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VABILO NA PREDAVANJE
V OKVIRU DOKTORSKEGA ŠTUDIJA
KEMIJSKE ZNANOSTI

Prof. dr. Eduardo Agosin Trumper

Department of Chemical and Bioprocess Engineering, School of Engineering, Pontificia Universidad Católica de Chile, Santiago, Chile

z naslovom:

Understanding and managing oxygen content during alcoholic fermentation

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v predavalnici 1 v 1. nadstropju Fakultete
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Vljudno vabljeni!

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Abstract

Oxygen addition is common practice during winemaking. However, it is still mostly carried out empirically and intuitively, and not as the result of the oxygen demanded at each stage of the oenological fermentation. After a description of the instrumentation employed to quantify oxygen dissolution achieved during pump-over operations, I will show the results obtained at different scales, *i.e.* industrial (40,000L fermentors), laboratory (1L) and cellular (metabolic flux analysis) for the evolution and control of this critical variable and its impact on wine quality.

At industrial scale, we designed a set of experiments with different pump-over modes (closed, open, and with Venturi) to evaluate oxygen dissolution and consumption during industrial-scale fermentations. Closed pump-overs incorporate negligible amounts of oxygen, while open pump-overs with Venturi incorporate the highest, *i.e.,* 3 mg/L (approximately twice more oxygen than the conventional open pump-overs). A highly heterogeneous vertical distribution of dissolved oxygen was also found, with approx. 80 % of the total concentrated at the top of the tanks

At laboratory and cellular scales, we simulated the range of dissolved oxygen concentrations that occur after a pump-over during the winemaking process by sparging nitrogen-limited continuous cultures with oxygen-nitrogen gaseous mixtures. When the dissolved oxygen concentration increased from 1.2 to 2.7 □M, yeast cells changed from a fully fermentative to a mixed respirofermentative metabolism. This transition is characterized by a switch in the operation of the tricarboxylic acid cycle (TCA) and an activation of NADH shuttling from the cytosol to mitochondria. Nevertheless, fermentative ethanol production remained the major cytosolic NADH sink under all oxygen conditions, suggesting that the limitation of mitochondrial NADH reoxidation is the major cause of the Crabtree effect.