I completely support the need for unifying the terminology in the field of mitochondrial physiology and clarifying the underlying concepts, as suggested in this manuscript. I am glad to be part of this endeavour, which reflects important considerations of transdisciplinary unity and general rigor. Of course, I don’t have much to say to improve the manuscript at this point, after so many inputs. Nonetheless, a few things are listed below.

I think my main concern pertains to the ROX coupling state. It is suggested that ROX is either obtained in absence of fuel substrate or in presence of inhibitors for all ET-pathways. I may not have conducted a formal experiment to test this claim, but working with isolated mitochondria over the last 8 years had me noticed that these are not always the same. The rate of oxygen consumption by mitochondria in absence of fuel substrate seems much lower than those I obtain when conducting a typical SUIT protocol and adding inhibitors of CI (rotenone) and CIII (Antimycin A) later in the run, when substrate and ADP are present. My thinking, for explaining this difference is that inhibitors not always perfectly inhibit complexes, depending on temperature, species and maybe other factors, and they are added during OXPHOS state, when conditions are reunited for a high *J*kO2.

Hence, sometimes, *ROX* as determined with inhibitors seems much higher, in particular, when relying on inhibitors for the AOX. This is especially important for measuring *L* in organisms with very slow mitochondrial capacities, such as bivalve mitochondria at 10°C. Accordingly, it may be interesting to distinguish between ROXn (no substrates) from ROXDi, obtained in the presence of saturating concentrations of ADP and inhibitors.

A few minor points are not completely clear to me:

* Fig. 2 : we are presented with the equation *J*rO2 = *J*kO2 + *J*rox, however,in Fig. 1, catabolic reactions occurring in the cell are suggested to be part of *J*rO2, which are clearly distinguished from *J*rox of either mitochondrial or cytosolic origin.
* The concept of matrix ETS (matrix components of the ETS) is new to me and I am not sure if I like it. When referring to the traditional definition of the ETS, only the complexes exchanging electrons (with or without translocation of protons) are included, which makes sense for the term electron transfer system. The dehydrogenases of the TCA cycle are very different in nature, they are not solely interacting with continuously recycled intermediate electron carriers (*e.g.* NADH and Q), but are also involved in a vast array of catabolic and anabolic pathways. It may be best restricting the use of the term ETS to the traditional CI, CII, CIII and CIV.
* Table 1. I wonder if LEAKOmy is affected by the presence of adenylates. Maybe LEAKOmy could be distinguished into LEAKOmy(n) (no ADP added yet), LEAKOmy(t) (all ADP has been phosphorylated, high concentration of ATP), and LEAKOmy(A) ADP has not yet been consumed entirely, becauseof possible allosteric activation of the ETS complexes by adenylates, but I am no expert in this matter.
* In the concept of net coupled *P* (*P-L*), it is assumed that *L* remains unchanged in the OXPHOS state, but the high protonmotive force in LEAK state will importantly increase proton leak through the membrane and mostly, the production of superoxide, as compared to the flux of these processes during OXPHOS state. This possible caveat has been addressed in the document, however, the means to address it (determination of *L* after adjusting the protonmotive force to that observed during the OXPHOS state) requires the use of fluorescence (not everyone has it) and molecular probes that (I think) have been shown to affect the activity of the complexes of the ETS. In other words, the determination of net *P* is not an easy task and may simply be ignored by the vast majority of practitioners.
* The concerns with the choice of markers have been addressed in the document. I would like to add that in our recent publication (Munro et al. 2019 *Aging Cell* e12916, e12916.), we show that three different conclusions could be reach for the difference between the naked mole-rat and the mouse, with respect to the flux of H2O2 formation by isolated mitochondria, with the use of three markers (CS activity, mg of mitochondrial proteins, and the activity of CIV).