displayed increased state 4 respiration accompanied by a decreased membrane potential, a diagnostic of mitochondrial uncoupling. The increased UCP3 expression and activity could be a physiological adaptation to perturbed cellular energy balance, leading to an increase in fatty acid oxidation and hence, the leaner phenotype observed in these Syt7 knockout mice.

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doi:10.1016/j.bbabio.2010.04.267

10P.12 Reduced cytotoxic $CD8^+$ lymphocytes in heterozygous UCP2-KO mice in experimental autoimmune encephalomyelitis

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Multiple sclerosis (MS) affects over 2.5 million people worldwide and the human uncoupling protein 2 (UCP2) promoter polymorphism −866G/A has been associated with susceptibility to multiple sclerosis [1]. Previous studies have also shown a protective function for UCP2 in Experimental Autoimmune Encephalomyelitis (EAE), a murine model of MS, with higher clinical scores in $UCP2^{-/-}$ mice [2]. We undertook a pilot study using the EAE model with 5 control and 5 $UCP2^{+/-}$ mice. The study showed reduced $CD8^+$ lymphocytes in brain (3.6-fold, $P<0.001$) and spleen (1.2-fold, $P<0.05$) of $UCP2^{+/-}$ mice after 21 days post immunization compared to wild-type controls while the percentage of $CD4^+$ lymphocytes remained unaffected. Our observation is not necessarily consistent with that of Vogler *et al.* (2006) who saw no effect of EAE on peripheral $CD4^+$ to $CD8^+$ ratios in a comparison of $UCP2^{-/-}$ and wild-type mice. Furthermore, $UCP2^{+/-}$ mice showed reduced clinical scores and therefore seemed to be less vulnerable to EAE when compared to wild-type mice. Future work will focus on a larger study comparing various parameters, including T-cell profiling, from *UCP2* deficient and wild-type mice.

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doi:10.1016/j.bbabio.2010.04.268

10P.13 UCP2 expression pattern in mouse tissue

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Uncoupling protein 2 (UCP2) is an inner mitochondrial membrane protein, which transports protons in the presence of fatty acids similarly to UCP1 [1]. Although the protein was discovered 1997 its function is still unclear. One reason for this is the obscurity of UCP2 protein expression among different tissues and cell types, caused by a discrepancy between mRNA and protein level [2]. Here, we applied a new designed antibody against UCP2 with proved specificity to the recombinant UCP2. Using this antibody we re-evaluated the protein expression pattern among various tissues of young, mature and adult mice. Additionally we measured the mRNA levels in these tissues and collated them with the protein expression levels. To evaluate the UCP2 expression in nervous system we analysed different brain regions, neuronal cell types and development stages. The obtained results are compared to the expression pattern of the brain specific UCP4 [3] and discussed in view of the hypothesized functions.

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doi:10.1016/j.bbabio.2010.04.269

10P.14 Thermogenically competent recruitment of uncoupling protein 1 in brown preadipocytes and in a subset of cell precursors from epididymal white adipose tissue by a PPARγ agonist

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Peroxisome proliferator-activated receptor-γ (PPARγ) activation pathway can competently recruit *brown* adipose tissue (BAT), independently of sympathetic stimulation. Here we studied the thermogenic capacity of brown pre-adipocytes chronically treated with the potent PPARγ agonist rosiglitazone. Mitochondriogenesis, an essential part of BAT recruitment, was significantly enhanced in treated brown adipocytes. Most importantly, these mitochondria were capable of thermogenesis, as rosiglitazone-treated brown adipocytes responded to the addition of norepinephrine with a large increase in oxygen consumption. This thermogenic response was not observable in rosiglitazone-treated brown adipocytes originating from *UCP1*-ablated mice; hence, it was UCP1 dependent. Thus the PPARγ pathway represents an alternative, potent, and fully competent mechanism for BAT recruitment, which may be the cellular explanation for the enigmatic recruitment in prehibernation and prenatal states. We also examined the cell precursors from the purest *white* adipose tissue depot (epididymal), and demonstrate that a similar chronic treatment with the rosiglitazone promotes not only the expression of PGC-1α and mitochondriogenesis in these cells but also *UCP1* gene expression in a significant subset of the cells, providing these cells with a genuine thermogenic capacity. Indeed the addition of norepinephrine to these white adipose tissue-derived cells induced high oxygen consumption. This thermogenic response to norepinephrine was significantly abolished in white adipocytes from *UCP1*-ablated mice, indicating true UCP1-dependent thermogenesis, which previously was considered as being unique brown adipocytes characteristic. However, although functional thermogenic genes are expressed, the cells are devoid of transcripts for the novel transcription factors now associated with classic brown adipocytes (Zic1, Lhx8, Meox2, and characteristically PRDM16) or for myocyte-associated genes (myogenin and myomirs (muscle-specific microRNAs)) and retain white fat characteristics such as Hoxc9 expression. These cells therefore constitute a subset of adipocytes ("brite" adipocytes) with a developmental origin and molecular characteristics distinguishing them as a separate class of cells.

doi:10.1016/j.bbabio.2010.04.270