

**6-я Международная научно-практическая конференция
«Научное издание международного уровня - 2017: мировая практика
подготовки и продвижения публикаций»,
(Москва, 18–21 апреля 2017 г.)**

**Каким образом очень плохие статьи
публикуются в очень хороших научных
журналах**

А.Н. Хохлов, Г.В. Моргунова, А.А. Клебанов

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«Вестник Московского университета. Серия 16. Биология»***

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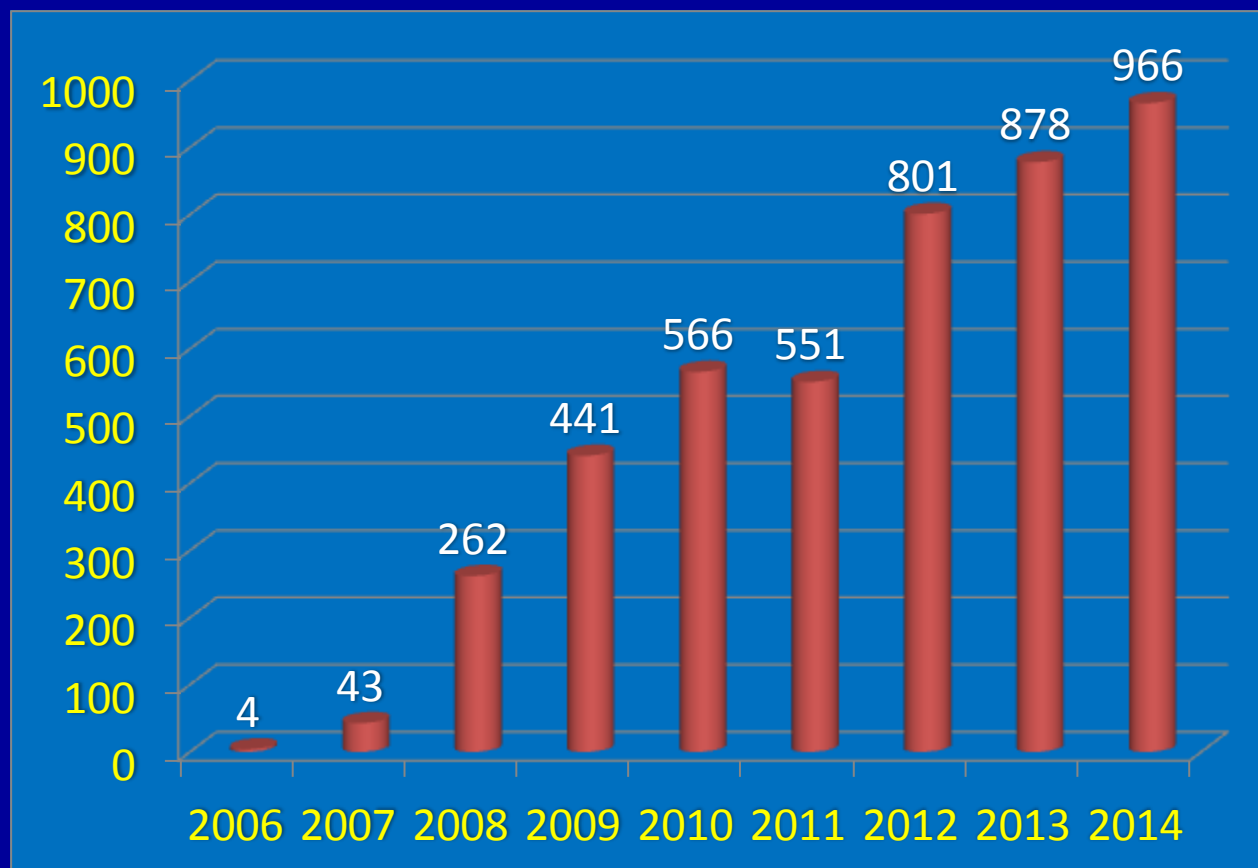
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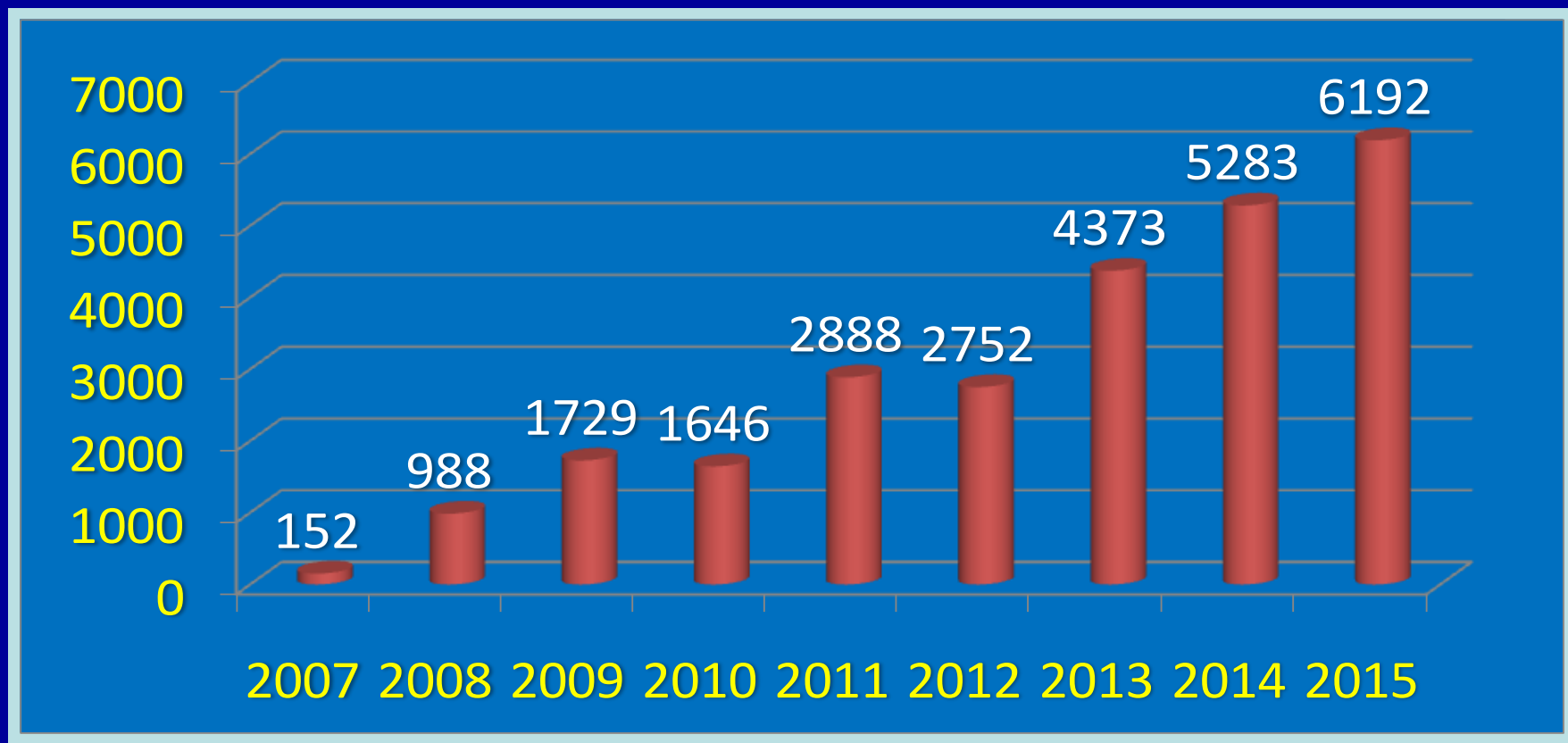
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Короткая ремарка (не можем удержаться):

О лжепатриотизме в издании научных статей

У кого из присутствующих на ногах – российская обувь? И у кого российский смартфон?

А почему вы непременно хотите печатать статьи на русском?

**Просим не рассматривать этот доклад как
руководство, хотя...😊**

О важности определений

Бабушка покупает рыбу:

- Скажите, а у вас рыба свежая?
- Бабушка, ну как же? Она же живая!
- Ну и что? Я тоже живая...

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Плохие журналы (Top-Less Journals[®])

Объективные и субъективные критерии
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**Опубликовать «плохую» статью в
хорошем журнале – значит, обойти
правила, многократно озвученные на этой
конференции**

**Впрочем, в последние дни мы
познакомились не только с такими
правилами, но и с разными способами
обходить требования к регламенту
конференции (ПРОГРАММА мероприятия
– это его свод согласованных со всеми
участниками правил)**

**О нарушениях грамматических и
стилистических ПРАВИЛ многими
докладчиками (простите, мое больное
место) я уж молчу**

**А.Ю. Гаспарян упомянул в одном из своих
замечательных докладов о странной
казахской статье в литовском журнале
(WoS)**

**Главный вопрос, интересующий нас, -
КАК она туда попала?**

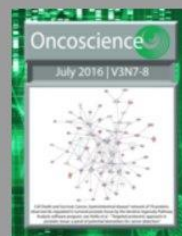
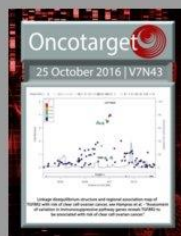
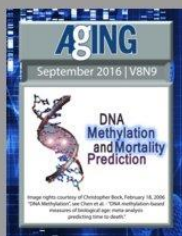
И каким образом попадают в Nature или Science те достаточно многочисленные статьи, которые потом подвергаются ретракции?

**Несколько вариантов
(исключительно НАШ взгляд на
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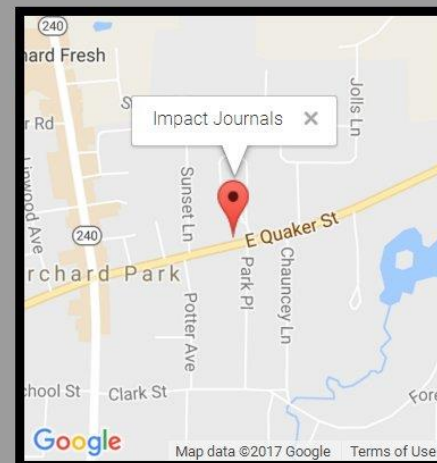
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Aging ranks #3 See here

Recent highly cited papers in **Aging**. See here

Aging a multi-disciplinary journal, publishes on diverse topics in Physiology&Medicine

Papers of Nobel winners in Physiology&Medicine: Elizabeth Blackburn (6 papers), Andrew V. Schally, and Shinya Yamanaka

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Introducing Oncoscience: from Oncotarget to the future

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Maximizing research impact via insightful peer-review

Oncotarget's rising popularity is due to insightful, constructive, punctual, multiple peer-review: helping our authors to increase the impact of their research

Members of Editorial Board Carlo M. Croce (former Editor-in-Chief of Cancer Research) and Andrew Schally (Nobel Prize winner) have published 27 and 10 papers in Oncotarget

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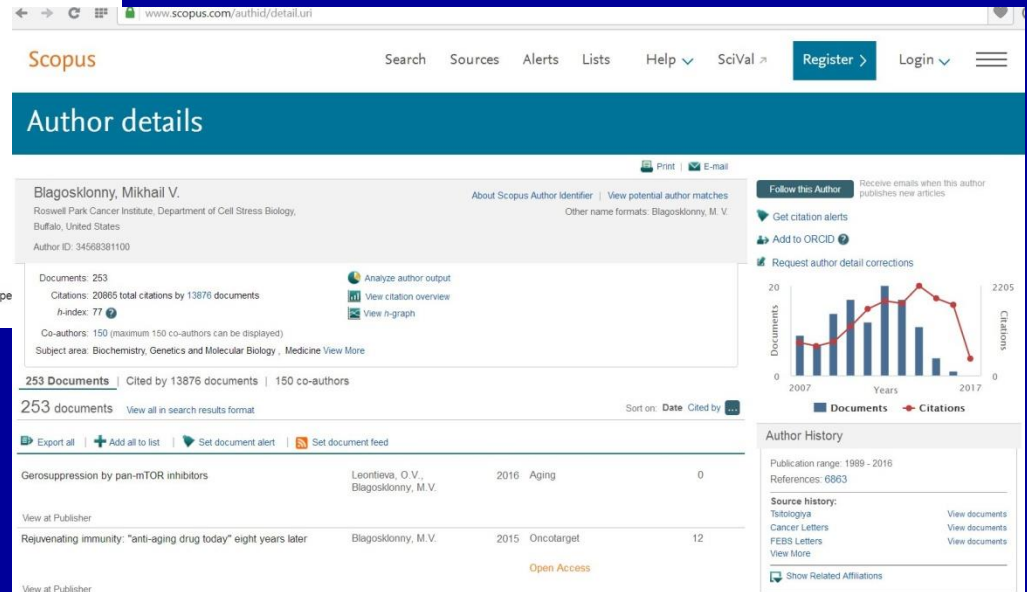
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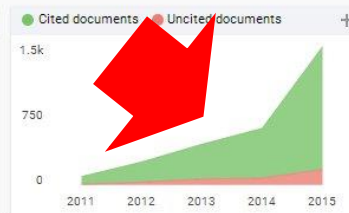
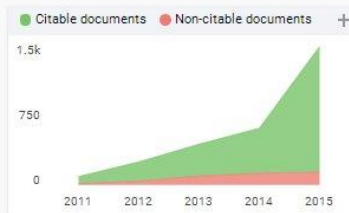
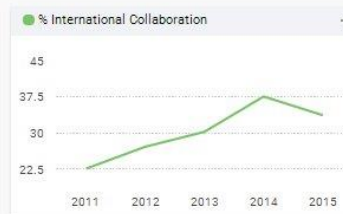
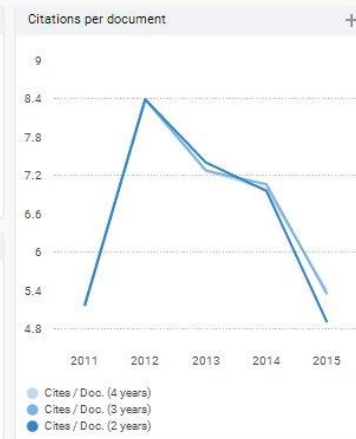
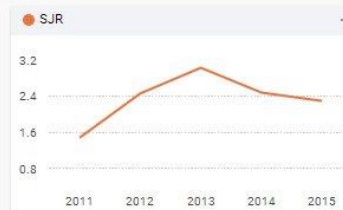
Blagosklonny is editor-in-chief of *Aging*,^[4] *Cell Cycle*,^[5] and *Oncotarget*.^[6] In addition, he is associate editor of *Cancer Biology & Therapy*^[7] and a member of the editorial board of *Cell Death & Differentiation*.^[8]

The reviewing process employed by *Oncotarget* has been criticized by Jeffrey Beall,^[9] who also included *Oncotarget* and *Aging* on his list of "potential, possible, or probable predatory scholarly open-access journals"^[10] in July 2015.^[9] Further reports on Beall's blog suggest that the substandard peer review processes for these journals are used by their respective editor-in-chief to entice prospective authors to include references to Blagosklonny's own publications in their articles (following the peer review), thereby raising his personal impact factor.^[11]

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FIG. 1. 2013/10/10

need to explore therapeutic strategies that target the identified pathways, not only in the striatum but also in other brain areas besides the basal ganglia which has been underestimated and are strongly involved in HD cognitive pathology

Silvia Ginés: Departament de Biologia Cel·lular, Immunologia i Neurociències, Facultat de Medicina, Universitat de Barcelona, Barcelona, Spain; Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Barcelona, Spain; CIBERNED, Spain

Correspondence to: Silvia Ginés, **email** silviagines@ub.edu

Keywords: Huntington's disease, cognitive deficits, hippocampus, p75 receptor, RhoA

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2. Brito V, et al. Cell Death Dis. 2013; 4:e595.
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СТАРЕНИЕ РАЗЛИЧНЫХ КЛЕТОКГ.В. Моргунова¹, А.А. Клебанов¹, Ф. Маротта², А.Н. Хохлов^{1,*}¹ Сектор эволюционной цитогеронтологии, биологический факультет, Московский государственный университет имени М.В. Ломоносова, Россия, 119234, Москва, Ленинские горы, д. 1, стр. 12;² ReGenera Research Group for Aging Intervention and San Babila Clinic, Corso Matteotti 1/A, 20121 Milano, Italy

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Существует точка зрения, согласно которой хронологическое старение (ХС) дрожжей и “стационарное” старение (СС) культивируемых клеток человека и животных являются следствием закисления ростовой среды. Однако целый ряд появившихся в последнее время работ свидетельствует о том, что этот процесс хотя и влияет в определенной степени на скорость “старения” клеток в стационарной фазе, но не определяет его полностью. По-видимому, определяющим фактором здесь является ограничение клеточной пролиферации, которое приводит к “старению” клеток даже в физиологически оптимальных условиях. При ХС дрожжей и при СС клеток млекопитающих ростовая среда закисляется до $\text{pH} \leq 4$. Если не допускать накопления кислоты в среде, можно увеличить продолжительность жизни культуры, однако клетки всё равно будут вымирать, только с меньшей скоростью. Наблюдаемые эффекты закисления среды при ХС и СС могут объясняться активацией высоко консервативных сигнальных путей роста, приводящих к развитию окислительного стресса, а эти процессы, в свою очередь, могут быть вовлечены в старение многоклеточных организмов и связаны с возникновением у них возрастных заболеваний. Ранее мы изучали влияние буферной ёмкости культуральной среды на СС трансформированных клеток китайского хомячка. Было установлено, что наличие в среде HEPES в концентрации 20 мМ не влияет на скорость роста клеток. При этом ростовые кривые выходили на “плато” в один и тот же день. Однако в среде с HEPES клетки, с одной стороны, достигали меньшей насыщающей плотности, чем в контроле (т.е. были “старше” согласно критериям геронтологической клеточно-кинетической модели), а с другой — претерпевали СС с гораздо меньшей скоростью (но все равно “старели”). Можно полагать, что внеклеточный рН, который, кстати, хорошо коррелирует с внутриклеточным, является хотя и важным (концепция И.А. Аршавского о роли ацидотической альтерации в старении), но не ключевым фактором, определяющим выживание клеток в стационарной культуре.

Ключевые слова: рН ростовой среды, буферная ёмкость, клеточные культуры, “стационарное старение”, хронологическое старение, обзор

Yeast-like chronological senescence in mammalian cells: phenomenon, mechanism and pharmacological suppression

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Key words: chronological aging, senescence, metabolism, rapamycin, mTOR, lactate

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Abstract: In yeast, chronological senescence (CS) is defined as loss of viability in stationary culture. Although its relevance to the organismal aging remained unclear, yeast CS was one of the most fruitful models in aging research. Here we described a mammalian replica of yeast CS: loss of viability of overgrown “yellow” cancer cell culture. In a density and time (chronological)-dependent manner, cell culture loses the ability to re-grow in fresh medium. Rapamycin dramatically decelerated CS. Loss of viability was caused by acidification of the medium by lactic acid (lactate). Rapamycin decreased production of lactate, making conditioned medium (CM) less deadly. Both deadly CM and lactate caused loss of viability in low cell density, not preventable by either rapamycin or additional glucose. Also, NAC, LY294002, U0126, GSK733, which all

Chronological lifespan in stationary culture: from yeast to human cells

Zoya N. Demidenko

Comment on: Leontieva OV and Blagosklonny MV. Yeast-like chronological senescence in mammalian cells: phenomenon, mechanism and pharmacological suppression. Aging 2011; 3: this issue

A decade ago, Mikhail Blagosklonny predicted that cellular senescence is driven by mitogenic pathways, when the cell cycle is blocked and actual growth is impossible [1]. In particular, the mitogen- and nutrient-sensing mTOR (Target of Rapamycin) pathway drives either cell mass growth or aging [2]. Rapamycin prevents conversion of reversible cell cycle arrest to senescence [3-11]. When the cell cycle is blocked, but mTOR is still active, then cells become senescent. This process was named *gerogenic conversion* or simply *geroconversion* [12]. Rapamycin did not by-pass arrest but suppress geroconversion. Cells remained resting but not senescent. The discovery of mTOR-dependent geroconversion allowed Blagosklonny to connect cellular aging to age-related diseases and organismal aging [13, 14]. Furthermore, this predicted that rapamycin is a gerosuppressant, which could be used to prevent age-related diseases by slowing down the aging

lytic cells can destroy cell culture. But like it was known to most researches (90 years ago) that fungi can destroy bacterial culture, it took a special insight to recognize the potential of this seemingly useless phenomenon. There is an intriguing parallel between penicillin and rapamycin. As described in this issue of *Aging*, mTOR pathway is involved in glycolytic phenotype, causing self-poisoning due to overproduction of lactic acid. By decreasing lactate production, rapamycin prevents chronological senescence (CS). CS can be manipulated genetically and pharmacologically. Most importantly, the same agents that suppress geroconversion, organismal aging and cancer also suppress CS.

This study does not break any dogma because there was no dogma as the field did not exist. This paper opens a new field in both aging and cancer research.

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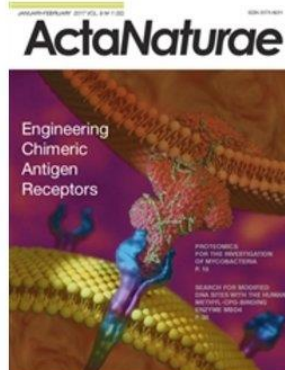
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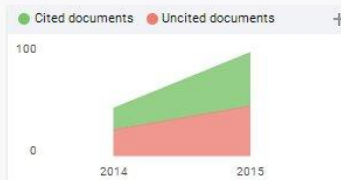
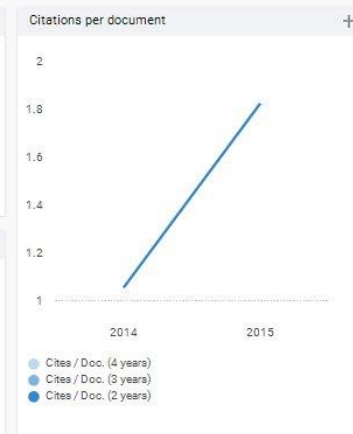
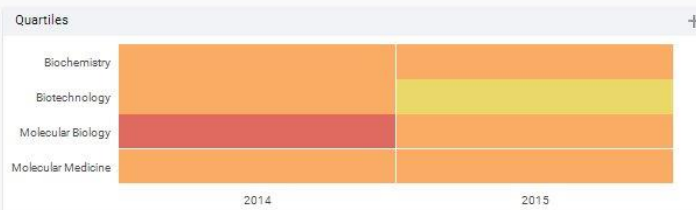
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Частные издательства в обоих случаях. Они не связаны с необходимостью соблюдать все правила больших международных издательств

При этом не очень важно, берут ли они за публикации деньги (Aging et al.) или нет (AN)

Кроме того:

- Спецвыпуски (приглашенные редакторы)
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- Поиск «хороших» журналов с «портфельными» проблемами (например, сезонными)
- Указание имен подходящих и неподходящих рецензентов

Впрочем, есть еще один вариант – улучшить «плохую» статью, приведя ее в соответствие с требованиями «хороших» журналов (стиль, данные, правильный английский язык, корректно оформленный список литературы и т.п.). Но об этом подходе на нашей конференции уже говорили сотни раз...

**Уметь говорить для оратора менее важное
достоинство, нежели уметь остановиться**

Сенека

Когда человек говорит мало, он кажется умнее

Максим Горький

Спасибо за внимание/терпение!

