hood of increasing problems with DHF and the risk of urban yellow fever in new areas. The global HIV epidemic will put a large number of people at risk for currently recognized and new opportunistic infections. The roles of hepatitis B and C viruses in chronic liver disease and hepatocellular carcinoma, of human papillomaviruses in cervical cancer, and of *Helicobacter pylori* infection in peptic ulcer disease and gastric cancer are now well-established. It is quite likely that more chronic diseases may be found to have an infectious aetiology.

References

- Lederberg J, Shope RE, Oaks SC (eds.). Emerging infections: Microbial threats to health in the United States. Washington, DC: The National Academy Press: 1992.
- McDade JE, Hughes JM. New and emerging infectious diseases. In: Mandell GL, Bennett JE, Dolin R (eds.). Basic principles in the diagnosis and management of infectious diseases Part 1. 5th ed. Churchill Livingstone Inc 2000:178-183.
- Thomas PA. Severe acute respiratory syndrome. *Indian J Med Microbiol* 2003; 21:152-60.
- WHO Communicable Disease Surveillance and Response website: Summary
 of probable SARS cases with onset of illness from 1 November 2002 to 31
 July 2003 (revised 26 September 2003). http://www.who.int/csr/sars/country/
 table2003_09_23/en/ (accessed 23 August 2004).
- UNAIDS/WHO global and regional HIV/AIDS estimates end-2002 (AIDS epidemic update: December 2002) http://www.unaids.org/html/pub/Topics/ Epidemiology/RegionalEstimates2002_en_pdf.htm (accessed 23 Aug.2004).
- 6. Kumar S. HIV cases rising sharply in India. BMJ 2003;327:245.
- LeDuc JW, Hughes JM. Surveillance for emerging infectious diseases. In: Maguire JH, Walker DH, Weller PF Guerrant RL, Krogstad DJ, Tropical infectious diseases: Principles, pathogens and practice. Churchill Livingstone Inc. 1999:25-60.

- Lakshmy AR, Siddiqi N, Shamim M, Deb M, Mehta G, Hasnain SE. Molecular characterisation of *Mycobacterium abscessus* strains isolated from a hospital outbreak. *Emerg Infect Dis* 2000;6:561-2.
- Sengupta S, Kumar P, Ciraj AM, Shivananda PG. Acinetobacter baumannii -An emerging nosocomial pathogen in the burns unit Manipal, India. Burns 2001:27:140-4.
- Vila J, Ruiz J, Gallardo F, Vargas M, Soler L, Figueras J, et al. Aeromonas spp. and Traveller's diarrhoea: Clinical features and antimcrobial resistance. Emerg Infect Dis 2003;9:552-5.
- Nataro JP, Steiner T, Guerrant RL. Enteroaggregative Escherichia coli. Emerg Infect Dis 1998;4:251-61.
- Basu A, Garg P, Datta S, Chakraborty S, Bhattacharya T, Khan A, et al. Vibrio cholerae 0139 in Calcutta: Incidence, antibiogrammes and genotypes. Emerg Infect Dis 2000:6:139-47.
- Chowdhury NR, Chakraborty S, Ramamurthy T, Nishibuchi M, Yamasaki S, Takeda Y, et al. Molecular evidence of clonal Vibrio parahaemolyticus pandemic strains. 2000;6:631-6.
- Fritz CL, Dennis DT, Tipple MA, Campbell GL, McCance CR, Gubler DJ. Surveillance for pneumonic plague in the United States during an international emergency: A model for control of imported emerging diseases. Emerg Infect Dis.1996;2:30-6.
- Dar L, Broor S, Sengupta S, Xess I, Seth P. The first major outbreak of dengue haemorrhagic fever in Delhi, India. 1999;5:589-90.
- Maitreyi RS, Dar L, Muthukumar A, Vajpayee M, Xess I, Vajpayee RB, et al. Acute haemorrhagic conjunctivitis due to enterovirus 70 in India. Emerg Infect Dis 1999:5:267-9.
- Chaudhry R, Premlatha MM, Mohanty S, Dhawan B, Singh KK, Dey AB. Emerging leptospirosis, North India. *Emerg Infect Dis* 2002;8:1526-7.
- Thakare JP, Rao TL, Padbidri VS. Prevalence of West Nile virus infection in India. Southeast Asian J Trop Med Public Health 2002; 33:801-5.
- Mehta A, Rodrigues C, Kumar R, Rattan A, Sridhar H, Mattoo V, et al. Pilot programme of MRSA surveillance in India. J Postgrad Med 1996;42:1-3.
- Threlfall EJ, Ward LR. Decreased susceptibility to ciprofloxacin in Salmonella enterica serotype typhi, United Kingdom. Emerg Infect Dis 2001;7:448-50.
- Joshi S, Wattal C, Sharma A, Oberoi JK and Prasad KJ. Quinolones Drug of choice for Enteric fever? IJMM 2004;22(4):271-2.

Drug Profile

DUTERSTERIDE

Dutersteride, 4 azasteroid is a selective and potent inhibitor of type 1 and 2 isoforms of 5x reductase. This drug is used in the treatment of BPH (Benign prostatic hyperplasia) current medical treatments of BPH include the use of after X-adrenoceptor antagnonists and 5x reductase inhibitors to relieve symptoms and improve urinary flow. How X-adrenoceptor antagonists acts directly on smooth muscle to decrease muscle tone; 5X redutase inhibitors decrease the size of the prostate.

Mechanism of action: Dutasteride is a dual 5X reductase inhibitor. The enzyme 5X reductase is central to the conversion of test-osterone to DHT. Daily doses of dutasteride result in a dose dependent reduction of serum DHT that is greater than finasteride.

<u>Pharmacokinetics</u>: Following single dose of 0.5 mg, peak serum concentration occurs within 1-3 hours. It is well absorbed, with bioavailability of approximately 60%. It is highly bound to plasma proteins (>99.5%). The volume of distribution is large (app. 300-5001). Drug is extensively metabolised in the liver by the human cytochrome P450, isoenzyme-CVP3A4 & CYP3A5. Single doses of <5.0 mg are eliminated more rapidly than doses of more than 5.0 mg by both concentration-dependent and independent elimination pathways and have a half life of 3-9 days. Dutasteride and it metabolities are mainly excreted in faces.

- Pharmacokinetics in special patient groups Dutasteride is contraindicated for use in children and adolescents; elderly patients-no dose adjustment is required.

-<u>In renal failure patients</u>-No adjustment in dsage is anticipated for these patients as less than 0.1% of unchanged drug is excretedin urine.

-In <u>hepatic impairment</u> patients-caution should be exercised in administering to patients with mild to moderate hepatic impairment. Drug is contraindicated in patients with severe hepatic impairment

<u>Drug interactions</u>: Blood concentrations of dutersteride may increase in the presence of inhibitors of CYP3A4 such as ritonavir, ketoconazole, verapamil, diltiazime, ciprofloxacin. There are no pharmacokinetic or pharmacodynamic interactions between dutersteride and tamsulosin, terazosin, warfarin, digoxin and cholestyramine.

Indications and Usage - Dutasteride is indicated for the treatment of symptomatic benign prostatic hyperplasia (BPH) in men to i) improve symptoms, and (ii) reduce the risk of acute urinary retention (iii) reduce the risk of the need for BPH related surgery.

<u>Contraindication</u> - The drug is contraindicated for use in women and children and in patients with known hypersensitivity to drug.

<u>Warnings</u> - Dutasteride is absorbed through the skins; therefore women who are pregnant or may be pregnant should not handle because of the possibility of absorption of dutasteride and the potential risk of a congenital anomaly in the male fetus.

Men being treated with dutasteride should not donate blood until a least months have passed following their last dose.

<u>Dosage and administration:</u> The recommended dose of Dutasteride is 0.5 mg daily orally. The capsules should be swallowed whole, can be given with or without food.

<u>Effect on PSA-PSA</u> levels decrease following dutasteride treatment. To interpret PSA value in a man treated with dutasteride for 6 month or more, the PSA value should be doubled for comparison with normal values in untreated man.