My Words or Your Words? Detecting and Investigating Plagiarism

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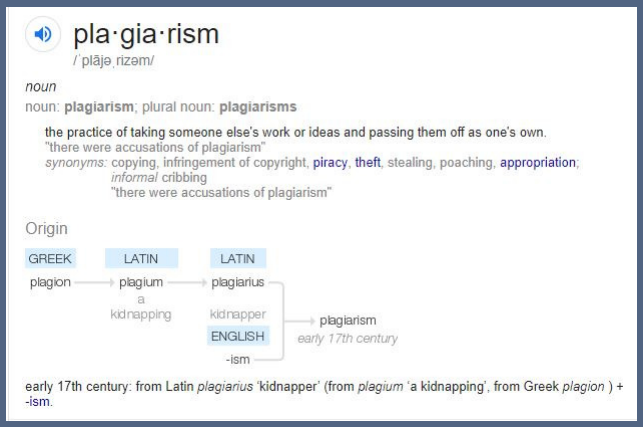
Simple Definition



pla·gia·rism
/ˈplājəˌrizəm/

noun
noun: **plagiarism**; plural noun: **plagiarisms**

the practice of taking someone else's work or ideas and passing them off as one's own.
"there were accusations of plagiarism"
synonyms: copying, infringement of copyright, **piracy**, **theft**, stealing, poaching, **appropriation**; *informal* cribbing
"there were accusations of plagiarism"

Origin

GREEK: plagion — a kidnapping
LATIN: plagium → plagiarius — kidnapper
LATIN: -ism
ENGLISH: plagiarism — *early 17th century*

early 17th century: from Latin *plagiarius* 'kidnapper' (from *plagium* 'a kidnapping', from Greek *plagion*) + -ism.

Copyright Infringement

Definition in Scholarly Publishing

Reusing someone else's words or work without permission and proper attribution

AND

Reusing your own published work without permission and proper attribution

Two Tales of Potential Plagiarism

Style differences are key to identification.

Tale 1:

Invited Review

Japanese author

Clue 1

In search for reviewers found article in a review journal with a very similar title and abstract…

by the same author!

Clue 2

Submitted manuscript text was very different in style and English usage than was all correspondence with author.

Investigation

Step 1: Obtain full text copy of the highly similar review article

Step 2: Compare overall organization of the two articles (submitted and published)

Step 3: Read and compare an entire section

Step 4: Examine reference list and order of cited references

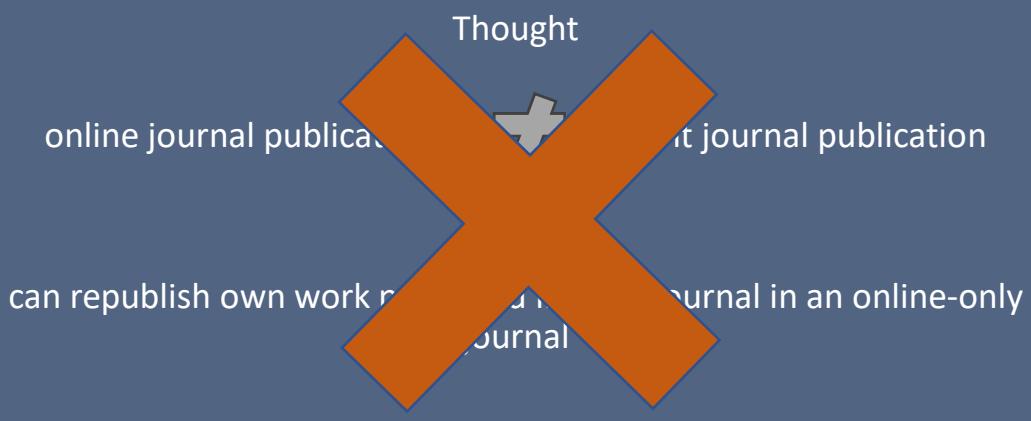
Findings

- Structure and organization identical
- Beginning and ending sentences in paragraphs identical
- Text identical within a section
- References mostly identical
- References presented in same order

Self-Plagiarism Confirmed: Next Steps

1. Document issue and place copy of plagiarized review in tracking system with manuscript record
2. Reject manuscript with clear explanation of issue to authors
3. Place copy of author response in tracking system with manuscript record

Author's Response



Tale 2: Primary Research Article

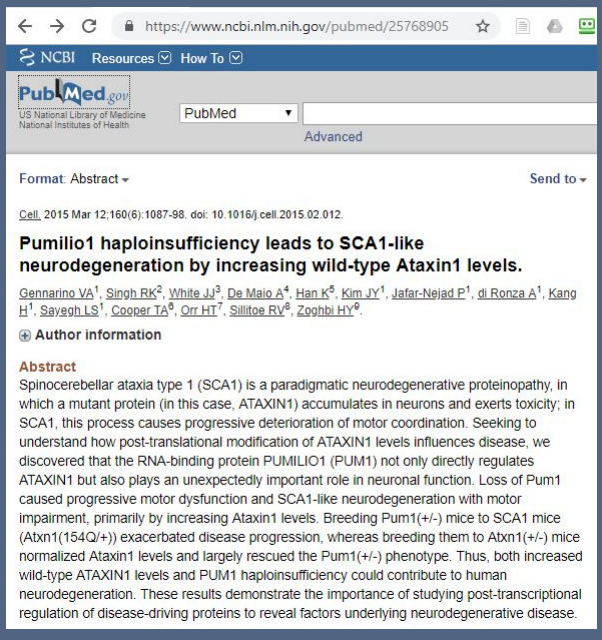
Clues

- Odd phrasing of a sentence in the Introduction
- Undefined abbreviation
- Awkward sentence on a topic related but not central to authors' expertise
- Phrasing contrasted with that in the rest of the paragraph

In published article's abstract:
"Loss of Pum1 caused progressive motor dysfunction and SCA1-like neurodegeneration with motor impairment, primarily by increasing Ataxin1 levels."

In submitted article's introduction:

Mouse and human PUM1, one of two PUMs encoded in mammalian genes, repress *ATXN1* mRNA and loss of PUM1 leads to SCA1-like neurodegeneration with motor impairment[16]. Other report showed that PUM2 controls axon potential in rat neurons by maintaining proper amount of voltage-gated sodium channel transcript (NaV1.6)[17]. PUM1 or PUM2 knockout mice showed reduced body mass and defect on fertility[18,19].



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Cell. 2015 Mar 12;160(6):1087-98. doi: 10.1016/j.cell.2015.02.012.

Pumilio1 haploinsufficiency leads to SCA1-like neurodegeneration by increasing wild-type Ataxin1 levels.

Gennarino VA[1], Singh RK[2], White JJ[3], De Maio A[4], Han K[5], Kim JY[1], Jafar-Nejad P[1], di Ronza A[1], Kang H[1], Sayegh LS[1], Cooper TA[6], Orr HT[7], Sillitoe RV[8], Zoghbi HY[9].

Author information

Abstract

Spinocerebellar ataxia type 1 (SCA1) is a paradigmatic neurodegenerative proteinopathy, in which a mutant protein (in this case, ATAXIN1) accumulates in neurons and exerts toxicity; in SCA1, this process causes progressive deterioration of motor coordination. Seeking to understand how post-translational modification of ATAXIN1 levels influences disease, we discovered that the RNA-binding protein PUMILIO1 (PUM1) not only directly regulates ATAXIN1 but also plays an unexpectedly important role in neuronal function. Loss of Pum1 caused progressive motor dysfunction and SCA1-like neurodegeneration with motor impairment, primarily by increasing Ataxin1 levels. Breeding Pum1(+/-) mice to SCA1 mice (Atxn1(154Q/+)) exacerbated disease progression, whereas breeding them to Atxn1(+/-) mice normalized Ataxin1 levels and largely rescued the Pum1(+/-) phenotype. Thus, both increased wild-type ATAXIN1 levels and PUM1 haploinsufficiency could contribute to human neurodegeneration. These results demonstrate the importance of studying post-transcriptional regulation of disease-driving proteins to reveal factors underlying neurodegenerative disease.

Plagiarism?

Technically, no.

But could be a red  flag

Follow Up

Check a few other cited articles for copied content

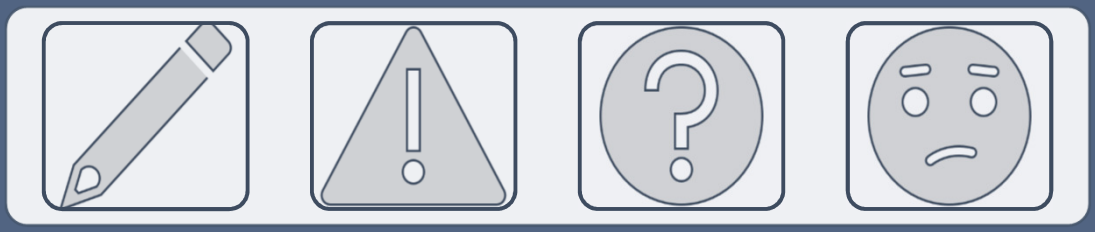
If no

- recommend to authors that they rephrase
- or consider citation adequate

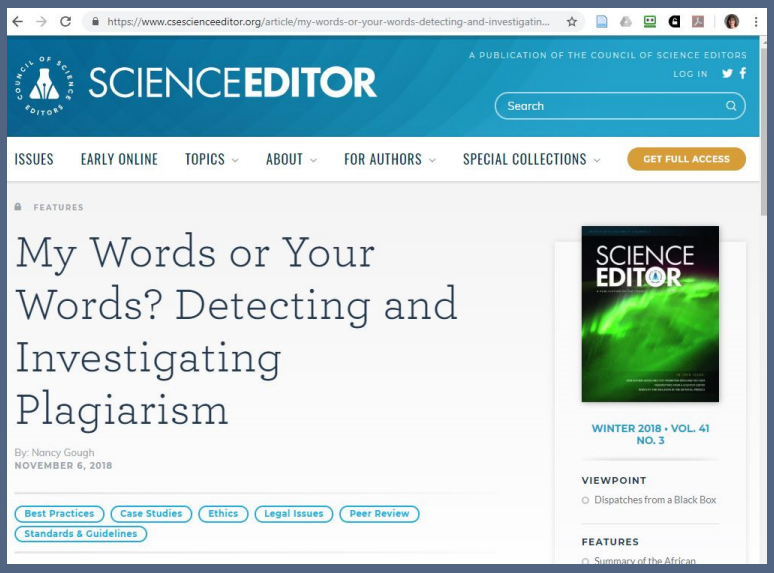
If yes

- document in tracking system
- alert authors to problem
- require rephrasing
- or reject

Common Clues



Mismatched writing style between -correspondence and article -within paragraphs of article	Introduction of a new abbreviation for a term consistently abbreviated elsewhere in manuscript	Statements with citations but lacking appropriate context or interpretation	Inability of authors to rephrase or clarify upon request



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