population (98 individuals, *p*-value of 0.001; χ^2 value of 58.6 and a 95% CI: 0.678-1.2).[2] The Japanese and Chinese populations had a higher prevalence of the 326C mutant allele than any other population. The Saudi population also showed a marked difference with African American ethnic group (24 individuals, *p*-value of 0.0262 using Fisher's exact test, CI 1.135-7.85, NCBI, rs1052133). The prevalence of the 326C allele in the African American sample was very low and the 326C/326C allele frequency was 0%, a variation of what was reported in other populations, but the small number of samples assayed might contribute to the lack of identifying the mutant homozygous allele.

The Kingdom of Saudi Arabia is a vast and an ethnically diverse country. Although, there are pockets of ethnically homogenous populations where there is very little population drift, inter-population differences have been reported for several other genetic loci including sickle cell gene and ß-thalassemias.[5] This study showed a significant difference in the genotype allele frequency of *hOGG1* S326C in Saudis compared to Chinese and Japanese populations. Further studies are required to genotype the *hOGG1* S326C in different regions of Saudi Arabia to document any regional variations and the association of this polymorphism and increased risk of different types of cancer in Saudi Arabia remain to be investigated.

Acknowledgment. *This work was entirely supported by SABIC via the Research Center Deanship in King Saud University, Riyadh, Kingdom of Saudi Arabia. The authors would like to thank Mr. Kalid Alzeyad for his help with the sample collection and consent forms.*

Received 9th November 2006. Accepted 30th December 2006.

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Is Saudi Arabia a fertile land for exchanging infectious diseases?

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Over 2 million people from around the world visit Saudi Arabia every year to perform the Muslim Pilgrimage, Hajj. All of these people congregate at the 2 Holy Mosques in the cities of Mecca and Madina, remaining in crowded environments for up to 3 weeks. A further 2 million people visit the Kingdom to perform Omra in the 2 Holy Mosques. In addition, the country is the home of more than 6 million working expatriates. Most of these visitors, pilgrims and expatriate laborers come from impoverished third world countries where tuberculosis (TB) is endemic.[1] This huge number of expatriates and pilgrims associated with Hajj means that there is a lot of contact between people; more than enough to transfer, spread and exchange communicable diseases. In the past, several outbreaks of meningitis and cholera have occurred, demonstrating transmission of infectious diseases via human-to-human contact.[2] During these visits, a number of people suffer from minor upper respiratory tract infections but there is little consideration for the involvement of serious contagious diseases.[3] One isolated report highlighted the fact that *Mycobacterium Tuberculosis*, a re-emerging communicable disease, was responsible for cases of pneumonia during Hajj.[4] This report was strengthened by Wilder-Smith et al,[5] when they measured the immune response to TB antigen prior to departure and 3 months after return from Hajj pilgrimage. At the end of Hajj, Pilgrims return to their home countries taking with them any contagious disease they may have acquired. These observations suggest that Saudi Arabia is a fertile environment for the spread and exchange of several indigenous and imported diseases. Previous observations have been reinforced by recent results during an on-going nationwide epidemiological study. For the last 2 years, we have been able to focus our research efforts on finger-printing *M. Tuberculosis* in Saudi Arabia. More than 1,400 isolates have been collected and typing is in the final stage. Preliminary data shows many imported clades in Saudi Arabia (previously identified in other parts of the world) such as Beijing, Manila, Latin-America-Mediterranean, Delhi and many others. The presence of several of these families in one country is a strong evidence that the crowdness of Hajj and Omra is facilitating the exchange of communicable diseases. There is a paucity of information regarding other diseases but it is unlikely that TB is unique. Further data is required in order to study other communicable

diseases. It is our belief that a committee should be established to monitor this issue that should include any countries from which Pilgrims and immigrants originate. The task of such committee should monitor, raise awareness and potentially initiate screening to minimize the transmission of communicable diseases.

Received 29th January 2006. Accepted 23rd December 2006.

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Spontaneous recovery of propylthiouracil-induced fulminant hepatic failure in an 8-year old child

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Hepatotoxicity was first reported as a side effect of propylthiouracil for the treatment of hyperthyroidism in 1947.[1] Subsequently, several case studies have been reported.[2] Symptomatic propylthiouracil-induced hepatic injury is rare in thyrotoxic adults and less than 10 cases have been published in childhood.[3] The clinical course is usually benign once the drug is withdrawn, however, fatal cases have been encountered.[4,5]

We describe an 8-year-old girl with hyperthyroidism treated with PTU, developed fulminant hepatic failure and ended with spontaneous recovery. An 8-year-old girl presented with 3-week history of heat intolerance, tremor, restless sleep and weight loss. She was noted having diminished school performance due to poor concentration and progressive prominence of her eyes. On examination, her vital signs showed a heart rate of 120 beats/min and blood pressure of 140/89. She had tremor, exophthalmos, and palpable thyroid. The rest of the examination was unremarkable. Laboratory evaluation at presentation showed white blood cell 11.1, neutrophils 40%, lymphocyte 43%, alanine aminotransferase (ALT) 25 U/L (10-35), aspartate aminotransferase (AST) 32 U/L (10-45), alkaline phosphatase (ALK) 203 U/L [normal range (NR) 100-300], gamma glutamyltransferase (GGT) 10 IU/L (NR 7-23), total bilirubin 13 umol/L (NR 0-21), direct bilirubin 3 umol/L (NR 0-5), ammonia 23 umol/L (NR 0-55), prothrombin time (PT) 6 seconds (NR 11.9-14.3), prolonged thrombin time (PTT) 35 seconds (NR 34.7-42.2), free thyroxine (FT4) 91 pmol/L (NR 12-22), total triiodothyronine (TT3) 4.1 nmol/L (NR 1.3-3.1), thyroid stimulating hormone (TSH) 0.02 mU/L (NR 0.27-4.2). Antithyroperoxidase antibody was 189 (normal <12), thyroid stimulating immunoglobulin (TSI) was 150% (normal <120%). Thyroid scan showed diffuse homogenous uptake. She was diagnosed with Grave's disease and started on PTU 50 mg 3 times per day and propranolol 10 mg 3 times per day.

One month later, FT4 was 30 pmol/L, TT3 was 3.1 nmol/L, and TSH was 0.07 mU/L. 2 months later on the above-mentioned treatment, she presented with progressive yellowish discoloration of sclera, lethargy and fluctuation of her consciousness. Laboratory evaluations showed FT4 24 pmol/L, TT3 2.8 nmol/L, TSH 0.07 mU/L, WBC 8.4, neutrophils 26%, lymphocyte 40%, ALT 161 U/L, AST 144 U/L, ALK 233 U/L, GGT 143 IU/L, total bilirubin 645 umol/L, direct bilirubin 512 umol/L, ammonia 290 umol/L, PT 20.2 seconds and PTT 43.4 seconds. A PTU-induced hepatoxicity was suspected and PTU was stopped. Potassium iodide (SSKI) 300 mg 3 times a day was started, in addition to hydrocortisone 10 mg 3 times a day. Ultrasound liver showed hepatomegaly with increased echogenicity and normal hepatic blood vessels flow. Liver biopsy showed cholestatic hepatitis with acinar necrosis. Other causes of cholestatic jaundice were ruled out. Serological studies for hepatitis A, B, C, E viruses, cytomegalovirus and Epstein-Barr virus were negative. Metabolic work-up including serum ceruplasmin level, alpha-1-antitrypsin level, 24-hour urinary copper level and tandem mass spectrometry was unremarkable. Immunological studies indicating autoimmune hepatitis including antinuclear antibody, anti-smooth muscle antibody