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Letter to the Editor

Comments on "Use of Malondialdehyde as a Biomarker for Assesing Oxidative Stress in Different Disease Pathologies: A Review"

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Dear Editor-in-Chief

In a recent article in this journal (1), a limited number of papers dealing with variations of malondialdehyde (MDA) in different pathological conditions were reviewed. After a brief introduction on the importance of oxidative stress, MDA production pathways, and analytical techniques, Singh et al. (1) concluded as "MDA is a useful biomarker for lipid peroxidation and oxidative stress." The aim of this communication is not to criticize the review article, but rather is to point out some further considerations, the validity of MDA measurements and reliability of MDA as a biomarker in clinical studies.

MDA values are collected from a limited number of investigations. Concerning case/control comparisons, significant and non-significant changes were observed (for details please see references of Table 1 of Singh et al. (1)). We are not interested in further details of these comparisons, however would like to pay more attention to the reported MDA levels of healthy controls which differ from 1.08 ± 0.33 nM (2) to 7500 ± 2700 nM (3) or even more, as an example 47180 ± 6960 nM (4). With this wide variation, the main concern is that; which MDA level should be considered as "normal" level against pathological conditions?

From a practical viewpoint, the commonly used methods for MDA determination suffer from a number of limitations including poor reproducibility (5), low repeatability (5), non-specificity (6), lack of full validation data and most of these problems were ignored by many researchers (7). In addition, there are problems regarding biological sample preparation (7), storage (8) and pretreatment (9) procedures. There are also some concerns on the stability of MDA standard solutions, its reactions with various compounds and its quick metabolism. On the other hand, its main derivatizing agent, i.e. thiobarbituric acid (TBA) reacts non-specifically with MDA and a number of other compounds. These cross-reactions of MDA and TBA are some reasons for discrepancies for reported MDA values.

Beside these points, MDA is formed from peroxidation of polyunsaturated fatty acids, reaction of deoxyribose with a hydroxyl radical, as a by-product of prostaglandin synthesis, and a by γ–irradiation of carbohydrates. It could be found in foods and be absorbed from gastrointestinal tract, which alters MDA levels. MDA levels are also affected by cigarette and intake of some drugs.

In conclusion, concerning the above-mentioned points and the characteristics of an ideal bi-

omarker (10), we believe that using MDA as an oxidative stress biomarker needs to be re-evaluated by an expert panel and more reproducible, repeatable and valid analytical methods should be developed.

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