See our full scientific file (40 publications).

SOD B® & AMD Correction

Age Macular Degeneration (AMD) affects 50 millions of people worldwide, and is the leading cause of severe vision loss in Western societies. The macular area of the retina is overexposed to numerous pro-oxidant factors which damage its structure and function. Retina impairments result from the accumulation of Reactive Oxygen Species (ROS), leading to oxidative damages and inflammation. As the first line of antioxidant defenses, and thanks to its anti-inflammatory properties, SuperOxide Dismutase (SOD) plays a key scientifically demonstrated role in AMD prevention and correction.

A universal visual alteration

Age-Macular Degeneration (AMD) is an age-related, progressive degeneration of photoreceptors and their underlying Retinal Pigment Epithelium (RPE) in the macular area of the retina (Figure 1). AMD is responsible for central vision alteration which significantly impacts people's ability to read, watch television or drive.

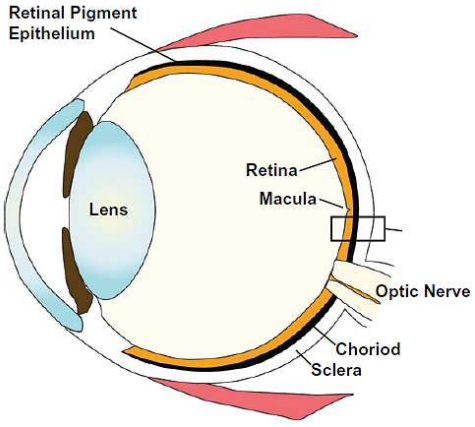

Figure 1: Eye structure and area affected by AMD

AMD: an oxidative process

The eye retina is one of the highest oxygen-consuming tissues. Highly exposed to oxygen, the retina continuously generates high levels of Reactive Oxygen Species (ROS), because of[1, 2]:

- Locally elevated oxygen-tension;
- Direct interface with oxidative products generated by photoreceptor outer segment phagocytosis,
- Direct exposure to exogenous pro-oxidant factors (UV rays, smoking, radiations);
- High polyunsaturated lipid content: risk of lipid peroxidation;
- The aged-related increase of photosensitizers ROS generator (lipofuscin).

Cumulative oxidative damage to proteins, lipids, and DNA leads to AMD development and progression (Figure 2)[3]. Without preventive approach, retina oxidation leads to inflammatory reactions, which lead to RPE cells apoptosis and retinal degeneration[4].

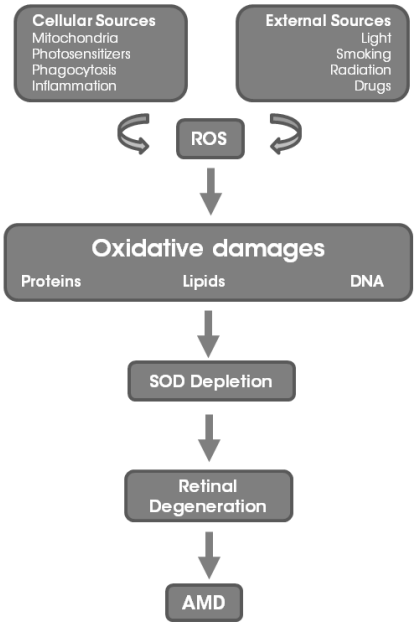

Figure 2: The involvement of oxidative stress in AMD[5].

Bionov, site Agroparc, Bâtiment Orion ; Chemin des Meinajaries, CS 80501 ; 84908 Avignon Cedex 9
Tel: +33(0)490.843.170 – Fax: +33(0)490.840.047 – contact@bionov.fr – www.bionov.fr

SOD depletion favors AMD

As we age oxidative damage increases, while the retina endogenous antioxidant capacity decreases. Epidemiological studies have demonstrated a 30% depletion of SOD levels in the retina of AMD subjects compared to controls (Figure 3)[6].

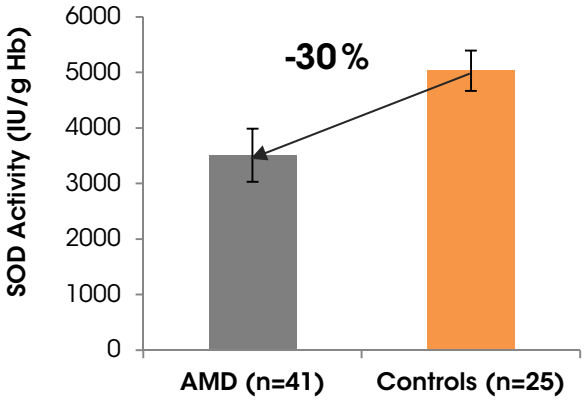

Figure 3: SOD levels in AMD subjects and controls[6]

Additional supports have demonstrated that disruption of the Nuclear Factor Nrf2 gene increases the susceptibility of outer retina to AMD development. Nrf2 is a transcription factor that plays a central role in expression of primary antioxidant enzymes, including SOD. Nrf2-deficient mice developed AMD-like phenotype[7].

The retina SOD depletion participates in[8, 9]:

- Favoring retina oxidation and inflammation;
- Increasing RPE cell apoptosis;
- Altering retinal sensibility;
- Affecting retina structure;

SOD corrects retina oxidation

Superoxide anion ($O_2^{\bullet-}$) is a major source of ROS produced in the mitochondria of retinal cells. As the first line of antioxidant defenses, and due to its mitochondria localization, SOD is the only antioxidant agent that eliminates $O_2^{\bullet-}$ and contributes to maintain a balanced ROS production in the retina.

SOD prevents retinal degeneration

Due to its mitochondrial localization, Mn-SOD plays a critical role in protecting retinal cells against ROS-induced apoptotic cell death.

An overexpression of Mn-SOD protects against ROS-induced apoptosis in mouse retinal cells[10, 11].

SOD preserves retinal sensibility

The SOD protective effect on RPE cells preserves retina thickness and sensibility and prevents retina degeneration, as demonstrated in mice with and without SOD depletion (Figure 4)[12].

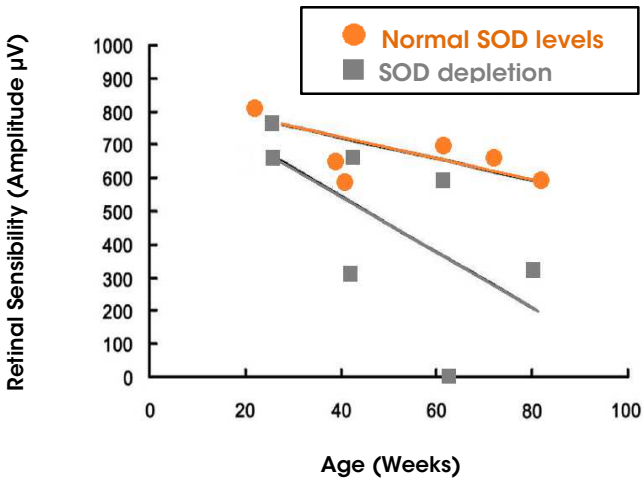

Figure 4: Age changes in retinal sensibility with SOD levels[12].

As the first and most important line of antioxidant defenses, SOD prevents and corrects AMD by acting at different levels. Optimal SOD levels:

- **Prevent retina oxidation**
- **Inhibit retina degeneration**
- **Preserve retinal sensibility**

DMLA affected subjects have a retinal SOD concentration reduced by 30% on average. According to its demonstrated way of action, SOD B® activates Nrf2, which promotes the expression of endogenous SOD in the retina. An oral SOD B® supplementation is an adapted strategy to inhibit AMD development.

Daily recommended dosage: 400 IU SOD.

Bibliography

1. Organisciak DT *et al.* 1996.
2. Mukherjee PK *et al.* 2004.
3. Zarbin M. 2004.
4. Nakazawa T *et al.* 2006.
5. Jarrett S. 2012.
6. Evereklioglu C *et al.* 2003.
7. Zhao Z *et al.* 2011.
8. Imamura Y *et al.* 2006.
9. Justilien V *et al.* 2007.
10. Kasahara E *et al.* 2005.
11. Kanwar M *et al.* 2007.
12. Hashizume K *et al.* 2008.