Magazinov)*

Type III: Duplication with Alteration



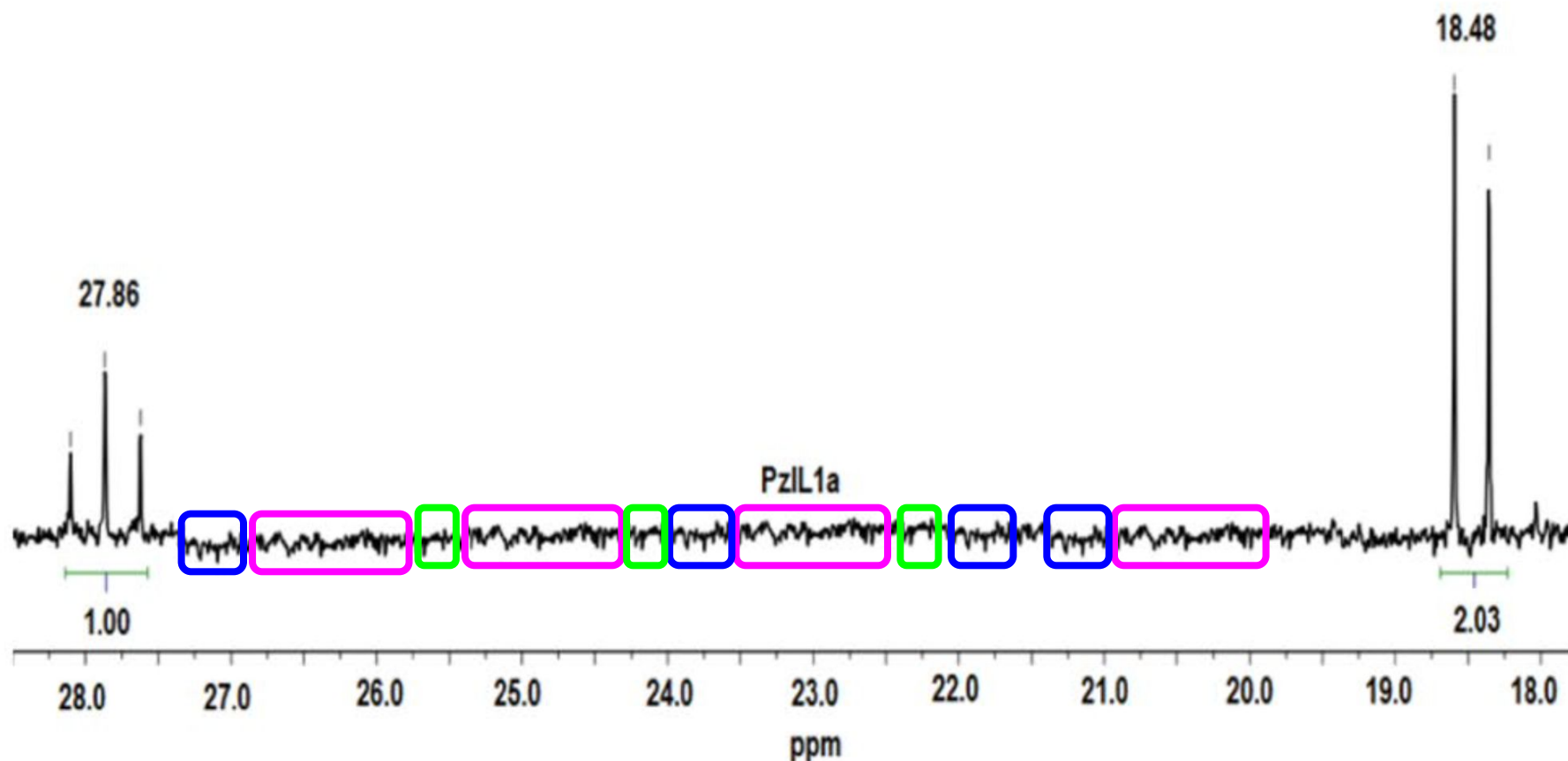
*National University of Science and Technology, Moscow, Russia
Materials (2022), DOI: 10.3390/ma15207366
Reported online September 2023 (first by Alexander Magazinov)*

Type III Duplication: Western blot art



NF- κ B-mediated signaling
School of Dentistry, University of California, Los Angeles, CA
PLOS ONE (2014), DOI: 10.1371/journal.pone.0096211
Reported online July 2023

Type III Duplication: NMR spectrum



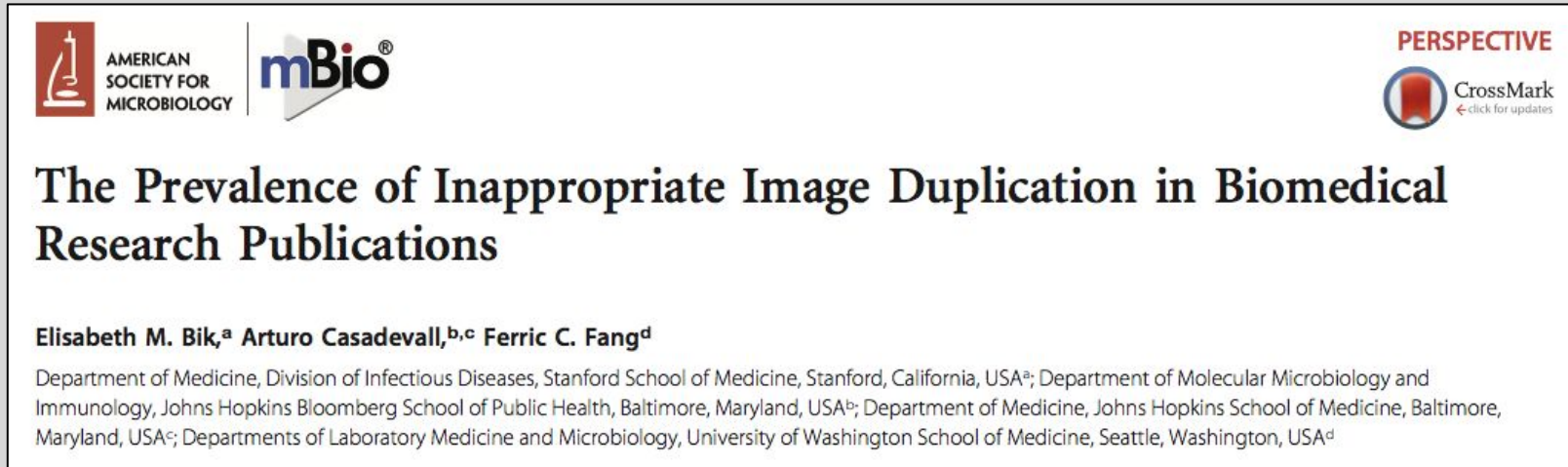
Trimeric phosphazene-based ionic liquids with tetrafluoroborate anions

Dumlupınar University, Turkey

Scientific Reports (2020), DOI: 10.1038/s41598-020-68709-5, cited by 2

Reported August 2020, retracted March 2021

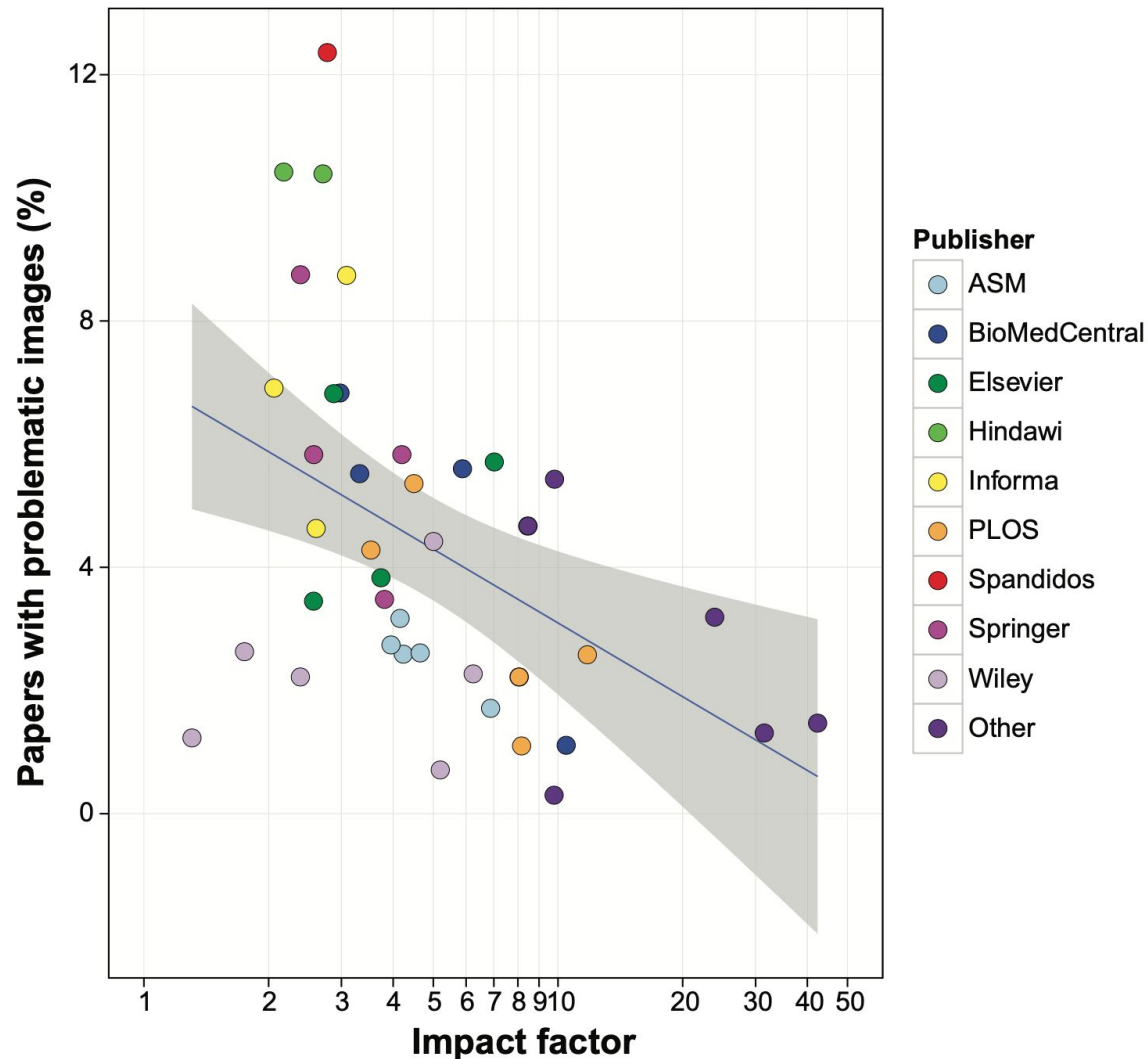
Inappropriate image duplication



- I scanned 20,621 papers from 1995-2014 - by eye
- 40 journals from 14 publishers
- Found ~ 800 papers with duplicated figures (4%)
- 3 types: Simple - Repositioned - Altered
- Not all are misconduct! About half intentional: 2%
- Alteration in other data types much harder to detect

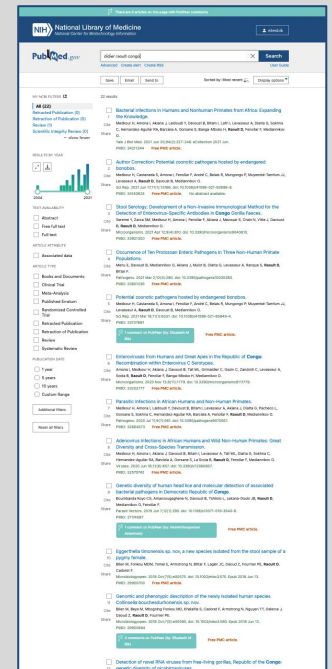
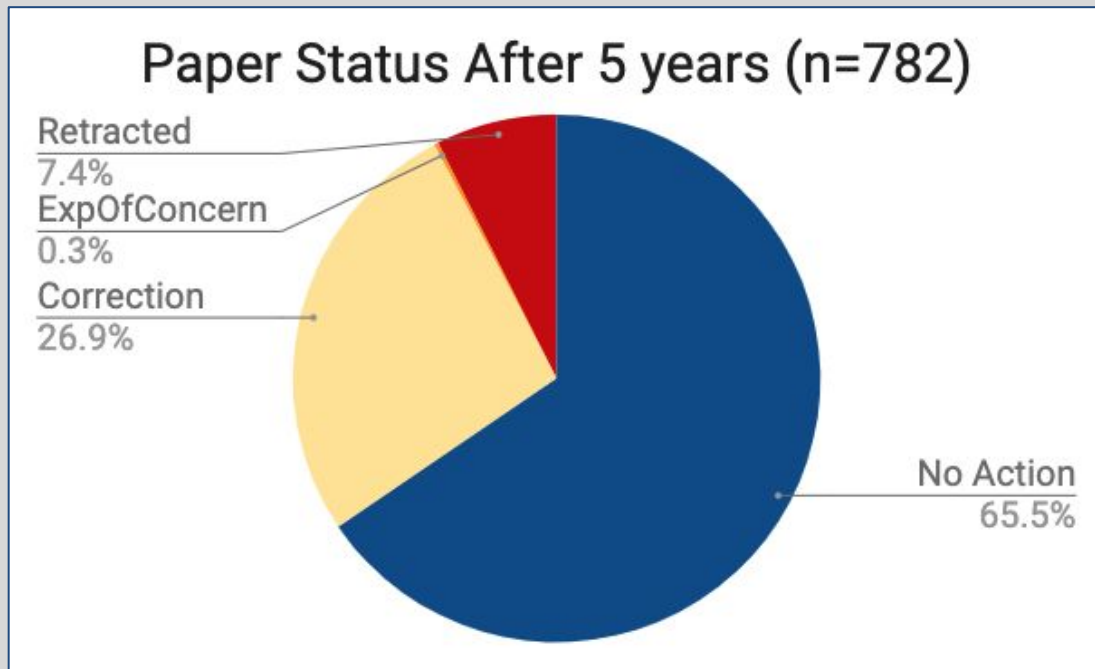
Higher Impact Factor, fewer problems

Bik EM et al. mBio 7(3):e00809-16 (2016), DOI: 10.1128/mBio.00809-16.



Journals are very slow to respond

- 2015: 782 papers reported to journals
- 2020: 66% of papers not corrected/retracted five years after reporting
- September 2023: 7,307 papers found
- 2,837 reported to journals/institutions; and 7,006 on PubPeer.com
- 1049 retractions, 999 corrections



Institutions vary widely in addressing fraud

The New York Times

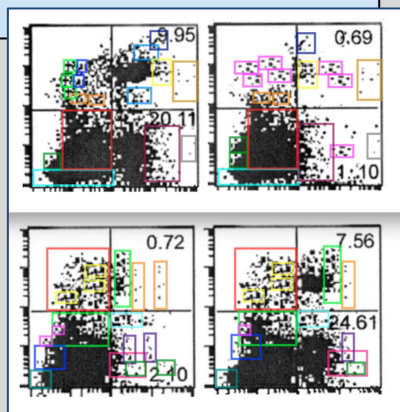
Years of Ethics Charges, but Star Cancer Researcher Gets a Pass

Investigation conclusions and handling opinions on 63 papers by academician X: "After investigation, no fraud, or plagiarism was found...."

www.most.gov.cn



www.complianceandethics.org



The Stanford Daily

Stanford president's research under investigation for scientific misconduct, University admits 'mistakes'

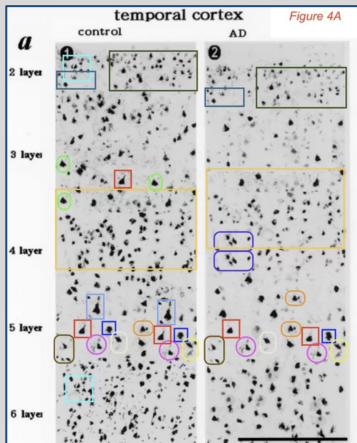
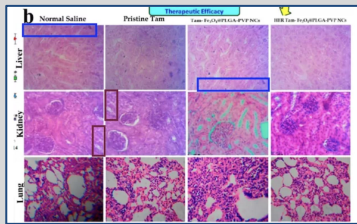
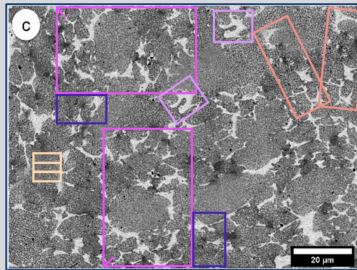
Additional papers by University president in question, structure of Board's investigation criticized

Stanford president resigns over manipulated research, will retract at least three papers

Marc Tessier-Lavigne failed to address manipulated papers, fostered unhealthy lab dynamic, Stanford report says

By Theo Baker

Authors' explanations



- "these similarities are entirely anticipated and are caused by (non-uniform) potent applied current within the material"
- Tissues "may look similar due to the same organ cell architecture"
- With software at that time "neurons from the previous image remained in the next image, probably because they were not completely erased, and that they were scattered in the next image"

Artificial intelligence can create fake papers

FORBES > BUSINESS

BREAKING

Fake Scientific Abstracts Written By ChatGPT Fooled Scientists, Study Finds

Brian Bushard Forbes Staff
I cover breaking news for Forbes

Follow

Jan 10, 2023, 01:44pm EST

Science journals ban listing of ChatGPT as co-author on papers

Some publishers also banning use of bot in preparation of submissions but others see its adoption as inevitable

Ian Sample Science editor

@iansample

Thu 26 Jan 2023 19:00 GMT

The Guardian

The New York Times

Tiffany Hsu and Stuart A. Thompson, February 2023

Disinformation Researchers Raise Alarms About A.I. Chatbots

Researchers used ChatGPT to produce clean, convincing text that repeated conspiracy theories and misleading narratives.

Proceedings of the Second International Conference on Applied Artificial Intelligence and Computing (ICAAIC 2023)
IEEE Xplore Part Number: CFP23BC3-ART; ISBN: 978-1-6654-5630-2

Design and Implementation of Smart Hydroponics Farming for Growing Lettuce Plantation under Nutrient Film Technology

Raspberry Pi 3 was provided. The information and the forecast were saved in a database called Firebase, which is hosted in the cloud [16].

As an AI language model, there is no access to the specific database details of any particular research study. However, in general, a well-designed database for a hydroponics system should include the following:

Tables: Tables should be created to store data related to each aspect of the hydroponics system. For example, there may be a table for sensor readings, a table for system settings, and a table for user data.

Columns: Each table should have relevant columns that store specific types of data. For example, the sensor readings table may have columns for data and time, temperature

time data and system control.

E. The Results Provided

Discussions and the supporting illustrations on the research findings observed. As an AI language model, I don't have access to the specific results and findings of any particular research study. However, some general guidance is provided on how a research study should report and discuss its findings. In general, the results section of a research study should provide a clear and concise presentation of the data and findings. This can include tables, figures, and statistical analysis to support the results. The discussion section should then provide a more detailed interpretation and explanation of the results, including any limitations of the study and implications for future research.

Artificial intelligence can create fake images



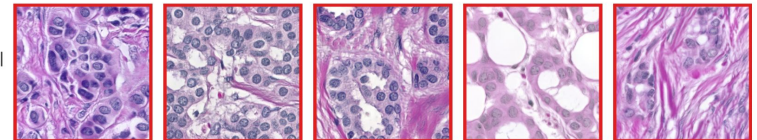
AI-Enabled Image Fraud in Scientific Publications

Jinjin Gu^a, Xinlei Wang^a, Chenang Li^b, Junhua Zhao^{b,c,1}, Wei Jin Fu^{d,2}, and Gaoqi Liang^b

^aSchool of Electrical and Information Engineering, University of Sydney, Sydney, Australia; ^bSchool of Science and Engineering, The Chinese University of Hong Kong, Shenzhen, China; ^cShenzhen Institute of Artificial Intelligence and Robotics for Society (AIRS), China; ^dDepartment of Urology, the First Affiliated Hospital of Guangxi Medical University, China

This manuscript was compiled on December 2, 2021

histopathological
images



cell
immunostaining
images

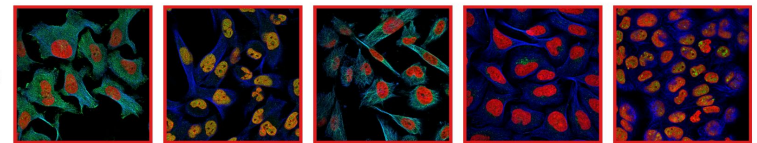
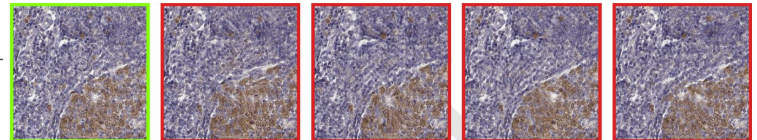


Fig. 1. Sampling fake images from a well-trained generative model. These fake images are created by an advanced generation technology called StyleGAN (6). All these images are fake and meaningless in science.

immunohistoche-
mistry images



scanning
micrographs of
materials

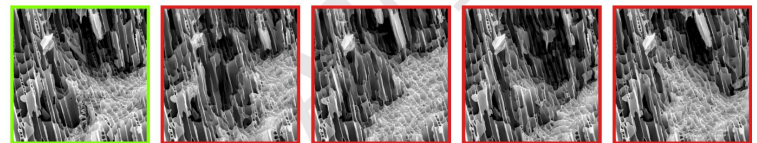


Fig. 2. Regenerating images using generative model learned based on a single image. For each group, the last four images are regenerated from the first real image. These images can escape the duplication detection methods based on the comparison of details because they have totally different local details. The images with the red border are all computer-generated, while the images with the green border are real ones.

Scientific Paper Mills: Authorship for Sale

- Already accepted papers - Special Issues
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- Fabricated data: fake patients, re-used / AI-generated images
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NEWS FEATURE | 23 March 2021

The fight against fake-paper factories that churn out sham science

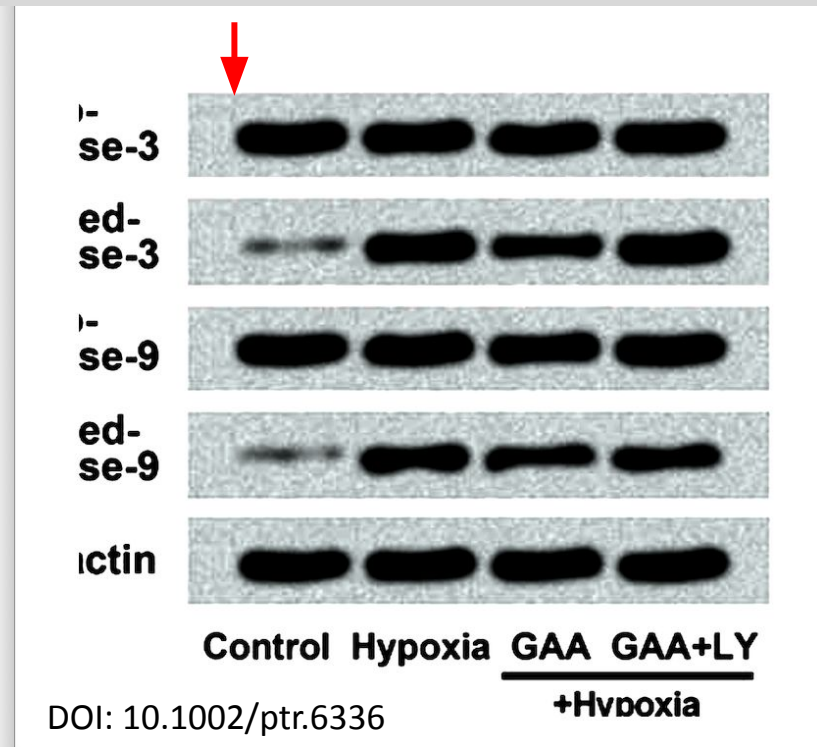
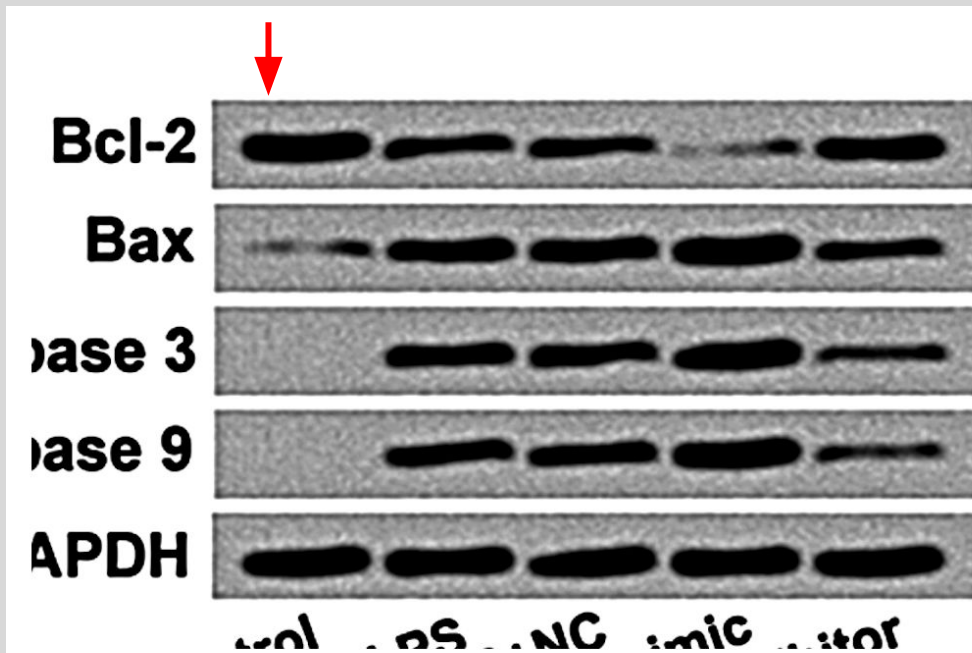
Some publishers say they are battling industrialized cheating. A *Nature* analysis examines the 'paper mill' problem – and how editors are trying to cope.

Publishers grapple with an invisible foe as huge organised fraud hits scientific journals



BY KATRINA KRÄMER | 25 MAY 2021

Tadpole Paper Mill: same blot background



- ~600 papers found so far
- Same blot background across all papers
- Bands generated through Generative Adversarial Networks (GAN)?

Non-coding RNA Paper Mill articles

Fu et al. *Cancer Cell Int* (2019) 19:338
https://doi.org/10.1186/s12935-019-1055-z

Cancer Cell International

PRIMARY RESEARCH

Open Access

Circular RNA ABCB10 promotes hepatocellular carcinoma progression by increasing HMG20A expression by sponging miR-670-3p

Yu Fu¹, Limin Cai², Xuexue Lei¹ and Dunwei Wang^{2*}

Abstract

Background/aims: The dysregulation of circABCB10 may play an critical role in tumor progression. However, its function in liver cancer (HCC) is still unclear. Therefore, this experimental design was based on circABCB10 to explore its pathogenesis of HCC.

Methods: The expression of circABCB10 and miR-670-3p in HCC tissues was detected by RT-qPCR. CCK-8, BrdU incorporation, colony formation and transwell assays were used to determine the effect of circABCB10 on HCC cell proliferation and migration. Target gene prediction and dual-luciferase reporter assays were used to validate downstream target genes of circABCB10 and miR-670-3p. HMG20A expression was detected by RT-qPCR and Western blotting. The tumor changes in mice were detected by IHC and H&E.

Results: CircABCB10 was significantly increased in HCC tissues and cell lines, and high circABCB10 expression was directly associated with low survival in HCC patients. Silencing of circABCB10 inhibited proliferation and invasion of hepatocellular carcinoma. In addition, circABCB10 acted as a sponge of miR-670-3p to upregulate HMG20A expression. In addition, overexpression of miR-670-3p knockdown of HMG20A reversed the carcinogenic effects of circABCB10 in HCC. There was a negative correlation between the expression of circABCB10 and miR-670-3p, and a positive correlation between the expression of circABCB10 and HMG20A in HCC tissues.

Conclusion: circABCB10 promoted HCC progression by modulating the miR-670-3p/HMG20A axis, and circABCB10 may be a potential therapeutic target for HCC.

Trail registration JLI1384739, registered at Sep 09, 2014.

Keywords: circABCB10, miR-670-3p, HMG20A, Liver cancer, Proliferation

Background

Primary liver cancer is the third leading cause of cancer-related death [1, 2]. The most common type of primary liver cancer is hepatocellular carcinoma (HCC) [3]. Currently, the main root therapy includes surgical resection and liver transplantation [4, 5]. However, due to the aggressive biological characteristics of liver cancer, the

current first-line and second-line treatments are relatively ineffective, and the number of deaths is basically the same every year [6]. In addition, because liver cancer is characterized by rapid growth of tumor cells, metastasis can occur early, tumor malignancy rate is high and many are multidrug resistant, and its 5-year survival rate is generally within 5% [7]. How to more effectively intervene in the occurrence of liver cancer and patients with liver cancer has become a major and urgent problem. Therefore, it is extremely important to further explore the pathogenesis of liver cancer and find effective diagnosis and treatment methods.

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miR-208b targets Bax to protect H9c2 cells against hypoxia-induced apoptosis

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ARTICLE INFO

Keywords: Myocardial infarction, miR-208b, Bax, H9c2 cell, Hypoxia, PI3K/AKT pathway

ABSTRACT

Background: miR-208 family members have been considered as promising biomarkers in myocardial infarction (MI). Among which, miR-208a and miR-208b are reported to function as ischemic injury promoting miRNAs. This study aimed to explore the in vivo effect of miR-208b in MI, which has not been widely studied.
Methods: RT-qPCR was conducted to determine the expression of miR-208b in MI patients, MI mouse model and H9c2 cells stimulated with hypoxia. H9c2 cells were subjected to hypoxia before which miR-208b expression was altered by transfection. CCK-8, flow cytometry and Western blot were performed to detect cell survival. Besides, the regulatory mechanism of miR-208b, Bax, and PI3K/AKT was tested by luciferase reporter, RT-qPCR and Western blot.
Results: Serum level of miR-208b in MI patients was significantly higher than those in the healthy controls. Also, miR-208b was highly expressed in mouse model and cell model of MI. Overexpression of miR-208b protected H9c2 cells against hypoxia-induced apoptosis, as the viability was increased, apoptosis rate was decreased, Bax and Cytochrome c were decreased, and Bcl-2 was up-regulated. Bax was a target gene of miR-208b. And miR-208b protected H9c2 cells from hypoxia-induced apoptosis. PI3K/AKT pathway was activated by hypoxia, and the activated PI3K/AKT pathway could further repress Bax expression. Finally, blockage of PI3K/AKT pathway by using LY294002 eliminated the myocardial protective effects of miR-208b. miR-208b was highly expressed during MI, and miR-208b protects H9c2 cells against hypoxia-induced apoptosis. miR-208b exerts myocardial protective effect by targeting Bax and activating PI3K/AKT pathway.

1. Introduction

Myocardial infarction (MI) is the most common cardiovascular disease, which results in disability or irreversible ischemic heart damage. Patients with MI often experience chest pain, acute pericarditis, acute pulmonary edema, and other symptoms. Changes on ST segment, new or deepened Q waves and the motion of heart wall are recognized as the main criteria of MI diagnosis [1]. Echocardiography is a main method to detect MI in the heart, and indicates a MI based on the abnormal motion of heart walls. Management of MI is mainly focused on early resuscitation [2], anticoagulation [3], and anti-fibrinolysis [4]. Thrombolytic therapy and arterial bypass surgery are two generally used treatments for MI [5–7]. These therapies can effectively re-establish coronary blood flow in a short period of time, but are limited in the repairment of myocardial damage. Thus a better understanding of

MI is urgently needed, which will be helpful for improving the diagnosis and treatment of this disease.

microRNAs (miRNAs) are a kind of non-coding RNAs with length of approximately 22 nt. They negatively regulate gene expression at the post-transcriptional level, and thus participate in almost all biological processes, including cell proliferation, differentiation, apoptosis, and even tumorigenesis [8,9]. It has been mentioned that miRNAs are paving the way for future researches on human diseases, from cancer to macular degeneration [10]. Besides, massive miRNAs have been reported to be associated with the disease state of MI. For example miR-34 family members, i.e., miR-34a, -34b and -34c are highly expressed in a mouse model of MI [11], while miR-150 is low expressed in this animal model [12]. miR-210 and miR-145 aggravate hypoxia-induced injury in cardiomyocyte H9c2 via respectively targeting CXCR4 [13] and Rac1 [14]. These examples demonstrated the significance of

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RETRACTED ARTICLE: Upregulation of long non-coding RNA OGFRP1 facilitates endometrial cancer by regulating miR-124-3p/SIRT1 axis and by activating PI3K/AKT/GSK-3β pathway

Yueguo Lv, Shaorong Chen, Jingling Wu, Ruilin Lin, Limei Zhou, Guimin Chen, Huiqing Chen and Yumin Ke

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ABSTRACT

We planned to investigate the possible influences of long non-coding RNA (lncRNA) growth factor receptor pseudogene 1 (OGFRP1) in endometrial cancer and its potential regulatory mechanism. We measured the level of OGFRP1 in endometrial cancer tissues and evaluated the influences of OGFRP1 on the tumour cell biological processes of endometrial cancer cells. Further, the regulatory relationships between OGFRP1 and miR-124-3p, between miR-124-3p and SIRT1 (SIRT1) were, respectively, investigated. The interaction between OGFRP1 dysregulation and activation of PI3K/AKT/β pathway was revealed by Western blotting. OGFRP1 was up-regulated in endometrial cancer cells. OGFRP1 suppression inhibited the malignant behaviour (inhibited cell viability, proliferation, cell apoptosis, and suppressed cell migration and invasion) of the Ishikawa cells via negatively regulating miR-124-3p. SIRT1 was a target gene of miR-124-3p, and miR-124-3p regulated tumour growth and metastasis by the down-stream signal of SIRT1. Moreover, suppression of OGFRP1 needed the activation of PI3K/AKT/GSK-3β signals in the Ishikawa cells via miR-124-3p/SIRT1 axis. Experiments revealed that upregulation of OGFRP1 may enhance the progression of endometrial cancer by regulating miR-124-3p/SIRT1 axis and by activating PI3K/AKT/GSK-3β pathway. OGFRP1 may significance in illustrating the biology of endometrial cancer.

ARTICLE HISTORY

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KEYWORDS

Endometrial cancer; long non-coding RNA; OGFRP1; miR-124-3p; Sirtuin1; PI3K/AKT/GSK-3β pathway

Introduction

Endometrial cancer remains to be a lethal gynaecological disease with a tremendous increase in the incidence over the past decades [1]. Although the survival rate is high if patients are diagnosed at an early stage, the percentage of patients diagnosed at a relative advanced stage is still high, i.e., about 30% [2]. The prognosis remains poor in the advanced stage or with a high risk of recurrence. Moreover, the molecular mechanisms of endometrial cancer have been poorly illustrated.

Non-coding RNAs (lncRNAs), some non-coding RNAs longer than 200 nucleotides in length, is widely pointed out as a new type of non-coding RNA in many biology of various diseases [4–6]. Expression of lncRNAs has been widely discovered in cancer and is considered as a character in cancer development. lncRNAs, including HOTAIR, BANCR, and have been identified to be crucial in the progression of endometrial cancer [10–12]. OGFRP1, a newly reported lncRNA has been shown to induce autophagy and growth in human coronary artery endothelial cells [13]. In addition, dysregulation of OGFRP1 was demonstrated to be involved in the biology of non-small cell lung cancer (NSCLC) and hepatocellular carcinoma [15]. However, OGFRP1 in endometrial cancer still remains incomplete reported. In

addition, Dong et al. pointed out that miR-124 was lowly expressed in tumour tissues of endometrial cancer [16]. However, there was no study focusing on investigating the regulatory pattern between OGFRP1 and miR-124 in endometrial cancer.

During this research, we elucidated the function and possible mechanism of OGFRP1 in endometrial cancer. We determined the expression of OGFRP1 in endometrial cancer and assessed the effect of OGFRP1 dysregulation on the malignant behaviour of endometrial cancer cells. Further, we analyzed the regulatory relationships between OGFRP1 and miR-124-3p, and those between miR-124-3p and SIRT1 (SIRT1). Moreover, the interaction between OGFRP1 dysregulation and activation of PI3K/AKT/GSK-3β pathway was also revealed. All of these data may offer a theoretical basis for designing novel strategies for illustrating the biology of endometrial cancer.

Materials and methods

Patients

Between April 2015 and July 2018, 48 patients diagnosed with endometrial cancer were recruited. Table 1 shows the characteristics of endometrial cancer patients. Forty-eight

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Iranian Plants Paper Mill

Lejing Lin et al., *Genetika* (2021), DOI: 10.2298/GENSR2102507L

Table 1. List of the investigated taxa including origin of voucher specimens

No	Taxa	Locality	Latitude	Longitude	Altitude(m)
Sp1	<i>E. gruinum</i> (L.) L'Hér.	Hamedan, 20km s of Nahavand	37° 07' 48"	49° 54' 04"	165
Sp2	<i>E. cicutarium</i> (L.) L'Hér.	Razavi Khorasan, Kashmar, Kuhstorkh District	37° 07' 08"	49° 54' 11"	159
Sp3	<i>E. moschatum</i> (L.) L'Hér.	Esfahan, ardestan on road to taleghan	38° 52' 93"	47° 25' 92"	1133
Sp4	<i>E. ciconium</i> (L.) L'Hér.	Semnan, 20km NW of shahrud	38° 52' 93"	47° 25' 92"	1139
Sp5	<i>E. oxyrrhynchum</i> M. Bieb.	Mazandaran, 40 km Tonkabon to janat abad	35° 50' 36"	51° 24' 28"	2383
Sp6	<i>E. hoefftianum</i> C. A.Mey	West-Azarbaijan, Urumieh, Silvana	35° 42' 29"	52° 20' 51"	2421
Sp7	<i>E. neuraefolium</i> Delile ex Godr.	Tehran, Damavand	35° 42' 29"	52° 20' 51"	2421

Juan Yin, *GENETIKA* (2022), DOI: 10.2298/GENSR2201173Y

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GENETIKA, Vol. 54, No1, 173-186, 2022

Table 1. List of the investigated taxa including origin of voucher specimens

Taxa	Locality	Latitude	Longitude	Altitude(m)
<i>Rindera albida</i> (Wettst.) Kuhn.	Kurdistan, Sanandaj	37° 07' 48"	49° 54' 04"	165
<i>Rindera hangei</i> (Boiss.) Gürke	Hamedan, 20km s of Nahavand	37° 07' 08"	49° 54' 11"	159
<i>Rindera lanata</i> (Lam.) Bunge	Razavi Khorasan, Kashmar, Kuhstorkh District	38° 52' 93"	47° 25' 92"	1133
<i>Rindera cyclodonta</i> Bunge	Kurdistan, Sanandaj	38° 52' 93"	47° 25' 92"	1139
<i>Rindera regia</i> Kuhn v	Esfahan, ardestan on road to taleghan	35° 50' 36"	51° 24' 28"	2383
<i>Rindera media</i> (Turill) Riedl n	Bojnord, Ghorkhod protected area Semnan, 20km NW of shahrud	35° 42' 29"	52° 20' 51"	2421

Dezhong Bi et al., *Genetika* (2021), DOI: 10.2298/GENSR2101393B

Table 1. List of the investigated taxa including origin of voucher specimens.

Taxa	Locality	Latitude	Longitude	Altitude(m)
<i>H. dogonbadanicum</i> Assadi	Hamedan, 20km s of Nahavand	37° 07' 48"	49° 54' 04"	165
<i>H. androsaceum</i> L.	Razavi Khorasan, Kashmar, Kuhstorkh District	37° 07' 08"	49° 54' 11"	159
<i>H. tetrapetrum</i> Fries.	Esfahan, ardestan on road to taleghan	38° 52' 93"	47° 25' 92"	1133
<i>H. perforatum</i> L.	Hamedan, Alvand	38° 52' 93"	47° 25' 92"	1139
<i>H. triquetrifolium</i> Turra	Mazandaran, 40 km Tonkabon to janat abad	35° 50' 36"	51° 24' 28"	2383
<i>H. elongatum</i> Ledeb	West-Azarbaijan, Urumieh, Silvana	35° 42' 29"	52° 20' 51"	2421

Juan Yin et al., *Genetika* (2021), DOI: 10.2298/GENSR2101363Y

Plant materials

Thirteen *Geranium* species were collected from different regions of Iran (Table 1).

Table 1. *Geranium* species and populations, their localities and voucher numbers.

Species	Locations	Latitude	Longitude	Altitude (m)
1. <i>G. dissectum</i>	Esfahan, Ghameshtou, Sanjab Lorestan, Oshorankah, above Tihun village	37° 07' 48"	49° 54' 04"	165
2. <i>G. collinum</i>	East Azarbaijan kalebar cheshme ali akbar	37° 07' 08"	49° 54' 11"	159
3. <i>G. rotundifolium</i>	East Azarbaijan kalebar, Shojabad	38° 52' 93"	47° 25' 92"	1133
4. <i>G. columbinum</i>	Tehran, Tushal	38° 52' 93"	47° 25' 92"	1139
5. <i>G. sylvaticum</i>	Ardabil, Khaikhal	35° 50' 36"	51° 24' 28"	2383
	East Azarbaijan kalebar cheshme ali akbar	35° 42' 29"	52° 20' 51"	2421
6. <i>G. pratense</i>	East Azarbaijan kalebar, Shojabad	38° 52' 39"	47° 25' 92"	1133
7. <i>G. platyptalum</i>	Hamedan, Nahavand	38° 52' 39"	47° 25' 92"	1137
8. <i>G. gracile</i>	Mazandaran, Tonkabon-janat radbar	36° 48' 47"	50° 53' 68"	1144
9. <i>G. ibericum</i>	Mazandaran, Haraz road, Emam Zade Hasben	36° 38' 05"	51° 29' 05"	1600
10. <i>G. kotschy</i>	Alborz, Karaj-Qazvin	35° 49' 23"	51° 00' 04"	1250
11. <i>G. tuberosum</i>	Kermanshah, Islamabad	38° 52' 39"	47° 25' 92"	1365
12. <i>G. stepporum</i>	Esfahan, Fereydon shahr	35° 50' 03"	51° 24' 28"	1133
13. <i>G. persicum</i>	Tehran, Damavand	35° 43' 15"	52° 04' 12"	2383

Jialing Li et al., *Genetika* (2021), DOI: 10.2298/GENSR2103369L

J. Li et al.: GENETIC DIVERSITY IN *Stellaria* L.

1371

Table 1. List of the investigated taxa including origin of voucher specimens.

Taxa	Locality	Latitude	Longitude	Altitude(m)
<i>S. pallida</i> (Dumort.) pie	Kurdistan, Sanandaj	37° 07' 48"	49° 54' 04"	165
<i>S. holostea</i> L.	Hamedan, 20km s of Nahavand	37° 07' 08"	49° 54' 11"	159
<i>S. media</i> (L.) VILL.	West-Azarbaijan, Urumieh, Silvana	38° 52' 93"	47° 25' 92"	1133
	Esfahan, ardestan on road to taleghan	38° 52' 93"	47° 25' 92"	1139
<i>S. persica</i> Boiss.	Bojnord, Ghorkhod protected area Semnan, 20km NW of shahrud	35° 50' 36"	51° 24' 28"	2383
<i>S. graminea</i> L.	Mazandaran, 40 km Tonkabon to janat abad			
	Mazandaran, Nowshahr			

Xixi Yao et al., *Genetika* (2021), DOI: 10.2298/GENSR2103229Y

X. YAO ET AL.: GENETIC RELATIONSHIPS IN *Erodium*

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Table 1. List of the investigated taxa including origin of voucher specimens.

Taxa	Locality	Latitude	Longitude	Altitude(m)
<i>E. malacoides</i> Bovi ex Deene.	Kurdistan, Sanandaj	37° 07' 48"	49° 54' 04"	165
<i>E. gruinum</i> (L.) L'Hér.	Razavi Khorasan, Kashmar, Kuhstorkh District	37° 07' 08"	49° 54' 11"	159
<i>E. glaucophyllum</i> (L.) L'Hér.	Esfahan, ardestan on road to taleghan	38° 52' 93"	47° 25' 92"	1133
<i>E. hoefftianum</i> C. A.Mey	Semnan, 20km NW of shahrud	38° 52' 93"	47° 25' 92"	1139
<i>E. neuraefolium</i> Delile ex Godr.	Mazandaran, 40 km Tonkabon to janat abad	35° 50' 36"	51° 24' 28"	2383
<i>E. moschatum</i> (L.) L'Hér.	West-Azarbaijan, Urumieh, Silvana	35° 42' 29"	52° 20' 51"	2421

Xixi Yao et al., *Caryologia* (2022), DOI: 10.36253/caryologia-1380

Table 1. List of the investigated taxa including origin of voucher specimens. All material is collected by Majid Khayatneshad.

Taxa	Locality	Latitude	Longitude	Altitude(m)
<i>Rindera albida</i> (Wettst.) Kuhn.	Kurdistan, Sanandaj	37° 07' 48"	49° 54' 04"	165
<i>Rindera bungei</i> (Boiss.) Gürke	Hamedan, 20km s of Nahavand	37° 07' 08"	49° 54' 11"	159
<i>Rindera lanata</i> (Lam.) Bunge	Razavi Khorasan, Kashmar, Kuhstorkh District	38° 52' 93"	47° 25' 92"	1133
<i>Rindera cyclodonta</i> Bunge	Kurdistan, Sanandaj	38° 52' 93"	47° 25' 92"	1139
<i>Rindera regia</i> Kuhn v	Bojnord, Ghorkhod protected area Semnan, 20km NW of Shahrud	35° 50' 36"	51° 24' 28"	2383
<i>Rindera media</i> (Turill) Riedl n	Mazandaran, 40 km Tonkabon to Janat abad			
	Mazandaran, Nowshahr			
	West-Azarbaijan, Urumieh, Silvana	35° 42' 29"	52° 20' 51"	2421

Unexpected findings - can you spot it?

MiR-605-3p inhibits malignant progression of prostate cancer by up-regulating EZH2

Table I. Association of miR-605-3p expression with clinicopathologic characteristics of prostate cancer.

Parameters expression	Number of cases	miR-605-3p		<i>p</i> -value
		High (%)	Low (%)	
Age (years)				0.964
<60	20	12	8	
≥60	32	19	13	
Gender				0.236
Male	25	17	8	
Female	27	14	13	
T stage				0.043
T1-T2	31	22	9	
T3-T4	21	9	12	
Lymph node metastasis				0.002
No	33	25	8	
Yes	19	6	13	
Distance metastasis				0.033
No	38	26	12	
Yes	14	5	9	

Synonymized Plagiarism Paper Mill

Tortured phrase:

"Chest peril [...] is a remarkable kind taking all into account, and basic part of ladies' ruins world-wide"

Original text: Ganesan et al., 2020 ICOT,
DOI: 10.1109/ICOT51877.2020.9468772

"Breast cancer [...] is the most usual kind of cancer and also one of the main reasons for women's deaths globally. "

Materials Today: Proceedings
Available online 3 February 2021
In Press, Corrected Proof

Analysis of bosom malignancy using supervised machine learning classifier

Vaibhavraj Nath Chauhan ^a, Sanjana Purbia ^a, Pankaj Chittora ^a, Prasun Chakrabarti ^b, Sandeep Poddar ^c

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<https://doi.org/10.1016/j.matpr.2020.12.442> Get rights and content

Abstract

Chest peril tends to one of the deceases that makes a huge amount of passing's dependable. It's a remarkable kind taking all into account, and basic part of ladies' ruins world-wide. Plan and data attraction approaches are a convincing method to portray data. Target in medic field, where assumptions are ordinarily considering and evaluation to pick. In this research, appraisal preposition various AI figuring: Random Tres, Deep Neural Net (DNN), Logistic Regression, Support Vector Machine (SVM), K-Nearest Neighbours (K-NN), Bayes Net, C5.0. The standard goal is to survey the models in get-together all information concerning productivity and reasonableness of each figuring as for accuracy, precision, affectability and unequivocally. Our research shows that KNN gives the basic exactness (97.13%) with low slip up rate. All outputs are runs inside an entertainment air and drove in SPSS information mining contraption.

arXiv:2107.06751

Tortured phrases: A dubious writing style emerging in science
Evidence of critical issues affecting established journals

Guillaume Cabanac · Cyril Labbé · Alexander Magazinov

Mass retractions of 'special issues'

Retraction Watch, April 2023

Wiley and Hindawi to retract 1,200 more papers for compromised peer review

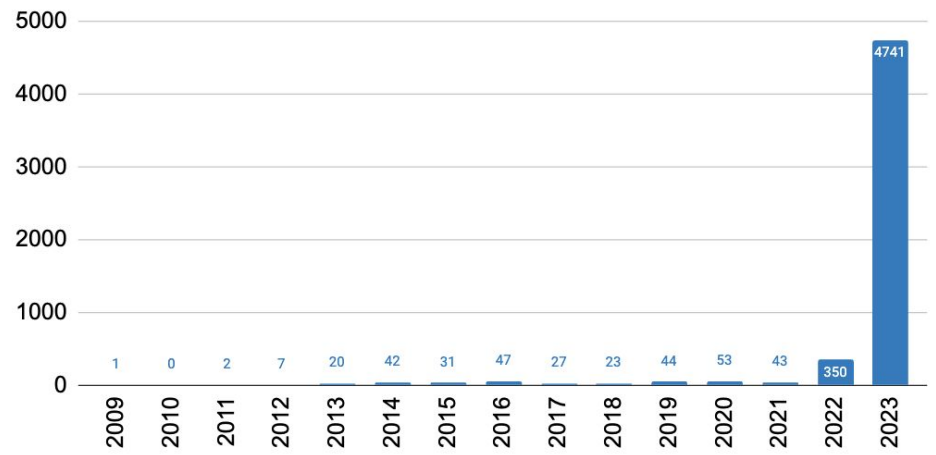
Hindawi and Wiley, its parent company, have identified approximately 1,200 articles with compromised peer review that the publishers will begin retracting this month.

Jay Flynn, executive vice president and general manager of the research division at Wiley, which acquired Hindawi in 2021, wrote about the forthcoming retractions in a [blog post](#) at Scholarly Kitchen yesterday.



Special Issues have been inundated by paper mill articles and peer review / citation rings

Hindawi Retractions Per Year



A screenshot of the Hindawi website search results page. The search query is "retracted:" OR "retraction". The results show 4741 items. The first result is from the Journal of Healthcare Engineering, Volume 2023, Article ID 9856721, titled "Retracted: Clinical Study of Neuromuscular Electrical Stimulation in the Prevention of Deep Venous Thrombosis of Lower Extremities after Anterior Cruciate Ligament Reconstruction". The second result is from Wireless Communications and Mobile Computing, Volume 2023, Article ID 9782149, titled "Retracted: Research on the Theory and Value Analysis Algorithm of Marx's Outlook on the People Based on Deep Learning under the Background of the Internet of Things".

Hindawi's mass retraction of "Special Issues" papers



eliesbik
August 10, 2023
Easy Journals, Large problematic paper sets, Paper mills, Peer Review Fail

Hindawi — and its parent company, Wiley — have recently announced that they will retract hundreds of papers from journals targeted by paper mills. The papers were all published in 'special issues', with the guest editors being either asleep at the wheel or perhaps knowingly looking the other way.

In this blog post, I will take an in-depth look at some of these papers.

Science Misconduct - Discussion

- Science is about discovering the truth
- Rewards of fraud are high, consequences are small
- Focus less on quantity; more on reproducibility
- It takes a village: role of reviewers, journals, institutions
- Need faster correction of the scientific literature
- Better legal protection for those who raise concerns
- Tremendous cost of science misconduct (*scientists, science*)



Science Integrity Digest

A blog about science integrity, by Elisabeth Bik, for Harbers-Bik LLC.