Leading articles

Leptin and puberty

Our knowledge of the potential genetic link between fat metabolism and the reproductive axis dates back to the 1950s when the obese (*ob*/*ob*) mouse strain was first described. These mice were not only distinctly hyperphagic and rapidly developed obesity associated with hyperglycaemia and insulin resistance, but they were also infertile.[1] It was not until 1994 when the leptin (*ob*) gene was postionally cloned and a mutation was identified in the coding sequence of murine leptin in *ob*/*ob* mice that the cause of their obesity was recognised.[2] As predicted from the phenotype of ob/ob mice, leptin, which is principally expressed in adipocytes, had potent actions to suppress appetite and stimulate energy expenditure. It rapidly became clear that leptin could also influence the reproductive system. The sterility of male and female *ob*/*ob* mice could be reversed when recombinant leptin was administered.[3 4]

In humans, initial studies focused on the possible role that leptin may play in obesity. The hypothesis that leptin deficiency may contribute to common human obesity was soon rejected. An exponential relation between serum leptin concentration and body mass index or percentage body fat was described,[5] implying that, as a person became fatter, so insensitivity to the anorexigenic action of leptin developed. However, the relation between body fat and reproductive ability in humans has long been recognised. Both anorexia nervosa and intense physical training are associated with reduced gonadotrophin levels,[6] while Frisch had proposed that a certain amount of body fat must be accrued to achieve regular menstruation.[7] There is therefore a clear link between peripheral energy stores (in fat) and central regulation of physical development and reproductive capacity. Studies in animals and humans over the last four years have provided compelling evidence that leptin may be a neurohumoral mediator capable of signalling between the extent of nutritional intake and body fat store to the central nervous system.

Examination of the link between leptin and hypothalamic-pituitary-gonadal function has been undertaken mainly in the murine model. The relation in higher species is less well defined. In addition, the role that leptin may play in the onset of puberty, when the gonadotrophin releasing hormone (GnRH) pulse generator is being re-activated, has not been clarified. A review of data from the mouse, rat, monkey, and man and discussion of the possible neural networks involved will be presented.

The murine model

In mice, the relation between leptin and gonadal function has been tested in two situations: fasting and by direct administration of leptin. Normal mice when fasted not only develop low leptin levels as expected but also hypogonadotrophic hypogonadism, reduced thyroxine levels, and elevated adrenocorticotrophin (ACTH) and corticosterone.[8] These pituitary abnormalities could be partially reversed by leptin administered to fasted mice at a dose that returned their low leptin levels back to normal. Leptin can therefore be implicated in adaptation to starvation. Low leptin levels in times of food deprivation would result in an appetite drive, but a reduced reproductive capacity, protecting the female from conceiving and hence the energy demands of pregnancy. In addition, leptin administered to male and female ob/ob mice increased gonadotrophins (luteinising hormone in the female, follicle stimulating hormone in the male) and reproductive organ weights (ovary and uterus, testes and seminal vesicles).[9]

Intraperitoneal injections of leptin into normal mice in a dose that reduced appetite and hence body weight or in a smaller dose that had no effect on weight have been reported to bring forward the timing of normal puberty as defined by vaginal opening.[10 11] However, injections of leptin into normal prepubertal rats did not alter pubertal timing. In this model, the animals needed to be starved for an effect of leptin on puberty to be observed.[12 13] The time of first vaginal opening in leptin treated rats, with food intake reduced to 80% of normal, was similar to that in control ad libitum fed rats, with both having vaginal opening earlier than rats pair fed to the leptin group.[12] However, if the food intake in the leptin treated and pair fed control groups was restricted further down to 70% of normal, then leptin only partially reversed the delay in vaginal opening. This suggested that leptin plays a permissive rather than initiating role, allowing puberty to proceed only in favourable circumstances.

The rhesus macaque monkey

In the monkey, investigation of the role of leptin in puberty has been observational rather than interventional. In the male monkey, serum leptin levels have been measured throughout the juvenile period.[14] Leptin levels paralleled changes in testosterone, with high levels in infancy, a prepubertal nadir, then elevation through puberty. In a separate study in normal and castrated male monkeys, leptin levels were frequently monitored around the time of puberty.[15] No change in leptin concentration was found either before or during the time when luteinising hormone levels were increasing, indicating re-activation of the GnRH pulse generator and the initiation of puberty. This would imply that leptin was certainly not a trigger to puberty. Experiments in which leptin is administered to monkeys in order to assess its effect on the timing of puberty have, however, not been reported.

Human data

Data in childhood have been by necessity observational. Most studies have been cross sectional in design.[16–19] Many investigators have now reported that leptin increases gradually in both sexes over the prepubertal years. At each age, girls tend to have higher levels than boys. The leptin peak is reached at Tanner genital stage (G) 2–3 in boys, but in girls leptin continues to rise through puberty with a particular increase after menarche. In boys, leptin decreases back to early childhood levels by G5. Therefore, from late puberty and thereafter, there are strikingly discordant leptin levels between the sexes. Measures of body fatness (body mass index (BMI), BMI SD score, percentage body fat) are the most significant determinants of leptin through childhood. However, in both sexes before and during puberty (Tanner stages (TS) 1 and 2), age is a further independent determinant of leptin, implying that there is a maturational influence on leptin independent of body composition.[16] In the later stages of puberty (TS 3–5), age

remains a significant positive influence in girls, but, in boys, age is replaced by a negative effect related to increasing testicular volume. The latter is likely to reflect the inhibitory effect that testosterone has on leptin secretion. High affinity leptin binding activity, as measured by specific binding of ^{125}I-leptin in serum, varies considerably from birth through childhood. It is relatively low, at 5%, in cord blood of normal neonates, has risen to 18% at age 5, then decreases to 6% in both sexes[20] by completion of puberty. Leptin binding activity remains at the slightly higher level of 7.5% in normal adults, in whom its level does not fluctuate with age. This would suggest that leptin may become progressively more available to bind long form leptin receptors over childhood. It could then exert enhanced biological action over the period that a child is progressing towards and entering puberty.

All these data have provided further evidence that leptin has a permissive role in puberty rather than acting as a trigger. However, in one report in which leptin levels were assessed longitudinally in boys as they entered puberty, leptin appeared to show pronounced individual elevation just before the rise in testosterone.[21] This may imply a triggering role. However, other reports have not confirmed this observation.[22]

The most compelling evidence for a role for leptin in human puberty comes from those very rare families with deleterious mutations in either leptin or the leptin receptor.[23 24] In adulthood, homozygous subjects with either condition remain substantively, although not completely, hypogonadal. In a peripubertal child with leptin deficiency, treatment with leptin has led not only to pronounced effects on satiety and fat loss but also acute increases in nocturnal gonadotrophin secretion.[25] In addition, in boys with constitutional delay in growth and puberty (CDGP), a common disorder of the tempo of puberty, leptin levels at pubertal onset were lower than predicted for age and BMI.[26] In normal boys, an increase in leptin between G1 and G2 occurred as indicated above. However, leptin levels in prepubertal boys with constitutional delay in growth were not different from those in early puberty with CDGP. This suggested that the increase in leptin over the prepubertal years was not necessary to achieve puberty, but its absence was associated with a delay in entering puberty.

Leptin is clearly required for appropriate pubertal development and maintenance of secondary sexual characteristics. The combined murine and human data would infer that leptin has a permissive rather than triggering role in puberty.

Table 1 Examples of hypothalamic neurotransmitters implicated in the control of gonadotrophin releasing hormone (GnRH) neurones and/or in appetite regulation

Neurotransmitter	*GnRH regulation*	*Appetite regulation*
γ-Aminobutyric acid	Y (−)	Y (+)
Glutamate	Y (+)	Y (+)
Neuropeptide Y*	Y (− & +)	Y (+)
Galanin		Y (+)
Melanin concentrating hormone		Y (+)
Orexins		Y (+)
Leptin	Y (+)	Y (−)
CART*	Y (+)	Y (−)
Pro-opiomelanocortin*	Y (+)	Y (−)
Melanocyte stimulating hormone		Y (−)
Corticotrophin releasing hormone		Y (−)
Agouti related protein		Y (−)
Urocortin		Y (−)
Glucagon-like peptide-1		Y (−)

*Factors that may act to mediate the relation between leptin and GnRH secretion.
The sign in parentheses denotes activation/increase or inhibition/decrease.
CART, cocaine and amphetamine regulated transcript.

Leptin and hypothalamic control of GnRH

Circulating leptin is transported into the central nervous system to signal through long form receptors, located on cell bodies, such as neuropeptide Y (NPY) neurones, in the lateral hypothalamus. Many neurotransmitters and neural pathways in the hypothalamus are being linked to the control of appetite and hence body weight (table 1). Likewise, many neurotransmitters have been implicated in the control of GnRH neurones, and potentially in the control of the onset of puberty (table 1). Some of these factors affect both processes. In particular, NPY, a potent orexigenic factor and regulator of GnRH secretion, is thought to be a mediator of the central actions of leptin on appetite.[8] NPY has differing effects on GnRH secretion.[27] Both activation and inhibition have been described, depending on the model (fasted versus fed, acute versus chronic administration) and the age of the animal. In the fed and nourished state, NPY increases GnRH pulses, but in the undernourished state it inhibits GnRH neurones. In this situation, leptin levels will be low, reducing inhibition on NPY neurones. NPY levels will rise inhibiting GnRH secretion. It is not clear how these opposing actions of NPY on GnRH are mediated. Nevertheless, NPY can clearly act as a central link between nutrition and reproductive function, just as leptin fulfils this role as a peripheral factor.

Leptin also acts on cell bodies, which express cocaine and amphetamine regulated transcript (CART), another anorectic peptide. In in vitro experiments using retrochiasmatic hypothalamic explants of GnRH neurones from prepubertal female rats, leptin can stimulate CART expression, which in turn reduces the interval between pulses of GnRH secretion.[28] However, effects of NPY on GnRH pulse interval were not affected by antibodies to CART, implying that the leptin-CART pathway was independent of NPY. It is likely that a number of pathways that can link leptin through to GnRH neurones will be found.

Conclusion

There has been disappointment that leptin deficiency was not the answer to common obesity and that leptin treatment was unlikely to make a significant contribution to improving the health burden resulting from obesity. It appeared that serum leptin in adults was principally a marker of fat mass. In children, however, evidence is mounting that leptin has an important permissive role in the progression into puberty and the maintenance of normal hypothalamic-pituitary-gonadal function thereafter. The central networks in the hypothalamus that mediate this relation are complex and as yet not fully defined. Nevertheless there are neurotransmitters that impact on appetite and GnRH neurones. It will be important to understand these networks as the pharmaceutical drive to develop specific anorectic agents may have repercussions for pubertal and reproductive function.

At present the measurement of leptin in relation to puberty does not have a clinical application. However, further investigation of the exact relation between nutritional intake, body composition, growth, and development may be key to characterising mechanisms that control the tempo of growth.

P E CLAYTON
J A TRUEMAN
Academic Unit of Child Health and Endocrinology, University of Manchester, Royal Manchester Children's Hospital, Pendlebury, Manchester M27 4HA, UK
email: peter.clayton@man.ac.uk

1 Coleman DL. Obese and diabetes: two mutant genes causing diabetes-obesity syndromes in mice. *Diabetologia* 1978;**14**:141–8.
2 Zhang Y, Proenca R, Maffei M, Barone M, Leopole L, Friedman J. Positional cloning of the mouse obese gene and its human homologue. *Nature* 1994;**372**:425–32.

3 Chehab FF, Lim ME, Lu R. Correction of the sterility defect in homozygous obese female mice by treatment with the human recombinant leptin. *Nat Genet* 1996;**12**:318–20.
4 Mounzih K, Lu R, Chehab FF. Leptin treatment rescues the sterility of genetically obese ob/ob males. *Endocrinology* 1997;**138**:1190–3.
5 Considine RV, Sinha MK, Heiman ML, *et al*. Serum immunoreactive leptin concentrations in normal weight and obese humans. *N Engl J Med* 1996;**334**:292–5.
6 De Souza M, Metzger D. Reproductive dysfunction in amenorrheic athletes and anorexic patients: a review. *Med Sci Sports Exerc* 1991;**23**:995–1007.
7 Frisch RE, McArthur J. Menstrual cycles: fatness as a determinant of minimum weight for height necessary for the maintenance or onset. *Science* 1974;**185**:949–51.
8 Ahima RS, Prabakaran D, Mantzoros C, *et al*. Role of leptin in the neuroendocrine response to fasting. *Nature* 1996;**382**:250–2.
9 Barash I , Cheung CC, Weigle DS, *et al*. Leptin is a metabolic signal to the reproductive system. *Endocrinology* 1996;**173**:3144–7.
10 Ahima RS, Dushay J, Flier SN, Prabakaran D, Flier JS. Leptin accelerates the onset of puberty in normal female mice. *J Clin Invest* 1997;**99**:391–5.
11 Chehab FF, Mounzih K, Lu R, Lim ME. Early onset of reproductive function in normal female mice treated with leptin. *Science* 1997;**275**:88–90.
12 Cheung CC, Thornton JE, Kuijper JL, Weigle D, Clifton DK, Steiner RA. Leptin is a metabolic gate for the onset of puberty in the female rat. *Endocrinology* 1997;**138**:855–8.
13 Aubert ML, Pierroz DD, Gruaz NM, *et al*. Metabolic control of sexual function and growth: role of neuropeptide Y and leptin. *Mol Cell Endocrinol* 1998;**140**:107–13.
14 Urbanski HF, Francis Pau K-Y. A biphasic developmental pattern of circulating leptin in the male rhesus macaque (Macaca mulatta). *Endocrinology* 1998;**139**:2284–6.
15 Plant TM, Durrant AR. Circulating leptin does not appear to provide a signal for triggering the initiation of puberty in the male rhesus monkey (Macaca Mulatta). *Endocrinology* 1997;**138**:4505–8.
16 Clayton PE, Gill MS, Hall CM, Tillmann V, Whatmore AJ, Price DA. Serum leptin through childhood and adolescence. *Clin Endocrinol* 1997;**46**: 727–33.
17 Blum W , Englaro P, Hanitsch S, *et al*. Plasma leptin levels in healthy children and adolescents; dependence on body mass index, body fat mass, gender, pubertal stage and testosterone. *J Clin Endocrinol Metab* 1997;**82**: 2904–10.
18 Argente J, Barrios V, Chowen JA, Sinba MK, Considine RV. Leptin plasma levels in healthy Spanish children and adolescents, children with obesity and adolescents with anorexia nervosa and bulimia nervosa. *J Pediatr* 1997; **131**:833–8.
19 Garcia-Mayor R , Andrade MA, Rios M, Lage M, Dieguez C, Casanueva FF. Serum leptin levels in normal children: relationship to age, gender, body mass index, pituitary-gonadal hormones and pubertal stage. *J Clin Endocrinol Metab* 1997;**82**:2849–55.
20 Quinton N , Smith RF, Clayton PE, *et al*. Leptin binding activity changes with age: the link between leptin and puberty. *J Clin Endocrinol Metab* 1999;**84**:2336–41.
21 Mantzoros CS, Flier JS, Rogol AD. A longitudinal assessment of hormonal and physical alterations during normal puberty in boys. V. Rising leptin levels may signal the onset of puberty. *J Clin Endocrinol Metab* 1997;**82**:1066–70.
22 Ahmed ML, Ong KKL, Morrell DJ, *et al*. Longitudinal study of leptin concentrations during puberty: sex differences and relationship to changes in body composition. *J Clin Endocrinol Metab* 1999;**84**:899–905.
23 Strobel A, Issad T, Camoin L, Ozata M, Strosberg AD. A leptin missense mutation associated with hypogonadism and morbid obesity. *Nat Genet* 1998;**18**:213–15.
24 Clement K, Vaisse C, Lahlou N, *et al*. A mutation in the human leptin receptor gene causes obesity and pituitary dysfunction. *Nature* 1998;**392**: 398–401.
25 Farooqi IS, Jebb SA, Langmack G, *et al*. Effects of recombinant leptin therapy in a child with congenital leptin deficiency. *N Engl J Med* 1999;**341**:879–84.
26 Gill MS, Hall CM, Tillmann V, Clayton PE. Constitutional delay in growth and puberty (CDGP) is associated with hypoleptinaemia. *Clin Endocrinol* 1999;**50**:721–6.
27 Kalra SP, Kalra PS. Nutritional infertility: the role of the interconnected hypothalamic neuropeptide Y-galanin-opioid network. *Front Neuroendocrinol* 1996;**17**:371–401.
28 Lebrethon M-C, Gerard A, Vandersmissen E, Kristensen P, Bourguignon JP. Cocaine- and amphetamine-regulated transcript (CART) mediation of leptin stimulatory effect on the rat GnRH pulse generator. *Horm Res* 1999; **51**(suppl 2):O38.

Mental health must be "centre stage" in child welfare

The size of the problem

The alarmingly high incidence of behaviour and emotional disorders in children in the United Kingdom was established 30 years ago.[1] A minimum annual incidence of 5–10% for children living in relatively stable semirural communities and 10–20% for those in inner cities was found. Recently, an overall annual national incidence of 10% has been reported.[2] Authoritative reviews suggest that there has, if anything, been a rise in the incidence of at least some of these disorders over the past 40 years.[3] Illustrative case reports have shown that, among mainly undiagnosed, young, untreated children identified with psychiatric disorders in the community, even those with less serious levels of disturbance are suffering major impairment of social functioning.[4]

The community response

Until about five years ago, the official response to these striking epidemiological findings was disappointing. From 1970 to 1995, there was a slow increase in the number of consultant posts in child and adolescent psychiatry and in the number of training positions, but the need to improve services for mental health problems in the school setting, for children receiving paediatric care, and for children in contact with social services (especially those in public care), and for young offenders was not clearly recognised, and, insofar as it was, the measures taken to improve the situation were inadequate and sometimes, as in the case of young offenders, calculated to do more harm than good.

However, in the past five years there has been a very substantial change in the attitudes of central government, beginning with the previous administration and gathering pace under the present one. The degree to which the change in attitude will be reflected in willingness to bring about improvement in the relevant structures, tackle difficult interprofessional issues, and provide increased resources is unclear, but a definite and promising start has been made. Child mental health is a great deal nearer centre stage than it was five years ago.

In 1994 Zarrina Kurtz and her colleagues published what amounted to a consumer survey of child and adolescent mental health services (CAMHS).[5] The findings were, to put it mildly, not complimentary to the service. For example, 67% of hospital paediatricians stated "their local service was woefully inadequate, very limited, overwhelmed by referrals, barely adequate, or with enormous waiting lists". Community paediatricians, who reported that a substantial amount of their work was psychiatric in nature, were reported to feel even more strongly that CAMHS resources were quite inadequate. Among social services respondents, almost a half reported that the "service was virtually nil, inadequate or limited with long waiting times". Although the opinions of family doctors were not canvassed in this survey, it is improbable that the replies would have been substantially different. For the sake of balance, it should be added that, at the same time, surveys of attendees at child psychiatric clinics suggested a reasonably high level of satisfaction.[6] The problem appeared to be the inadequacy and uneven distribution of resources rather than the quality of the service received by those who did, in fact, receive it.

Kurtz *et al*[5] made a number of recommendations including the need for more sophisticated purchasing, greater use of child psychiatrists for consultation and liaison, regular audit, and a wider and more appropriate use of community child psychiatric nurses. Interestingly, in line with the philosophy of the time, they did not mention the need for increased resources.

In 1995 the Departments of Health and Education and the Social Services Inspectorate produced a *Handbook on child and adolescent mental health*.[7] This document introduced the concept of a tiered CAMHS, proposed earlier by P Hill (personal communication, 1999), with tier 1 providing primary care, tier 2 being represented by uniprofessional groups relating to others through a network, tier 3 providing a locally accessible specialist service, and tier 4 a more specialist service for children with unusual needs. In the same year, the NHS Executive commissioned a thorough *Health care needs assessment in child and adolescent mental health*,[8] and the health advisory service produced a substantial document entitled *Together we stand* giving guidance on the commissioning, role, and management of CAMHS.[9]

In 1997, the House of Commons select committee on health produced a *Report on child and adolescent mental health services*,[10] which noted that "the current provision of child and adolescent services is inadequate both in quality and in geographical spread", and supported the four tier model of services. In 1999, the Mental Health Foundation published the report of a committee of enquiry chaired by Tessa Baring, entitled *Bright futures: promoting children and young people's mental health*.[11] The title of this annotation is taken from that report. In the same year the Audit Commission, whose brief is to promote the best use of public money, came out with *Children in mind*,[12] a report on CAMHS which disclosed that there was as much as a sevenfold difference in CAMHS resources in different parts of England and Wales.

This volume of attention given to CAMHS is impressive, but, as Dr Seuss's cat in the hat[13] exclaimed, that is not all, that is not all! Various other relevant government initiatives and reports must be cited, including: Home Office legislation on young offenders setting up multiagency youth offending teams; Department for Education and Employment (DfEE) guidance on reducing the risk of disaffection among pupils by, for example, rewarding achievement, supporting behaviour management, and working with parents[14]; "Quality protects", a Department of Health programme setting standards for the care of children in public care as well as draft guidance from the same source on the multidisciplinary management of child abuse,[15] and a report from the Social Exclusion Unit on children excluded from school.[16]

The attention given to child mental health problems and CAMHS over the past five years is staggering. But what, if anything, has really happened to improve the lot of children suffering from behaviour and emotional disorders? What has occurred to help those in non-CAMHS professions, such as paediatricians, to deal more effectively with such children?

Here there is inevitably less positive information, but nevertheless it would be ungrateful to fail to note really noteworthy progress. The approach that the present administration has taken is largely, but not exclusively, to put extra resources into non-CAMHS services that can be seen as preventive in relation to the development of mental health problems.

Prevention: relevant government initiatives

None of the preventive initiatives that the government has set in train have been given a specific mental health "spin".

However, virtually all of them can be seen as having potential preventive possibilities in child mental health. They include:

- SureStart. This is by far the most substantial initiative. In introducing it, the ministers responsible stated "All the evidence shows that early intervention and support can help to reduce family breakdown; strengthen children's readiness for school; . . .Inside the home we want to offer support to enable parents to strengthen the bond with their children; outside it, we want to help families make the most of the local services on offer". The cost of the programme is £540m (£452m in England) over three years. The money will go to provide outreach services, support for families, and a range of other activities. Initial contact with families is expected to be made before birth and to continue for the first three years.
- The Family and Parenting Institute. This is a recently established body intended to fund research and publicise information about relationships and parenting. Part of its remit will be to identify what services should be available to families.
- Health Action Zones. This initiative provides extra resources for particularly deprived areas to enable new initiatives to be undertaken in areas that require a multiagency response. Many of the funded initiatives have involved children's services, especially those concerned with abuse and mental health problems.
- Education Action Zones. Again extra resources are provided for schools in deprived areas to enable them to improve support and other services, especially for children with special education needs. Many of these will be at risk of developing emotional and behaviour disorders, or will already have them.
- Literacy initiatives. These include the National Curriculum, the introduction of the literacy hour in primary schools, and other attempts to improve the teaching of reading, for example, by the publication of league tables.
- Personal, Social and Health Education (PSHE). The DfEE is promoting this component of the school curriculum. Although sadly it is not insisting that it forms part of the National Curriculum, nor providing additional resources for implementation, OFSTED inspections are now expected to cover an evaluation of the PHSE curriculum. This could go beyond the currently favoured topics of sex education, diet, smoking, and exercise to include the development of "emotional literacy". The "Healthy Schools Initiative", to which many schools are now signed up, requires that the school openly deals with "issues of emotional health and well-being by enabling students to understand what they are feeling . . ."[17] Children who are more "in touch" with their feelings may be readier to accept and understand that, when they are under stress, they may experience bodily sensations that are signs of emotional strain rather than physical illness, and that, if their family doctors are unable to identify a physical cause for their headaches and abdominal pain, there may be other useful explanations.

These initiatives have been generally welcomed, but have met with some significant reservations. Apart from SureStart, they have generally been introduced with an inadequate increase in resources. Even the generously funded SureStart has only been funded for three years, and there is concern that its budget will not be "built in" to the system after that time. The effectiveness of the initiatives has been queried. SureStart has been allocated a considerable sum for evaluation, but the other initiatives have not. It is unclear what evidence will be obtained to decide whether the new initiatives provide value for money. Some of the initiatives, especially those involving teachers, have been introduced without adequate concern for the sensitivities of the professionals concerned. Finally, as many of those in the field will be aware, there is increasing concern over the degree to which the activities undertaken by these initiatives are being coordinated, and little guidance to achieve joint working between them. At last, government is providing a better example of joint interdepartmental working. For example, the DfEE funded SureStart initiative is directed by a group based at the DfEE, but chaired by the minister of public health. However, there is often a different story at the local level. The need for local authority children's services plans has improved local joint working, but child health is still too often excluded from the planning processes. Most of the documents put out by the DfEE and many of those put out by the Home Office fail to mention the possible role of CAMHS in the assessment and management of intractable behaviour problems. Mental health may have come centre stage, but, to pursue the theatrical analogy, the play performed might be appropriately called *Six characters in search of an author*.

If, however, these initiatives are successful in improving parent-child relationships, in reducing educational underachievement, and in preventing the development of mild behavioural difficulties in school from escalating, then the rate of emotional and behaviour disorders should be lowered, and, of course, this would have an impact on the work of paediatricians. To suggest that it will have such an effect may seem overoptimistic, but there is a strong evidence base for thinking there will be a positive effect.

Treatment services: government initiatives

In February 1999, the government announced the allocation of an extra £84m over three years for child and adolescent mental health services. The extra resources are to be distributed between the NHS modernisation fund for authorities with pressing development needs in this field and initiatives promoting joint working between health trusts and local authority departments.

A number of other initiatives have been launched in areas that can be regarded as within the child and adolescent mental health fields. For example, the establishment of youth offending teams at community level will result in more coordinated arrangements for adolescents, most of whom, if seen by psychiatrists, would be regarded as showing conduct disorders. Drug action teams are expected to pay special attention to the young drug user. Mechanisms recommended for dealing with children excluded from school are intended to reduce the numbers of children who are excluded and increase the rate at which they can be returned to mainstream education. Although there is a deplorable lack of research in this area, it is known that many of these children are in the care of social services departments, and consequently it is certain that they suffer from high rates of conduct, attention deficit, and emotional disorders.

Implications for paediatricians

Few, if any, of these initiatives are directly targeted at the children who are of most concern to general hospital and community paediatricians. Yet the significance of child mental health and CAMHS for the work of paediatricians is well recognised and has been so for many years. The Diploma in Child Health has had a strong psychiatric input for about 15 years. The Royal College of Paediatrics and Child Health strategy document[18] makes specific reference to the need to "promote child and adolescent mental health by the development of specialist psychiatrist and

psychologist posts". It is not clear if the intention is to create a new type of psychiatrist or psychologist dedicated to paediatric liaison work, or to increase the input of existing specialists in the field. Whatever the mechanism, it is clearly desirable to increase the CAMHS input into paediatric services. How might this best be achieved?

The lesson from the allocation of extra resources announced in February 1999 is clearly that monies are most likely to be made available for initiatives that involve collaborative working. Paediatricians, through the College, might consider how they could use this approach for making bids relevant to child mental health. It could be proposed that monies are earmarked for initiatives that link CAMHS to paediatric facilities, especially where CAMHS is delivered through community mental health trusts. Because of their administrative separation, such trusts are likely to find it more difficult to provide good paediatric liaison and back up. The provision of evidence based CAMHS services using, for example, cognitive behavioural techniques for the non-organic pain syndromes so commonly encountered in paediatric practice, could be an attractive option. Such resources could provide increased support for new and existing joint initiatives, for example in the management of attention deficit hyperactivity disorder.

The training of paediatricians also requires further consideration. A survey of paediatricians recently appointed to consultant posts carried out a few years ago showed that the areas of greatest lack were in the management of child mental health problems and in experience of administration. Those involved in laying down the curriculum for specialist registrars in paediatrics and the paediatric specialties really do need to reconsider their priorities in the light of this information. Current attempts to improve links between the training of paediatricians and child and adolescent psychiatrists (D Cottrell, personal communication, 1999) need strong official backing if they are not to founder in the same way as previous attempts.

Mental health is already much more centre stage than could possibly have been envisaged only five years ago. The fact that paediatrics has so far been given such a small part in the unfolding drama should be seen as a challenge to the specialty and to its College.

PHILIP GRAHAM

Chair, National Children's Bureau, 8 Wakley Street, London EC1V 7QE, UK
email: philipgraham1@compuserve.com

1 Rutter M, Graham P. Epidemiology of psychiatric disorder. In: Rutter M, Tizard J, Whitmore K, eds. *Education, health and behaviour*. London: Longmans, 1970:178–201.
2 Meltzer H, Gatward R, Goodman R, *et al*. *Development and wellbeing of children and adolescents in Great Britain*. London: Stationery Office, 2000.
3 Rutter M, Smith DJ (eds). *Psychiatric disorders in young people*. Chichester: Wiley, 1995.
4 Richman N, Stevenson J, Graham P. *Pre-school to school: a behavioural study*. London: Academic Press, 1982.
5 Kurtz Z, Thornes R, Wolkind S. *Services for the mental health of children and young people: a national review*. London: Department of Public Health, South Thames RHA, 1994.
6 Moukaddem S, Fitzgerald M, Barry M. Evaluation of a child and family centre. *Child Psychology and Psychiatry Review* 1999;**3**:161–8.
7 Department of Health, Department for Education, Social Services Inspectorate. *A handbook on child and adolescent mental health*. London: Department of Health, 1995.
8 Wallace S, Crown J, Cox AD, *et al*. *Health care needs assessment: child and adolescent mental health*. Winchester: Wessex Institute of Public Health Medicine, 1995.
9 Health Advisory Service. *Together we stand: child and adolescent mental health services*. London: HMSO, 1995.
10 House of Commons Health Committee. *Child and adolescent mental health services*. 4th report. London: The Stationery Office, 1997.
11 Mental Health Foundation. *Bright futures: promoting children and young people's mental health*. London: Mental Health Foundation, 1999.
12 Audit Commission. *Children in mind: child and adolescent mental health services*. London: Audit Commission, 1999.
13 Dr Seuss. *The cat in the hat*. 40th anniversary ed. London: Harper-Collins, 1999.
14 Department for Education and Employment. *School inclusion: pupil support*. Circular no 10/99. London: Department for Education and Employment, 1999.
15 Department of Health. *Working together to safeguard children: new government proposals for inter-agency co-operation*. Consultation paper. London: Department of Health, 1998.
16 Social Exclusion Unit. *Truancy and school exclusion*. London: Stationery Office, 1998.
17 Department of Education and Employment. *National healthy school standard: guidance*. Annesley, Nottingham: DfEE Publications, 1999.
18 Royal College of Paediatrics and Child Health. *A children's health service*. London: Royal College of Paediatrics and Child Health, 1998.

Public health

Taking a population perspective on child health

Inferior doctors treat the patient's disease
Mediocre doctors treat the patient as a person
Superior doctors treat the community as a whole
Huang Lee, 2600 BC

We have long recognised that many health issues affecting children and their families cannot be addressed solely by health service workers. The major health gains of the last century have been determined by changes in life quality, sanitation, and living standards affecting whole populations.[1] However, for many years paediatricians working in both community and hospital systems have developed alliances with both statutory agencies (social services and education), government, and voluntary groups to plan and develop appropriate services. The child public health movement is growing in Europe, the USA, and Australia.[2 3] In the UK, a number of initiatives have been developed, including a special joint group of the British Association of Community Child Health and the Faculty of Public Health, an advocacy committee within the Royal College of Paediatrics and Child Health, and an advocacy internet based discussion group (CHANT). Paediatricians are fully participating in the health inequalities debate, both in their contribution to the research,[4] and in helping to influence wider health policy development.[5]

The purpose of this article is to explore the value of taking a population perspective on child health and placing paediatric care in context with the "total picture" of child health and illness. It is aimed at the general paediatrician. Other articles in the series explore whether there is a role for a specific "child public health" specialist.

In the UK, the majority of paediatric specialists work in a hospital setting, with their clinical activities usually being carried out in a ward or outpatient setting.[6] The service provided is one of a secondary care service, with referrals being made by local general practitioners (GPs), usually in the geographical area surrounding the hospital. This is in marked contrast to the European and North American system of paediatric care with its emphasis on the primary care paediatrician, often based in an "office" setting with or without local hospital admitting rights. Approximately 60% of GPs receive a formal postgraduate training in paediatrics with some partners in the practice taking a special interest in the subject. In the UK, GPs are widely considered to be the primary care providers of paediatric care. Consultant community paediatricians in the UK are also secondary care specialists who are often based in a geographical patch or locality,[8] working closely with local agencies, usually with a more social, educational, and developmental bias in their work.[9 10] The number of paediatricians in European countries varies from 18 to 120 per 100 000 population. In the UK it is approximately 20 per 100 000,[11] and this figure reflects the differences in models of service provision described above.

Much of the work of the acute paediatrician working in a hospital setting is reactive and depends very much on the prevailing epidemiology of childhood morbidity in the community and the skills and abilities of the main carers and primary care services in managing illnesses being presented. Hospital admissions and discharges have increased considerably in the past 10 years, with shorter stays and a higher turnover leading to ambulatory and short stay unit models of care being developed.[12] The workload relating to complex behavioural, social, and educational problems is also increasing. How should we react to these trends? I would argue that it is helpful to look at the problem from a population perspective using a number of public health tools. Firstly, this article describes a number of public health concepts including the pyramid of morbidity and types of disease prevention, and how they apply to paediatrics and child health. It goes on to explore what is available in terms of measuring disease and health status and how the data might be used to plan services, and to inform an education and research agenda for future paediatricians.

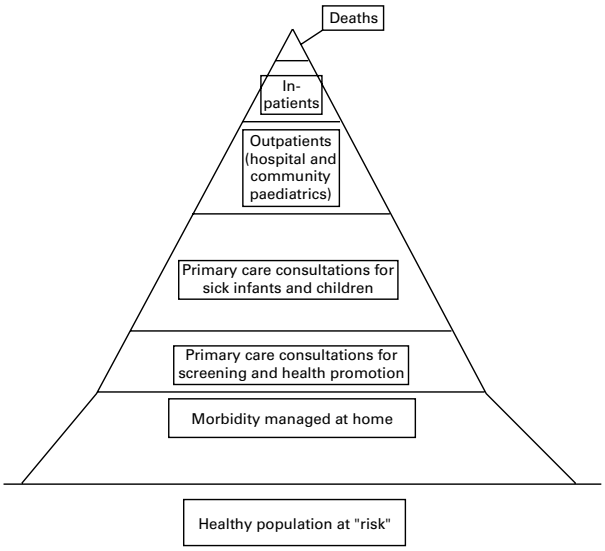

Figure 1 The pyramid of health and disease.

Pyramid of care

A useful concept used by public health physicians is the "pyramid" of disease (fig 1). If data are added to this pyramid, in terms of GP consultations and paediatric secondary care consultations in outpatients and inpatients, then it is easy to see that a very small increase in GP referral rates or an even smaller increase in primary care consultations, can easily overwhelm the capacity of the system further up the pyramid (table 1). Most paediatricians will appreciate how the annual bronchiolitis epidemic often overwhelms the system as described. This has led to a growing concern about patient demand management.[13] The nurse led telephone advice service, NHS Health Direct,[14] is one response to this. An understanding of this dynamic does place preventive care as a high priority, in an attempt to reduce the inexorable demand on secondary paediatric care services.

Types of prevention

Three forms of disease prevention are described.

Primary prevention—in which the bodily impairment, whether biochemical, physical, or psychological, is prevented from initiation; such as antenatal folate supplemen-

Table 1 General paediatric inpatient, outpatient, and GP consultations (age <16) in Nottingham (approximate figures for illustration), 1997 data

Deaths	80
Inpatient consultations	4000
Outpatient consultations	17 000 (9000 community; 8000 hospital)
General practice (preventive and treatment consultations)	300 000
Home based care	??

tation, childhood immunisation, fluoridation of drinking water, and road safety legislation.

Secondary prevention—in which the disease is detected at an early preclinical or clinical stage, and is reversed or ameliorated as a result; such as biochemical screening of the neonate, otoacoustic emmission screening for hearing loss, physical examination for cardiovascular disease, and structured questionnaires, for example, the Edinburgh postnatal depression screen.

Tertiary prevention—in which the impact of an established clinical disease is minimised; such as minimising handicap for the individual, for example, constructing a ramp in the playground for a wheelchair bound schoolchild, or teaching independence self catheterisation skills for an adolescent with spina bifida.

This framework has led to my considering every encounter with a patient as an opportunity for health promotion and preventive care, even in the most acute setting. The epidemiological ward round has been a success in the adult setting and could easily be transferred to a paediatric one.[15]

What problems affect my "community"?—describing the health status of the population

I am referring here to the local community within which we operate as paediatricians and from which we receive most of our referrals. This can be divided up into data collected centrally on a continuous basis, data collected by regular survey, and data collected by special survey. In addition there are district based and general practice caseload as well as local survey data. The General Registrar's Office was established over 150 years ago and led to the development of the Office of Population Census and Surveys (OPCS), and the more recent Office of National Statistics.[16] This organisation collects birth registration and death data as well as hospital admission data in the UK. The General Household Survey carries out a regular three yearly survey of households and collects data on childhood disability. The decennial UK wide census due in 2001 is a prime source of demographic data for accurate "denominator" data and will contain a question on long standing and life limiting illness. The GP sentinel practices collect morbidity data every 10 years. Data on infectious disease are regularly disseminated by the Centre for Disease Surveillance and provide a timely assessment of the patterns of disease in the community. A review of all these data sources is given elsewhere,[17 18] and a regular annual review of statistics is produced in this journal.[19]

Lifestyle surveys have been invaluable for collecting data on adolescent health (Trent).[20] District based child health systems have been in operation since 1975 and collect immunisation and paediatric screening data, including anthropometric data at key ages.[21] Approximately 70% of districts have such a system and although the quality of the data is variable, some valuable information on national child growth and screening has been collected.[22 23]

Can the data be used for planning services?

Increasingly paediatricians are having to make business cases for the services which they perceive are required and information from the sources above can be very helpful as part of that process. Unfortunately, NHS information strategies so far have not delivered the data required accurately or quickly enough, certainly in the secondary care sector. Child health professionals and others have worked hard to readdress this, with increased involvement in defining clinical terms and lobbying for enhanced system functionality and improved system communication.[24] Special needs registers can be used to help plan services for disabled children and their families.[25] They are probably best used as sampling frames for further detailed research on the needs of the disabled child population. Comparisons between districts are difficult because of differences in case eligibility, definitions, and recording. A core dataset has been developed by the Child Health Informatics Consortium, an umbrella organisation in the UK for child health information systems, with paediatric and primary care medical, nursing, and public health representation. This dataset is intended to act as an interagency baseline for describing the health of a district's children, to provide the basis for future trend data and some basic outcome data to ascertain the effectiveness of interagency interventions and programmes, for example, child health surveillance, Quality Protects.[27] It is becoming evident that outcome measures for services are needed in the area of child health, both from a health service evaluation perspective and also to help inform social care.[28] School entry still provides an opportunity for universal coverage of a screening or health status schedule, but it is not being fully utilised using evidence based validated tools. There is a real need to re-examine the data being collected at this age and review its utility for planning and epidemiological purposes.

Community profiling

Community nurses and others have used community profiling and rapid assessment methods to evaluate the needs of a local community and plan services responsively.[29] The utility of this approach is in effect to make a community diagnosis as opposed to an individual patient diagnosis, and put into place further investigations and interventions at the population level. A recent review of the health needs of the school aged child has highlighted the value of an annual report at school level with school nurse profiling as its basis, collated at school, district, and national level.[30] Interestingly, this idea has existed ever since the inception of the school health service in 1906 and is having resurgence at the end of the century!

Implications for education and research

Taking a population perspective is an important driver for the GMC recommendation of "Tomorrow's doctor". As Stone and Campbell point out: "the main challenge facing medical educationalists is one which the GMC may have underestimated: to provide students with practical preventive and health promotional skills that they can use in clinical settings, rather than merely theoretical knowledge of epidemiology and related fields".[31] As the acute paediatric workload intensifies in terms of greater numbers of admissions and shorter stays, it is easy to forget the public health perspective and the preventive perspective. As the "new" morbidities impact on our services, there is a need to take a broader view of how we can help children and families, and this is increasingly in a multidisciplinary context and across primary, secondary, hospital, and community interagency boundaries. Multidisciplinary education initiatives and training placements of paediatricians in primary and community secondary care settings can only help to build up the trust required between professionals in tackling these problems.

In response to the increasing demands made on the emergency acute services, a number of different service models are being explored in the UK and elsewhere, including ambulatory care and short stay observation units. Evaluation methods are developing alongside these, including the use of tools to assess appropriateness of admission,[32] and illness severity scoring methods[33]; we are beginning to learn more about the way in which parents and professionals deal with a child's illnesses,[34 35] and the patterns of morbidity in the population.[36] There is a great deal of potential for parent and professional education as a means of reducing demands on services. On a population level, work is progressing in defining functional outcomes in a number of areas, including mental health, against which population based interventions can be measured in future research programmes.[37 38]

Conclusion

Taking a population perspective on child health is a step in evolving our paediatric care and services in the millennium. An integrated well functioning, well trained paediatric and child health workforce should allow us to provide care for all children by the most appropriately trained person in the most appropriate location. Training and research needs to be informed by the needs of the community in its widest sense.

I would like to thank Mary Rossiter for the quote, Euan Ross for introducing me to the subject, and Leon Polnay for his long standing support.

M BLAIR

Senior Lecturer in Paediatrics and Child Health,
Department of Paediatrics,
Imperial College School of Medicine and Northwick Park Hospital (North West London Hospital NHS Trust),
Harrow, Middlesex HA1 3UJ, UK

1 McKeown T. *The role of medicine—dream, mirage or nemesis*. London: Nuffield Provincial Hospitals Trust, 1976.
2 Kohler L. Child public health—a new basis for child health workers. *European Journal of Public Health* 1998;**8**:253–5.
3 Waters E, Oberklaid F. Data collection in community child health. *Ambulatory Child Health* 1998;**3**:373–5.
4 Spencer N. *Poverty and child health*. Oxford: Radcliffe Medical Press, 1996.
5 RCPCH. *Annual report 1999–2000.* London: RCPCH.
6 RCPCH. *Manpower survey 1999*. London: RCPCH.
7 Polnay L, Pringle M. General practitioner training in paediatrics in the Trent region. *BMJ* 1989;**298**:1434–6.
8 Polnay L. Community paediatrics in the UK. *Ambulatory Child Health* 1996; **4**:407–14.
9 Blair M, Horn N, Polnay L. General practitioners use of hospital and community based paediatric out-patient services in Nottingham. *Public Health* 1997;**111**:97–100.
10 Blair M, Pullan CR, Rands CE, Crown N. Community paediatrics moves on—an analysis of changing work patterns 1994–1997. *Public Health* 2000; **114**:61–4.
11 UNESPA. *Proceedings of UNEPSA workshop on Primary Care Paediatrics*. Warsaw: UNESPA, 1997.
12 Heller DR. Ambulatory paediatrics: stepping out in a new direction? *Arch Dis Child* 1994;**70**:339–42.
13 Edwards N, Hensher M. Managing demand: managing demand for secondary care services: the changing context. *BMJ* 1998;**317**:135–8.
14 McLellan N. NHS Direct: here and now. *Arch Dis Child* 1999;**81**:376–9.
15 Stone DH. The clinical epidemiology ward round: can we teach public health medicine at the bedside? *J Public Health Med* 1998;**20**:377–81.
16 Nissel M. *People count. A history of the General Register Office*. London: HMSO, 1987.
17 Blair ME. Information. In: Polnay L, Hull D, eds. *Community paediatrics*, 2nd edition. London: Churchill Livingstone, 1993:73–94.
18 Dunnell K. Monitoring children's health. *Popul Trends* 1990;**60**:16–22.
19 Platt MJ. Child health statistics review, 1998. *Arch Dis Child* 1998;**79**:523–7.
20 Roberts H, Dengler R, Magowan R. *Young people's survey results 1992–1994.* Trent: Trent Health, NHS Executive Trent, 1995.
21 Ross E. Child health computing. *BMJ* 1991;**302**:727.
22 Chinn S, Hughes JM, Rona RJ. Trends in growth and obesity in ethnic groups in Britain. *Arch Dis Child* 1998;**78**:513–17.
23 Streetly A, Grant C, Pollitt RJ, Addison GM. Survey of scope of neonatal screening in the United Kingdom. *BMJ* 1995;**311**:726.
24 Blair M. Information strategy and its impact on child health. *Arch Dis Child* 1995;**72**:355–7.
25 Woodroffe C. Abra A. A special conditions register. *Arch Dis Child* 1991;**66**: 927–30.
26 Blair ME, Hutchison T. Special needs registers—dreams and nightmares. In: Spencer N, ed. *Progress in community child health 2*. London: Churchill Livingstone, 1997:57–67.
27 Child Health Informatics Consortium. *Monitoring the nation's health—key indicators and the underlying core data set*. In press.
28 Waters E, Wright M, Wake M, Landgraf J, Salmon L. Measuring the health and well-being of children and adolescents: a preliminary comparative evaluation of the Child Health Questionnaire in Australia. *Ambulatory Child Health* 1999;**5**:131–41.
29 Murray S. Experiences with "rapid appraisal" in primary care involving the public in assessing health needs, orientating staff, and educating medical students. *BMJ* 1999;**318**:440–4.
30 Polnay L, ed. *Health needs of the school age child—report of a Joint Working Party of the British Paediatric Association*. London: BPA, 1995.
31 Stone D, Campbell H. Child health promotion and its challenge to medical education. *BMJ* 1997;**315**:694–5.
32 Werneke U, Smith H, Smith IJ, Taylor J. MacFaul R. Validation of the paediatric appropriateness evaluation protocol in British practice. *Arch Dis Child* 1997;77:294–8.
33 Hewson PH, Gollan RA. A simple hospital triaging system for infants with acute illness. *J Paediatr Child Health* 1995;**31**:29–32.
34 Kai J. Parents' difficulties and information needs in coping with acute illness in preschool children: a qualitative study. *BMJ* 1996;**313**:987–90.
35 Stewart M, Werneke U, MacFaul R, Taylor-Meek J, Smith HE, Smith IJ. Medical and social factors associated with the admission and discharge of acutely ill children. *Arch Dis Child* 1998;**79**:219–24.
36 Spencer NJ, Coe C. The development and validation of a measure of parent-reported child health and morbidity: the Warwick Child Health and Morbidity Profile. *Child Care Health Dev* 1996;**22**:367–79.
37 Verrips E, Vogels T, Koopman H, *et al*. Measuring health-related quality of life in a child population. *European Journal of Public Health* 1999;**9**:188–93.
38 Waters E, Wake M, Toumbourou J, Wright M, Salmon L. Prevalence of emotional and physical health concerns amongst young people in Victoria. *J Paediatr Child Health* 1999;**35**:28–33.