Hong Kong Physiotherapy Journal (2012) 30, 2—3

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LETTER TO THE EDITOR

# Clinical implications for the effect of glucosamine sulfate iontophoresis on fasting plasma glucose levels

Vikram Mohan, MPT [a], Leonard Joseph, MSPT [b,*]

[a] Department of Physiotherapy, Faculty of Health Sciences, Universiti Technologi MARA, Puncak Alam, Selangor, Malaysia
[b] Programme of Physiotherapy, Faculty of Allied Health Sciences, Universiti Kebangsaan Malaysia, Bangi Selangor, Malaysia

We enjoyed reading the recently published paper "Acute effects of combination of glucosamine sulfate iontophoresis with exercise on fasting plasma glucose of participants with knee osteoarthritis" by Onigbinde et al [1]. Glucosamine sulfate is one of the most common drugs prescribed to strengthen cartilage in patients with osteoarthritis. Due to speculation that glucosamine sulfate might increase blood glucose levels, the study by Onigbinde et al generated appreciable clinical interest among readers. However, there are certain points and suggestions that we would like to share with readers before the study's findings are applied clinically.

First, we believe that the quasi experimental design weakens the clinical implications of this study. A quasi-experimental design would typically be adopted in situations where true experiments were not possible. A randomized controlled trial might have been a stronger design to consider for this study given that a recent systematic review supported mixed evidence about the effect of glucosamine on glucose metabolism in humans [2]. The quality of evidence generated in the study by Onigbinde et al would certainly be higher if a controlled and randomized design was embraced.

Secondly, the small sample size used in the study by Onigbinde et al could be another potential limitation for applying the results to clinical practice. As a result of small sample size, the scientific evidence generated by the study outcome may be challenged. In such a case, the power and effect size of the obtained results would be factors to consider. However, these factors were not reported in their study.

We would also like to suggest a few additional thoughts on the intervention adopted in the study by Onigbinde et al. The authors acknowledged that they were not certain about the quantity of glucosamine administered through iontophoresis which could affect plasma glucose concentrations in the blood stream. The authors mentioned detectable glucosamine in the blood stream after iontophoresis and, in our opinion, it would be interesting to evaluate the bioavailability of glucosamine in the blood stream after iontophoresis. It is possible that a drug could be detectable in the blood stream but remain physiologically inactive until it reached a specific level of bioavailability. Therefore, pre-testing the bioavailability of glucosamine sulfate through iontophoresis might be important before starting the intervention.

Similarly, aerobic exercises used as intervention in this study could be well-controlled. Grading and predetermining the intensity of exercise may reduce variations in exercise intensity among the study groups. In addition, the effect of reverse iontophoresis could be verified by having an additional population of matched controls in the study. We would also like to mention that there were two current modes cited by the authors in the study. They had stated

* Corresponding author. Programme of Physiotherapy, School of Rehabilitation Science, Faculty of Allied Health Sciences, Universiti Kebangsaan Malaysia, Jalan Raja Muda Abdul Aziz, UKM Selangor 43600, Kuala Lumpur 50300, Malaysia.
*E-mail address:* leonardjoseph85@hotmail.com (L. Joseph).


doi:10.1016/j.hkpj.2011.11.001

that interrupted galvanic current was used for the purpose of iontophoresis but that they had turned on the direct current when starting iontophoresis. It is not known if the selection of current mode had any association with reverse iontophoresis and this issue might be worth considering when doing interventions with iontophoresis.

Although the author's hypothesis suggested that glucosamine sulfate had not raised the plasma glucose level, the results showed significantly lowered plasma glucose levels in the second group. In our view, lowering of plasma glucose levels in the second group after glucosamine iontophoresis should have been discussed because of significant clinical importance. We hope our suggestions will be of assistance for future studies in this field. We congratulate the authors of this study for their interesting work and suggest well-controlled and randomised trials. We also compliment the editor of the journal for publishing a good article of interest to the public.

## References

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