

Proposal for Controlled Trial to Support the Hypothesis that Competitive Eating may be Protective against Development of Obstructive Sleep Apnea

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Competitive Eating (CE), in recent times, has developed an international reputation (particularly in the United States, Canada, Japan and Australia) as a progressive sporting interest with burgeoning groundswell participatory and spectator support [1]. Speed Eaters or “Wolfers” such as the notable Joey “Jaws” Chestnut, multiple title holder of the July 4 Nathan’s Hot Dog Eating Contest [2], have had to suffer the ignominious “slings and arrows” of assumptions regarding the dangers of the sport, and the messages it sends in the context of rising obesity concerns [3], without any documented controlled trials to confirm such charges. Contrarily, we propose the hypothesis that CE may in fact be protective against onset of OSA, a condition that affects as many as 24% of men and 9% of women [4]. Such an hypothesis could be tested via a randomised controlled clinical trial, with recruited participants keen to transition from amateur consumption (or ‘best available eating practice’) to CE, randomly allocated to immediate entry to training and competition or to ongoing usual eating for 6 months. Both groups would undergo formal in laboratory polysomnography at commencement and 12 months, and then control group participants could still contract to CE thereafter. Such a design would permit support or refutation of our hypothesis. The study could perhaps also incorporate Electromyography (EMG) assessment of masticatory muscles and upper airway dilators (such as Genioglossus) in both groups (performed via needle electrode placement on the evening of polysomnography), to elucidate potential underlying mechanisms and for accurate physiological phenotyping [5]. MRI imaging for anatomical assessment would add further supportive data. Others have published on unusual upper airway muscle strengthening modalities as in didgeridoo playing [6] in controlled trials, and treatment effects have been noted. A negative may be progressive weight gain, and height, weight, body mass index and neck circumference would need to be recorded in both groups at 0, 6 and 12 months.

Conclusion

CE, like other irregular or quirky therapies for enhancing upper airway muscle tone, may be worthy of scrutiny and evaluation as a mechanistic pathway to protect against OSA. The authors welcome input from other OSA researchers and members of the CE fraternity into such evaluation.

References

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