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The tropospheric level of the phytotoxic air pollutant ozone has considerably increased during the last century and is expected to continue to rise. Long-term exposures of higher plants to low ozone concentrations affect biochemical processes prior to any detectable symptoms of visible injury. On the other hand, the current critical level of ozone used to determine the threshold for damaging plants (biomass loss) is still based on the seasonal sum of the external concentrations of the pollutant above 40 nl l^{-1} (AOT40). Taking into account the stomatal conductance, a more relevant concept is based upon the actual ozone flux in the leaf through the stomata (cumulative uptake of ozone = CUO). CUO however ignores the internal capacity of leaf defense, which led to the concept of "effective ozone flux", balance between stomatal flux and the intensity of cellular detoxification, with the aim to propose an improved threshold for ozone risk. Although the direct detoxification of ozone (and ROS issued from its decomposition) can primarily be carried out by cell wall ascorbate, the existing level of this antioxidant is not sufficient to indicate the degree of cell sensitivity. The capacity for regeneration of the antioxidant barrier is needed, implying the knowledge of the increased production of reducing power (NAD(P)H), primary supplier for detoxifying processes. It is made possible through the increased participation of the catabolic pathways and associated shunts which can provide NAD(P)H. In addition, the large change in the rubisco/PEPcase ratio, due to a huge increase in activity of the latter enzyme, leads to changes in carbon isotopic discrimination, which could be related to water use efficiency. Some results will be presented knowing that the challenge is to integrate the possible indicators in a leaf model to be used, through an upscaling process, in a tree and forest stand model.

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