sIgA level in saliva (μg/mg) of increased significantly (Day 0: 208.11±168.20; Day 28: 298.66±154.71). On the other hand, the levels of cortisol and catecholamine did not change.
Conclusion: When the ETAS intake for 28 days raised the results of mental arithmetic as dysphoria stimulation, increased the levels of sIgA promoting a stress response. These data suggest that ETAS intake may reduce a tired feeling in daily living and have beneficial effects in the response to dysphoria stress.

Disclosure of Interest: H. Waki: None Declared, S. Miyazaki: None Declared, T. Miura: None Declared, K. Uebaba: None Declared, T. Hisajima Grant/Research Support from: Amino Up Chemical Co., Ltd.

LB005-MON
ENERGY TARGETED NUTRITION STRATEGY DOES NOT PROVIDE ADEQUATE PROTEIN IN CRITICALLY ILL PATIENTS

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Rationale: It is important to monitor the energy and protein requirements and the actually given amount during the initiation and maintenance of nutrition support in critically ill patients. In general practice, nutritional support is usually energy targeted. Despite adequate energy supply, this strategy might cause protein deficiency.
In this retrospective study, we aimed to compare the targeted and actual energy/protein supply and to evaluate nitrogen balance in ICU patients with enteral, parenteral or combined nutritional support.
Methods: 217 ICU patients with a mean APACHE II score of 19.5 and having a complete nitrogen balance calculation records were included. The patients were divided into three groups as enteral, parenteral and combined nutritional support.
Results: See Table 1.

Table 1. Energy and protein balance (median values)

	All patients (n = 217)	Enteral (n = 149)	Parenteral (n = 46)	Combined (n = 22)
Energy (kcal/d)				
Target	1650	1660	1535	1751.50
Actual	1688	1680	1616	1896*
Protein (g/d)				
Target	97.4	98.3	97.42	93.4
Actual	80.6*	86.4*	63.8*	89.8
Nitrogen balance (g/d)	−2.59	−2.4	−5.33 †/††	0.03
Volume (ml/d)	1680	1560	1680 ††	2038 †/†††

*p < 0.05 target vs actual values; †p < 0.05 parenteral vs combined; ††p < 0.05 parenteral vs enteral; †††p < 0.05 enteral vs combined.

Conclusion: Energy targeted nutrition support may cause inadequate protein delivery and increased nitrogen deficiency especially in parenterally fed patients. The same strategy resulted in over provision of calories and volume in combined nutrition group.

Disclosure of Interest: None Declared

LB006-MON
INFLUENCE OF LIPOPOLYSACCHARIDES ON THE EXPRESSION OF HUMAN PEPTIDE TRANSPORTER (PEPT1) IN INTESTINAL CACO-2 CELL MONOLAYERS

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Rationale: The intestinal oligopeptide transporter, PEPT1, is upregulated both in the distal colon in patients with inflammatory bowel disease (IBD); it is also upregulated in the distal colon and downregulated in the distal small intestine in colitis rats. Although the detailed mechanism regulating these effects remains unclear, it was reported that one transcription factor, Nuclear Factor-kappa B (NF-κB), is activated in the intestinal tissue of patients, suggesting the possibility that this activated signal is associated with the onset of IBD. Therefore, the aim of this study was to investigate the influence of lipopolysaccharides (LPS), which are known NF-κB activators, on human PEPT1 expression in intestinal Caco-2 cell monolayers.
Methods: Caco-2 cells were cultured on 6-well Transwell permeable membranes for two weeks. LPS was then added in the culture medium on the apical or basolateral side of the cell monolayer at 100 ng/ml, and the cells were further incubated for 24 or 48 h. For NF-kB inhibition studies, the cells were pretreated with 10μM BAY-11 1 h before the addition of LPS. The expression levels of human PEPT1 were evaluated by real-time RT-PCR and Western blot analyses.
Results: Although no changes were observed in mRNA levels, PEPT1 protein levels were significantly decreased by the addition of LPS to the basolateral side of Caco-2 cell monolayers for 48 h. Furthermore, PEPT1 protein levels were restored by pre-treatment with basolateral BAY-11.
Conclusion: These findings suggest that PEPT1 protein expression specifically responds to LPS stimuli from the basolateral membrane. This effect may be due to a reduction in protein synthesis and/or an increase in proteolysis downstream of NF-κB signaling. Further study is required to elucidate the regulatory mechanisms controlling PEPT1 expression in greater detail.

Disclosure of Interest: None Declared

LB007-MON
SERUM 25-HYDROXYVITAMIN D3 LEVELS IN MODERATE ALZHEIMER'S DISEASE PATIENTS

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Rationale: Hypovitaminosis D has been associated with Alzheimer's disease. Vitamin D (VitD) supplementation should be initiated in the absence of sun exposure, inadequate diet or for people living above 33° latitude, according to guidelines. However, Portugal is on 37–42° latitude and serum 25-hydroxyvitamin D3 [25(OH)D3] is not routinely assessed. The main purpose of this study was to describe serum 25(OH) D3 levels in patients with moderate Alzheimer disease living in Porto, Portugal.
Methods: A cross-sectional study was conducted among 50 patients with moderate Alzheimer's disease (18 men/32