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Corrigendum

Corrigendum to "Mammalian mitochondrial nitric oxide synthase: Characterization of a novel candidate" [FEBS Lett. 580 (2006) 455–462]☆

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Since the time of our report on the mitochondrial localization of mouse ortholog of AtNOS1, mAtNOS1 [1], the following data has been acquired.

Importantly, both ours and Dr. Durner's labs have failed to detect any NO-synthase activity in purified AtNOS1 protein [2]. Similarly, purified mAtNOS1 and hAtNOS1 did not produce NO in our assays [2]. In response to that, Dr. Crawford reported that his data no longer supports the view that AtNOS1 is an arginine-dependent NOS enzyme and suggested renaming AtNOS1 to AtNOA1 (nitric oxide associated 1) [3]. Following that, we rename mAtNOS1 to mAtNOA1.

Based on information learned from data mining, we have hypothesized an involvement of eukaryotic orthologs of AtNOA1 in mitochondrial ribosome biogenesis and/or processes of translation [2]. mAtNOA1 (and AtNOA1) is an evolutionary conserved protein that contains a circularly permuted GTP-binding domain [4]. Its bacterial ortholog, YqeH protein (CAB14509, *Bacillus subtilis*), was shown to bind GTP and GDP. Deletion of this gene revealed its essentiality for *B. subtilis* viability [5]. YqeH was assigned to a YlqF/YawG protein family (characterized by a circularly permuted GTPase domain) that includes members involved in ribosomal biogenesis and/or the translation process [6]. Recently, work on *YqeH* gene has been reported which suggested YqeH protein to be required for proper 70S ribosome formation and, in particular, 30S subunit assembly/stability in *B. subtilis* [7]. Moreover, the yeast genome contains a homolog of mAtNOA1, YOR205c, that localizes to the mitochondria and co-purifies with mitochondrial ribosomal proteins of the small subunit [8]. *YOR205C*-deficient yeast mutant has a severe defect in phosphorylative oxidation [9]. An inner membrane localization of mAtNOA1 [1] overlaps with that of mitochondrial ribosomes and might imply a function within the mitochondrial translational machinery. In this light, an observed deregulation in levels of reactive oxygen species (ROS) in AtNOA −/− plant [10] might result from defective translation of the mitochondrial respiratory chain subunits that are encoded by the mitochondrial genome.

Further works undertaken in our laboratories, including a generation of *mAtNOA1* K.O. mouse, are expected to shed some more light on the role of this evolutionary conserved gene.

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