A Manuscript Microscope 'Sentence Audit' of:

MG53 is not a critical regulator of insulin signaling pathway in skeletal muscle

What is the Manuscript Microscope Sentence Audit?

The Manuscript Microscope Sentence Audit is a research paper introspection system that parses the text of your manuscript into minimal sentence components for faster, more accurate, enhanced proofreading.

Why use a Sentence Audit to proofread your manuscript?

- Accelerated Proofreading: Review long technical papers in a fraction of the usual time.
- Superior Proofreading: Detect subtle errors that are invisible to traditional methods.
- Focused Proofreading: Inspect each individual sentence component, one at a time.
- Easier Proofreading: Take the hardship out of examining complex academic text.
- Safer Proofreading: Ensure every single word of your manuscript is correct.

+ Bonus 1: Improved Productivity: Rapidly refine rough drafts to polished papers.
+ Bonus 2: Improved Authorship: Cultivate a clear, concise, consistent, writing style.
+ Bonus 3: Improved Reputation: Become known for rigorously precise publications.

Manuscript Source: https://www.biorxiv.org/content/10.1101/2020.12.24.424288v1

Manuscript Authors: Clothilde Philouze, Sophie Turban, Béatrice Cremers, Audrey Caliez, Gwladys Lamarche, Catherine Bernard, Nicolas Provost & Philippe Delerive

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Features of the Sentence Audit:

The Sentence Audit combines two complementary proofreading approaches:

1. Each sentence of your text is parsed and displayed in isolation for focused inspection.
2. Each individual sentence is further parsed into Minimal Sentence Components for a deeper scrutiny of the clarity, composition and consistency of the language you used.

The Minimal Sentence Components shown are the smallest coherent elements of each sentence of your text as derived from it's conjuctions, prepositions and selected punctuation symbols (i.e. commas, semicolons and rounded brackets).

The combined approaches ensure easier, faster, more effective proofreading.

Comments and Caveats:

• The sentence parsing is achieved using a python natural language processing pipeline and may result in occasional instances where two sentences are displayed as one.

• Depending on the source of the input text, the Sentence Audit may contain some html artefacts that are parsed as sentences (E.g. "Download figure. Open in new tab").

• Always consult the original research paper as the reference source of the text.

Contact Information:

To get a Manuscript Microscope Sentence Audit of any other research paper, simply forward any copy of the text to John.James@OxfordResearchServices.com.

Any queries, feedback or suggestions are also very welcome.

Research Paper Sections:

The sections of the input text parsed in this audit.

Section No.	Headings	Sentences
Section: 1	Abstract	11
Section: 2	Introduction	35
Section: 3	Materials and Methods	153
Section: 4	Conclusions	2
Section: 5	Acknowledgments	2
N/A	--- --- --- --- --- --- ---	0
N/A	--- --- --- --- --- --- ---	0
N/A	--- --- --- --- --- --- ---	0
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The Sentence Audit Of The Research Paper

Title # MG53 is not a critical regulator of insulin signaling pathway in skeletal muscle

S1 [001] Abstract

S1 [002] In type 2 diabetes (T2D), both muscle and liver are severely resistant to insulin action.

In type 2 diabetes ...
... (T2D), ...
... both muscle ...
... and liver are severely resistant ...
... to insulin action.

S1 [003] Muscle insulin resistance accounts for more than 80% of the impairment in total body glucose disposal in T2D patients and is often characterized by an impaired insulin signaling.

Muscle insulin resistance accounts ...
... for more than 80% ...
... of the impairment ...
... in total body glucose disposal ...
... in T2D patients ...
... and is often characterized ...
... by an impaired insulin signaling.

S1 [004] Mitsugumin 53 (MG53), a muscle-specific TRIM family protein initially identified as a key regulator of cell membrane repair machinery has been suggested to be a critical regulator of muscle insulin signaling pathway by acting as ubiquitin E3 ligase targeting both the insulin receptor and insulin receptor substrate 1 (IRS1).

Mitsugumin 53 ...
... (MG53), ...
... a muscle-specific TRIM family protein initially identified ...
... as a key regulator ...
... of cell membrane repair machinery has been suggested ...
... to be a critical regulator ...
... of muscle insulin signaling pathway ...
... by acting ...
... as ubiquitin E3 ligase targeting both the insulin receptor ...
... and insulin receptor substrate 1 ...
... (IRS1).

S1 [005] Here, we show using in vitro and in vivo approaches that MG53 is not a critical regulator of insulin signaling and glucose homeostasis.

Here, ...
... we show ...
... using ...
... in vitro ...

... and ...
... in vivo approaches ...
... that MG53 is not a critical regulator ...
... of insulin signaling ...
... and glucose homeostasis.

S1 [006] First, MG53 expression is not consistently regulated in skeletal muscle from various preclinical models of insulin resistance.

First, ...
... MG53 expression is not consistently regulated ...
... in skeletal muscle ...
... from various preclinical models ...
... of insulin resistance.

S1 [007] Second, MG53 gene knock-down in muscle cells does not lead to impaired insulin response as measured by Akt phosphorylation on Serine 473 and glucose uptake.

Second, ...
... MG53 gene knock-down ...
... in muscle cells does not lead ...
... to impaired insulin response ...
... as measured ...
... by Akt phosphorylation ...
... on Serine 473 ...
... and glucose uptake.

S1 [008] Third, recombinant human MG53 does not alter insulin response in both differentiated C2C12 and human skeletal muscle cells.

Third, ...
... recombinant human MG53 does not alter insulin response ...
... in both differentiated C2C12 ...
... and human skeletal muscle cells.

S1 [009] Fourth, ectopic expression of MG53 in HEK293 cells lacking endogenous MG53 expression fails to alter insulin response as measured by Akt phosphorylation.

Fourth, ...
... ectopic expression ...
... of MG53 ...
... in HEK293 cells lacking endogenous MG53 expression fails ...
... to alter insulin response ...
... as measured ...
... by Akt phosphorylation.

S1 [010] Finally, both male and female mg53 –/– mice were not resistant to high fat induced obesity and glucose intolerance compared to wild-type mice.

Finally, ...
... both male ...
... and female mg53 –/– mice were not resistant ...
... to high fat induced obesity ...
... and glucose intolerance compared ...

... to wild-type mice.

S1 [011] Taken together, these results strongly suggest that MG53 is not a critical regulator of insulin signaling pathway in skeletal muscle.

Taken together, ...
... these results strongly suggest ...
... that MG53 is not a critical regulator ...
... of insulin signaling pathway ...
... in skeletal muscle.

S2 [012] Introduction

S2 [013] Type 2 diabetes (T2D) is a global epidemic affecting more than 370 million people worldwide.

Type 2 diabetes ...
... (T2D) ...
... is a global epidemic affecting more than 370 million people worldwide.

S2 [014] It is a systemic and progressive disease characterized by hyperglycemia arising, at least in part, from beta cell dysfunction and peripheral insulin resistance [1,2].

It is a systemic ...
... and progressive disease characterized ...
... by hyperglycemia arising, ...
... at least ...
... in part, ...
... from beta cell dysfunction ...
... and peripheral insulin resistance [1,2].

S2 [015] Obesity is a major risk factor for T2D [3].

Obesity is a major risk factor ...
... for T2D [3].

S2 [016] Indeed, more than 80% of patients with T2D are overweight or obese which is a major root cause for the development of insulin resistance [4].

Indeed, ...
... more than 80% ...
... of patients with T2D are overweight ...
... or obese ...
... which is a major root cause ...
... for the development ...
... of insulin resistance [4].

End of Sample Audit

This is a truncated Manuscript Microscope Sample Audit.

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