Too much O₂?

Bloomfield et al. (2) make an interesting and persuasive case for a central role for oxidative damage in generating organisms which, although not dead, cannot be cultured. They also suggest that ‘we must better understand the biochemistry and physiology of the interactions between growth, respiration and the devastatingly destructive power of oxidative damage’. As microbiologists we have become adept at the rigorous exclusion of O₂ from our cultures of strict anaerobes. We are also careful to supply O₂ above the ‘critical O₂ level’ for our highly populated aerobic populations. We would like to emphasize here that from the crowd-protected herd in the ecosystem and on the loop and spreading the individuals to the lonely solitude of the spread plate suddenly elevates their ambient O₂. From dense population foci can easily be shown in microaerophilic protozoa (1); in the natural environment many protozoa migrate vertically in response to the oxygen stratification of stratified lakes to reach their preferred O₂ concentration (4). Even in more aerobic organisms, provision of O₂ in excess can be lethal. In mammalian cells, early events leading to apoptosis include detachment of cytochrome c from the cytosolic face of the inner mitochondrial membrane, opening of a mitochondrial permeability pore, massive release of stored Ca²⁺ and production of reactive O₂ species (5). Provision of reducing power (as respiratory substrate) can protect as effectively as radical scavengers (3). In general, the balance between demand and supply of oxidizing equivalents has to be controlled, and O₂ excess may lead to elevated intracellular concentrations to a level where reactive O₂ species accumulate more rapidly than depletion by cellular defences (7). Intracellular O₂ concentration in Acanthamoeba castellani has been measured, and rarely exceeds 2 μM (i.e. less than 1/100 of its atmospheric value). Steep gradients of O₂, e.g. occur in the plasma membrane, and especially near O₂ sinks (the mitochondria) (8). One mechanism whereby intracellular O₂ concentration is kept low is by means of the cyanide-insensitive electron transport chain (6); operation of this mechanism can adjust redox balance and optimize intracellular O₂ without the involvement of phosphorylating electron transport. It may be especially important in well-known microaerophiles, e.g. Plasmodium falciparum (9), as well as in a host of organisms not recognized as preferring to live at low O₂.

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GUIDELINES

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