

- 3 Peden A, McCardle L, Head MW, et al. Variant CJD infection in the spleen of a neurologically asymptomatic UK adult patient with haemophilia. *Haemophilia* 2010; **16**: 296–304.
- 4 National Blood Service, Scottish National Blood Transfusion Service, Welsh Blood Service, Northern Ireland Blood Transfusion Service, National CJD Surveillance Unit. Transfusion medicine epidemiology review (TMER). <http://www.cjd.ed.ac.uk/TMER/TMER.htm> (accessed Dec 24, 2010).
- 5 The National Creutzfeldt-Jakob Disease Surveillance Unit. Variant Creutzfeldt-Jakob disease: current data. December, 2010. <http://www.cjd.ed.ac.uk/vcjdworld.htm> (accessed Dec 24, 2010).
- 6 Edgeworth JA, Farmer M, Sicilia A, et al. Detection of prion infection in variant Creutzfeldt-Jakob disease: a blood-based assay. *Lancet* 2011; published online Feb 3. DOI:10.1016/S0140-6736(10)62308-2.
- 7 Zobeley E, Flechsig E, Cozzio A, Enari M, Weissmann C. Infectivity of scrapie prions bound to a stainless steel surface. *Mol Med* 1999; **5**: 240–43.
- 8 Edgeworth JA, Jackson GS, Clarke AR, Weissmann C, Collinge J. Highly sensitive, quantitative cell-based assay for prions adsorbed to solid surfaces. *Proc Natl Acad Sci USA* 2009; **106**: 3479–83.
- 9 Gregori L, Gray BN, Rose E, Spinner DS, Kacsak RJ, Rohwer RG. A sensitive and quantitative assay for normal PrP in plasma. *J Virol Methods* 2008; **149**: 251–59.
- 10 Chen B, Morales R, Barria MA, Soto C. Estimating prion concentration in fluids and tissues by quantitative PMCA. *Nat Methods* 2010; **7**: 519–20.
- 11 Brown P, Cervenakova L, Diringier H. Blood infectivity and the prospects for a diagnostic screening test in Creutzfeldt-Jakob disease. *J Lab Clin Med* 2001; **137**: 5–13.

Restricted elimination diet for ADHD: the INCA study

See **Articles** page 494

Psychopharmacological and psychosocial treatments are evidence-based treatments for attention-deficit hyperactivity disorder (ADHD). However, concerns about side-effects of psychoactive drugs, and barriers to access to and commitment needed for psychosocial treatments, often lead to consideration of other interventions.¹ One such intervention relates to the tenet that hypersensitivity or intolerance to foods or additives is a risk factor for ADHD.²

In *The Lancet*, Lidy Pelsser and colleagues³ report a two-phase randomised trial (INCA) with a control or a diet group in 100 children diagnosed with ADHD, who were aged 4–8 years and unselected for any food sensitivities. After a 2-week baseline period, controls were placed on a waiting list and continued normal eating, and their parents received healthy food advice and kept a diary of their child’s behaviour. The diet group received a 5-week open trial with a restricted

elimination diet of oligoantigenic few foods (rice, meat, vegetables, pears, water) complemented with specific foods such as potatoes, fruits, and wheat. Of the 41 diet-group children who completed phase 1, 17 (41.5%) had no behavioural response to the diet by the end of week 2 and their diet was further restricted to few foods only. At the end of phase 1, symptoms of ADHD and oppositional defiant disorder significantly improved in 64% children in the diet group compared with no improvement in the controls. Phase 1 clinical responders then had a double-blind crossover food challenge in random order with 2 weeks each of three high IgG and three low IgG foods added to the elimination diet or the few-foods diet. Selection of the high and low IgG foods was based on individual total IgG levels to 270 different foods. Relapse of ADHD symptoms occurred with the first, second, or both food challenges in 19 of the 30 children entering the crossover phase (phase 2). IgG levels against foods did not predict which foods might lead to a negative effect on behaviour because an equal number of low and high IgG food challenges resulted in relapse of ADHD symptoms.

Studies with restricted elimination diets are complex and challenging. Pelsser and colleagues’ study was well-designed and carefully done, showed benefit with a supervised elimination diet, and provides an additional treatment option for some young children with ADHD. The study also provides evidence against the benefit of using IgG blood levels (a common practice in complementary medicine) to determine which foods are triggering ADHD symptoms. However, it is important to note that 36% of children either did not respond to the elimination diet or were non-compliant in phase 1. Additionally, there were at least 16 other



Corbis

eligible children who were not motivated to enter the study. To help provide guidance to practitioners and families about appropriate options for their child, it will be helpful to know which children can be predicted to respond to the diet.

The blinded assessments in Pelsser and colleagues' study were based on information provided by parents. However, the parents and teachers were aware whether the children received the elimination diet or not in phase 1, and that the children entering phase 2 received the challenge foods (the only information the parents and teachers were blind to pertained to whether the challenge foods were low or high IgG foods). Therefore, in both the control and diet groups, the beliefs and expectations of parents and teachers about changes in the ADHD symptoms could have been influenced by this knowledge. Hence, it is important to use more objective measures for treatment outcomes in these investigations.

In phase 2, ADHD symptoms relapsed in 19 of 30 (63%) children in response to the food challenge. We do not know which of the six foods in the food challenge caused the hypersensitivity, nor whether some of the other 264 remaining foods might also cause hypersensitivity in the 19 children who relapsed and 11 who did not relapse in phase 2. To provide guidance to families and to avoid unnecessary dietary restrictions over long periods, identifying the incriminated foods is important. Also, Pelsser and colleagues reported only short-term benefit from the dietary restriction; however, maintenance of benefits over time and any long-term effects of dietary elimination on the child's nutritional status are unknown.

Feingold² first introduced the idea that many children are sensitive to dietary salicylates and artificially added food colours, flavours, and preservatives, and that eliminating the offending substances could ameliorate learning and behavioural problems, including ADHD. Population-based studies have reported behavioural sensitivity to artificial food colours and preservatives in children with or without ADHD.^{4,5} Food manufacturers are under increasing pressure from consumer groups and researchers to avoid these additives, to include a warning on the label about adverse effects on activity and attention of children, or both.⁶

Elimination diet studies suggest behavioural sensitivity to common salicylate and non-salicylate foods. Parents of children with ADHD should be made aware of

the research about behavioural sensitivity to common foods and additives in some children. For interested parents, a careful dietary elimination strategy can be implemented especially in younger children, because dietary elimination can be more practical and more effective in younger children because of better control of the diet by the caregiver.^{7,8} An elimination diet trial should be implemented only under the supervision of the child's primary health-care provider and a nutritionist to ensure that growing children do not suffer from nutritional deficiencies with the restricted diet.^{7,8} On the basis of parental preference, dietary elimination can be done by itself or with standard recommended treatments for ADHD.

Diagnosing food sensitivity is complex, can take several weeks, and can be burdensome for families to implement. The restricted diet can be tried for 2–5 weeks.^{3,8} If there is benefit, the restricted foods can be added back weekly, one food component at a time, to identify the problem foods to be excluded from a less restrictive permanent diet. In my opinion, a stringent elimination diet should not continue for more than 5 weeks without obvious benefit because of the time, effort, and resources required to implement the restricted diet and because long-term effects of dietary elimination on the child's nutritional status are not known.

To advance the field and provide clinical guidance to practitioners and parents, future studies should identify the specific incriminated foods responsible for the hypersensitivity reaction, include more objective and functional outcome measures, address predictors of response and non-response, address long-term effectiveness and tolerability of the dietary restriction, evaluate the nutritional composition of the elimination diet, investigate the impact of long-term dietary elimination on the child's nutritional status, and report on compliance, acceptance, and level of ease or difficulty in maintaining the dietary restriction.

Jaswinder Kaur Ghuman

Child and Adolescent Psychiatry, University of Arizona, Tucson, AZ 85724, USA
jkghuman@email.arizona.edu

I declare that I have no conflicts of interest.

1 Dulcan MK, Benson RS. AACAP official action: summary of the practice parameters for the assessment and treatment of children, adolescents, and adults with ADHD. *J Am Acad Child Adolesc Psychiatry* 1997; **36**: 1311–17.

- 2 Feingold BF. Hyperkinesis and learning disabilities linked to artificial food flavors and colors. *Am J Nurs* 1975; **75**: 797–803.
- 3 Pelsser L, Frankena K, Toorman J, et al. Effect of a restricted elimination diet on the behaviour of children with attention-deficit hyperactivity disorder (INCA study): a randomised controlled trial. *Lancet* 2011; **377**: 494–503.
- 4 Bateman B, Warner JO, Hutchinson E, et al. The effects of a double blind, placebo controlled, artificial food colourings and benzoate preservative challenge on hyperactivity in a general population sample of preschool children. *Arch Dis Child* 2004; **89**: 506–11.
- 5 McCann D, Barrett A, Cooper A, et al. Food additives and hyperactive behaviour in 3-year-old and 8/9-year-old children in the community: a randomised, double-blinded, placebo-controlled trial. *Lancet* 2007; **370**: 1560–67.
- 6 European Parliament and Council of the European Union. Regulation (EC) No 1333/2008 of the European Parliament and of the Council of 16 December 2008 on food additives. Dec 31, 2008. <http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2008:354:0016:0033:en:PDF> (accessed Jan 29, 2011).
- 7 Ghuman J, Arnold L, Anthony B. Psychopharmacological and other treatments in preschool children with attention-deficit/hyperactivity disorder: current evidence and practice. *J Child Adolesc Psychopharmacol* 2008; **18**: 413–47.
- 8 Stevens L, Kuczek T, Burgess J, Hurt E, Arnold L. Dietary sensitivities and ADHD symptoms: thirty-five years of research. *Clin Pediatr (Phila)* 2010; published online Dec 2. DOI:s10.1177/0009922810384728.

Universal health care in India: the time is right

Published Online
January 12, 2011
DOI:10.1016/S0140-6736(10)62044-2
See Series page 505
See Series *Lancet* 2011; **377**: 252, 332, and 413
See Online/Series
DOI:10.1016/S0140-6736(10)61894-6,
DOI:10.1016/S0140-6736(10)61888-0,
DOI:10.1016/S0140-6736(10)61884-3, and
DOI:10.1016/S0140-6736(10)61960-5

India has supported the ideal of health for all since it become an independent nation more than 60 years ago. The Bhore Committee report¹ in 1946 recommended a national health system for delivery of comprehensive preventive and curative allopathic services through a rural-focused multilevel public system, financed by the government, through which all citizens would receive care irrespective of their ability to pay. However, a newly independent India faced monumental challenges in 1947. The country had been divided by a bloody partition, poverty was widespread, the economy was weak, and the administrators were new. The population's health was grim. Memories of the Bengal famine of 1943, which killed 2–3 million people, were still fresh, health services were concentrated in urban areas, and health indicators were universally poor with a life expectancy at birth of 37 years. Much progress has been recorded since then. Life expectancy is greater than 60 years, and the

India of 2011 is a thriving democracy with a diversified production base, a large scientific community, and an impressive information technology sector.

During the same period, however, India's record in expanding social opportunities has been uneven. The health and nutritional status of children and women remains poor, and India is routinely ranked among countries performing weakly on overall health performance.^{2,3} But there is good reason for hope. The country has withstood the recent global financial crisis and quickly returned to rapid economic growth. There is a refreshing openness to participation by civil society and to the power of ideas to improve performance and governance. We are enthused by India's recent commitments to invigorate the public health-care system to address health disparities. Furthermore, we are encouraged by a vibrant economic climate that has propelled the nation into the ranks of middle-income countries, and by the advocacy for health from civil society organisations that speak for people. The growing confidence manifest in bold social-policy initiatives (such as the Right to Information Act of 2005, the Right to Free and Compulsory Education Act of 2009, and the proposed Right to Health Bill) offers an opportunity to revisit the case for universal health care.

Unsurprisingly, this Series in *The Lancet* shows both achievements and failures in health. India has one of the most fragmented and commercialised health-care systems in the world, where world-class care is greatly outweighed by unregulated poor-quality health services. Because public spending on health has remained low, private out-of-pocket expenditures on health are among the highest in the world.⁴ Health care,

The printed journal includes an image merely for illustration