## G-RC-10

### MULTICENTRE PHASE II TRIAL OF TAXOTERE AND CISPLATIN IN ADVANCED NON-SMALL CELL LUNG CANCER

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Non-small cell lung cancer is the commonest cause of cancer death in Hong Kong and frequently presents itself at advanced stages. Combination chemotherapy has been used in advanced stages with unsatisfactory response. We have performed a phase II study on the response rate and safety profile of Taxotere and cisplatin given in combination in patients with metastatic or locally advanced non-small cell lung cancer (NSCLC). Chemotherapy naïve patients with histologically or cytologically proven stage III or IV unresectable NSCLC and good performance state were recruited consecutively. Exclusion criteria included brain or leptomeningeal involvement, major organ failure, previous malignancies, active uncontrolled infection, or definite contraindications for the use of corticosteroids. Taxotere 75mg/m$^2$ as 1 hour intravenous infusion and cisplatin 75mg/m$^2$ as 30 minutes intravenous infusion were given in 3-weekly intervals for 6 cycles. Interim results were obtained from 20 patients (10F) with age 55.8± 7.34 years. There were 14 adenocarcinomas, 3 squamous cell carcinoma and 3 undifferentiated NSCLC with 2 in TNM stage IIIA, 10 in stage IIIB and 8 in stage IV. The partial response rate after 3 courses of chemotherapy was 42% and 47% had stable disease. After completion of 6 courses of chemotherapy, the overall response rates were 53%, 26% had stable disease, and 21% progressive disease. Grade 3 or 4 neutropenia occurred in 55% of chemotherapy cycles in which 4 out of 20 patients required dose reduction and 3 of them required granulocyte-colony stimulating factor support. Neutropenic fever occurred in 17%. Grade 3 thrombocytopenia occurred in 3%. Significant toxicities (grade 3 or 4) included nausea (1%), vomiting (10%), diarrhoea (2%), infection (3%), asthenia (2%) and allergy (1%). There had been no treatment-related deaths. The combination of Taxotere and cisplatin appears to be a fairly well tolerated and highly effective regime in the treatment of advanced NSCLC. Further studies are warranted to evaluate the impact of this combination on the median term response and survival of these unfortunate patients.

## G-RC-11

### SERUM LEPTIN AND VASCULAR RISK FACTORS IN OBSTRUCTIVE SLEEP APNOEA

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**Study objectives**: To define the metabolic profile relevant to vascular risks in obstructive sleep apnoea (OSA) and the role of leptin resistance in this risk profile
**Design**: Case control study
**Setting**: Sleep Laboratory, Queen Mary Hospital, University of Hong Kong
**Methods**: Thirty OSA subjects were compared with 30 non-OSA subjects, matched for body mass index (BMI), age, sex and menopausal status, in the following parameters : girth of neck, waist and hip, skinfold thickness, fasting serum levels of lipids, glucose, insulin and leptin.
**Results**: Compared to control subjects without OSA, despite a similar BMI, the OSA group had a significantly more adverse vascular risk factor profile including dyslipidemia, higher diastolic blood pressure, insulin resistance, and greater adiposity reflected by skinfold thickness. OSA subjects also had higher circulating leptin levels (9.2± 4.2 vs 6.5± 3.8 ng/ml, mean ± SD, p=0.001). Serum leptin levels correlated positively with BMI, skinfold thickness, serum cholesterol, LDL-cholesterol, insulin, insulin:glucose ratio, apnoea-hypopnea index and oxygen desaturation time, and multiple stepwise regression analysis identified skinfold thickness, waist : hip ratio, serum LDL-cholesterol, and diastolic blood pressure as independent correlates, while only serum insulin and diastolic blood pressure were independent correlates in OSA subjects. After treatment with nCPAP for 6 months, there was a significant decrease in circulating leptin (p =0.01) and triglyceride levels (p=0.02) without change in anthropometric and other metabolic characteristics.
**Conclusion**: Despite controlling for BMI, OSA subjects showed a distinct profile with clustering of vascular risk factors. Hyperleptinaemia was present in OSA subject but it can be normalised by treatment with nCPAP, suggesting that increased leptin resistance was not the cause of OSA or its associated vascular risks.