factor (BDNF) and neurotrophins like neurotrophin3 (NT3) were upregulated from monocyte/macrophages infected with *Chlamydophila pneumoniae* which is a microorganism considered among environmental factors. In this study we aimed to show at the worldwide level, for the first time, the relation of C.*pneumoniae infection*, BDNF and NT3 levels.

Methods & Materials: In this crossectional retrospective study 50 patients with shisophrenia and 35 healthy controls(HC) were included. The *C.pneumoniae DNA* was investigated by RT-PCR from PBMC and IgA, IgG, IgM were investigated by immunofluorescense in both patients and HC's serum samples. Additionnaly in serum samples BDNF and NT3 levels were determined by ELISA .Chi square and student's t-tests were used for statistical analyses.

Results: A past *C.pneumoniae* infection was detected in 36 persons(>1/16)(72%) of our patient group and in 14 patients(40%) of our HC group ($p < 0.05$). *C.pneumoniae* DNA was not found in both groups.BDNF level in the patient group was between 811-3422pg/ml (average 1408 pg/ml). In the HC this lewel was between 1084-3171 pg/ml (average 2736 pg/ml)($p < 0.05$). NT3 level, in the patient group, was between 81-1312 pg/ml(average 525pg/ml) and in the HC this level was between 378-1750 pg/ml (average 860 pg/ml)($p < 0.05$)

Conclusion: In conclusion,although in cases with schizophrenia, compared to control group,the presence of *C.pneumoniae* infection was found remarquably high,the DNA of *C.pneumoniae* was not found in any cases. Otherwise, from neurotrophins, the BDNF and NT3 levels, were found significantly low in the patients compared to healthy controls.This findings suggested us that this is the result of their long term medication with antipsychotics. Although, in this study, which is the first international study in the basis of *C.pneumoniae*, schizophrenia relation, and the detection of BDNF and NT3 levels, a conviction was'nt reached and we are thinking that new studies, especially based on cohort and performed with large series including these tree parameters (schizophrenia, *C.pneumoniae*, neurotropic factors) supported by molecular methods are needed.

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Management of non-malarial fevers in outpatients – when is there a need of antibiotics?

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Background: Recent years' decreased malaria transmission, the implementation of rapid diagnostic tests for malaria and the roll out of vaccines against common pneumonia causing bacteria have affected validity of guidelines and there is evidence of increasing over-use of antibiotics.

Methods & Materials: We studied the aetiologies of non-severe febrile illness among children and adults, 1087 patients, 3 months to 50 years old, with a history of fever during the past 48 hours were enrolled. A combined throat and per nasal swab, urine sample and aerobic blood culture were taken on each case. Patients were followed up 2, 7 and 14 days after enrolment to follow progress and to take a convalescent blood sample.

Results: Upper respiratory tract infection (34.5%) and urinary tract infection (23.3%) were the most common diagnoses followed by unspecific fever (16.5%). 133 (12.2%) patients fulfilled criteria for IMCI/IMAI pneumonia. 19% of throat/NP cultures were positive with most (63%) growing S. pneumonia. A positive swab had no relation with being classified as WHO-defined pneumonia ($p > 0.05$). For urine cultures, 9% of children less than one presented significant bacterial growth, all with E.coli. In adults, 6% had significant bacteria in urine. 48 (4.4%) of blood cultures flagged positive, whereof 33 (3.0%) contaminants while 15(1.4%) grew illness-causing pathogens. 8 (1.6%) of these were in children below five ($n = 487$). Seven blood cultures flagged positive for S. typhi, four children grew streptococci spp, one child grew Acinetobacter Baumanii and two adults and one child were positive for E.coli. IMCI/IMAI pneumonia or the presence of danger signs at assessment did not predict a positive blood culture ($p = 0.19$ and 0.6, respectively). There were no case fatalities during the study and 1010 patients were seen on day 14 whereof 2% reported no improvement or worsening, non of these were among patients with positive blood cultures.

Conclusion: Invasive bacterial illness is uncommon in outpatients with limited relation between clinical classification and positive culture. Results from PCR and the aetiological correlation with IMCI/IMAI diagnosis will be presented and the possible impact on antibiotic use discussed.

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Ferritin-iron acquisition in the emerging pathogen Burkholderia pseudomallei

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Background: The ability to acquire iron from the host environment is essential for the virulence of pathogenic bacteria. Previous studies with the globally emerging Gram-negative pathogen *Burkholderia pseudomallei* revealed that mutants defective in siderophore synthesis and transport were still capable of using mammalian ferritin as iron source and remained virulent in a murine melioidosis model. At present, essentially nothing is known about the role of iron acquisition in the pathogenesis of *B. pseudomallei* infection and ferritin-iron acquisition pathways have never been defined in Gram-negative pathogens.

Methods & Materials: Previous studies with *B. cenocepacia* indicated involvement of a serine protease in ferritin iron acquisition. *B. pseudomallei* cells were grown in the absence and presence of a soluble protease inhibitor cocktail or its individual constituents. Putative genes encoding secreted and non-secreted serine protease were deleted. In both instances, growth in iron-depleted growth medium was employed as a measure of the ability to utilize ferritin as sole iron source.

Results: Addition of a soluble cocktail containing six protease inhibitors to a culture abolished the ability of *B. pseudomallei* to grow on ferritin as sole iron source. Addition of individual proteases contained in the cocktail revealed that two of them, AEBSF (4-[2-aminoethyl] benzene sulfonyl fluoride) and EDTA exhibited the most potent effect. These data are consistent with the previous finding that *B. cenocepacia* employs a serine protease for ferritin iron acquisition. Mutants that had all seven serine proteases individually deleted were successfully constructed. All mutants were still able to utilize ferritin as sole iron source at rates indistinguishable from the parent strain. The most plausible explanation for this observation is that *B. pseudomallei* does not employ a serine protease for ferritin iron acquisition.

Conclusion: We have provided conclusive evidence that ferritin-iron acquisition by *B. pseudomallei* involves proteolytic degradation of ferritin but the protease(s) and other factors, e.g. transporters, involved in this process remain to be identified. Defining a ferritin-iron acquisition pathway will substantially alter our current view of iron acquisition mechanisms in Gram-negative bacteria and provide a more complete picture of the pathogenesis of infection with *B. pseudomallei*.

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Invasive *Salmonella typhimurium* populations from Sub-Saharan Africa: Transmission and adaptation dynamics

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Background: The emergence of invasive non-typhoidal *Salmonella* (iNTS) disease across sub-Saharan Africa has been associated with two related lineages of *S.* Typhimurium which are of multi locus sequence type ST313. The expansion of both lineages has a temporal association with the HIV pandemic and antibiotic usage.

Methods & Materials: Whole genome sequences of ST313 *S.* Typhimurium were probed for evidence of genome degradation in the form of non-synonymous mutations, frameshift mutations and deletions. Phenotypic microarrays, RNA-sequencing and murine infection models were used to investigate differences in the phenotype of the isolates from the sub-Saharan African lineages in comparison to gastroenteritis-associated strains.

Results: The results provide evidence that lineage-specific genome degradation, with some similarities to that observed in human *S.* Typhi, occurred throughout the evolution of these two lineages. Both lineages exhibited altered metabolic potential exemplified by the ability to metabolise alternative carbon sources and different virulence potential in mice.

Conclusion: These results identify likely adaptive changes that may have underpinned the emergence of these lineages and aided their adaptation to immunocompromised humans.

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Healthcare-associated, community-acquired and hospital-acquired Gram-negative bacteremias in a Turkish referral hospital

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Background: Bloodstream infections (BSIs) due to gram-negative bacteria are an important cause of sepsis and septic shock that are associated with a high rate of morbidity and mortality. The aim of the present study was to evaluate the differences in the clinical and microbiological characteristics and outcomes of community-acquired, hospital-acquired and healthcare-associated gram-negative bacteremias (GNBs).

Methods & Materials: A prospective cohort study was carried out over a 36-month period in a tertiary referral hospital inTurkeyand patients with gram-negative bacteremia were included. Those patients with a prior hospitalization within 90 days, ongoing chemotherapy, hemodialysis, home intravenous therapy, wound care and residence in a long-term care facility were classified as having healthcare-associated BSIs.

Results: Among 151 patients, 57 (37.7%) had bacteremia within two days of admission and classified as having community-acquired BSIs. Forty (70%) of these patients were found to be healthcare-associated. The remaining 94 patients (62.3%) were considered to have hospital-acquired infections.

Malignancies were more common in patients with healthcare associated and hospital-acquired GNBs than in patients with community-acquired infections (42.5%, 45.7% and 5.8% respectively).

Nearly all (88.2%) cases of community-acquired bacteremia were originated from urinary tract infections (UTIs). UTIs were the origin of bacteremia in 55% of health-care associated and 20% of hospital-acquired GNBs.

E. coli was the most common pathogen in both community-acquired and health-care associated GNBs (64.7% and 72.5%, respectively), whereas accounted for 34% of hospital-acquired cases. Acinetobacter spp. (22.3%) was the second most frequent pathogen in patients with hospital-acquired GNBs.

The third-generation cephalosporin resistance rates were similar in patients with community-acquired and health-care associated GNBs (52.9% and 55%, respectively) and carbapenem resistance was not detected among these patient groups. Ceftriaxone and imipenem resistance was found to be 74.4% and 32.9% respectively in patients with hospital-acquired GNBs.

Fatality rates were not significantly different between community-acquired and health-care associated GNBs (11.7%