How to Write a Strong Discussion

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I M R A D

- ✓ **I**ntroduction : **Why** did you do it? What did you/others do?
- ✓ **M**ethods : **How** did you do it?
- ✓ **R**esults : **What** did you find?
- ✓ **A**nd
- ✓ **D**iscussion : What does it all **mean**?

(Borja, 2014; elsevier.com)

Aims

- To state authors interpretations and opinions,
- To explain the implications of author findings, and make suggestions for future research.
- To answer the questions posed in the Introduction, explain how the results support the answers and, how the answers fit in with existing knowledge on the topic.

(www.sfedit.net; Drotar, 2009)

Importance

- Organized the Discussion.
- Develop an outline to organize author thoughts in a logical form.
- Use a cluster map, an issue tree, numbering, or some other organizational structure.

(www.sfedit.net)

Importance

- Kept as short as possible while clearly and fully stating, supporting, explaining, & defending author answers and discussing other important and directly relevant issues.
- Provide commentary and not reiteration of the results.
- Side issues should not be included, as these tend to obscure the message.
- Help the reader determine what can be positively learned and what is more speculative.

(www.sfedit.net)

General Guidelines

- Organize Discussion from specific to general: author findings to literature, theory, to practice.
- Use same key terms, same verb tense (present tense), & same point of view with questions in Introduction.
- Begin by re-stating hypothesis authors were testing and answering questions posed in introduction.
- Support answers with results.

General Guidelines

- Address all results relating to questions, regardless of statistical significance.
- Describe patterns, principles, & relationships shown by each major finding/result and put them in perspective.
- Defend author answers, if necessary, by explaining both why author answer is satisfactory & why others are not.
- Discuss and evaluate conflicting explanations of results.

General Guidelines

- Discuss any unexpected findings.
- Identify potential limitations and weaknesses.
- Summarize concisely principal implications of the findings, regardless of statistical significance.
- Provide recommendations (no more than two) for future study.

General Guidelines

- Explain how results and conclusions of the study are important & how they influence our knowledge or understanding of problem.
- Discuss everything, but be concise, brief, & specific.
- Describe novel contribution of findings relative to previous research

EXAMPLE

Re-stating Hypothesis

reported in the literature [18,19]. Our study clearly shows that the HAEC frequency was significantly higher in the Duhamel group (28%) than the Soave group (10%). The incidence of enterocolitis following the Duhamel procedure in our study was higher than in previous studies [7,18,20] but with similar results reported by Kim et al. [21], while those of in the Soave group were comparable with other findings

(Parahita et al., 2017)

Discussion

In this study, we have compared two methods, PCR-RFLPs and the TaqMan PCR assay, for genotyping the *RET* rs2435357 polymorphism as a genetic risk for HSCR in the Indonesian population. Our study clearly demonstrated that the PCR-RFLP method is 100% accuracy for correct genotyping and is comparable to the TaqMan assay results.

(Gunadi et al., 2016)

Our study demonstrates that *RET* rs2435357 individually is a strong risk factor with a background allele frequency of ~50% in Indonesia and a relative risk of 4.5 (Table 1). This value is consistent with the genetic effect observed in both European ancestry [7] and Chinese [9] HSCR

(Gunadi et al., 2014)

Weakness

It is well known that either *NRG1* variant has a weaker effect on HSCR with relative risks of 1.68 and 1.98 for rs16879552 and rs7835688, respectively [9]. The results in Table 1 are entirely consistent with these observations with relative risks of 1.6 and 2.0 for rs16879552 and rs7835688, respectively. Moreover, these values predict that we should observe transmission rates in families of 0.62 and 0.67, respectively, also consistent with the observations of 0.62 and 0.63, respectively (Table 2). The lack of statistical significance, except for *NRG1* rs7835688, is most likely caused by the small numbers of trios used for transmission analyses.

(Gunadi et al., 2014)

The finding of epistasis between *NRG1* and *RET*, at the disease penetrance level, suggests that the activity of *NRG1* is downstream of *RET*. However, this epistasis could arise in one of two ways, namely, from functional direct molecular interactions (biological) or from indirect effects amplifying the combined effects on penetrance (epidemiological). The available data suggest that these interactions are direct since a recent study demonstrated functional *Ret–Nrg1* interactions in neural crest isolated from mouse embryonic guts, which are enteric neuron precursors, when treated with *Gdnf* (*Ret* ligand) and *Nrg1* (*ErbB2* ligand) [10]. Specifically, they showed that *Gdnf* negatively

(Gunadi et al., 2014)

Conflicting explanation

Comparison of accuracy between PCR-RFLP and the TaqMan method is still inconclusive and debating although the TaqMan technology is often claimed to be superior to the PCR-RFLP technique [13-16]. One of the disadvantages of the PCR-RFLP method is that more time is needed for the post-amplification steps including a first gel electrophoresis to verify the presence of the PCR product, the restriction endonuclease digestion, and a second gel electrophoresis for restriction pattern visualization. In contrast, the TaqMan method is less time-consuming because an analysis of the amplification can be performed within minutes of completing the thermocycling using the SDS software [13,17]. However, the TaqMan method requires a special and

(Gunadi et al., J Surg Res 2016)

The evolving evidence in HSCR is that epistasis between *RET* and *EDNRB* [9] and between *RET* and *NRG1* [9] (this study) is important to ENS development [23]. Therefore, we hypothesize that compromising either *RET* or *EDNRB* or *NRG1* function or their interactions is detrimental to normal gangliogenesis. In other words, a key set of early developmental genes are the primary target for mutations in HSCR. This result is now clear from studies of both European and Asian

(Gunadi et al., J Ped Surg 2014)

Recommendation for Future Study

Our study revealed that there were no statistically significant differences between HE staining and S100 IHC, however, notably the small sample size of the study implies that a significantly larger sample of patients needs to be involved to better clarify and confirm the results.

(Setiadi et al., BMC Surgery 2017)

Summary

- Organize and focus
- Present novel contribution to findings
- Describe study limitation
- Implication findings for new study, clinical care/policy

THANK YOU

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Mengakar Kuat
Menjulang Tinggi