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ACHE OFFICE OF RESEARCH

GUIDE TO WRITING A SUCCESSFUL ABSTRACT

Use these steps and subsequent example of a student abstract to guide you as you create your abstract. **This guide is designed in a way to help you think through the process of how to effectively organize all of the knowledge you know about your topic and present it to the public.** Remember, your faculty mentor and the ACHE Office of Research are here to support you and are committed to your success!

1. Create a Title
    a. Your title should be descriptive and engaging enough that a reader can tell what your project is about and WHY it matters/is unique.
2. Include Authors and Affiliations
    a. Include all authors (include credentials) that were involved in your project and note the organizations/departments to which they belong.
3. Establish the importance of your research
    a. There is a reason that you (and your team) conducted this research project. WHAT is it? How is the research you completed unique? Including this helps to engage the reader in your research and to continue reading your abstract/come see your poster.
4. Introduce the subject model
    a. Who or what was this research completed on? If the subject is unique then explain the significance of conducting research on this specific subject type.
5. Briefly outline the methods
    a. Explain the design for the project including specifics regarding control and experimental groups (what variables were introduced to the experimental groups, etc.) as well as factors that were ruled out (ex: diet was kept the same for each group).
6. Summarize key results
    a. Present data that are the most significant to your study. Keep in mind that this might include any results that show “no change” between your experimental groups.
7. Highlight significance of the results
    a. Refer back to the importance of conducting the research. What is the impact that your specific research project (and its findings) made to the topic as a whole? Did we learn something valuable? Did we rule out that “x” is not a cause of “y”?

STUDENT EXAMPLE

*Abstract submitted and presented at 2022 Experimental Biology Conference as a member of American Physiological Society. Publicly published abstract.

Renal Derived Human sPRR Increases Plasma Osmolality and Aquaporin 2 Expression in Male but not Female Mice

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Several studies have shown that soluble prorenin receptor (sPRR) plays an important role in fluid and electrolyte balance. In rodent models, water deprivation led to an increase in sPRR in the kidney. Our laboratory previously showed that infusion of mouse recombinant sPRR increased urinary vasopressin but did not change water intake and urine flow rate in female mice. Moreover, a counter regulatory increase in plasma sPRR due to knockdown of prorenin receptor (PRR) in the adipose tissue decreased urine flow rate in both male and female mice. However, there is a gap of knowledge concerning the functional role of locally produced sPRR from the kidney. Additionally, the role of human sPRR in fluid and electrolyte balance has not been evaluated. Therefore, we evaluated the role of renal derived human sPRR in fluid and electrolyte homeostasis.

Human sPRR-Myc-tag transgenic mice were bred with mice expressing Hoxb7/Cre to selectively express human sPRR in the collecting duct (RHsPRR). RHsPRR and control (CTL) male and female mice were fed a standard diet for 10 months (n=8- 11/group). Body weight, body composition and water balance were assessed. Western blot analysis depicted the presence of human sPRR-Myc-tag (28 KDa) in the cortex and medulla of RHsPRR male and female mice validating the humanized sPRR mouse model

Renal derived human sPRR did not change body weight (BW) in male or female mice (Male: CTL: 34±1, RHsPRR: 33±1g; Female: CTL: 28±1, RHsPRR: 30±1g) and kidney function (eGFR: Male: CTL: 817±83, RHsPRR: 1088±163µl/min/100gBW; Female: CTL: 1057±75, RHsPRR: 875±89µl/min/100gBW). Renal derived human sPRR did not significantly increase circulating sPRR (Male: CTL: 3995±643, RHsPRR: 4342±500pg/ml; Female: CTL: 3479±194, RHsPRR: 3948±238pg/ml) suggesting that kidneys are not a source of circulating sPRR. Renal derived human sPRR tended to increase plasma osmolality in males but not females (Male: CTL: 328±3, RHsPRR: 407±55mOsm/kg; Female: CTL: 327±3, RHsPRR: 327±2 mOsm/kg) but did not change urine osmolality in both males and females (Male: CTL: 3855±153, RHsPRR: 3881±275 mOsm/kg; Female: CTL: 3730±341, RHsPRR: 3247±256 mOsm/kg). Moreover, renal derived human sPRR tended to increase urinary vasopressin in males but not females (Male: CTL: 373±70, RHsPRR: 971±369pg/day, P=0.056; Female: CTL: 584±140, RHsPRR: 1873±567pg/day). In line with those results, urine flow rate decreased by ~50% in males but not females (Male: CTL: 1.03±0.2, RHsPRR: 0.57±0.2ml/day; Female: CTL: 0.42±0.1, RHsPRR: 0.47±0.1ml/day).

This title concisely mentions the key/significant findings of the research project (two ways- (1) increase is a key physiological function AND (2) sex difference) without adding unneeded or extra information.

Authors are organized with the presenting author first and then followed by collaborators in alphabetical order. Superscripts are used to identify the different departments of each author. Note: if all authors are from the same department and institution then superscripts are not needed but organization should still be listed beneath author list.

Immediately introduces the overall topic of the research. Mentions what work has already been conducted on this topic and where there are still knowledge gaps. Succinctly describes the REASON for conducting this research (see last sentence).

The subject (in this case animal model) is introduced as mice. Mice are not an unusual subject model so no explanation is needed for why mice are used, BUT the specific mouse model is unique with the insertion of a human gene expression (which is why it is mentioned).

Briefly outlines the data that was collected and how this data was analyzed. Notice that this section does not need to go in depth regarding the specifics of the design. Save the details for your poster!

Results are organized in a way that they tell a story. First, the data is given followed by a summary sentence of what this data shows. Notice that it includes results that show a significant difference as well as results that show no difference. Both are important!

Renal AQP2 expression increased in males while in females, AQP2 expression decreased (Male: CTL: 9.1±3.0, RHsPRR: 43.8±14.0 AU, P<0.05; Female: CTL: 6.0±3.0, RHsPRR: 0.9±0.4 AU). Renal derived human sPRR also significantly increased phosphorylation of ERK1/2 in the cortex ofboth males and females (Male: CTL: 0.42±0.1, RHsPRR: 0.81±0.2 AU; Female: CTL: 0.40±0.1, RHsPRR: 0.63±0.1 AU). Overall, our data suggest that human sPRR exerts an antidiuretic effect in a sex-dependent manner and could contribute to the increase in plasma tonicity by promoting the activation of ERK1/2-AQP2 pathway in male mice.

Briefly summarizes all key results and refers back to the purpose of the study to now identify what strides have been made in the field due to the current study.