muscular atrophy are allelic mutations at the *SMN* locus, as suggested by Brahe and colleagues. Thus, all patients should be tested for *SMN* gene deletion. In addition, the search for intragenic mutations should be carried out in non-deleted typical type IV cases, as reported by Zerres and co-workers. Indeed, patients retaining this gene carry small deletions of consensus splice sites, point mutations, or frame-shift deletions.[1,2] This observation also emphasises the variable clinical expression of *SMN* gene deletions, which causes a continuum of clinical phenotypes ranging from early-onset severe form (type I, Werdnig-Hoffmann disease) to the mildest adult form of the disease (type IV).

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Melanin, melanocytes, and melanoma

SIR—Balding professors of dermatology know that hair provides nature's most effective sunscreen. The evolutionary counterpart of this familiar observation was the extension of the follicular melanocyte's role to provide interfollicular protection against ultraviolet radiation (UVR). Studies showing low melanoma rates in albinos living in areas such as equatorial Africa merit thought.[1] Albino blacks are particularly sensitive to the effects of UVR and show an increased frequency of sunburn, actinic damage, and basal-cell and squamous-cell carcinomas compared with non-albinos, yet they have a low incidence of melanoma.[1] There are two points. Firstly, in the absence of (black type) pigment, the major selective drive for pigmentation comes from death and illness from sunburning and squamous-cell malignancy, not melanoma; after all there is little virtue in developing interfollicular pigmentation if the sole purpose is to protect the melanocytes themselves. Secondly, in black skin, melanocytes without melanin appear much more resistant than keratinocytes to malignant transformation. In white populations living in areas with relatively low UVR exposure, the situation has changed. Low absolute UVR levels relax the drive for effective sun protection, and other selective pressures such as vitamin-D deficiency due to lack of UVR may become important.[2] But why, if in black African populations, melanocytes without pigment are so resistant to UVR, are melanocytes so sensitive to such (relatively) low UVR exposure in northern European populations?

The answer may lie in the type of pigmentation and in particular the type of melanin. The argument for the red-hair and skin type-1 phenotype (which we have recently shown to be associated with variants of the melanocyte stimulating hormone receptor),[3] is that the elevated melanoma risk is not just due to lack of photoprotection, but must reflect some of the characteristics of the melanocyte or type of melanin. If this were not the case we would expect melanoma rates, at least in terms relative to UVR exposure, to be as low as in albinos in Africa. The argument to a lesser degree must also hold for those without red hair. In high-UVR areas melanoma is a trade off for protection against squamous cancer, whereas by contrast, in low-UVR areas evolutionary pressures must have driven, or reduced rates of squamous malignancy allowed to pass by, the development of multiple variant melanocyte stimulating hormone-receptor alleles and a type of pigmentary system that is inherently more prone to melanoma. It is also relevant to this argument that although sunburn has been associated with melanoma,[4] sunburn per se would not appear to be a causative factor because albinos, who experience frequent episodes of sunburn, do not show a markedly increased risk of melanoma. Rather sunburn, which is caused mainly by UVB radiation could merely be a marker of high exposure to other regions of the solar spectrum such as UVA or visible light.

If melanin is the primary chromophore and the wavelengths responsible for melanoma induction are allied to the absorption spectrum of melanin, what are the consequences? Firstly, because the UVA component of sunlight is about 20 times the UVB component but melanin absorbs UVB only slightly more than UVA, solar UVB would contribute only about 10% towards the induction of melanoma and any changes in stratospheric ozone (which only affects the terrestrial UVB flux) would have a negligible effect on melanoma incidence. Secondly, people who used sunscreens containing mainly UVB-absorbing ingredients and spend longer in the sun because of the reduced risk of sunburn, would increase their UVA dose and possibly their risk of melanoma. Might this be a contributory factor to the observation from some case-control studies[5] that sunscreen use appears to be a risk factor for melanoma?

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1 Lookingbill DP, Lookingbill GL, Leppard B. Actinic damage and skin cancer in albinos in northern Tanzania: findings in 164 patients enrolled in an outreach skin care program. *J Am Acad Dermatol* 1995; **32:** 653–58.

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Influenza types and patient population

SIR—Watson and colleagues (Aug 19, p 515) have commented on our report on influenza types (July 15, p 180). In the 1994/95 influenza season the Netherlands National Influenza Centre had observed that among specimens from patients participating in a sentinel network of general practitioners the ratio between influenza B and influenza A(H3N2) virus isolates was 93/34 (73% type B) and among clinical samples examined in hospital-based diagnostic laboratories it was 29/55 (35% type B). Watson and colleagues could not confirm this with data for England and Wales for the same season, suggesting that other factors played a part in the differences we described. The ratio for England and Wales was 46/1 (98% type B) for isolates derived from the sentinel surveillance and 281/12 (96% type B) among hospital laboratory-derived isolates.

We do not see a contradiction between Watson's data and ours. In 1994/95 in the Netherlands, but not in England and Wales, significant numbers of influenza A(H3N2) viruses were isolated so only the Dutch data permit a conclusion to be drawn about the different proportions of type A and type B viruses isolated in the two patient populations.

Watson and colleagues may be right in their supposition that hospital-based diagnostic laboratories also examine specimens for GPs. However, this would imply that among Dutch hospital patients influenza B was even less frequent than our data suggest, strengthening the message we tried to convey. We do agree that both surveillance systems provide valuable information on the nature of the influenza viruses circulating in the community.

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Breastfeeding as prophylaxis against atopic disease

SIR—Saarinen and Kajosaari (Oct 21, p 1065) suggest that breastfeeding for longer than 1 month about halves the risk of respiratory allergy at 17 years of age. We have followed up a national cohort of British adults born in 1958.[1] All cohort members who had been contacted at ages 7, 11, 16, 23, and 33 and who had a lifetime history of asthma, wheezy bronchitis, or wheezing reported at any of these follow-ups were included. A 20% sample of cohort members with complete follow-up data but no history of wheezing illness at any age were also included. Skin prick reactions to *Dermatophagoides pteronyssinus*, mixed grass pollen, and cat fur were measured at age 34–35 years on 1400 subjects for whom a history of infant feeding had been ascertained from parents. Subjects with any detectable weal to one or more allergen (after 10 min) were classified as atopic.

Our table shows the relation of atopy and prospectively ascertained wheezing history with duration of breastfeeding. Comparison of past and present wheezers with the control group who had never wheezed showed that the proportions who were breastfed for at least 1 month were very similar. The odds ratios (with 95% CI) associated with breastfeeding for at least 1 month were 0·94 (0·71–1·23) for past wheeze versus controls, and 1·00 (0·76–1·32) for current wheeze versus controls. The corresponding odds ratio comparing atopic with non-atopic individuals was 1·12 (0·91–1·39).

These findings do not exclude a small protective effect of prolonged breastfeeding on the development of respiratory allergy, but they seem to be more consistent with a modest increase in risk of allergic sensitisation among breastfed infants, as shown (at the 5% level of significance) for self-reported hay fever at age 23 in the whole cohort.[2] Selective perseverance with breastfeeding by atopic mothers is unlikely to have affected these results, as the cohort were born some years before the suggestion that breastfeeding might protect against allergic disease.

There are many other reasons to promote breastfeeding, but we conclude that it is unlikely that it offers substantial protection against subsequent allergy, as suggested by Saarinen and Kajosaari.

History of asthma or wheezing*	Atopic†	Non-atopic	Total
None	46 (58/126)	48 (102/214)	47 (160/340)
Past	46 (122/263)	45 (131/294)	45 (253/557)
Current	50 (171/345)	42 (66/158)	47 (237/503)
Total	48 (351/734)	45 (299/666)	46 (650/1400)

*Lifetime history of asthma or wheezy bronchitis reported by parents at age 7, 11, or 16; self-report of asthma or wheezy bronchitis at ages 16–23; self-report of asthma or wheezing by age 34–35. Subjects reporting wheeze in the year before examination are included in current group.
†Skin prick reactivity (≥1 mm weal diameter) to *D pteronyssinus*, mixed grass pollen, or cat fur, tested at age 34–35.

Table: **Percentage (numbers) of subjects in the British 1958 birth cohort breastfed for at least 1 month, by history of asthma or wheezing, and atopy**

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Authors' reply

SIR—Strachan and colleagues conclude that breastfeeding is unlikely to offer substantial protection against subsequent allergy, on the basis of findings for individuals with none, past, and current wheezing, and those with and without skin prick reactivity, among whom the numbers breast-fed for 1 month or longer did not differ. However, there are many difficulties in the interpretation of their data. Their original design was not to conduct a breastfeeding study, and consequently the data cannot be considered reliable. Also, several major factors influencing the development of atopic disease were not included in the evaluation. We make the following points.

The history of infant feeding was taken by Strachan and colleagues retrospectively from the parents as late as the age of 7 years, so that the duration of breastfeeding may—at best—be an arbitrary estimate (in our experience mothers tend to remember the exclusive breastfeeding period quite inaccurately). To be protective, breastfeeding needs to be exclusive, with no dairy-milk-based supplements added. No information was given about supplemental milk (which could not possibly be obtained retrospectively at age 7). No information was given about early solid-food intake. The diagnosis was based on self-reporting or reporting by parents, being divided into none, past, or current wheezing; this is inaccurate, although understandable, in a large cohort. Eczema and food allergy were not included, so their analysis did not cover the broad picture of atopic disease. Atopic heredity, a major factor, was not taken into account. Finally, the prick tests were performed as late as age 35, leaving the possibility that part of the prick positivity had faded away with age.

The study design of Strachan and co-workers may well serve other—eg, epidemiological—purposes but it is certainly not convincing regarding early, exclusive human milk feeding.

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Fatal air embolism resulting from gastroscopy

SIR—A 56-year-old man had had abdominal pain for 14 days and was admitted for gastroscopy. 13 years previously he had a perforated gastric ulcer that was surgically treated. He was otherwise healthy. Sonography of the upper abdominal region was normal.

The patient was premedicated with midazolam intravenously. Lidocaine was used to anaesthetise the pharynx A gastroscope was inserted into the stomach without difficulty. Diffuse reddening of the gastric mucosa as well as a rather tight pyloric canal was observed. The lining of the stomach bled on contact with the gastroscope. During examination of the pyloric canal a marked lividity of the mucosa was seen. The patient became suddenly restless and a cardiac arrest occurred. Cardiopulmonary resuscitation was started immediately but was unsuccessful. On