Urine metabolome in women with Chlamydia trachomatis infection

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Background: Chlamydia trachomatis (CT) represents the agent of the most common bacterial sexually transmitted infection worldwide. Although several in-vitro studies have investigated the metabolic adaptation of CT to host cells, no data are available about the metabolic profile that can be found in human biological fluids during an ongoing CT infection. The aim of this study was to compare the urine metabolome in women with or without Chlamydia urethritis.

Materials/methods: From January to July 2016, all the pre-menopausal non-pregnant Caucasian women attending the STI Outpatients Clinic of Sant’Orsola-Malpighi Hospital in Bologna (Italy) and meeting one of the following criteria were enrolled: presence of uro-genital symptoms and/or presence of risk factors for CT infection. After a clinical examination, an urine sample was collected from each woman and used for CT and Neisseria gonorrhoeae molecular detection (Versant CT/GC DNA 1.0 Assay; Siemens Healthineers). Moreover, 1 mL of urine specimen was subjected to the metabolomic analysis, by means of a proton-based nuclear magnetic resonance (¹H-NMR) spectroscopy. In case of a CT positive result, CT molecular genotyping was performed by omp1 gene semi-nested PCR followed by RFLP analysis.

A written consent was obtained by all the patients and the study protocol was approved by the Ethics committee of the Hospital.

Results: During the study period, a total of 119 subjects were enrolled: 98 women were negative for a CT infection, whereas 21 were assigned to the CT-positive group. We were able to identify 3 metabolites whose concentrations were significantly higher in the urine samples of CT-infected women: sucrose (P=0.0005), mannitol (P=0.003) and lactate (P=0.002). These results were comparable for all the CT serovars detected (D, E, F, G, K).
**Conclusions:** Since the presence of sugars can increase the stability of chlamydial proteins, higher levels of sucrose and mannitol in the urethral lumen could have favoured CT infection acquisition or persistence. Lactate could result from CT-induced immunopathology, as a product of the inflammatory microenvironment. Further studies are needed to understand the potential role of these metabolites in the pathogenesis of CT infection, as well as their diagnostic/prognostic use.