如何写好SCI论文的投稿信 (Cover Letter)及实例模板

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如何写好SCI论文的Cover Letter (投稿信)?本文以Cover Letter 写作实例模板的形式介绍一下Cover Letter的写作要点和如何写好Cover Letter。

Cover Letter虽然不属于论文的一部分，但是Cover Letter对于一篇需要投稿的论文来说，其重要性与论文不相上下。有时编辑可能看一下Cover Letter就决定是否继续看一下论文，有的论文看一下Cover Letters可能就给退稿了。因此，如何写好Cover Letter对于一篇论文来说非常重要。

Cover Letter 写作要点

Cover Letter的内容必须能够激励编辑继续去读你的文章全文。因此，你工作中的重要内容(例如创新性和在领域内的意义等)需要在Cover Letter中被呈现出来。一般情况下，Cover Letter的长度在五段以内。太长的话会让编辑感觉作者心虚，太短的话不足以充分表达文章所做工作的重点内容。我们现在根据Cover Letter的写作顺序来介绍相关要点。

首先，向编辑简要介绍一下论文的内容以及参与此项研究工作的作者名单。

在这里不需要太详细，即使是工作中包含了一些复杂的实验部分。

其次，重点解释为什么该研究是有创新性的而且会受大家感兴趣。

在这里，你可以介绍有哪些创新性的技术被用到、你的实验样本数量足够多、研究成果将对这个领域有较大影响等。 一些临床国际期刊还要求你介绍这项工作会对将来的实际治疗应用有什么意义。需要注意的是写作时包含要点而不用仔细解释。同时，尽量让编辑看到你的工作是和所投期刊非常相关的。最好是你的工作也刚好验证了别的研究团队在这个期刊以前发表的成果，或者与他们结果相反，而你也用很好的证明。

再次，你可以介绍你和你的研究团队：主要研究人员，实验室的研究方向，以及与这篇文章的关系。

同样，你可以解释为什么要选择这个期刊来投稿。如果此项工作有与其他实验室合作完成，你也可以在这里介绍相关合作团队。

最后，声明你的文章没有任何利益方面的冲突(查询各期刊在利益冲突政策方面的描述，因为每

个期刊有不同的相关规定)。

同时，你需要表明这项研究没有发表过，而且也没同时在别的期刊投稿。

当然，整篇Cover Letter的写作要没有语法和拼写错误，所以你要再三检查。如果你的母语不是英语，可以让朋友或专业编辑服务公司帮忙较对和润色。总之，始终带着积极的态度来完成一篇能说服编辑去仔细进行审阅你的论文全文的Cover Letter。

Cover Letter 写作实例模板

范例1：《欧洲心血管病杂志》

Dear editorial board of European Journal of Cardiology,

Please find enclosed the manuscript: “ The angiotensin-converting enzyme is not a risk factor for myocardial infarction in French individuals ”， by Sarah H., et al., to be submitted as a Short Communication to the European Journal of Neurology for consideration of publication. All co-authors have seen and agree with the contents of the manuscript and there is no financial interest to report. We certify that the submission is original work and is not under review at any other publication.

In this manuscript, we report the results of the first study on the genetic and functional roles of the ACE on the risk of suffering a myocardial infarction in the French population. Indeed, we genotyped the rs4341 polymorphism in 531 IS cases and 549 healthy controls, and then performed functional studies by measuring serum ACE protein level and activity in healthy controls, stroke patients at baseline and stroke patients 24h after stroke symptoms onset. The results from our study did not reveal any association of the ACE variant with myocardial infarction, although it affected ACE protein level, and ischemic stroke patients showed lower ACE level than controls in the acute phase but not in the chronic phase.

We believe that our findings could be of interest to the readers of European Journal of Cardiology because they bring new and strong evidence that the ACE gene and protein are not a risk factor for myocardial infarction.

We hope that the editorial board will agree on the interest of this study.

Sincerely yours,

Sarah H. on behalf of the authors.

Corresponding author: Sarah Hamilton at Cardiovascular Research Laboratory, Marie Curie Research Institute, 75000, Paris, France, xxx@mariecurie.fr, phone number: +33582246xxx, fax number: +33582246xxx.

范例2：《动脉粥样硬化》的修改稿回复

Dear Dr Enzo Montanero,

Thank you for considering the revised version of our manuscript ACE variants and risk of Cardiovascular Disease, by Sarah H. et al. for publication in Atherosclerosis. We are thankful to the referees and the Editor for pointing out some important modifications needed in the report. We have thoughtfully taken into account these comments. The explanation of what we have changed in response to the reviewers ’ concerns is given point by point in the following pages.

We believe that the comments have been highly constructive and very useful to restructure the manuscript. We also believe that the new data included in the article really improved the quality of both our genetic and functional analysis. Indeed, we now show that the A allele of the rs10947 SNP of the ACE gene is a risk factor for all etiologies of cardiovascular disease (the association in the overall population resisted correction for multiple testing by Bonferroni)。 Moreover, we now show that mRNA levels of the ACE gene are higher in MI cases during the acute phase than in healthy controls or MI cases 3 months after the event. and IS cases in the stable condition.

We hope that all these changes fulfil the requirements to make the manuscript acceptable for publication in Atherosclerosis.

Looking forward to hearing from you soon.

Sincerely yours,

Sarah H. and Lucas Delphino on behalf of the authors.

Corresponding author: Lucas Delphino at Alzheimer ’ s Disease Laboratory, Marie Curie Research Institute, 75000, Paris, France, xxx@mariecurie.fr, phone number: +33582246xxx, fax number: +33582246xxx.

范例3：《老年神经生物学》

Dear editorial board of Neurobiology of Aging,

Please find enclosed the manuscript: ACE variants and risk of Alzheimer ’ s Disease, by Sarah Hamilton et al., to be submitted as an Original Research Article to Neurobiology of Aging. All co-authors have seen and agree with the contents of the manuscript and there is no financial interest to report. We certify that the submission is not under review at any other publication.

In this manuscript, we report the results of a genetic and functional study in a white population of sporadic Alzheimer ’ s Disease patients on the risk of suffering cognitive impairments.

We believe that our findings could be of interest to the readers of Neurobiology of Aging, because they could have a great impact on the diagnostic, prognostic and treatment of patients with Alzheimer ’ s Disease. Indeed, the ApoE gene is the only well recognized risk factor for Alzheimer ’ s Disease at the moment (Goder et al. 1998), and we report here evidence that the ACE gene could also be involved in this disease. Moreover, we show that the variant studied modulates ACE levels and increase the predictive value of the ApoE gene.

This study could thus have a great pharmacogenetic interest and bring new and important light in the field of

Alzheimer’s Disease management and we hope that the editorial board and the reviewers will agree on the interest of this study.

Sincerely yours,

Sarah H. and Lucas Delphino on behalf of the authors.

Corresponding author: Lucas Delphino at Alzheimer’s Disease Laboratory, Marie Curie Research Institute, 75000, Paris, France, xxx@mariecurie.fr, phone number: +33582246xxx, fax number: +33582246xxx.

范例4：《临床化学》

Dear Dr Broderick,

Please find enclosed the manuscript: Association between Estrogen Receptor Alpha (ESR1) genetic variants and risk of Stroke, by Sarah H., Isaac S., Marta L., Marc C. and Julien S. to be submitted as an new article to Clinical Chemistry.

In this manuscript, we report the results of a nested case-control study in a French population on the risk of suffering an ischemic stroke associated with genetic variants in the gene coding for the protein Estrogen Receptor Alpha.

We believe that our findings could be of interest to the readers of Clinical Chemistry because they replicate partially the results observed by Rexrode et al. in a white American population and published in Clinical Chemistry in October 2007. Indeed, we demonstrate an association of the rs1271673 SNP T variant and a very common haplotype encompassing the rs1271673 variant with an increased risk of ischemic stroke.

We hope that the editorial board and the reviewers will agree on the interest of this study.

Sincerely yours,

Sarah H

范例5：《脑血管疾病》

Dear editorial board of Cerebrovascular Diseases,

Please find enclosed the manuscript: Association of a SNP in the ALOX5AP gene with risk of Ischemic Stroke, by Sarah H., et al., to be submitted as a Original Research Article to Cerebrovascular Diseases for consideration of publication. All co-authors have seen and agree with the contents of the manuscript and there is no financial interest to report. We certify that the submission is original work and is not under review at any other publication.

In this manuscript, we report the results of the first study on the genetic and functional roles of the ALOX5AP and PDE4D genes on the risk of suffering an ischemic stroke in the French population. We believe that our findings could be of interest to the readers of Cerebrovascular Diseases because they bring

new light on the controversial role of the ALOX5AP and PDE4D genes in stroke and replicate some of the results observed by Helgadottir et al. in an Icelandic population and published in Nature Genetics in 2004. Indeed, we demonstrate an association resistant to Bonferroni of the SG13S114 (rs10507391) T allele of the ALOX5AP gene with an increased risk of ischemic stroke in white populations through an actualized meta-analysis, and in the never tested before Iberian population through case-control study. Moreover, we investigated and showed for the first time that ALOX5AP mRNA levels depended on the SG13S114 genotypes and stroke patients had higher ALOX5AP mRNA levels than healthy controls. These results thus support a role for the ALOX5AP gene in stroke and uncover diagnostic and therapeutic expectations.

We hope that the editorial board will agree on the interest of this study.

Sincerely yours,

Sarah H. and Lucas D. on behalf of the authors.

Corresponding author: Lucas D. at Stroke Laboratory, Marie Curie Research Institute, 75000, Paris, France, xxx@mariecurie.fr, phone number: +33582246xxx, fax number: +33582246xxx.