ERRATUM

A technical error occurred in the Discussion of the paper entitled "Oxaloacetate and Adenosinetriphosphate Levels during Inhibition and Activation of Succinate Oxidation" by Peter Schollmeyer and Martin Klingenberg, which was published in BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS 4, 43-47 (1961). The Discussion is reprinted below in its correct form.

Discussion. In many of the reported experiments oxaloacetate is present at a level sufficiently high to inhibit the succinate oxidation, particularly if one considers that the oxaloacetate concentration inside of the mitochondria could be still much higher. The activation of the succinate oxidation by ATP, which is not accompanied by a decrease of the oxaloacetate level, could then be interpreted, for example following Pardee and Potter (1948) and Tyler (1955), that oxaloacetate has been transformed by ATP to a noninhibitory state. This explanation would have to be extended to the activation found on addition of phosphate, albumin or DPN linked substrates. In fact, in all these cases of activation the level of the endogenous ATP is increased. However, it is difficult to reconcile with this concept that the respiration is not reactivated when, in the presence of dinitrophenol, oxaloacetate is nearly completely removed by cysteine sulfinate. In this context we might recall previously conducted experiments on liver mitochondria (Azzone, Ernster and Klingenberg 1960) where in an inhibited state of the succinate oxidation the concentration of oxaloacetate was found to be within the limits of accuracy of the measurements, i.e. 5×10^{-8}M. It should be added however, that in the pre-treated liver mitochondria with a very low level of oxaloacetate, succinate oxidation could not be inhibited to such an extent as in pigeon breast muscle mitochondria, where a high level of oxaloacetate is observed.

We may summarize that the total results do not support the concept of an inhibition by oxaloacetate: But the results cannot exclude this mechanism. For example, it is conceivable that the oxaloacetate concentration is high at the inhibition

site and still very low as an overall concentration in the suspension of the mitochondria. On the other hand, the level of ATP is elevated in all cases of an activated succinate oxidation. This result is in agreement with an energy requirement for the succinate oxidation as proposed by Azzone and Ernster (1960). If one still accepts the theory of direct inhibition of succinate oxidation by oxaloacetate, one would have to postulate, that the succinate oxidation becomes sensitive to even very low amounts of oxaloacetate when ATP decreases below a certain level.

Attention may be directed to the finding, that dinitrophenol inhibits the activation of the succinate oxidation by ATP to about 60 to 70 % but does not inhibit the activation by ketoglutarate and also by glutamate. It is known and confirmed in our experiments by ATP analysis that dinitrophenol does not prevent the substrate phosphorylation of ketoglutarate oxidation. The experiment shows further that dinitrophenol also does not prevent ATP formed by substrate phosphorylation from activating succinate oxidation.