The human gene SLC25A17 encodes a peroxisomal transporter of coenzyme A, FAD and NAD⁺

Gennaro AGRIMI*, Annamaria RUSSO*, Pasquale SCARCIA* and Ferdinando PALMIERI*†

*Department of Biosciences, Biotechnology and Pharmacological Sciences, University of Bari, 70125 Bari, Italy, and †CNR Institute of Biomembranes and Bioenergetics, 70125 Bari, Italy

INTRODUCTION

Peroxisomes are virtually ubiquitous organelles of eukaryotic cells that are involved in several metabolic pathways including fatty acid α- and β-oxidation, the biosynthesis of ether phospholipids and bile salts, and the catabolism of purines, polyamines and L-pipolic acid [1]. Although the peroxisomal membrane was originally thought to be freely permeable to solutes, various studies have demonstrated the existence of peroxisomal transporters in both Saccharomyces cerevisiae and mammalian cells [2–6]. Moreover, a pH gradient between the cytosol and peroxisomal matrix has been shown to exist, indicating that even the transport of protons between these two compartments is regulated [7–9]. Despite the considerable progress made in peroxisomal solute transport, as mentioned above, the function of many PMPs (peroxisomal membrane proteins) is yet to be determined. Furthermore, some of the already identified peroxisomal transporters are still relatively poorly characterized. For example, except for the yeast Pxa1p/Pxa2p and the human PMP70, which most probably transport long-chain acyl-CoA esters, much remains to be determined about transported substrates and mechanisms of other peroxisomal ABC (ATP-binding-cassette) transporters [3].

Previously known as PMP34, SLC25A17 (solute carrier family 25 member 17) was first described by Wylin et al. [10] as a likely counterpart of the Candida boidinii PMP47, and by Honsho and Fujiki [11] as a model PMP to investigate topogenic signals. Both of these studies demonstrated that SLC25A17 is localized to peroxisomes and belongs to the mitochondrial carrier family [10,11], in humans known as the SLC25 solute carrier family [12,13]. Later, SLC25A17 was confirmed to be targeted to peroxisomes and found to possess some unquantified ATP transport activity [14]. Because this latter study reported only the percentage of radioactivity, and not the amount of [14C]ATP taken up by liposomes reconstituted with SLC25A17, and employed a limited set of nucleotides, we set out to perform a more in-depth study on the activity of SLC25A17.

In the present study we provide direct evidence that SLC25A17 is a transporter for CoA and FAD, and to a lesser extent NAD⁺. SLC25A17 was expressed in Escherichia coli, purified, reconstituted into liposomes and shown to transport CoA, FAD, NAD⁺, PAP (adenosine 3',5'-diphosphate), FMN, AMP and ADP, but not many other compounds. The transporter nearly exclusively catalysed a counter-exchange of substrates, was saturable and was inhibited by pyridoxal 5'-phosphate and other inhibitors; its mRNA was found in all of the tissues investigated. This is the first time that a carrier for multiple free cofactors has been identified and characterized at the molecular level.

EXPERIMENTAL

Construction of the expression plasmid

The coding sequence for SLC25A17 (GenBank® accession number NM_006358) was amplified from HepG2 cell cDNA by PCR using primers corresponding to the extremities of the coding sequence with additional BamHI and XhoI sites. The amplified product was cloned into the pET-21b expression vector.

Bacterial expression and purification of SLC25A17

SLC25A17 was produced as inclusion bodies in the cytosol of E. coli as described previously [15], except that the host cells were E. coli strain Rosetta gami B (Novagen). Control cultures with the empty vector were processed in parallel. Inclusion bodies

Abbreviations used: ORC, human ornithine carboxylase; PAP, adenosine 3',5'-diphosphate; PMP, peroxisomal membrane protein; SLC25A17, solute carrier family 25 member 17; TY, tryptone/yeast extract.

1 To whom correspondence should be addressed (email fpalm@farmbiol.uniba.it).
were purified on a sucrose density gradient [15], washed at 4°C with TE buffer [10 mM Tris/HCl and 1 mM EDTA (pH 6.5)], then twice with a buffer containing 3% (w/v) Triton X-114, 1 mM EDTA and 10 mM Pipes-NaOH (pH 7.0), and once again with TE buffer. SLC25A17 was solubilized in 1.7% (w/v) sarkosyl (N-dodecanoyl-N-methylglycine sodium salt), and a small residue was removed by centrifugation (59,000 rev./min for 1 h at 4°C, rotor Beckman Coulter Type 70 Ti). In some experiments, SLC25A17 with a C-terminal His<sub>6</sub>-tag was purified from <i>S. cerevisiae</i> ant1Δ cells transformed with a single copy vector containing the SLC25A17–His<sub>6</sub> downstream of the catalase A promoter, exactly as described by Visser et al. [14].

**Reconstitution of SLC25A17 into liposomes and transport assays**

The recombinant protein in sarkosyl was reconstituted into liposomes in the presence and absence of substrates, as described previously [16]. External substrate was removed from proteoliposomes on Sephadex G-75 columns, pre-equilibrated with 50 mM NaCl and 10 mM Mops-NaOH at pH 7.4 (buffer A). Transport at 25°C was started by adding [14C]AMP (ARC), [14C]ADP (PerkinElmer), [14C]ATP (Hartman Analytic), [3H]NAD<sup>+</sup> (PerkinElmer), FAD and FMN to proteoliposomes and transport was terminated by addition of 10 mM pyridoxal 5'-phosphate and 10 mM bathophenanthroline (the ‘inhibitor stop’ method [16]). In controls, inhibitors were added at the beginning together with the substrate. The external substrate was removed, and the entrapped substrate was measured [16]. The experimental values were corrected by subtracting control values. The initial transport rate was calculated from the substrate taken up by the protein added to the reconstitution mixture. FAD and FMN were measured fluorimetrically using an LS 50B fluorescence spectrometer (PerkinElmer) at 450 nm excitation and 530 nm emission wavelengths [19]; all data were corrected by subtracting the background fluorescence.

**RESULTS**

**Bacterial expression of SLC25A17**

The SLC25A17 gene was expressed in <i>E. coli</i> Rosetta gami B (DE3) cells (Figure 1). The gene product accumulated as inclusion bodies and was purified on a sucrose discontinuous gradient followed by centrifugation and washing (Figure 1, lane 5). The identity of the purified protein was confirmed by MS analysis of the trypsin digests. The protein was not detected in bacteria harvested immediately before induction of expression (Figure 1, lane 2), or in cells harvested after induction but lacking the coding sequence in the expression vector (Figure 1, lane 4). Approximately 10 mg of purified protein per litre of culture was obtained.

**Functional characterization of SLC25A17**

The recombinant purified SLC25A17 was reconstituted into liposomes and its transport activity for potential substrates was tested in homo-exchange experiments (i.e. with the same substrate inside and outside). Using external and internal substrate concentrations of 0.1 and 10 mM respectively, the reconstituted protein catalysed [14C]AMP/AMP and [14C]ADP/ADP exchanges that were inhibited completely by a mixture of pyridoxal 5'-phosphate and bathophenanthroline. In contrast, despite the long incubation period (i.e. 30 min), virtually no homo-exchange activity was observed for ATP, GTP, TTP, CTP, phosphate, malate, citrate, glutamate, glutamine, ornithine, arginine and 5'-adenosylmethionine (results not shown). Furthermore, no [14C]AMP/HOMO-exchange activity was detected when SLC25A17 had been boiled before incorporation into liposomes or if proteoliposomes were reconstituted with sarkosyl-solubilized material from bacterial cells either lacking the expression vector for SLC25A17 or harvested immediately before induction of expression.
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Figure 2  Substrate specificity of SLC25A17

Liposomes reconstituted with SLC25A17 were preloaded internally with various substrates (concentration 5 mM). Transport was started by the addition of 0.2 mM [14C]AMP and terminated after 2 min. Values are means ± S.E.M. for at least three independent experiments. Actyl-CoA, acetyl-CoA; Pan, pantothentic acid; Pi, phosphate; PPi, pyrophosphate; Ppnyl-CoA, propionyl-CoA; Th, thiamine; ThPP, thiamine pyrophosphate.

To investigate further the substrate specificity of SLC25A17 the initial rate of [14C]AMP uptake into proteoliposomes that had been preloaded with a variety of potential substrates was measured (Figure 2). The highest activity of [14C]AMP uptake into proteoliposomes was observed with internal AMP, CoA, dephospho-CoA and FAD. To a lesser extent, [14C]AMP was also exchanged with internal acetyl-CoA, FMN, ADP, PAP and NAD$^+$. In contrast, a marginal exchange of [14C]AMP was observed with internal 3′-AMP, cAMP, ATP, propionyl-CoA, NADH, NADP$^+$, NADPH, NMN, GMP, IMP, CMP, TMP, UMP, phosphate, pyrophosphate, thiamine, thiamine pyrophosphate and pantothenate (Figure 2), and GTP, TTP, CTP, malate, citrate, glutamate, glutamine, ornithine, arginine and S-adenosylmethionine (results not shown). The residual activity in the presence of these compounds was approximately the same as that observed in the presence of NaCl (Figure 2). In addition, no uptake of [14C]AMP into pure liposomes, e.g. without incorporated protein, was observed (results not shown).

In another set of experiments we measured the uptake of the fluorescent FAD and FMN, and of the radioactively labelled NAD$^+$ and ADP into proteoliposomes in the presence and absence of internal substrate. The results shown in Figure 3 demonstrate that FAD and FMN were taken up by liposomes reconstituted with SLC25A17 in exchange with internal AMP, CoA or PAP, and [14C]NAD$^+$ and [14C]ADP with internal AMP, FAD, FMN, CoA or PAP to a considerable extent. In contrast, the uptake of FAD, FMN, NAD$^+$ or ADP into proteoliposomes containing no substrate (NaCl present) was very low (Figure 3); the uptake of [14C]ATP and [14C]CTP in the presence of internal FAD, FMN, NAD$^+$ or ADP was negligible; and finally, the uptake of FAD, FMN, NAD$^+$, AMP or ADP into pure liposomes was virtually nil, despite the long incubation period (i.e. 30 min) (results not shown). Therefore, in the presence of a suitable counter-substrate, FAD, FMN, NAD$^+$ and ADP are transported by reconstituted SLC25A17 not only when present inside proteoliposomes, but also when added externally.

Given that SLC25A17 (PMP34) was proposed to be an ATP transporter [14] in contrast with the results reported above, we measured the uptake of [14C]ATP in proteoliposomes using the same experimental conditions used in [14]. In these experiments we found an extremely low uptake of [14C]ATP, equal to 0.73 ± 0.2 mmol/15 min per g of protein, into proteoliposomes containing ATP. Therefore ATP is virtually not transported by SLC25A17.

The effect of inhibitors on the [14C]AMP/AMP exchange reaction catalysed by reconstituted SLC25A17 was also examined (Figure 4). The exchange reaction was inhibited strongly by pyridoxal 5′-phosphate, bathophenanthroline, HgCl$_2$ and mersalyl, and partially by α-cyano-4-cinnamate and p-hydroxymercuironazoate. In contrast, little effect was observed with N-ethylmaleimide, butylmalonate, 1,2,3-benzenetricarboxylate, carbonylaxtractylside and bongkrekic acid, which are known inhibitors of other mitochondrial carriers (Figure 4).

Kinetic characteristics of recombinant SLC25A17

In Figure 5(A) the time-course is compared for the uptake of 1 mM [14C]AMP into reconstituted liposomes measured either as uniport (in the absence of internal substrate) or as exchange (in the presence of one of the indicated substrates). The uptake of [14C]AMP by exchange with internal AMP CoA, FAD, PAP or FMN substantially increased with time, equilibrium being approached at or after 60 min incubation. In comparison, the uniport uptake of [14C]AMP was very low (Figure 5A). The efflux of [14C]AMP from prelabelled active proteoliposomes is shown in Figure 5(B). In the absence of external substrate, a marginal efflux was observed. However, upon addition of external AMP, PAP, CoA or FAD a substantial efflux of radioactivity occurred,
which was prevented by the inhibitors pyridoxal 5'-phosphate and bathophenanthroline. Also the efflux of fluorescence from proteoliposomes preloaded with FAD was pronounced upon addition of CoA or FAD to the reaction medium, whereas it was much lower and slower upon addition of buffer alone, i.e. in the absence of externally added substrates (Figure 6). In control assays, there was no FAD efflux when transport inhibitors were added at time zero. These results indicate that, at least under the experimental conditions used, reconstituted SLC25A17 functions essentially by a counter-exchange mechanism.

The kinetic constants of recombinant SLC25A17 were determined by measuring the initial transport rate at various external [14C]AMP concentrations in the presence of a fixed saturating internal concentration of 10 mM AMP. The transport affinity (K_m) and specific activity (V_max) values for [14C]AMP/AMP exchange at 25°C were 0.19 ± 0.03 mM and 74 ± 9 μmol/min per g of protein respectively. Several external substrates were competitive inhibitors of [14C]AMP uptake as they increased the apparent K_m without changing the V_max (results not shown). The K_i values of these substrates for SLC25A17 were as follows: CoA, 19.6 ± 3.8 μM; diphospho-CoA, 14.4 ± 5.4 μM; FAD, 2.6 ± 1.3 μM; FMN, 10.2 ± 3.3 μM; NAD^+, 0.85 ± 0.12 mM; PAP, 0.78 ± 0.1 mM; and ADP, 0.6 ± 0.08 mM.

Expression of the SLC25A17 gene in human tissues

The tissue distribution of SLC25A17 mRNA was determined by real-time PCR. The results shown in Figure 7 indicate that the SLC25A17 gene was expressed in all 21 human tissues investigated. This finding is in line with the fact that peroxisomes are present in virtually all eukaryotic cells, except in erythrocytes and male germ cells [20]. It should be noted that, as post-transcriptional mechanisms may operate, the levels of expression presented in Figure 7 do not necessarily reflect the ratios of transport activities.

Figure 4 Effect of inhibitors on the [14C]AMP/AMP exchange by SLC25A17
Proteoliposomes were preloaded internally with 5 mM AMP and transport was initiated by adding 0.2 mM [14C]AMP to liposomes reconstituted with SLC25A17. The incubation time was 2 min. Thiol reagents and α-cyanojcinamate were added 2 min before the labelled substrate. The final concentrations of the inhibitors were 10 μM (BKA, bongkrekic acid; CAT, carboxyatractylsulfate), 10 mM (BAT, bathophenanthroline; PLP, pyridoxal 5′-phosphate), 0.2 mM (MER, mersalyl; pHMB, p-hydroxymercuribenzoate), 1 mM (CCN, α-cyanojcinamate; NEM, N-ethylmaleimide) and 2 mM (BMA, butylmalonate; BTA, 1,2-benzene dicarboxylate). Values are extents of inhibition (%). ± S.E.M. for at least three duplicate independent experiments.

Figure 5 Kinetics of [14C]AMP transport into proteoliposomes reconstituted with SLC25A17
(A) Uptake of AMP. [14C]AMP (1 mM) was added to liposomes reconstituted with SLC25A17 and preloaded internally with 10 mM AMP ( ), CoA ( ), FAD ( ), PAP (Δ), FMN ( ), NAD^+ ( ), NMM (□) or NADPH ( ). Additionally, 1 mM [14C]AMP was added to pure liposomes preloaded internally with 10 mM AMP ( ). (B) Efflux of [14C]AMP. The efflux of [14C]AMP was started by adding buffer A alone ( ■ ), AMP ( ), 5 mM CoA ( ), FAD ( ), PAP (Δ), NAD^+ ( ), FMN ( ) or CoA, 10 mM pyridoxal 5′-phosphate and 10 mM bathophenanthroline ( ◊ ) in buffer A. Values are means of at least three duplicate independent experiments. S.E.M. of all the values were < 10%. In (A) differences between all of the values with internal AMP, FAD and PAP, with internal CoA from 10 to 60 min, and with internal FMN at 30 and 60 min, and controls (samples with internal NaCl) were significant (P < 0.01, one-way ANOVA followed by the Bonferroni t test). In (B) differences between all of the values with added AMP and PAP, with added CoA from 10 to 40 min and with added FAD at 20 and 40 min, and controls (with added inhibitors) were significant (P < 0.01, one-way ANOVA followed by the Bonferroni t test).

DISCUSSION

In the present study, the function of SLC25A17 was investigated by direct transport assays upon expression in E. coli, purification and incorporation of the protein into phospholipid vesicles. The transport properties and kinetic characteristics of recombinant SLC25A17, reported in the present paper, unequivocally demonstrate that this protein is a transporter of CoA and FAD, and to a lesser extent NAD^+. Given that the subcellular localization of SLC25A17 to peroxisomes has been proven by three different groups [10,11,14], this is the first time that a peroxisomal carrier for the free cofactors CoA, FAD and NAD^+ has been identified from any organism. Of note, the specific activity of reconstituted SLC25A17 is comparable with the activities of other SLC25 proteins [15,17,21–24], and the K_i values of CoA, FAD and NAD^+ for SLC25A17 are lower, or not much higher, than the cytosolic-free concentrations of these cofactors [25–27].
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SLC25A17 functions almost exclusively by a counter-exchange mechanism, the carrier-mediated uptake of the free cofactors CoA, FAD and NAD\(^+\) requires efflux of a counter-substrate. Our transport measurements in reconstituted liposomes indicate that the most likely candidates to exchange with external CoA, FAD and NAD\(^+\) are PAP, FMN, AMP and ADP, which are effective reactants of SLC25A17. Thus, in the intraperoxosomal matrix, PAP is generated from CoA and CoA derivatives by the Nudix hydrolases NUDT7 and NUDT19 [28–30], FMN is generated from FAD by NUDT12 [31], AMP is generated from ATP by acyl-CoA synthetases and from NAD(H) by NUDT12 [32], and ADP is generated from ATP by PsLon, an ATP-dependent protease [33]. The results of the present study do not agree with the previous conclusion that SLC25A17 (PMP34) is an ATP transporter [14]. This discrepancy cannot be attributed to methodological differences because, using the same experimental conditions used in [14], we did not detect any significant ATP transport. Furthermore, in the present study we have shown that SLC25A17 does not significantly catalyse any of the \([^{14}C]ATP/ATP, [^{14}C]ATP/FAD, [^{14}C]ATP/NAD^+\) or \([^{14}C]ATP/ADP\) exchanges. It is worth mentioning that the authors of the previous study [14] reported only the percentage of radioactivity without giving numbers, i.e. not the counts of radioactivity per 15 min and the specific radioactivity (counts of radioactivity/15 min per nmol of \([^{14}C]ATP\)). Therefore it is impossible to calculate the extent of ATP transport activity from their results [14].

In a phylogenetic tree of Homo sapiens, S. cerevisiae and Arabidopsis thaliana mitochondrial carriers (see Figure 3A in [13]), SLC25A17 clusters with the carriers for NAD\(^+\) [23,34], pyrimidine nucleotides [35,36], FAD/folate [37–39], the peroxisomal Ant1p [40] and its homologues At3g05290 and At5g27520 [41], and the peroxisomal At2g39970 (see below). With all of these transporters, except yeast Ant1p and its homologues in Arabidopsis, SLC25A17 shares the distinct feature of having a tryptophan residue, instead of an acidic residue, in the mitochondrial carrier signature motif PX(D/E)XX(R/K) present in the second repeat of these proteins (see Figure 2 in [13]). Nevertheless, the substrate specificity of SLC25A17 is distinct from that of any other carrier present in the above-mentioned cluster, although some overlapping functions (i.e. substrates transported) occur.

During the revision of the present paper, two papers have been published on the closest relative of SLC25A17 in Arabidopsis, i.e. the PMP encoded by At2g39970, also known as PMP38. Importantly, in the first paper it was found that PMP38 transports NAD\(^+\), AMP and ADP, but not ATP [42]. In the second paper, in which the APEM3 gene encoding PMP38 was knocked out, the authors concluded that this protein plays an important role in peroxisomal proliferation and stated that they were unable to detect transport of ATP catalysed by PMP38 [43]. It should be added that PMP38, i.e. At2g39970, was previously found to be incapable of complementing the growth defect of an ant1-null strain, whereas At3g05290 and At5g27520 exhibited this ability [41]. It is also interesting to recall that the yeast NAD\(^+\) carrier [23] and Arabidopsis mitochondrial and plastidial NAD\(^+\) carriers [34] transport FAD and FMN as well, although to a much lesser extent than NAD\(^+\), AMP and GMP.

The closest relative of SLC25A17 in yeast, Ant1p, was found to transport ATP, AMP and ADP with high efficiency, and to a lesser extent UDP, UTP, CDP and CTP [40]. Unfortunately, most of the substrates found to be transported by SLC25A17 in the present study, i.e. FAD, FMN, CoA, NAD\(^+\) and PAP, were not tested in the previous study [40]. However, there are some important sequence differences between SLC25A17

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**Figure 6** Efflux of FAD from liposomes reconstituted with SLC25A17

The efflux was started by adding buffer A alone (A), 5 mM CoA (B), 5 mM AMP (C) or 10 mM pyridoxal 5'-phosphate and 10 mM bathophenanthroline (X). Values are means ± S.E.M. for four duplicate independent experiments.

**Figure 7** Expression of SLC25A17 in human tissues

The relative quantification of SLC25A17 mRNA in the indicated tissues was performed according to the comparative method (2\(^{-\Delta\Delta C_t}\)). The liver \(\Delta C\), for SLC25A17 was used as an internal calibrator. For the internal calibrator, the \(\Delta\Delta C\) equals zero and 2\(^0\) equals one. For the remaining tissues, the values 2\(^{-\Delta\Delta C}\) on the abscissa indicate the fold-change in gene expression relative to the liver. Values are means ± S.D. for three duplicate independent experiments.

Besides CoA, FAD and NAD\(^+\), SLC25A17 transports AMP, dephospho-CoA, acetyl-CoA, FMN, ADP and PAP. Because in humans the biosynthesis of CoA, FAD and NAD\(^+\) takes place entirely outside the peroxisomes, a primary function of SLC25A17 is to catalyse the import of CoA, FAD and NAD\(^+\) into the peroxisomal matrix where they are indispensable for the activity of various intraperoxosomal enzymes. Moreover, because...
and Ant1p. Apart from the presence of a tryptophan residue in the signature motif of the second repeat in SLC25A17 and not in Ant1p, both proteins share 26% identical amino acids; this value is too low to draw definite conclusions about the substrate specificity of mitochondrial carrier family members. Furthermore, the residues of the even-numbered transmembrane α-helices, which have been proposed to form the similarly located substrate-binding site of mitochondrial carriers [44], markedly differ between SLC25A17 (Ser74 and Ser78 in H2, Leu75 and Val176 in H4, and Gin276 in H6) and Ant1p (Ala47 and Gin85 in H2, Leu91 and Thr195 in H4, and Lys286 in H6). All of these differences between SLC25A17 and Ant1p suggest that they may display differences in the substrates they transport. In general, low homology between mitochondrial carriers, as found in the closest relatives of distant organisms, should be regarded with caution when drawing conclusions about their substrate specificity in the absence of direct transport assays involving a wide range of potential substrates. A clear example is provided by the closest Arabidopsis mitochondrial carriers has recently been pointed out [13]. Even identified isoforms of mitochondrial carriers with a high percentage of identical amino acids may display clear differences in substrate specificity. For example, ORC1 (human ornithine carrier 1) transports the L-forms of ornithine, lysine and arginine, whereas ORC2 has a broader specificity, also transporting the D-forms of these amino acids as well as histidine and homoarginine, despite the fact that they share 87% identical amino acids [46]. Historically, the first mitochondrial carrier found to be localized in the peroxisomal membrane was PMP47 of Candida boidinii [47]. Nothing is known about the substrate(s) transported by PMP47, although it was speculated to be involved in ATP transport without any direct empirical evidence [48].

Apart from the relatively low homology that SLC25A17 displays with the transporters discussed above (23–31% identical amino acids), SLC25A17 does not exhibit significant sequence homology with any other known carrier greater than the homology existing among the different members of the mitochondrial carrier family. However, several protein sequences available in databases are likely to be orthologues of SLC25A17 in other organisms. These sequences include (National Center for Biotechnology Information accession numbers): XP_531726.1 from Canis familiaris (97% identity), NP_001119741.1 from Rattus norvegicus (95%), NP_001039413.1 from Bos taurus (94%), NP_035529.1 from Mus musculus (94%), NP_00108333.1 from Xenopus laevis (73%), NP_001092731.1 from Danio rerio (72%), XP_623636.1 from Apis mellifera (47%), NP_728982.1 from Drosophila melanogaster (43%) and XP_001662175.1 from Aedes aegypti (46%). To our knowledge, none of these proteins has been characterized biochemically. Future studies are warranted to investigate the effects of knock-down of SLC25A17 by RNAi (RNA interference) or SLC25A17-knockout mice, and to ascertain a potential regulatory role of SLC25A17 in peroxisomal function, as well as its involvement in peroxisome-related disorders.

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AUTHOR CONTRIBUTION

Gennaro Agrimi and Ferdinando Palmieri designed the study. Gennaro Agrimi, Annamaria Russo and Pasquale Scarcia performed the experiments. Gennaro Agrimi and Ferdinando Palmieri analysed the data. Ferdinando Palmieri and Gennaro Agrimi wrote the paper.

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