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A child with *Salmonella enterica* serotype Paratyphi B infection acquired from a fish tank

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TO THE EDITOR: Keeping fish as pets, as with other animals, carries the risk of zoonotic infections. A 2000 review of bacterial zoonoses that can be acquired topically from fish commented on infection with *Aeromonas hydrophila*, *Edwardsiella tarda*, *Erysipelothrix rhusiopathiae*, *Mycobacterium marinum*, *Streptococcus iniae*, *Vibrio vulnificus* and *Vibrio damsela*.¹ Our public health unit was recently notified of a case of *Salmonella* infection acquired from a fish tank.

In October 2003, a 14-month-old boy was admitted to hospital with a 2-day history of fever, vomiting and diarrhoea. Culture of three stool specimens revealed a *Salmonella* isolate. This was identified by serotyping (at the Institute of Medical and Veterinary Science, Adelaide, South Australia) and phage typing (at the Microbiological Diagnostic Unit, Melbourne, Victoria) as *Salmonella enterica* serotype Paratyphi B var Java phage type Dundee.

The child had no recent history of overseas travel or overseas visitors. Other family members were well, and their stool specimens were negative for *Salmonella* spp. However, the family kept a tropical fish tank, and the child's parents reported that he would place his hands in the water to help feed the fish. Culture of water from the tank revealed a *Salmonella* isolate identical to that in the stool specimens.

We believe that the boy became infected after touching the water while feeding the fish. The fish tank contained red-eyed tetras, bala sharks, silver dollars and angel fish, which had been purchased from three local aquariums several years previously. None appeared sick at the time of the child's illness.

It is important for clinicians to recognise that *Salmonella* infections are not always foodborne in origin. Salmonellosis is a well-known zoonosis that can be found in a variety of pets, including cats, dogs, birds, rodents and even reptiles.² In fact, an estimated 90% of all reptiles shed *Salmonella* spp. in their faeces.³ *Salmonella* spp. have been isolated from tropical fish aquariums previously; unusual *Salmonella* serotypes were found in eight of 100 tropical aquariums sampled in Wales.⁴ Fish can excrete *Salmonella* without appearing unwell.⁵

Our patient was a 14-month-old child. In a Canadian outbreak of *S. enterica* serotype Paratyphi B linked to aquariums, five of seven cases were also in children aged under 10 years.⁵ This may reflect a combination of immature immunity and behaviour — young children may not wash their hands properly (or at all) before eating or touching their mouths.

This case highlights the importance of good handwashing at all times after contact with an aquarium, regardless of the appearance of the fish. It may also be worthwhile recommending close supervision of children under 5 years of age around aquariums.

Acknowledgements: We wish to acknowledge laboratory staff at the Division of Analytical Laboratories, Sydney, NSW. SNS is funded by the Master of Applied Epidemiology Program through the Australian Department of Health and Ageing.

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Inappropriate use of food quality standards for seafood-derived complementary medicines

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TO THE EDITOR: Seafood is not only consumed as food, but also as dietary supplements and complementary medicines. Examples are capsules of freeze-dried oysters and mussels, or freeze-dried extract of shellfish meat, sold as reputed antihypertensives, cardioprotectants, and anti-inflammatories, among other medical claims. However, oysters and mussels can become dangerously toxic after they ingest poisonous microscopic algae. If these molluscs are sold as food in Australia, they are subject to the Food Standards Code,¹ under which their sale is prohibited if biotoxin levels per kilogram of wet shellfish meat exceed 800 µg of paralytic shellfish poisons, 200 mouse units of neurotoxic shellfish poisons, 200 µg of diarrhoeic shellfish poisons, or 20 mg of amnesic shellfish poisons.

Shellfish capsules can be simply manufactured by milling dried meat and encapsulating the powder, a process unlikely to degrade shellfish biotoxins, which are stable to heat, pressure and freeze-drying.^{2,3} Such capsules may then become subject to regulation by the Australian Therapeutic Goods Administration (TGA), which distinguishes therapeutics from food, on the basis of whether there is a "tradition of use as a food in the form presented", especially if there is an associated health claim. Such complementary medicines can be either "registered" or "listed". Registered medicines require extensive

Correspondents

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There should be no more than 5 references. The reference list should not include anything that has not been published or accepted for publication. Reference details must be complete, including: names and initials for up to 4 authors, or 3 authors et al if there are more than 4 (see mja.com.au/public/information/uniform.html#refs for how to cite references other than journal articles).

safety, quality and efficacy data. Listed medicines are considered to pose a lower risk than registered medicines, and regulations allow product sponsors to “self-assess” products. Listing is a route commonly taken for complementary medicines. A pertinent example is the TGA listing of therapeutic goods containing dried green-lipped mussel (*Perna canaliculus*).⁴

Where manufacturers of shellfish capsules have undertaken the responsibility of ensuring product safety, they invariably adopt existing biotoxin testing protocols developed for food safety. However, as the allowable biotoxin level is based on wet weight, and the dry weight of bivalve shellfish is only 10%–15% of the wet weight,⁵ safety limits for shellfish meat as food are incorrect by an order of magnitude, and potentially more for capsules containing extracts of shellfish meat.

While important for acute exposure to these toxins, this may be even more relevant in chronic exposure. Okadaic acid, the cause of diarrhoeic shellfish poisoning, is a tumour promoter,⁶ and epidemiological studies suggest that rates of cancer have increased in regions with regular dietary exposure to low levels of this toxin.⁷ Capsules are available that contain 500 mg of dried shellfish meat, which may equate to 5 g of wet shellfish meat.⁵ Unlike a shellfish meal, which may be considered equivalent to a single acute exposure, recommended doses for shellfish capsules can be as many as five capsules a day for many weeks, if not months, therefore magnifying the risk of chronic exposure. It is known that different classes of biotoxins can co-occur in shellfish, adding to the potential hazard outlined here.⁸ Further complications arise because some shellfish capsules include other natural extracts (such as ginseng) or pharmaceutical formulations that might affect toxin uptake.

While this situation needs to be subjected to risk assessment, testing products in accordance with an inappropriate standard can make them seem safe when they might not be. This is especially so for products which are usually self-prescribed, and where patients can exceed recommended doses in the belief that more is better. For products such as shellfish capsules that straddle the food/

therapeutic divide, it is better for manufacturers to test the final consumer product and *not* the raw supply.

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Intragam can interfere with blood glucose monitoring

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TO THE EDITOR: Intravenous immunoglobulin preparations containing maltose (eg, Intragam P [CSL Limited]) can interfere with the readings of blood glucose monitors that use test strips with glucose dehydrogenase, such as Advantage (Roche).¹ Glucose dehydrogenase is an enzyme of the *Pyrrroloquinolinequinone* class which reacts with the disaccharide isomer maltose present in Intragam P at concentrations of 10 g/100 mL,² resulting in falsely elevated blood glucose level results. Blood glucose monitors, such as Precision (Medisense products), Medisense (Medisense products)³ or Accutrend (Roche), that use the glucose oxidase system, do not react with maltose and can be safely used for patients receiving Intragam P.

We have had two patients in whom capillary blood glucose levels were over-estimated.

One was a 64-year-old woman with type 2 diabetes who was being treated

with haemodialysis, and who received Intragam P for immune-mediated thrombocytopenic purpura. Before being admitted, her diabetes was reasonably controlled by 10 units of prothophane twice daily.

She received prednisone (100 mg/day) from Day 1 to Day 13 and Intragam P intravenously each day from Day 2 to Day 6 and from Day 14 to Day 18.

While the patient was receiving Intragam P, the Advantage monitor gave persistently higher readings than both the Precision monitor and plasma glucose level measurements. The following paired readings were obtained:

- Advantage capillary glucose reading of 9.3 mmol/L while the plasma glucose level was 2.3 mmol/L; and
- Advantage capillary glucose reading of 24.4 mmol/L while the plasma glucose level was 10.4 mmol/L.

During the second course of Intragam P we noted that readings with the Precision monitor were equivalent to the measured plasma glucose level.

This patient developed hypoglycaemia because her insulin doses were increased on the basis of falsely elevated capillary glucose readings as measured on Advantage blood glucose strips. A Precision monitor was used for this patient until after the haemodialysis treatment which followed the final dose of Intragam P. After this time Advantage blood glucose readings approached those obtained with the Precision monitor.

A second patient — a 35-year-old woman who was not known to have diabetes, but was having her glucose levels monitored while undergoing total parenteral nutrition — was given Intragam P for idiopathic thrombocytopenic purpura. While receiving Intragam P, capillary glucose levels by the Advantage monitor were elevated by 13–20 mmol/L compared with concurrent (and normal) plasma glucose level measurements.

It has previously been reported that icodextrin, used in some peritoneal dialysis fluids, can have similar effects, as it is hydrolysed to oligosaccharides, including maltose, maltotriose and maltotetraose.^{4,5}

The reaction with maltose has important clinical implications. Although it is

stated in the product information for Advantage test strips that glucose readings may be affected by maltose levels over 16 mg/dL (0.89 mmol/L), clinicians may not be aware that Intragam P is in a maltose solution.

For patients receiving Intragam P, the Advantage monitor is not suitable, and the Precision, Medisense or Accutrend blood glucose monitors, which use glucose oxidase which does not react with maltose, should be used instead.

We have reported our experience to the Australian Red Cross Blood Service as an adverse event.

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Coronary heart disease risk prediction by general practitioners in Victoria

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TO THE EDITOR: Coronary heart disease (CHD) risk prediction for primary prevention now focuses on multifactorial risk,¹⁻³ and various risk calculation tools exist.¹⁻³ The need for such tools depends on the degree to which risk status can be estimated by healthcare professionals. We performed a study to assess general practitioners' intuitive calculation of multifactorial CHD risk for patients likely to be considered for lipid-lowering therapy.

In 1999 we posed a survey to a random sample of 400 GPs in Victoria, and received back 155 completed surveys (39% response rate). The respondent population was demographically

similar to the Australian GPs and trainees who billed Medicare in 1998-99. GPs were asked to estimate 5-year absolute risk of CHD for four vignettes (two primary prevention and two secondary prevention), each based on the average characteristics of a published lipid-lowering clinical trial cohort⁴⁻⁷ (vignette descriptions are available from the authors). For the primary prevention vignettes, GPs were also requested to estimate the risk relative to other Australians of the same age and sex. Estimated risks were compared with reported risks to derive risk ratios.

GPs accurately estimated the relative CHD risk for the two vignettes of patients with no prior CHD, with mean risk ratios of 0.84 (95% CI, 0.77-0.91) and 1.23 (95% CI, 0.97-1.49).

However, they overestimated absolute risk, with risk ratios ranging between 2.79 (95% CI, 2.61-2.96) and 6.43 (95% CI, 5.59-7.27). The proportion of GPs estimating within 10% of the actual risk was 9% for a 62-year-old man with prior CHD and average cholesterol level, 13% for a 60-year-old woman with prior CHD and high cholesterol level, 17% for a 55-year-old man with no prior CHD and high cholesterol level, and 43% for a 58-year-old man with no prior CHD and average cholesterol level. Although the two primary prevention vignettes had 5-year absolute CHD risks below 10%, most GPs' estimates (93% and 71%, respectively) were greater than 10%.

In conclusion, GPs in Victoria have a good understanding of a patient's relative risk of CHD, but they consistently overestimate absolute risk. The problem with absolute risk estimation may not be the sophisticated multifactorial calculations required, but rather a general overestimation of risk within the population, at least for middle age. The GPs' estimates of absolute risk for the four vignettes were correctly ranked, suggesting that they recognised the degree to which a risk was lower or higher, but were unfamiliar with the scale. This overestimation led most GPs to categorise patients such as those from the AFCAPS/TexCAPS trial, with only a 3.3% 5-year risk of CHD,⁶ as having a risk of greater than 10%. GPs would therefore incorrectly consider these patients appropriate for lipid-lowering

therapy according to national and international guidelines.¹⁻³ Education may improve understanding and accuracy of risk communication for CHD in middle-aged patients, but tools for accurate assessment of coronary risk are needed in routine clinical practice.

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Prevalence of use of hip protectors in NSW residential aged-care facilities

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TO THE EDITOR: Hip protectors may be an effective means of preventing hip fracture in residents of aged-care facilities, if there is adequate compliance with their use.¹ Research studies conducted in the Northern Sydney and Illawarra health regions^{2,3} have found that, if a hip protector is worn at the time of a fall, the chance of hip fracture is reduced by about 80%. We wished to determine the extent to which hip protector use had become a routine part of healthcare in residential aged-care facilities generally, and whether there was a greater uptake of their use in areas where the research had been conducted.

A brief questionnaire about hip protector use was sent to all residential

aged-care facilities in the Northern and South Eastern Sydney health regions, and the region covered by the New South Wales Southern Area Health Service. The response rate was 60% in each region, and responses are summarised in the Box. Only 30 residential aged-care facilities participated in the previous study, and the results indicate that use is now much more widespread. Another contrast is that, in the research study, hip protectors were provided free of charge, whereas most current users pay for hip protectors.

Overall, the survey indicates that 61% of aged-care facilities across three regions of NSW are either using hip protectors or have used them in the past. Hip protector use was similar in Northern Sydney and Southern NSW; however, comparing Northern Sydney (where previous research had been conducted) with the other two regions combined showed a greater prevalence related to conducting previous research.

Issues such as cost, laundering of the hip protectors, and comfort are seen as major barriers to their use, but many aged-care facilities continue to use them for selected residents at high risk of hip fracture. It is our impression that the more residents within a facility who wear hip protectors, the better the adherence to hip protector use and the management of these issues. It is also evident that hip protector research encourages use of hip protectors. We are unsure of the mechanism involved, but believe that knowledge and enthusiasm for use of hip protectors is spread by word of mouth between health professionals, staff working in nursing homes and hostels and, in some cases, through

relatives of the residents of residential aged-care facilities.

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Evidence-based guidelines for fixing broken hips

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TO THE EDITOR: Chilov and colleagues have presented an updated set of guidelines for management of hip fracture, which included the statement "regional anaesthesia is recommended for most patients".¹ The evidence for this recommendation was graded as Level I (National Health and Medical Research Council) and was supported by a single reference, namely a systematic review from the Cochrane Database by Parker et al.²

Parker et al performed a meta-analysis of the published trials examining the effect of regional versus general anaesthesia on a variety of outcomes after surgery for hip fracture. A possible difference in 1-month mortality was found in favour of regional anaesthesia, but this difference was borderline using one statistical model (relative risk, 0.7; 95% CI, 0.5–1.0) and non-significant using another model. There was no significant difference in mortality at 3 months or 1 year, and no significant difference in a variety of other outcomes. Appropriately, the authors concluded that "both regional and general anaesthesia produce comparable results and therefore anaesthetists should choose which technique is most appropriate for each individual patient".²

One of the many difficulties in interpreting meta-analyses of regional anaesthesia is that most of the published trials were performed some decades ago. For example, one study that contributed a large proportion of the data within the Cochrane meta-analysis was conducted between 1980 and 1982, and patients

were explicitly excluded if they were receiving low-dose anticoagulation therapy.³ The relevance of such trials to patients receiving general anaesthesia today is highly questionable, given the improvements in general anaesthetic drugs and techniques and the importance now placed on routine thromboprophylaxis.

There is a wide range of opinion within the specialty of anaesthesia regarding the place of major regional blockade, with little outcome-based evidence to support any particular advantage of these techniques. Although medical practitioners can benefit greatly from the efforts of reviewers to develop guidelines based on the best available evidence, care must be taken to ensure that recommendations do not go beyond what is supported by available data. Particular care needs to be taken when recommendations are made for areas of practice outside the reviewers' expertise. The authors of these guidelines might consider withdrawing their recommendation regarding choice of anaesthesia.

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IN REPLY: We thank McCulloch for his comments regarding the use of regional anaesthesia in the surgical management of hip fracture. He makes the point that surgical and anaesthetic techniques have improved and implies that the advantage seen for regional anaesthesia in published studies may no longer be present. Given that controversy still exists, we would recommend that further randomised controlled trials be conducted. However, for the following

Hip protector use in residential aged-care facilities in three health service regions of New South Wales

Region (no. of residential aged-care facilities)	Any hip protector use*
Northern Sydney (99)	74%
South Eastern Sydney (104)	43%
Southern NSW (35)	75%

* $\chi^2 = 7.5$, $df = 1$, $P = 0.006$ for comparison of frequency of use in Northern Sydney with use in the other two area health services combined.

reasons, we stand by our recommendation that the available evidence supports the use of regional anaesthesia for most patients with this condition.

Our current recommendation is unchanged from the earlier version of the guideline (published in the *Journal* in 1999),¹ and is also consistent with at least one other published guideline.² A number of the concerns raised by McCulloch were addressed in the response to a letter by another correspondent after the publication of the original guidelines.³

While we acknowledge that the review by Parker et al⁴ only found the reduction in mortality at 1 month to be of borderline significance, when our review team reassessed the original articles using the Cochrane Collaboration protocol we reached a summary odds ratio for mortality of 0.68 (95% CI, 0.49–0.96). With time and further studies we expect that this estimate of effect will become more precise as the power of the meta-analysis is increased. This view is supported by a systematic review of all randomised studies comparing regional anaesthesia with general anaesthesia across surgical specialties. The study of Rodgers et al found a statistically significant reduction in mortality (odds ratio, 0.70; 95% CI, 0.54–0.90) when regional anaesthesia was compared with general anaesthesia.⁵ This overall point estimate is very similar to that of Parker et al in their meta-analysis of patients with hip fracture. Although lack of power meant that statistical significance did not exist within individual surgical specialties, there was, in fact, little difference in the effect across surgical groups, with no significant heterogeneity between studies.

Serious complications of regional anaesthesia (eg, spinal haematoma) are extremely rare, as shown in the recent PEP study in Australia and New Zealand that reported no cases in 4603 patients undergoing regional blockade.⁶ This should be compared with the number needed to treat with regional anaesthesia to prevent one death of 38, according to the data of Parker et al.⁴

There is no doubt that our recommendation needs to be considered in the context of individual patient characteristics and, while the recommendation

may not apply to all patients with hip fracture, we feel that the available evidence supports the use of regional anaesthesia.

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Public funding of large-scale clinical trials in Australia

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TO THE EDITOR: I strongly support the editorial comments and recommendations of McNeil et al¹ on public funding of clinical trials. Having worked in the Australian healthcare system for 11 years, and having returned to a changed National Health Service in Scotland a few months ago, I can vouch for the benefits that accrue from adequate funding for clinical trials.

While McNeil and colleagues focus on large-scale trials, their comments apply equally to smaller trials. Certainly, in oncology, several trials organisations in Australia have struggled for years to continue conducting trials in spite of inadequate government funding. The ANZ Breast Cancer Trials Group and the Trans Tasman Radiation Oncology Group are but two organisations with which I am familiar. In addition to precarious funding, I believe the consequences in the past 2 years of upheaval in the insurance industry have placed all such groups on an uncertain and untenable footing.

Clinical trials must be ethical, scientific and well managed. Clinicians entering patients into trials need support from essential data managers and

clinical nurse specialists. Governments encourage and, in fact, demand evidence-based medicine. The only effective way to produce the evidence is to conduct clinical trials.

That costs money. In Victoria, cancer trial management was supported by about \$800 000 per annum in grants from the Cancer Council Victoria. Those funds provided start-up assistance to institutions new to clinical trials and supported the others that could not rely on pharmaceutical company largesse. However, only part of that money was state government funded and then only for rural and regional centres or for breast cancer. The diagnosis-related-group-based casemix funding of Victorian hospitals included a notional element for research. That sop was lost in budget deficits.

In Scotland, where health matters are totally devolved to the Scottish Parliament, the latter's Scottish Executive Health Department has very recently enhanced funding for cancer care. This includes £500 000 (A\$1.25 million) annually to support clinical research in cancer. Each of the three cancer networks has a guaranteed share of that sum to resource clinical trials in all the associated health boards and cancer units.

This is in addition to the excellent clinical trials units in the main cancer centres, often funded by the charity Cancer Research UK and industry. There is also a national system of considering and approving clinical research in cancer. Such approval places obligations on health boards to support such trials. Lastly, accreditation of cancer centres can depend on clinical trial participation.

The evidence that this (still imperfect) system has an effect is seen in trial entry at our oncology centre, where 11% of patients are already entered into trials. The new funding should see that increase.

Government needs to put its money where its mouth is: evidence needs resourcing. The alternative is to rely on charity or on industry (whose eye is more often on marketing than science).

1. McNeil JJ, Nelson MR, Tonkin AM. Public funding of large-scale clinical trials in Australia [editorial]. *Med J Aust* 2003; 179: 519-520. □

Otitis media and ventilating tubes

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TO THE EDITOR: The study by Paradise et al on tympanostomy tubes for persistent otitis media,¹ which was expertly reviewed by Morris and Leach in the Journal recently,² has since been updated.³ The findings of both studies from Pittsburgh should only be applied with caution in Australia. Indications for inserting ventilating tubes (VTs) can be divided into three:

- bilateral hearing loss of more than 25–30 dB continuously for 3 months after failed non-operative management;
- structural damage to the tympanic membrane (TM) which may lead to irreversible hearing loss or cholesteatoma; and
- a miscellany which includes underlying sensorineural hearing loss or learning difficulties or similar conditions with deterioration associated with bilateral middle ear effusion (MEE), and recurrent middle ear infections with use of VTs as an alternative to antibiotic prophylaxis, among others.

The conclusion of the more recent article by Paradise et al³ — that there was no difference in expressive or receptive speech or cognition between children in whom VTs were inserted early or late — is valuable, but may not readily be extended to Australian practice. Although 6350 children were enrolled, only 397 were actually randomly allocated into the early or late treatment groups. Thus, the numbers are not large.

Of more concern is that only 18% of those analysed had bilateral continuous MEE (40 in the early and 32 in the late treatment group), with the remaining 82% having unilateral (continuous or discontinuous) or bilateral discontinuous MEE. Only the 18% with bilateral continuous MEE would ordinarily be candidates for VTs in Australia, as Paradise et al underline the fact that intermittent and/or unilateral MEE is not

associated with speech and language difficulties in the absence of other handicaps to learning. An abnormal hearing test result was identified by the study as a 15 dB loss. This could well fall in the normal range for the Australian Hearing Service for children wearing headphones, and a minimum threshold of 25–30 dB should typically be required for considering VTs in Australia. As Morris and Leach² pointed out for the earlier study,¹ the later study also excludes children “not otherwise healthy”,³ and the results cannot be generalised to such children, or to those with moderate rather than mild hearing loss.

Pointing to studies such as that of Paradise et al can be very helpful in reassuring parents who want VTs for their child with unilateral or intermittent hearing loss that not having VTs does not place the child's speech and language development at risk.

1. Paradise JL, Feldmann HM, Campbell TF, et al. Effects of early or delayed insertion of tympanostomy tubes for persistent otitis media on developmental outcomes at the age of three years. *N Engl J Med* 2001; 344: 1179–1187.
2. Morris PS, Leach AJ. Is early surgical referral for children with persistent otitis media with effusion (OME) appropriate? *Med J Aust* 2003; 179: 436–437.
3. Paradise JL, Dollaghan CA, Campbell TF, et al. Otitis media and tympanostomy tube insertion during the first three years of life: developmental outcomes at the age of four years. *Pediatrics* 2003; 112: 265–277. □

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