irrelevant changes is unproductive. It is far more productive to focus on the other possible uses of this drug.

OGT 918 might not be as worthless as it seems for the neuronopathic forms of Gaucher's disease (types 2 and 3). Enzyme therapy could affect the course of the disease by reducing the total body burden of the substrate. Such treatment might impact the brain if some lipid load is transferred to the nervous system. The enzyme cannot cross the blood-brain barrier, but OGT 918 and newer more potent and specific derivatives can.[3] Thus, the combination of reduced substrate synthesis and reduced total body lipid burden could have benefit for the most devastating forms of Gaucher's disease.

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Ethnic minorities ill served by health service

Sir—The National Health Service plan[1] is a watershed document in the 52-year existence of this much beleaguered and often cash-starved institution. Sustained financial investment is being made to provide the nation with a world-class health service. However, although this resource input over a clearly defined period is to be applauded, we are disappointed that there are major flaws.

Your editorial (Aug 5, p 441)[2] highlights the scheme's failings with regard to vulnerable groups such as children and adolescents, who make up a quarter of the UK population. Another important group that has been poorly served, in our view, is ethnic minorities. This group constitutes 6% of the UK population and up to 30% in some UK regions. In addition, a substantial proportion of the National Health Service workforce is from ethnic-minority backgrounds.

In 1997, the UK government stated that services for black and minority ethnic communities must be an integral part of the National Health Service.[3] Improvement of the health of ethnic-minority groups was underscored as a government priority, signalling determination to ensure their needs were properly taken into account throughout the whole of the National Health Service policy-making process, from service planning to delivery. The National Health Service aims to provide equitable health care to all, and the national plan is about redesigning health service around the needs of the individual patient, but falls short of the mark on these issues.

To eliminate inequalities in health-service provision, especially for ethnic-minority groups, efforts need to be geared towards preventing disease, promoting health (at culturally accessible levels), and delivering appropriate care (strategically located community outreach programmes). Collection and use of data must also be improved to facilitate identification of all high-risk groups, such as patients with sickle-cell disease and thalassaemia, and to regularly assess the success of health interventions targeting these various groups. A solid research base, which understands the crucial relation between health status and ethnic-minority attitudes and perceptions is necessary.

We need to pinpoint and address the underlying causes of higher rates of certain diseases in ethnic-minority populations. In addition, we need better understanding of the reasons for lack of access to and awareness of available health services, as well as environmental hazards in homes and neighbourhoods. The evidence points to the fact that ethnic origin generally correlates with chronic and frequently increasing health inequalities among the UK ethnic-minority populations.

The national plan is about health for all, and the future health of the nation will be influenced substantially by success in improving the health of ethnic minorities. That this issue has received little attention in the plan is, therefore, disappointing.

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Gestational thrombocytopenia

Sir—James N George (April 29, p 1531)[1] states that distinction between gestational thrombocytopenia and idiopathic thrombocytopenic purpura is imprecise and not important for management of pregnant patients. Severe, symptomatic thrombocytopenia during pregnancy can be treated in the same way as mild symptomless thrombocytopenia arising at other times. He suggests that gestational thrombocytopenia may simply be a mild transient manifestation of idiopathic thrombocytopenic purpura.

We disagree with this point of view and fear that it could lead physicians to overlook a serious threat to the health of pregnant women who are at risk of developing haemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome. This syndrome can develop in the absence of clinical signs of pre-eclampsia.[2]

Although the development of thrombocytopenia in women with pre-eclampsia is well documented, as acknowledged by George, changes in platelet count in an apparently normal pregnancy has not been adequately considered. The lack of change in mean platelet count in cohort studies of normal pregnancies does not mean that platelet counts cannot or do not change in such pregnancies. Conversely a cause for thrombocytopenia must be diligently sought when a low platelet count is detected in a woman with a normal pregnancy. When a specific cause cannot be identified, the diagnosis by exclusion becomes gestational thrombocytopenia. Thus, gestational thrombocytopenia has been imprecisely defined, as George points out.

We longitudinally assessed changes in platelet count in 592 consecutive women from before week 13 to week 37 of uncomplicated singleton pregnancies.[3] In the third trimester, a similar proportion of women had a gradual decrease in platelet count to that of women with a gradual increase. Magnitudes of decreases and increases were similar. This observation explains how mean platelet count can show no apparent change during normal pregnancy. More importantly, 22 (3·7%) of the women who had a gradual decline in platelet count to less than 150×10^9/L were at high risk for the HELLP syndrome.[4]

The observation that a gradual decline in platelet count can precede onset of the HELLP syndrome also

was confirmed in a prospective cohort study in twin pregnancies.[5] Thus, gestational thrombocytopenia can be a precursor of the HELLP syndrome, rather than simply a mild transient manifestation of idiopathic thrombocytopenic purpura.

HELLP syndrome is diagnosed by laboratory data but blood tests generally are done only when disorders such as pre-eclampsia and epigastralgia become evident. Patients at an advanced stage of the HELLP syndrome with severe thrombocytopenia require platelet transfusion.[2] Prompt delivery is the only intervention that improves the clinical course of HELLP syndrome; women who are at increased risk of HELLP or are at an early stage of the syndrome must be identified as early as possible to avoid serious complications such as disseminated intravascular coagulation. We therefore urge that women with gestational thrombocytopenia be carefully monitored.

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Author's reply

Sir—Hisanori Minakami and Ikuo Sato suggest that mild but progressive thrombocytopenia during the third trimester predicts a greater risk of the HELLP syndrome, and they urge a diligent search for the cause of thrombocytopenia when it is noted.

This recommendation is reasonable, but I would take assessment of mild thrombocytopenia during pregnancy to be sufficiently diligent if the patient is symptom-free, physical examination is normal, and the remainder of the blood counts and urine analysis are normal. In such women, I do not believe that more expensive laboratory assessment is necessary. If thrombocytopenia becomes more severe, such as with a platelet count less than 50×10^9/L, or abdominal pain or other symptoms develop, or if anaemia exceeds the anticipated physiological level, then more extensive laboratory assessment would be appropriate.

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Sir—James N George[1] states: "Although patients with essential thrombocythaemia (ET) do not have the Philadelphia chromosome t(9;22), which defines chronic myelocytic leukaemia, the chimeric *BCR-ABL* transcript mRNA from this translocation has been identified in patients with clinically typical essential thrombocythaemia".

These findings have been detected, by D Blickstein and colleagues[2] in 48% of 25 patients negative for the Philadelphia chromosome who had essential thrombocythaemia, investigated by two-step nested RT-PCR. Some workers have not, however, been able to confirm these data by the same technique, in 18 and 20 such patients, respectively.[3,4] Moreover, others have shown the absence of the chimeric transcript in 41 patients with essential thrombocythaemia studied by the fluorescence in-situ hybridisation with a *BCR-ABL* probe.[5]

We investigated 112 patients who had essential thrombocythaemia with long follow-up, by RT-PCR, and detected the *BCR-ABL* transcript in only one patient who progressed to myeloid blastic crisis 12 years after diagnosis. Actually, whether essential-thrombocythaemia patients negative for the Philadelphia chromosome express the *BCR-ABL* transcript has been a matter of controversy for several years. In studies, the apparent essential thrombocythaemia carrying the Philadelphia anomaly, cytogenetically or molecularly should probably be considered as a variant of chronic myelocytic leukaemia with thrombocytosis and often long survival, with obvious clinical and therapeutical implications.

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Global health status

Sir—Kasturi Sen and Ruth Bonita (Aug 12, p 577)[1] refer to the double burden of disease as the epidemics of non-communicable diseases in countries that are struggling with old and new infectious diseases.

They describe the increasing population of older people in less-developed and more-developed countries, stating that more than half of the population older than 65 years lives in less-developed countries. Sen and Bonita argue that these rapid demo-graphic and risk-factor changes will lead to an increase in the heavy burden of non-communicable diseases in the absence of preventative action.

We believe that non-communicable diseases are already well established in less-developed countries.[2] Our calculations using data from 1998[3] show that the rates of non-communicable diseases are similar in countries with high and low or middle incomes (table).[2] The rates, expressed as disease-adjusted life years per 100 000 population, take into account the fact that low-income or middle-income countries contain 85% of the world's population and account for at least 92% of the world's disease burden. The rates for communicable conditions (including maternal and perinatal conditions, and malnutrition) and injuries are, respectively, thirteen and three times higher in low-income and middle-income countries than in high-income countries.

Group	Low/middle-income countries	High-income countries
Communicable diseases, maternal, perinatal, and nutritional disorders	11 206	863
Non-communicable diseases	10 200	9664
Injuries	4198	1403

Burden calculated as disease-adjusted life years per 100 000 population.

Rate of burden of disease by disease group and country income in 1998