relation to the retrospective data. There was no significant change during 2005.

The susceptibility pattern during the 12-month duration from December 2005 to 2006 from outpatients and inpatients was not all diverse. Isolates had included E. coli, 1209 strains, Klebsiella, 650, Proteus, 379, Pseudomonas, 32 and Enterococcus fecalis, 6 strains. The contribution of inpatients was of E. coli, 269 strains, Klebsiella, 147, Proteus, 44, Pseudomonas, 12 and Enterococcus fecalis, 2 only. There was no significant difference for amoxicillin-clavulanic acid, chloramphenicol, nitrofurantoin, amikacin and netilmicin susceptibility among inpatient and outpatient isolates (Figure-1). There was a significant reduction in the prevalence of isolates susceptible to ceftazidime, accompanied by an increase in isolates susceptible to gentamicin (Figure-2).

The clinical course in neonates with urinary tract infections treated with short-term intravenous antibiotic followed by oral treatment is highly favorable, both in short and long terms (3). Selection of appropriate intravenous and oral antibiotics against isolates in local circulation should be a reality through updates to the susceptibility profile of urinary isolates (1) encountered among patients seeking treatment in the hospital premises (2). That would also encourage frequent dialogues amongst clinical microbiology personnel and those responsible for patient care and guarantee better empirical recipe in urinary tract infections.

References

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Re: Wet Heat Exposure: A Potentially Reversible Cause of Low Semen Quality in Infertile Men

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To the Editor:

Dry and wet heat differ mainly in the speed of energy transfer, as heat transfer by convection and contact in water is increased than in air, and because heat dissipation by sweat evaporation is blocked into
the water (1), thus this study expressed the concern of recreational immersion in hot water over male reproductive health.

The current article by Shefi and colleagues proves the reversible effect of this spermatogenic harm through the comparison of total motile count during wet heat exposure and after cessation. Heat effects over sperm quality are likely to happen within 3 days of exposure (2) and it seems to be reversible as in heat injury from other sources (3).

In spite of the important results demonstrated, this study does not privilege some issues as sperm DNA fragmentation, and other molecular pathways, like heat shock proteins (Hsp) expression. Heat stress produced by cryptorchidism and varicocele is well characterized as a DNA fragmentation inducer (4,5) even when normal morphology is observed (5), and may affect ART outcomes (4). Also, Hsp, reestablish a homeostatic mechanism and equilibrium between protein synthesis and degradation in the cells (6) and differential expression of HSP70 and HSP90 was shown in the heat-induced stress (7).

Investigation of these underlying factors process should be considered in further studies intending to elucidate the mechanisms and differences of wet heat gonadotoxicity and induced infertility.

References

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REPLY BY THE AUTHORs

We agree that the functional capacity of sperm should be evaluated after wet heat exposure to understand if the mechanisms of impairment are similar to that described for other forms of heat stress.