were homozygous for HLA-DW; a further ten from six families were HLA-DR3,DR7 and seven from four families were HLA-D3,DR2. These were single cases of HLA-D3,DRI; HLA-D3,DR4 and HLA-D3,DR8.

CONCLUSIONS: The results confirm an association of coeliac disease with HLA-D3,-DQw2. They suggest that this link is due to the presence of these alleles in a disease-associated extended haplotype. Further investigation of the individual alleles on this haplotype by DNA sequencing should clarify which of the MHC class II genes are responsible for this association. This would enable easier identification of individuals at risk.

30 INCREASED RISK OF TB AMONG HINDU ASIANS IN LONDON: AN EFFECT OF VEGETARIANISM AND VITAMIN D?
PJ FINCH, FJC MILLARD and JD MAXWELL
St. George's Hospital Medical School, London, SW17 ORE

We have examined all confirmed cases of TB in Wandsworth over a 15 year period (1973-1988), and selected the immigrant Asians and native Caucasians. Asians were of Hindus to Muslims in the localitv was estimated from the 1981 Census (Small Area Statistics). The proportion of 1052 cases of TB were studied:

<table>
<thead>
<tr>
<th>Religion</th>
<th>Hindu</th>
<th>Muslim</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAUCASIAN</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>India</td>
<td>420</td>
<td>217</td>
<td>3</td>
</tr>
<tr>
<td>PAKISTAN</td>
<td>350</td>
<td>200</td>
<td>3</td>
</tr>
<tr>
<td>AFRICA</td>
<td>200</td>
<td>200</td>
<td>3</td>
</tr>
</tbody>
</table>

The number of Hindus, Muslims and Caucasians in the Wardsworth Area were estimated at 129490, 5790, and 10065. The annual occurrence index for TB 1988, were:

- PULMONARY GLANDULAR BONE OTHER

The earlier presentation and excess numbers of Hindus at presentation (5.74, 5.31 - 6.17) than Muslims (7.28, 7.06 - 7.52) with TB shown here support the hypothesis that vitamin D deficiency associated with a vegetarian diet leads to an early and more pronounced impairment of cellular immunity, and an increased risk of TB, especially in extrapulmonary sites, after immigration.

31 CHLORIDE (Cl\(^{-}\)) CHANNELS IN A CULTURED HUMAN GASTRIC CELL LINE (HGT-1)
G I SANDLE and G WARHURST
Department of Medicine (University of Manchester School of Medicine), Hope Hospital, Salford M6 BHD

Secretion of HCl by gastric parietal cells depends, at least in part, upon an apical membrane Cl\(^{-}\) conductance which may be regulated by histamine-activated cyclic AMP-dependent protein kinases. We have studied Cl\(^{-}\) channel activity in the plasma membrane of cultured (non-polarized) cells (HGT-1) derived from a human gastric carcinoma. These cells possess H\(_2\) receptors and exhibit histamine-activation of the adenyl cyclase-cyclic AMP system.

Using patch clamp techniques, excited inside-out patches (n=15) from non-stimulated cells (i) showed non-linear single channel current-voltage relationships with outward rectification, the mean conductance increasing from 34t 5ps at -60mV to 116±14ps at +100mV (ii) exhibited low single channel open probabilities (P\(_o\)) between -100mV and +100mV, and (iii) shifted their reversal potential 2fmV in a hyperpolarizing direction when bath Cl\(^{-}\) was decreased from 149mM to 53mM, as predicted for a Cl\(^{-}\) channel. In cell-attached patches, 1mM histamine increased P\(_o\) from 0.044 to 0.340 (n=3) and 10mM forskolin increased P\(_o\) from 0.014 to 0.297 (n=9). Outward rectifying channels in cell-attached and inside-out patches from these agonist-treated cells were voltage-dependent. P\(_o\) increasing 3-fold during membrane depolarization. In excited inside-out patches, channel activity was inhibited by the Cl\(^{-}\) channel blocker diphenylcarboxylic acid (1mM). These results indicate that the plasma membrane of histamine-sensitive HGT-1 cells expresses cyclic AMP-activated Cl\(^{-}\) channels, which may be similar to apical Cl\(^{-}\) channels operating in gastric parietal cells during HCl secretion.

32 INTRACELLULAR pH (pHi) REGULATION IN RAT HEPATOCYTES: BICARBONATE (HCO\(_3^{-}\)) DEPENDENT AND INDEPENDENT MECHANISMS
D GLEESON, PD SHITH and JL BOYER
Liver Centre, Yale Univ Sch Med, New Haven, Connecticut, USA

Hepatocytes possess a Na\(^+/\)H\(^+\) exchanger which mediates pHi recovery from an intracellular acid load in HCO\(_3^{-}\}-free media (AJP 1987 252:G109). HCO\(_3^{-}\}-transport systems regulate pHi in many cells but have been incompletely characterised in hepatocytes. We therefore studied rat hepatocyte pHi recovery following intracellular acid loading (NH\(_4\) pulse) in both the presence (+) and absence (-) of HCO\(_3^{-}\)-using the pH sensitive dye BCECF and a continuously perfused subconfluent monolayer cell culture system.

RESULTS: pHi recovery (maximum pH efflux rates) was higher in +HCO\(_3^{-}\)-than in -HCO\(_3^{-}\)- (5.4±0.1 SDi.1 vs. 3.6±0±.17 mH\(_{mi}^{-}\)s, p<0.05). pHi recovery in -HCO\(_3^{-}\)- was inhibited 89±12% by acute Na\(^+\) replacement (with choline) and inhibited 75±9% by 1mM amiloride, suggesting mediation by Na\(^+/\)H\(^+\) exchange. In contrast, 1mM amiloride inhibited pHi recovery in +HCO\(_3^{-}\)- by only 27±5%. The amiloride independent pHi recovery in +HCO\(_3^{-}\)- was (a) inhibited 50±6% by preincubation of cells in 0.25-2.0 mM of the anion transport inhibitor DIDS (which did not affect pHi recovery in -HCO\(_3^{-}\)-), (b) inhibited 78±3% by acute Na\(^+\) replacement (o) unaffected by depletion of intracellular Cl\(^{-}\)- suggesting that this mechanism does not involve Cl\(^{-}\/-HCO\(_3^{-}\)- exchange. CONCLUSION: Rat hepatocyte pHi recovery following an intracellular acid load is mediated by two mechanisms: (i) Na\(^+/\)H\(^+\) exchange and (ii) a DIDS inhibitable, Na\(^+\) and HCO\(_3^{-}\)-dependent but Cl\(^{-}\)-independent mechanism, characteristic of Na\(^+\)/HCO\(_3^{-}\)- cotransport.
The prevalence of microalbuminuria was assessed in 149 consecutive newly diagnosed and untreated patients with Type 2 diabetes on initial presentation to the diabetic clinic. An albumin/creatinine ratio of > 3.0 in an early morning urine specimen was considered abnormal and, using this criterion, a total of 36 (24%) patients had evidence of microalbuminuria. The diabetic patients with microalbuminuria were older (64.8 (10.8) vs 58.1 (11.1) yrs; p<0.005) and the initial glycated haemoglobin was higher (13.1 (3.3) vs 11.4 (2.7)%; p=0.01). A significant correlation was observed between presence of microalbuminuria and systolic blood pressure (p<0.01) and there was also an association with evidence of ischaemic heart disease, peripheral or cerebrovascular disease (p<0.01). No association was observed with sex, diabetic retinopathy, diastolic or mean arterial blood pressure. In conclusion, microalbuminuria is commonly found in newly diagnosed patients with Type 2 diabetes. While this may be related in part to glomerular hyperfiltration associated with uncontrolled hyperglycaemia, the predictive value of microalbuminuria as a marker for macrovascular disease is again emphasized.

34 PLASMA VISCOITY, COAGULATION AND FIBRINOLYSIS IN MALE BODYBUILDERS: EFFECT OF HIGH DOSE ANALOGIC STEROIDs
McKillop G, McLaughlin K, Northcote RJ, Gallantyne D and Love GD

Department of Medical Cardiology, The Victoria Infirmary Glasgow and University Department of Medicine, Royal Infirmary, Glasgow

Plasma viscosity, coagulation and fibrinolysis were compared in three matched male groups: ten sedentary controls, 8 non-steroid-using bodybuilders and ten steroid-using bodybuilders (dose 8 - 20 times therapeutic). Non-steroid-using bodybuilders had lower viscosity than sedentary controls (1.16 vs 1.30 mPa.s, p<0.002), but no significant changes in fibrinogen, antithrombin, protein C, plasminogen or plasminogen activators. In steroid-using bodybuilders, this reduction in viscosity was abolished (1.27 mPa.s). Significant elevations (p<0.001) of protein C (70% mean increase) and plasminogen (26% mean increase) occurred in steroid users, similar to those previously induced by therapeutic doses in volunteers. These effects may be relevant to changes in cardiovascular risk in bodybuilders.

35 DO ABNORMAL PLASMA LIPIDS EXPLAIN ELEVATED SODIUM-LITHIUM COUNTERTRANSPORT IN HYPERTENSION?
S. CARR, T THOMAS, M LAKER* and R WILKINSON

Freeman Hospital, Newcastle Upon Tyne, University of Newcastle Upon Tyne

Elevated erythrocyte sodium-lithium countertransport (SLC) is related to a family history of not only essential hypertension (EHT) but also associated cardiovascular events. SLC has a strong inherited component but may be affected by environmental factors including plasma lipids which are a risk factor for cardiovascular disease. Plasma lipids could effect SLC by their interaction with erythrocyte membrane lipids. 30 patients with hyperlipidaemia and EHT (HTHT) 14 male,49.4 SD 12 years,20 with normotensive with hyperlipidaemia (HLNT) 9 male, 47.0 SD 11.6 years,13 with normal lipids and EHT (NLHT) 7 male, 49.2 SD 13.7 years) and 23 normal controls (NLNT 9 male 43.4 SD 17.6 years) were studied.24 patients had combined hyperlipidaemia, 11 hypercholesterolaemia,3 hypertryglyceridaemia and 2 hyper-alpha lipoproteinemia. None were taking lipid lowering drugs but some were taking anti-hypertensives. Analysis of variance showed a significant effect on SLC of both hypertension (p<0.01) and hyperlipidaemia (p<0.01). SLC was higher in HTHT (48.8 SD .15 mmol l/h per litre cells) than HLNT (.32 SD 14 p<0.02) and higher in NLHT (.34 SD .13 ) than NTNL (.26 SD .07 p<0.02). SLC was also higher in NLHT than NLNT (p<0.001) and in HLNT than NLNT (p<0.05).

SLC in all subjects was correlated with serum triglyceride (Rs=47 p<0.001) and high density lipoprotein (R=0.29, p<0.01). Elevated SLC may indicate a lipid related membrane changes that increase the risk of cardiovascular complications of hypertension.

36 SODIUM-LITHIUM COUNTERTRANSPORT: A GENETIC MARKER OF HYPERLIPIDEMIA AND HYPERTENSION?
S. CARR, T THOMAS, M LAKER* and R WILKINSON

Freeman Hospital, Newcastle Upon Tyne, University of Newcastle Upon Tyne

Elevated sodium-lithium countertransport (SLC) may identify normotensive individuals (NT) with an inherited predisposition to hypertension. We found SLC was associated with hypertension and hyperlipidaemia. Hypertension and hyperlipidaemia are risk factors for cardiovascular disease. We investigated 60 relatives (41.9 sd 16.9 years, mean diastolic blood pressure(DBP) 77.9 sd 15 mmHg, mean SLC 0.39 sd .14 mmol/L/h per litre cells) of 17 hypertensive patients (HT) with elevated SLC (54.6 sd 14.9 years, mean DBP 99.2 sd 13.4 mmHg, SLC .56 sd 0.09). The relatives were divided into two groups; those with SLC above the normal control range (0.02-0.4) and those within the control range.48% of relatives had elevated SLC. ANOVA showed a significant effect on SLC of hypertension (p<0.01) and hyperlipidaemia (p<0.01). In the families, SLC was significantly correlated with triglyceride(TG) p<0.001, cholesterol(Ch) p=0.003, low density lipoprotein (LDL) p=0.008, very low density lipoprotein (VLDL) p=0.001 and negatively correlated with high density lipoprotein (HDL) p<0.001.

6 Ht patients High rels Low rels Controls
mmol/L
CH 7.2 sd 1.4 6.6 sd 1.6 5.8 sd 1.0 5.6 sd 0.9
TG(range) 0.0 [1.7-5.3] 2.1 [1.4-4.7] 1.1 [1.4-5.8] 1.1 [1.4-5.2]
HDL 1.0 [1.8-2.3] 1.0 [1.8-2.3] 1.0 [1.8-2.3] 1.0 [1.8-2.3]
The HT patients and rels with elevated SLC had elevated CH p<0.001, TG p=0.001, LDL/VLDL and reduced HDL compared to rels with normal SLC and controls. The HT rels of HT patients with elevated SLC may be genetically predisposed to hypertension. If these individuals also have hyperlipidaemia they would be at increased risk cardiovascular complications of hypertension and it would be important to identify them at an early age.