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Bruxism is a continuously distributed behaviour, but disorder decisions are dichotomous (Response to letter by Manfredini, De Laat, Winocur, & Ahlberg (2016))

K. G. RAPHAEL[*], **V. SANTIAGO**[*], and **F. LOBBEZOO**[*,†]

[*]Department of Oral & Maxillofacial Pathology, Radiology, and Medicine, New York University College of Dentistry, New York, NY, USA [†]Department of Oral Health Sciences, Academic Centre for Dentistry Amsterdam (ACTA), MOVE Research Institute Amsterdam, University of Amsterdam and VU University Amsterdam, Amsterdam, The Netherlands

We were extremely pleased to see the letter composed by Manfredini, De Laat, Winocur and Ahlberg (1), many of the co-authors of the original consensus statement (2) on the defining and grading of bruxism. The authors state that they agree 'on almost all the arguments' raised in our recent commentary (3) written as a critique of the original consensus paper. We appreciate their support and agreement, but also welcome the opportunity to respond to several points that they raise, as these points indicate areas in which we need to provide further clarification. We never intended to imply that bruxism behaviour was dichotomous, and we did not comment on the relationship between aetiology and clinical consequences.

The primary issue raised in their letter may derive from a semantic misunderstanding. Stating that (sleep) bruxism is a 'behaviour' in no way precludes the possibility (at some to-be-specified and validated cut point) of it being *more than* a behaviour, either a risk factor or disorder. 'Activity' and 'behaviour' are virtually synonymous in English. To provide an example of an activity than can be both a behaviour and/or indicative of a disorder, compulsive washing of the hands is certainly a behaviour but it can also be part of an obsessive-compulsive disorder in psychiatry, a harmful dysfunction. Certainly, behaviour is not 'black/white'; in fact, we specifically refer to 'the continuum of sleep bruxism behaviour' (p. *pending*). However, the decision that something is a disorder is typically made using some kind of dichotomous cut point, because exceeding the cut point facilitates the decision about need for a clinical action or intervention. This is a matter of clinical efficiency, not inherently reflecting the natural distribution of the underlying behaviour. The chosen cut point may indicate the point at which benefits of further assessment or treatment exceed costs and treatment risks.

The letter writers' statements referring to aetiology are orthogonal to the points raised in our commentary. Behaviour is certainly a physiological phenomenon. The need to understand

Correspondence: K. G. Raphael, NYU College of Dentistry, 380 Second Ave., Suite 301, New York, NY 10010, USA. kgr234@nyu.edu.

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the aetiology of the physiological phenomenon/phenomena is arguably more urgent, when we are concerned that the behaviour represents a disorder, as understanding aetiology may provide clues to understand the nature of the underlying dysfunction and to develop an appropriate intervention. We fail to understand how the authors' groupings based on postulated aetiological factors help to clarify how bruxism is a behaviour, risk factor or disorder.

The letter writers propose that bruxism should be viewed as a disorder 'only when it has consequences'. According to the framework proposed in our commentary, bruxism which only occasionally leads to a harmful consequence but does not routinely lead to harm would be viewed as a risk factor, not a disorder. We believe that this definitional distinction is important. The concept of bruxism as a disorder 'only when it has consequences' is likely to lead to overtreatment of bruxism, in an effort to ward off potential *harmful* consequences that are likely to occur only in a fraction of individuals. It also creates difficulty in making correct treatment decisions in those situations when 'clinically obvious bruxism' (probably characterised by extreme wear) is unlikely to be followed up with polysomnographic verification for current (sleep) bruxism or other factors in isolation, or when other factors work synergistically with bruxism to cause harm. If bruxism is only harmful sometimes, it becomes critical that we work to identify co-risk factors increasing the probability of harm (e.g. an acidic (4) or xerostomic (5) oral environment combined with relatively high or even average levels of bruxism behaviour or functional attrition will be likely to cause more extreme tooth wear). Investigation into the probable multiple risk factors leading to specific harmful oral dysfunctions which were previously assumed to be caused by (sleep) bruxism needs to be conducted. We can then logically consider which of the statistically and clinically significant risk factors is most amenable to effective clinical intervention.

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