Recipe for safer sauces

SIR—Contamination of intact chicken eggs by *Salmonella enteritidis* has led to a notable rise in food poisoning in the West¹⁻⁴. Health authorities have named home-made mayonnaise, hollandaise and béarnaise sauces as dishes that pose some hazard⁵. Mayonnaise, prepared by emulsifying vegetable oil in raw egg yolks, has been implicated in several outbreaks of salmonellosis, including one at the House of Lords⁶. The acid ingredients in this sauce can eliminate salmonellae from raw yolks, but only over a period of hours to days⁷. Hollandaise and béarnaise sauces, butterfat-in-water emulsions thickened with lightly cooked yolks⁸, are usually heated briefly to 70 or 75 °C, but some salmonellae from an initially large population may survive this treatment^{9,10}. Egg yolk seems to increase the resistance of salmonellae to thermal killing¹¹.

The standard kitchen method for minimizing microbial contamination, thorough boiling, has not been considered applicable to emulsified sauces. Egg yolks harden and emulsions break well below 100 °C, so cooks never intentionally boil either the yolks alone or a finished sauce. But a sufficiently low pH can delay or prevent the heat coagulation of egg proteins^{8,12}. Under suitable biochemical and thermal conditions, egg yolks can be boiled without curdling them or fatally compromising their ability to produce smooth, stable emulsions.

The method is as follows. Raw egg yolks are mixed thoroughly with an equal volume of water and from one-third to an equal volume of lemon juice or vinegar. This mixture is then placed in a small glass bowl, covered and irradiated in a microwave oven at maximum power until it bubbles: for 1- and 2-yolk mixtures, 1 minute or less, depending on oven power. The unevenly heated mixture is then beaten with a fresh implement and cooked a second time until it has bubbled for 5 to 10 seconds. Beaten with another fresh implement as it cools down, the yolk mixture retains the consistency of a stirred custard. I find that this mixture can then be used to make an acceptable if slightly more heat-sensitive hollandaise or béarnaise sauce or, as long as unrefined olive oil is only a small fraction of the total oil volume, a stable mayonnaise.

To test the effectiveness of this method of eliminating large bacterial populations, a culture of *S. enteritidis* strain 1601E, phage type 4 (B. A. D. Stocker, Stanford University), grown in broth with shaking for 18 hours at 37 °C, was inoculated into egg yolks at about 5×10^6 colony-forming units per millilitre of yolk. The yolks, initial pH 6.2, were then mixed with water and one-third their volume of either lemon juice or vinegar; the final pH of the

mixtures were 4.0 and 4.8, respectively. After an initial sample was removed, the yolk mixtures were treated as above in a Quasar microwave oven rated at 600 W, and sampled again. Bacteria were estimated by dispersing yolk samples in saline and plating dilutions on standard media. Recovery of colony-forming units from the yolks sampled before heating was consistent with the known inoculum. No live bacteria were detected in samples taken after the second heating period. The surviving population, if any, thus was probably smaller than 5 cells per millilitre of yolk.

These results suggest that cooks can greatly reduce the health risk posed by yolk-based sauces. If public-health laboratories would verify that the combination of acidification and rapid boiling reliably eliminates salmonellae from yolks, then this method could provide a more palatable alternative to abstention from freshly made mayonnaise, hollandaise and béarnaise sauces⁵.

HAROLD MCGEE

838 La Jennifer Way,
Palo Alto, California 94306, USA

1. *Lancet* **ii**, 720–722 (1988).
2. Perales, I. & Audicana, A. *Lancet* **ii**, 1133 (1988).
3. Rodrigue, D. C., Tauxe, R. V. & Rowe, B. *Epidem. Inf.* **105**, 21–27 (1990).
4. St Louis, M. E. *et al.* *J. Am. med. Ass.* **259**, 2103–2107 (1988).
5. US Centers for Disease Control Morbidity Mortality Weekly Rep. **38**, 877–880 (1990).
6. Glynn, A. A. *Nature* **339**, 671–672 (1989).
7. Smittle, R. B. *J. Food Protect.* **40**, 415–422 (1977).
8. McGee, H. *The Curious Cook* (North Point, San Francisco, in the press).
9. Baker, R. C., Hogarty, S., Poon, W. & Vadehra, D. V. *Poultry Sci.* **62**, 1211–1216 (1983).
10. Humphrey, T. J., Greenwood, M., Gilbert, R. J., Rowe, B. & Chapman, P. A. *Epidem. Inf.* **103**, 35–45 (1989).
11. Humphrey, T. J., Chapman, P. A., Rowe, B. & Gilbert, R. J. *Epidem. Inf.* **104**, 237–241 (1990).
12. Small, D. M. & Bernstein, M. *New Engl. J. Med.* **300**, 801–802 (1979).

Radiation and Down's syndrome

SIR—Recent epidemiological evidence¹ reported in *Nature*² has suggested that paternal occupational exposure to repeated low doses of ionizing radiations may be responsible for a tenfold excess of leukaemia in children. It has been pointed out³ that, according to such an explanation, the postulated inheritable lesions determining leukaemia would be induced at a high rate, while no other genetic effect has been noticed in the progeny. Therefore, detection of those lesions in the exposed parents and in the progeny should provide the crucial epidemiological evidence and eventually some clue concerning their peculiarly high frequency and/or heritability. Otherwise, parental exposure to ionizing radiation might also be considered as an indicator of children's

exposure to the same or to another leukaemogenic factor.

In a similar context, we have evaluated⁴ the effect of cumulative parental exposure to abdominal diagnostic X-rays on non-disjunction of chromosome 21, as detected by a trisomic conceptus (Down's syndrome). Nondisjunctional parents have been defined as cases and their partners as controls, avoiding recall bias by applying an obvious double-blind interview concerning exposure. A significantly increased risk for nondisjunction has been detected in exposed fathers which, not surprisingly, is correlated with age. Results observed in exposed mothers are consistent with a bifactorial aetiology, ageing and exposure to ionizing radiation, acting according to an additive model.

PAOLO STRIGINI

RAFFAELE SANSONE

SERGIO CAROBBI

Section of Epidemiological Genetics
& Eco-Oncogenetics,
Ist. Naz. Ricerca sul Cancro,
Viale Benedetto XV, 10,
Genova 16132, Italy

MAURO PIERLUIGI

Regional Centres for Human Genetics
and Down's Syndrome Study,
Galliera Hospital,
Via A. Volta 6, Genova 16121, Italy

1. Gardner, M. J. *et al.* *Br. med. J.* **300**, 423–429 (1990).
2. Aldhous, P. *Nature* **343**, 679 (1990).
3. Evans, H. J. *6th Int. Clin. Genet. Sem.* (Corfu, May 1990).
4. Strigini, P. *et al.* *Am. J. med. Genet.* (in the press).

New tricks for old dogmas?

SIR—Gehring *et al.* suggested¹ a possible role of cytoplasmic calcium ($[Ca^{2+}]_{cyt}$) in higher plant tropisms. In our opinion, some physiological aspects of this work were less than ideal. Furthermore, the authors seemed to be guided in the interpretation of their results by some old dogmas, rather than considering contemporary work.

None of the cellular responses reported by the authors can be unambiguously linked to a tropic response. In the case of the phototropism experiments, white light was used and no evidence was presented that the changes in $[Ca^{2+}]_{cyt}$ were not the result of red or blue light acting non-phototropically (both known to occur in coleoptiles). In the case of the gravitropism experiments, the unilateral blue illumination to the lower side (due to the laser used as part of the measurement technique) could have produced a physiological response. One reason for questioning the relationship of the observed changes in $[Ca^{2+}]_{cyt}$ to any tropic response is the fact that the main changes in $[Ca^{2+}]_{cyt}$ observed after unilateral white-light treatment were at the shaded side, with no changes reported at the illuminated side. This is