Journal of Agricultural Science, Cambridge (1996), **126**, 117–122. © 1996 *Cambridge University Press*

ABSTRACTS OF COMMUNICATIONS

Proceedings of the Twenty-Seventh Meeting of the Agricultural Research Modellers' Group

EDITED BY

J. FRANCE

Institute of Grassland and Environmental Research, North Wyke Research Station, Okehampton,
Devon EX20 2SB, UK

This group, which is concerned with the applications of mathematics to agricultural science, is sponsored by the Biotechnology and Biological Sciences Research Council. It was formed in 1970, and has since met at approximately yearly intervals in London for one-day meetings. The twenty-seventh meeting of the group, chaired by Professor D. E. Beever of the Department of Agriculture, University of Reading, was held in the Wellcome Meeting Room at the Royal Society, 6 Carlton House Terrace, London on Friday, 13 October 1995, when the following papers were read.

The interaction of evaporation from soil and transpiration in a sparse crop. C. C. DAAMEN AND L. P. SIMMONDS. *Department of Soil Science, The University of Reading, Whiteknights, PO Box 233, Reading RG6 6DW, UK*

In a sparse crop system (0 < leaf area index < 4) the crop canopy does not cover the soil surface and a significant fraction of solar energy is incident on both crop leaves and the soil surface. This system can result in the 'clothes line effect' which is a name given to the increase in transpiration caused by hot dry air rising through a canopy of leaves from an underlying dry soil surface. Recent papers have used a network of aerodynamic resistances linking the soil and the crop to the overlying atmosphere in order to account for this type of interaction. In these studies, evaporation from soil is estimated using a soil aerodynamic resistance.

Measurements of evaporation from soil, E_s, in semi-arid environments show that this process is complex and difficult to characterize accurately with simple models. Use of two distinct and consecutive stages of evaporation following an initial wet soil condition is not realistic. A reduction in net radiation incident at the soil surface does not necessarily result in a proportional reduction in E_s. Use of a soil aerodynamic resistance (as a function of topsoil water content) is also unsatisfactory.

The model SWEAT (Daamen & Simmonds 1994) has been developed to simulate E_s and transpiration (T) in sparse canopies. It is a numerical model developed from a number of sources (e.g. Campbell 1985; Choudhury & Monteith 1988). SWEAT uses a network of aerodynamic resistances to simulate flow through the canopy air space and also simulates water and heat fluxes in the soil profile to provide an accurate simulation of E_s. It demonstrates the remarkably dynamic nature of the E_s and T fluxes and also shows a strong enhancement of T from a sparse crop when the underlying soil surface is dry.

This paper gives examples of measurement and simulation of E_s and T fluxes from sparse crop canopies in two semi-arid environments (India and Niger). The interaction of these fluxes and the effects of leaf area index on this interaction are discussed.

Campbell, G. S. (1985). *Soil Physics with BASIC: Transport Models for Soil-Plant Systems.* Amsterdam: Elsevier.

Choudhury, B. J. & J. L. Monteith (1988). A four-layer model for the heat budget of homogeneous land surfaces. *Quarterly Journal of the Royal Meteorological Society* **114**, 373–398.

Daamen, C. C. & Simmonds, L. P. (1994). *SWEAT, a numerical model of water and energy fluxes in soil profiles and sparse canopies.* Department of Soil Science, University of Reading.

A Bayesian network for yield response of winter wheat to fungicide programmes. F. TARI. *Silsoe Research Institute, Wrest Park, Silsoe, Bedford MK45 4HS, UK*

Bayesian probability networks (BN) present a powerful tool for incorporating uncertainty in decision support systems (Pearl 1988). They provide a basis for probabilistic inference, to calculate the changes in probabilistic belief as new evidence is entered into the model. In practice, for agricultural systems, the construction of a BN is challenging, as there are often insufficient data for computing the prior and conditional probabilities required for the network.

The model here is a BN for yield response of winter wheat to spraying programmes. Fungicides are used extensively in England and Wales, with over 90% of the winter wheat area receiving at least one application in the years 1985–89. Routine spraying treatments have often led to unnecessary fungicide use, poor sprays and financial waste. Experimental work has been carried out to study the effects of individual site factors (previous crop, sowing date, region and cultivar) on the yield response of winter wheat to fungicide programmes applied at growth stages GS39, GS31+39 and GS31+39+59 (Cook & Thomas 1990). The BN uses the experimental data from ADAS trials and makes some assumptions in order to complete these data.

HUGIN software (Hugin Expert A/S 1993) was used for editing and testing the BN. The technique extends the experimental results by studying the interactive effect of the site factors on the yield response, and making deductions on spraying policies that are not considered experimentally (GS31, GS59, GS31+59 and GS39+59). The model also shows the potential of BN for modelling uncertainty in agricultural systems.

COOK, R. J. & THOMAS, M. R. (1990). Influence of site factors on yield response of winter wheat to fungicide programmes in England and Wales, 1979–1987. *Plant Pathology* **39**, 548–557.

HUGIN EXPERT A/S (1993). *Hugin Explorer*®. Hugin Expert Ltd, Niels Jernes Vej 10, DK-9220 Aalborg Ø, Denmark.

PEARL, J. (1988). *Probabilistic Reasoning in Intelligent Systems: Networks of Plausible Inference*. Series in Representation and Reasoning. San Mateo, CA: Morgan Kaufmann Publishers, Inc.

Growth hormone response of the pituitary to stimulatory and inhibitory releasing factors from the brain.

E. A. STEPHENS[1], D. BROWN[1], G. LENG[2] AND R. G. SMITH[3]. [1] *Department of Neurobiology, Babraham Institute, Cambridge CB2 4AT, UK*, [2] *Department of Physiology, University of Edinburgh, Edinburgh EH8 9AG, UK*, [3] *Merck Research Laboratories, Rahway NJ, USA*

Growth hormone (GH) is released from the pituitary gland in a pulsatile manner, and this patterning of release is essential to its effectiveness in the body tissues. In the intact animal, release is controlled by the coordinated actions of stimulatory growth hormone-releasing hormone (GHRH) and inhibitory somatostatin (SST) which are, in turn, released by the hypothalamus. A key first step in understanding the control of GH release is to model the pituitary release mechanism. Synthetic growth hormone-releasing compounds, such as the GH-releasing peptides (GHRP), also stimulate release from the pituitary, and these have medical and agricultural applications, such as the treatment of dwarfism and in growth promotion. When applied together, GHRP and GHRH interact synergistically to produce a greater GH release than the sum of their individual effects, both *in vitro* and, to a greater extent, *in vivo*. In the prolonged absence of SST, a desensitization of the response to GHRH pulses occurs, resulting in smaller GH pulses. In addition to its powerful inhibitory effect, SST acts in a paradoxically positive manner to resensitize the pituitary cells to GHRH. After a period of high SST and low GHRH exposure, as is observed *in vivo* between GH pulses, the pituitary will respond optimally to a pulse of GHRH.

A nonlinear differential equation model for the action of GHRH and SST at the pituitary is presented. Simulations of the model are shown, and are interpreted in the light of data from *in vitro* experiments on isolated pituitary cells from the male rat. An approach to incorporating GHRP into the model is discussed.

The simulation of N fixation by white clover within a model describing N flows in grazed grassland.

D. SCHOLEFIELD[1], D. R. LOCKYER[1] AND S. F. LEDGARD[2]. [1] *Institute of Grassland and Environmental Research, North Wyke, Devon, EX20 2SB UK*, [2] *AgResearch, Ruakura Research Centre, Private Bag 3123, Hamilton, New Zealand*

There have been few attempts to model the flows of N within and between the soil, plant and animal components of a grazed grassland system. There are two major additions of N to such a system; one is manufactured fertilizer, and the other is fixation by the root nodules of legumes. While all existing models consider the former, not even the more mechanistic approaches (e.g. Thornley & Verberne 1989) consider the latter explicitly, except, as reported in a recent publication (Thornley *et al.* 1995), when the herbage is not harvested. A development of our empirical model N CYCLE (Scholefield *et al.* 1991), which is based on annual mass balance in N, to describe N fixation and transfer within grass/white clover swards under grazing, is reported here.

The N fixation sub-model is also empirical, but it includes three important mechanisms: (i) the inhibitory effect of soil inorganic N on fixation, and thus the proportion of clover N originating from the soil, (ii) the direct and indirect transfer of clover N to soil via senescence and grazing, respectively, and (iii) the effect of grazing preference on the proportion of clover in the sward. The sub-model starts with the dry matter (DM) yield of grass predicted by N CYCLE and, using a relationship derived from a national suite of experiments known as the GM series, calculates the equilibrium DM yield of clover in association with the grass. The total N in clover is then derived assuming a constant concentration of 3.75% N in the DM. The N in soil available to the clover component is calculated by subtracting grass N from the annual flux through the inorganic N pool. The amount of clover N derived from soil, and thus the proportion from fixation, is determined from a relationship obtained from results of a growth experiment using ^{15}N labelling under controlled conditions (Nesheim & Boller 1991).

The model can be used to investigate the combined influences of climate, soil type, land drainage, grazing pressure, sward age and previous management on N fixation and sward composition. The model shows that the equilibrium clover content of a sward is a function of these parameters and cannot be imposed by reseeding. However the model considers neither within-year effects nor the spatial arrangement of grass and clover patches. Its use is aimed at broad scale management issues for which the need is for numerically accurate predictions rather than further insight into fine-scale mechanisms. Independent tests of the model output using field data obtained from experiments conducted in the UK and New Zealand are presented. Discrepancies between predicted and measured values are discussed.

NESHEIM, L. & BOLLER, B. C. (1991). Nitrogen fixation by white clover when competing with grasses at moderately low temperatures. *Plant & Soil* **133**, 47–56.

SCHOLEFIELD, D., LOCKYER, D. R., WHITEHEAD, D. C. & TYSON, K. C. (1991). A model to predict transformations and losses of nitrogen in UK pastures grazed by beef cattle. *Plant & Soil* **132**, 165–177.

THORNLEY, J. H. M. & VERBERNE, E. I. J. (1989). A model of nitrogen flows in grassland. *Plant, Cell & Environment* **12**, 863–886.

THORNLEY, J. H. M., BERGELSON, J. & PARSONS, A. J. (1995). Complex dynamics in a carbon–nitrogen model of a grass–legume pasture. *Annals of Botany* **75**, 79–94.

Modelling bovine tuberculosis in badgers. G. D. RUXTON. *Biomathematics & Statistics Scotland, King's Buildings (JCMB), Mayfield Road, Edinburgh EH9 3JZ, UK*

The viral disease, bovine tuberculosis, is believed to be endemic in the European badger (*Meles meles*) population in Britain. It is thought that badgers pose a significant threat of cross-infection to domestic cattle, and hence controlled culling of badgers has been implemented in certain areas. In order to maximize the effectiveness of control measures, such as culling, there has been considerable field and theoretical research into the dynamics of the disease within the badger population. Here, the recent model of Bentil & Murray (1993) is re-analysed and found to be unable to reproduce the observed behaviour of the disease. The reasons for this discrepancy are discussed and suggestions made for modifications to the model.

Field monitoring suggests that the prevalence of tuberculosis in the British badger population is cyclic (Cheeseman *et al.* 1989). Bentil & Murray (1993) present an ordinary differential equation (ODE) model designed to represent bovine tuberculosis within a badger population. For parameter values estimated from the literature, their model predicts that the time series of the fraction of the population which is infected will exhibit damped cycles. However, this damping is very strong and the model can only be induced to produce cycles similar to field observations by initial conditions when almost all the population is uninfected. This is unrealistic for a disease which is known to be endemic in the population.

Bentil & Murray (1993) also suggest that the addition of seasonality and stochasticity would act to sustain cycles in the model. This speculation draws on the behaviour of similar models parameterized to represent childhood diseases in humans. For chickenpox, Rand & Wilson (1991) demonstrate that stochasticity and seasonality combine to produce model dynamics which are very far from the stable equilibrium due to non-linear amplification of noise. This amplification is caused by the presence of so-called chaotic repellors. It is demonstrated here that such repellors are likely to be present when the model is parameterized for chickenpox in humans but not when parameterized for tuberculosis in badgers. Hence the addition of noise and seasonality will not act to sustain limit cycles. This prediction is supported by direct integration of a modified model.

ODE models seem unable to predict the behaviour of this disease in natural populations. This may be because one of the fundamental assumptions of such models is unrepresentative of badger behaviour. ODE models assume perfect mixing where any two individuals in the population are equally likely to come into contact. This is not true in badgers who live in small social groups (clans) which defend a territory against other badgers. It seems likely that future modelling studies of tuberculosis infection must address the spatial structure of the badger population in more detail.

BENTIL, D. E. & MURRAY, J. D. (1993). Modelling bovine tuberculosis in badgers. *Journal of Animal Ecology* **62**, 239–250.

CHEESEMAN, C. L., WILESMITH, J. W. & STUART, F. A. (1989). Tuberculosis: the disease and its epidemiology in the badger, a review. *Epidemiology and Infection* **103**, 113–125.

RAND, D. A. & WILSON, H. (1991). Chaotic stochasticity: a ubiquitous source of unpredictability in epidemics. *Proceedings of the Royal Society of London B* **246**, 179–184.

Modelling the effect of disease aggregation on the rate of disease progress in plant disease epidemics.

N. McROBERTS[1] AND G. HUGHES[2]. [1] *Plant Science Department, Scottish Agricultural College, Auchincruive, Ayr KA6 5HW, UK,* [2] *Institute of Ecology & Resource Management, University of Edinburgh, Edinburgh EH9 3JG, UK*

The logistic and similar functions have frequently been used to describe plant disease progress with time. Corrections to the function to take account of the phenomenon of aggregation of disease have been suggested (Waggoner & Rich 1981; Yang & TeBeest 1992) in the case where lesion numbers per leaf (a severity assessment) determine the proportion of diseased plants (an incidence assessment). Here, the effect of disease aggregation on the rate of disease progress when disease assessments are based on incidence at both the leaf and plant scales is investigated. The proportion of diseased plants is denoted P and the proportion of diseased leaves is denoted L. N denotes the number of leaves per plant. The spatial component of the model specifies the frequency distribution of diseased leaves per plant, which is assumed to follow a binomial distribution when disease has a random pattern, or a beta-binomial distribution (Hughes & Madden 1993) when disease has an aggregated pattern. In both cases the proportion of plants which have no diseased leaves (P_0) is given by the first term of the frequency distribution. The proportion of diseased plants is given by $P = 1 - P_0$. The temporal component of the model assumes that the rate of disease progress over time (t) can be described by a logistic function $\mathrm{d}P/\mathrm{d}t = RP(1-P)$, in which R is a constant. The effect of aggregation of disease on the rate of disease progress can be examined either under the assumption that the beta-binomial aggregation parameter (θ) is constant or that θ varies with mean incidence of diseased leaves. This simple model, in which host growth is not considered, suggests that the effect of aggregation of disease would be to slow the rate of disease progress early in the epidemic. The derivative $\mathrm{d}P/\mathrm{d}L$, provided by the relationship between disease incidence of plants and of leaves, can be thought of as characterizing the rate of disease spread. This can be incorporated in a conceptual model $\mathrm{d}P/\mathrm{d}t = \mathrm{d}N/\mathrm{d}t.\mathrm{d}L/\mathrm{d}N.\mathrm{d}P/\mathrm{d}L$, in which the terms $\mathrm{d}N/\mathrm{d}t$ and $\mathrm{d}L/\mathrm{d}N$ can be thought of as rates of host growth and of colonization of the host by the pathogen, respectively.

HUGHES, G. & MADDEN, L. V. (1993). Using the beta-binomial distribution to describe aggregated patterns of disease incidence. *Phytopathology* **83**, 759–763.

WAGGONER, P. E. & RICH, S. (1981). Lesion distribution, multiple infection and the logistic increase of plant disease. *Proceedings of the National Academy of Science, USA* **78**, 3292–3295.

YANG, X. B. & TEBEEST, D. O. (1992). Dynamic pathogen distribution and the logistic increase of plant disease. *Phytopathology* **82**, 380–383.

Testing models for sensitivity to changes in the variances of their parameters.

T. M. ADDISCOTT AND G. TUCK. *Institute of Arable Crops Research, Rothamsted, Harpenden, Herts AL5 2JQ, UK*

Parameter variability is one of the curses of the soil modeller. Sensitivity analysis on a model usually assesses the effect of percentage changes in its parameters in terms of the effects on selected outputs. The values tested are unique, but if the parameter is obtained from replicated measurements on the soil it almost certainly needs to be represented by a probability distribution. Should the model therefore be tested for sensitivity not only to changes in the mean of the parameter but also to changes in its variance? The answer is yes, for two reasons (Addiscott 1993).

(i) If the model is non-linear with respect to a parameter, the mean of an output from the model will depend not only on the mean of the parameter but also its variance. This provides a means of testing for non-linearity.

(ii) It may be important to know to what extent the model transmits the variance of the parameter through to the output. Some models may suppress the variance while others exaggerate it.

These questions were explored with the SLIM leaching model (Addiscott & Whitmore 1991). This has two main parameters, α, which is a simplified rate parameter and provides a measure of the ease with which water percolates through the soil, and W_r a capacity parameter which is the quantity of water reckoned to be immobile in the soil. SLIM is known to be somewhat non-linear with respect to α but was expected to be linear with respect to W_r. Because both parameters and outputs have widely differing numerical values, the effects of changes in variance were evaluated in terms of changes in the coefficient of variation (C.V.), the ratio of the standard deviation to the mean. The results of changing the C.V.s of α and W_r on the means and C.V.s of three model outputs, water loss, solute loss and solute concentration in drainage were examined. The means of all three outputs changed, as expected, when the C.V. of α increased. Increasing the C.V. of W_r had only a small effect on the mean cumulative values of water and solute loss but more effect on mean solute concentrations on individual days. There was also an interaction between the change in the C.V. of α and that in the C.V. of W_r. The model transmitted the changes in C.V. from parameter to output in different ways for the two parameters, and the C.V.(output) to C.V.(parameter) relationships were not necessarily linear. The model tended to suppress the effects of the change in the C.V. of the α parameter but to exaggerate those of the change in W_r and there were interactions between these two effects.

ADDISCOTT, T. M. (1993). Simulation modelling and soil behaviour. *Geoderma* **60**, 15–40.

ADDISCOTT, T. M. & WHITMORE, A. P. (1991). Simulation of solute leaching in soils of differing permeabilities. *Soil Use and Management* **7**, 94–102.

An isotope dilution model for tyrosine uptake and release across the hindlimb of lambs.

L. A. CROMPTON AND M. A. LOMAX. *Growth Biochemistry Group, School of Animal & Microbial Sciences, University of Reading, Whiteknights, PO Box 228, Reading RG6 6AJ, UK*

A complete understanding of the nutritional and hormonal regulation of tissue protein turnover necessitates quantification of the rates of both protein synthesis and degradation. The anabolic process of skeletal muscle protein synthesis has been extensively studied in many species, but our knowledge of the catabolic process of protein degradation remains poor, due to the lack of suitable techniques for measurement of protein degradation *in vivo*. A model of tyrosine uptake and release across the hindlimb tissues of growing lambs is constructed and solved in the steady state. Model solution permits calculation of tyrosine uptake from the arterial plasma supply, tyrosine release into the venous drainage and the fractional rates of protein gain, synthesis and degradation in a mixture of tissues containing predominantly muscle, but also skin, bone and fat. The model assumes that the specific radioactivity of the precursor pool for protein synthesis is equal to either that of the artery or the vein. The experimental measurements required for model solution are plasma flow rate, and the plateau concentration and specific radioactivity of tyrosine in arterial and venous plasma.

In an effort to establish the validity of the hindlimb model for studies on the acute regulation of skeletal muscle protein synthesis in sheep, experiments were conducted to measure hindlimb protein synthesis and muscle protein synthesis simultaneously in nine growing lambs fed a range of dry matter intakes (Crompton & Lomax 1993). There was no significant difference between the mean values for the fractional rates of hindlimb (k_{sav}) protein synthesis (3.66 ± 1.50%/d) and muscle (k_{sh}) protein synthesis (3.35 ± 1.26%/d). Hindlimb and muscle protein synthesis (y) were both significantly related to dry matter intake (x) (k_{sav}, $r^2 = 0.667$, $P = 0.007$; k_{sh}, $r^2 = 0.968$, $P < 0.001$) and there was no significant difference between the slopes ($P = 0.945$) and intercepts ($P = 0.532$) of the two regression lines.

The model has been applied to investigate rapid changes in protein turnover across the hindlimb in fed (F), undernourished (U) and at four time points during 11 h of refeeding (R2, R5, R8 and R11) in lambs. Well-fed lambs had a net gain of protein across the hindlimb which changed to a net loss when undernourished, but returned to a net gain within 5 h of initiating refeeding. The changes in net protein gain were the result of specific changes in the fractional rates of hindlimb protein synthesis (F 5.46, U 3.38, R2 3.41, R5 3.64, R8 3.83 and R11 3.94 ± 0.86%/d) and degradation (F 3.78, U 4.30, R2 3.72, R5 3.09, R8 2.37 and R11 2.53 ± 0.67%/d). Changes in net protein gain (amino acid balance) across the hindlimb when under-fed are due to a decrease in protein synthesis, but the acute response to refeeding is mainly the result of a rapid decrease in protein degradation. Identification of the exact signals responsible for this change would be of use in the nutritional management of lean tissue mass both in agricultural and clinical applications. Preliminary evidence from further experiments has implicated amino acids, insulin, cortisol and thyroid hormones.

Therefore, the hindlimb model is capable of measuring the impact of nutrition on protein turnover. Although the responses to nutrition in hindlimb and muscle tissues were similar, the values derived from the model represent protein metabolism in the mixed tissues of the hindlimb. The major advantage of the hindlimb model is that it enables the rate of hindlimb tissue protein gain, synthesis and degradation to be determined simultaneously *in vivo*, and it can therefore be used serially for large animal and clinical studies.

CROMPTON, L. A. & LOMAX, M. A. (1993). Hindlimb protein turnover and muscle protein synthesis in lambs: a comparison of techniques. *British Journal of Nutrition* **69**, 345–358.

A modelling analysis to investigate the effects of environmental factors on the growth of winter wheat.

A. G. GILLETT[1], N. M. J. CROUT[1], R. K. SCOTT[1], D. T. STOKES[1], R. W. CLARE[2], R. SYLVESTER-BRADLEY[3], R. M. WEIGHTMAN[3], G. RUSSELL[4], P. S. KETTLEWELL[5] AND J. MACBETH[5]. [1] *University of Nottingham, Sutton Bonington Campus, Loughborough LE12 5RD, UK,* [2] *ADAS Arable Research Centre, Rosemaund, Preston Wynne, Hereford HR1 3PG, UK,* [3] *ADAS, Anstey Hall, Maris Lane, Cambridge CB2 2LF, UK,* [4] *Institute of Ecology and Resource Management, School of Agriculture, University of Edinburgh, West Mains Road, Edinburgh EH9 3JG, UK,* [5] *Harper Adams Agricultural College, Edgmond, Newport, Shropshire TF10 8NB, UK*

The relationship between biomass and absorbed photosynthetically active radiation (PAR) was investigated across six reference sites for two seasons (1993 and 1994) to determine if there were significant differences in the radiation conversion coefficient (ϵ) between sites. Where such differences existed, the most important environmental factor(s) controlling the growth of the winter wheat reference crop (*Triticum aestivum* L. cv. Mercia) was determined for each site. A series of simple models describing the observed variation in biomass accumulation across sites in relation to the environmental variable(s) was developed, using the same model across all sites. By developing a general and robust modelling approach appropriate for use across all sites, it is anticipated that the longer term objective of extension to include regions not included in this calibration will be facilitated.

The models derived were able to describe the growth of biomass with a weighted R^2 of at least 0.97. They were compared using weighted sums of squared residuals. It was found that the most important factors controlling biomass were light and nitrogen (within the standing crop) per unit ground area. Accounting for these factors across the course of the two seasons resulted in a mean percentage error, root mean square error and mean bias error over all sites of 12–18%, 1 t ha^{-1} and -0.2 t ha^{-1}, respectively. The final percentage differences between model output and observed data were within 10% for most sites, and it is hypothesised that the nitrogen per unit area provides a useful basis to predict biomass accumulation.

Using thresholds in population based modelling of seed germination.

K. PHELPS AND W. E. FINCH-SAVAGE. *Horticulture Research International, Wellesbourne, Warwick CV35 9EF, UK*

Prediction of the timing of seed germination, as with many other biological processes, is often made on the basis of thermal time requirements estimated from curves obtained in constant temperature experiments. A straight line is drawn relating the rate of germination of the 50th percentile to temperature. The parameters of the line are expressed in terms of a number of day-degrees above a base temperature extrapolated from the line. This base temperature is usually interpreted as the temperature below which radicle emergence (germination) ceases to occur. However the high levels of maximum percentage germination often observed at the extrapolated base temperature in laboratory experiments suggest this is an over-simplification. It is suggested here that the temperature at which 50% of viable seeds fail to germinate provides a more meaningful median base temperature. Furthermore the frequency distribution of base temperatures for the population of seeds can be inferred from the range of reductions in final percentage germination often observed at extreme temperatures.

Explicit determination of threshold temperatures, as described above, provides further insight into the causes of variability in the timing of germination. There appear to be two main sources: (i) the range of base temperatures and (ii) the inherent rates at which the seeds progress to germination. Hypothetically these sources can interact in different ways and it is not simple to infer the mode of interaction from constant temperature experiments. Interactions may mean that the observed frequency distributions of germination times give a misleading representation of the underlying distributions. Thus taking potential interactions into account may lead to better description of frequency distributions and hence a more robust representation of rate-temperature relationships.

Simulation models can be constructed which incorporate both the base and rate distributions. These allow both the timing of germination and the final percentage germinated to be estimated under conditions of fluctuating temperature. Such temperature conditions are needed to provide a proper laboratory test of the proposed models and are inevitable in field conditions. The methods described can be extended to incorporate varying water potentials.

We thank the Ministry of Agriculture, Fisheries and Food for funding this work.