A Manuscript Microscope 'Sentence Audit' of:

Procollagen C-Proteinase Enhancer 1 (PCPE-1) is a marker of myocardial fibrosis and impaired cardiac function in a murine model of pressure overload

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: Examine long technical texts in a fraction of the usual time.
- Superior Proofreading: Detect subtle errors that are invisible to traditional methods.
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- Reliable Proofreading: Ensure every single word of your manuscript is correct.
- Easier Proofreading: Take the hardship out of crafting academic papers.

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.

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Manuscript Authors: Priscillia Lagoutte, Alexandra Oudot, Mélissa Dussoyer, Victor Goncalves, Mélanie Guillemin, Olivier Bouchot, David Vandroux, Pierre-Simon Bellaye, Catherine Moali & Sandrine Vadon-Le Goff

Audit Date: 22/03/21 **Audit Identifier:** V83N2K3BF52988T **Code Version:** 3.6

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 review 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 conjunctions, prepositions and selected punctuation symbols (i.e. commas, semicolons, round and square brackets).

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

Comments and Caveats:

• The sentence parsing is achieved using a prototype natural language processing pipeline written in Python and may include occasional errors in sentence segmentation.

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

• Always consult the original research paper as the true reference source for 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.

All queries, feedback or suggestions are also very welcome.

Research Paper Sections:

The sections of the research paper input text parsed in this audit.

Section No.	Headings	Sentences
Section: 1	Abstract	14
Section: 2	(1) Introduction	16
N/A	--- --- --- --- --- --- ---	0
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The Sentence Audit Of The Research Paper

Title **Procollagen C-Proteinase Enhancer 1 (PCPE-1) is a marker of myocardial fibrosis and impaired cardiac function in a murine model of pressure overload**

S1 [001] Abstract

S1 [002] Aims

Aims

S1 [003] Procollagen C-proteinase enhancer 1 (PCPE-1) is an extracellular matrix protein and a major regulator of fibrillar collagen biosynthesis.

Procollagen C-proteinase enhancer 1 ...
... (PCPE-1) ...
... is an extracellular matrix protein ...
... and a major regulator ...
... of fibrillar collagen biosynthesis.

S1 [004] Previous work has shown that its abundance is often increased in the context of tissue repair and fibrosis.

Previous work has shown ...
... that its abundance is often increased ...
... in the context ...
... of tissue repair ...
... and fibrosis.

S1 [005] The present study was designed to evaluate its potential as a biomarker of myocardial interstitial fibrosis (MIF), a well-established pathogenic pathway leading to heart failure.

The present study was designed ...
... to evaluate its potential ...
... as a biomarker ...
... of myocardial interstitial fibrosis ...
... (MIF), ...
... a well-established pathogenic pathway leading ...
... to heart failure.

S1 [006] Methods and Results

Methods ...
... and Results

S1 [007] Cardiac fibrosis was induced in rats using an optimized model of chronic pressure overload triggered by angiotensin II and Nω-nitro-L-arginine methyl ester (L-NAME).

Cardiac fibrosis was induced ...
... in rats ...

... using an optimized model ...
... of chronic pressure overload triggered ...
... by angiotensin II ...
... and Nω-nitro-L-arginine methyl ester ...
... (L-NAME).

S1 [008] All treated animals suffered from heart hypertrophy and the increase in heart collagen volume fraction (CVF), evidenced by histology and 68Ga-Collagelin uptake, confirmed the development of cardiac fibrosis.

All treated animals suffered ...
... from heart hypertrophy ...
... and the increase ...
... in heart collagen volume fraction ...
... (CVF), ...
... evidenced ...
... by histology ...
... and 68Ga-Collagelin uptake, ...
... confirmed the development ...
... of cardiac fibrosis.

S1 [009] Functional analysis by simultaneous PET-MRI further showed that our model closely reflected the pathological features seen in human MIF, including left ventricle thickening and diastolic dysfunction associated with decreased ejection fraction.

Functional analysis ...
... by simultaneous PET-MRI further showed ...
... that our model closely reflected the pathological features seen ...
... in human MIF, ...
... including left ventricle thickening ...
... and diastolic dysfunction associated ...
... with decreased ejection fraction.

S1 [010] PCPE-1 mRNA and protein levels were augmented by factors of 3.4 and 6.1 respectively in the heart tissue of treated rats.

PCPE-1 mRNA ...
... and protein levels were augmented ...
... by factors ...
... of 3.4 ...
... and 6.1 respectively ...
... in the heart tissue ...
... of treated rats.

S1 [011] Moreover, protein abundance was well-correlated with CVF ($r=0.92$, $p<0.0001$) and PCPE-1 immuno-detection mainly localized the protein to fibrotic areas.

Moreover, ...
... protein abundance was well-correlated ...
... with CVF ...
... (r=0.92, ...
... p<0.0001) ...
... and PCPE-1 immuno-detection mainly localized the protein ...
... to fibrotic areas.

S1 [012] Finally, PCPE-1 plasma levels measured by ELISA were increased in fibrotic rats compared to controls.

Finally, ...
... PCPE-1 plasma levels measured ...
... by ELISA were increased ...
... in fibrotic rats compared ...
... to controls.

S1 [013] Conclusion

Conclusion

S1 [014] Together, our findings demonstrate that PCPE-1 levels in the heart and circulation tightly reflect the cardiac fibrosis status and heart function impairment in rats and suggest that it could be a very useful marker to monitor human heart diseases leading to fibrosis.

Together, ...
... our findings demonstrate ...
... that PCPE-1 levels ...
... in the heart ...
... and circulation tightly reflect the cardiac fibrosis status ...
... and heart function impairment ...
... in rats ...
... and suggest ...
... that it could be a very useful marker ...
... to monitor human heart diseases leading ...
... to fibrosis.

S2 [015] (1) Introduction

S2 [016] Heart failure (HF) is a major cause of morbidity and mortality worldwide and the most frequent source of hospitalization for patients over 65 years of age1.

Heart failure ...
... (HF) ...
... is a major cause ...
... of morbidity ...
... and mortality worldwide ...
... and the most frequent source ...
... of hospitalization ...
... for patients ...
... over 65 years ...
... of age1.

S2 [017] Myocardial interstitial fibrosis (MIF) is one of the main pathogenic factors that predispose to HF2,3.

Myocardial interstitial fibrosis ...

... (MIF) ...
... is one ...
... of the main pathogenic factors ...
... that predispose ...
... to HF2,3.

S2 [018] Resulting from a variety of acute or chronic injuries, it is characterized by an aberrant and persistent tissue remodeling response, leading to an excessive accumulation of extracellular matrix (ECM) produced by activated fibroblasts (myofibroblasts)4,5.

Resulting ...
... from a variety ...
... of acute ...
... or chronic injuries, ...
... it is characterized ...
... by an aberrant ...
... and persistent tissue remodeling response, ...
... leading ...
... to an excessive accumulation ...
... of extracellular matrix ...
... (ECM) ...
... produced ...
... by activated fibroblasts ...
... (myofibroblasts)4,5.

S2 [019] As opposed to the large scars resulting from myocardial infarction, interstitial fibrosis is characterized by diffuse and patchy areas located around cardiomyocytes or vessels6.

As opposed ...
... to the large scars resulting ...
... from myocardial infarction, ...
... interstitial fibrosis is characterized ...
... by diffuse ...
... and patchy areas located ...
... around cardiomyocytes ...
... or vessels6.

S2 [020] This extensive tissue remodeling impairs normal physiological heart function, and results in myocardial stiffening, mechanical, electrical and/or vasomotor dysfunctions that can ultimately lead to death2,7–9.

This extensive tissue remodeling impairs normal physiological heart function, ...
... and results ...
... in myocardial stiffening, ...
... mechanical, ...
... electrical ...
... and/or vasomotor dysfunctions ...
... that can ultimately lead ...
... to death2,7–9.

S2 [021] Regardless of the etiology of the disease, fibrillar collagens I and III are the major components of the interstitial ECM observed in the fibrotic myocardium5,10.

Regardless ...

End of Sample Audit

This is a truncated Manuscript Microscope Sample Audit.

To get the full audit of this text (or any other research paper),
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John.James@OxfordResearchServices.com
