S2-1

Modelling Microbial Growth and Inactivation through the Farm to Fork Chain within Quantitative Microbial Food Safety Risk Assessment

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Since 2001, Tom has held the post of Senior Lecturer in Food Microbiology at the University of Tasmania, and is also part of the Australian Food Safety Centre of Excellence, a joint venture between the University of Tasmania and Food Science Australia under the National Food Industry Strategy.

Tom started research work in the early 1990's on mathematical modelling of the microbial ecology of foods. The results of his team's studies are used to improve the safety and shelf life of foods. Some have been adopted into Australian food regulations and integrated into commercial computer programs for use by industry. Latterly, those techniques have been integrated into the development of microbial food safety risk assessments for government and industry, both within Australia and overseas. These studies also motivate new laboratory-based research projects.

Tom's contributions to food microbiology have been recognised within Australia and internationally through national and international awards. He has served on both Australian and international expert committees concerned with food safety, and currently collaborates in national and international food safety research projects.

Modelling microbial growth and inactivation through the Farm-to-Fork chain

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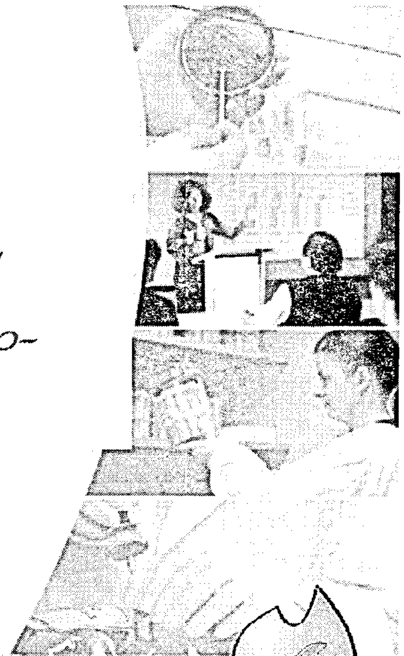

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Food Safety: the essential ingredient

Assessing food-borne risk...

- *need to know:*
  - *dose-response relationship*
    - *levels of pathogens* vs *probability of illness*
  - exposure to the pathogen in the food
    - *frequency of contamination*
    - *dose at consumption;*
      - *amount of the pathogen present in the food*
      - *size of meal serving*

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$$P_{ill} = f(\text{Dose}) \approx \text{Serving Size} \times [H_c]$$

- P_{ill} = probability of illness
- D = dose of pathogens ingested
- H_c = concentration of the hazard at the time of consumption

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$$(H_o - \Sigma R + \Sigma I) \times S = D$$

- H_o = initial level of the hazard
- ΣI = total increase (growth or recontamination)
- ΣR = total reduction (inactivation or removal)
- S = size of meal

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Predictive microbiology

"quantitative microbial ecology of foods"

- *responses of microbes to biotic and abiotic environment*
- *summary of KNOWLEDGE in model structure*
- *summary of DATA in models (determines specific parameters)*

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Primary models

- *sigmoid curve for batch growth*
  - Gompertz, log logistic, Richards, "three phase linear", etc
  - all feature lag time, exponential growth rate, maximum population density
- *various models for inactivation kinetics*
  - log linear, Weibull, Kamau and Pruitt, etc.
  - characterise decimal reduction times, non-linear behaviour

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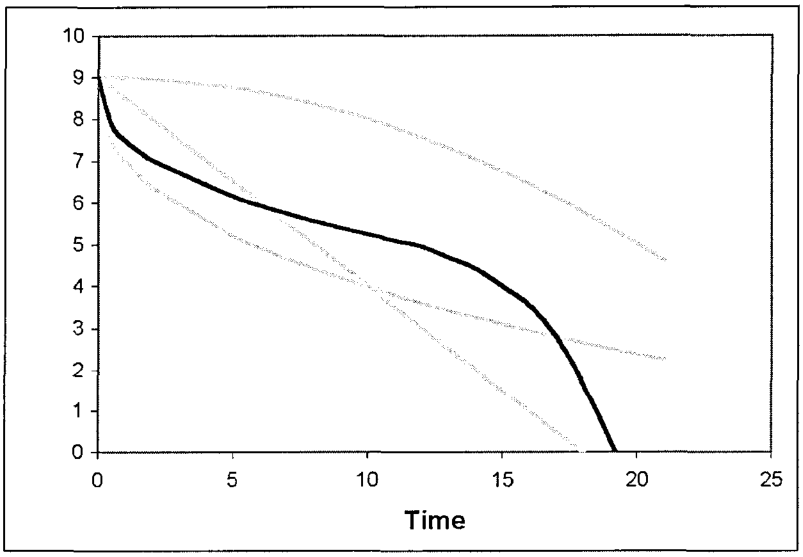
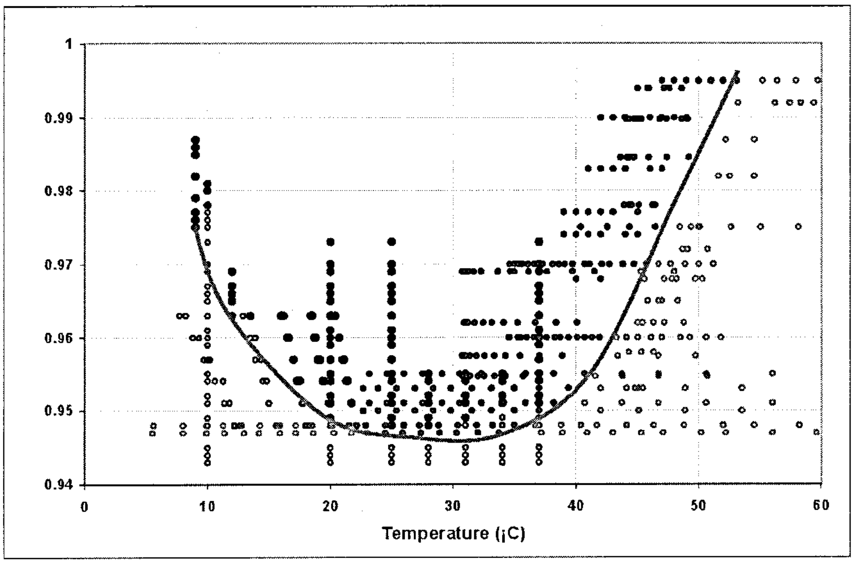
Microbial population inactivation



Microbial ecology of foods

- ***intrinsic***
- ***extrinsic***
- ***processing***
- ***implicit***
  - patterns of microbial behaviour
  - e.g. responses to temperature, pH, water activity, 'competition' etc.

Growth/no-growth interface for *E. coli*



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Secondary models

- ***describe the effect of environment on 'primary' responses'***
  - growth rate and lag time
    - ***square root type,***
    - ***polynomials***
    - ***Arrhenius type***
  - inactivation rate
    - ***polynomials***
    - ***DRT***
    - ***time for 4-log reduction***
  - growth-limits models
- ***cross-contamination***

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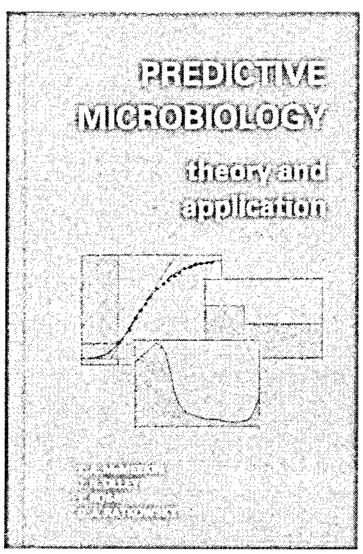
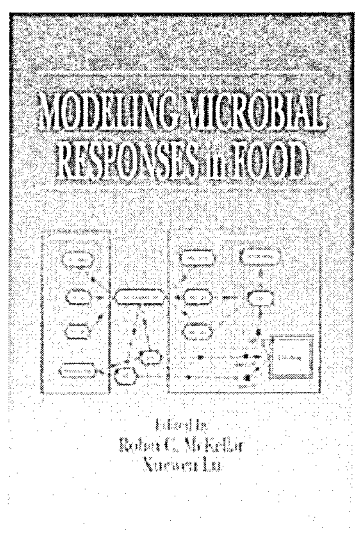
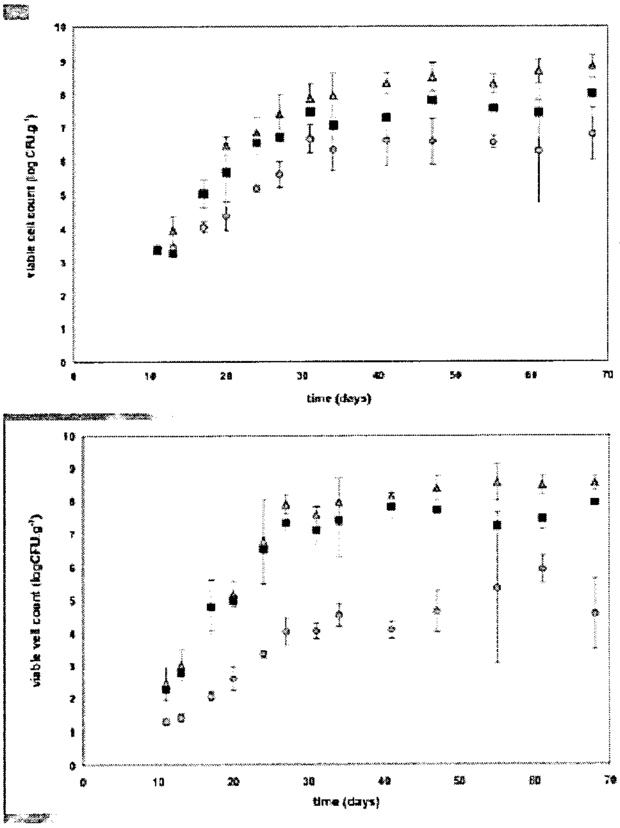
finding resources

- *on growth/inactivation rate predictions*
  - averages example
- *e.g from B. cereus in custard*
  - the importance of significant differences

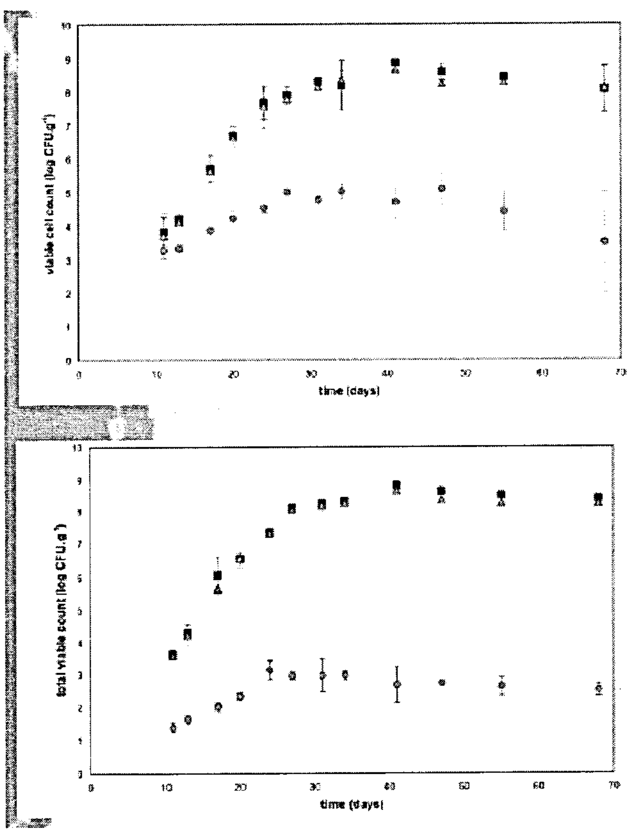






Demonstration of the Jameson Effect:

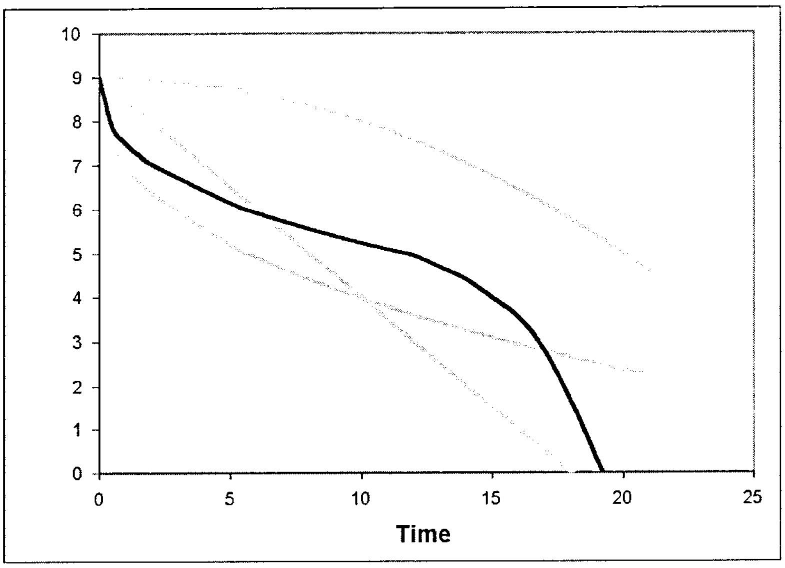
growth of *L. monocytogenes* inoculated onto commercial ham and stored at 8°C



Demonstration of the Jameson Effect:

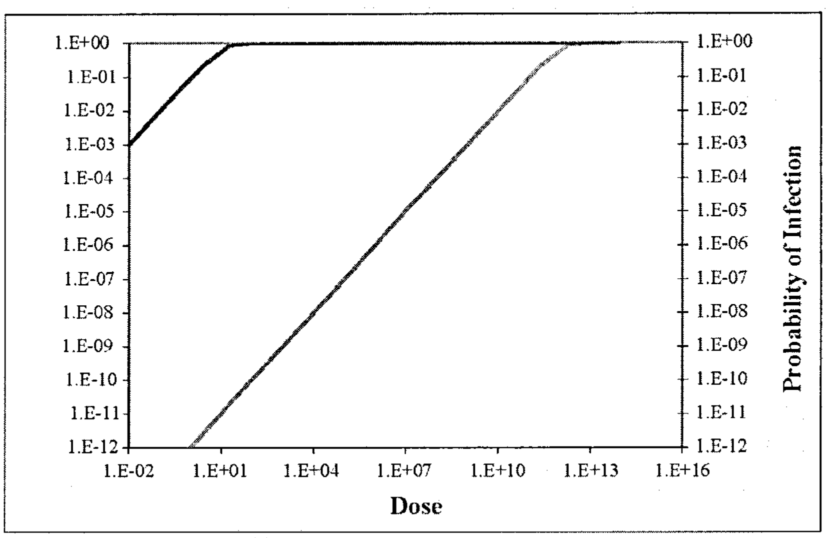
growth of *L. monocytogenes* inoculated onto commercial ham and stored at 4°C

Bacterial population Inactivation



Dose response relationships

- errors in prediction of H_c translate directly into errors in estimate of risk



Predictive microbiology in Australian food safety risk management

- PM and quantitative risk assessment results used by companies, industry, and government for decisions
- *E. coli* growth rate model incorporated into regulations ("Refrigeration Index")
- *E. coli* inactivation model endorsed by FSANZ and industry for salami process evaluation, development
- more and better models, ability to generate and apply models more quickly, integration with new technologies

Predictive micro. in risk assessment

- conditions change throughout the food supply chain...need to be able to
  - *model the effect of fluctuations*
  - *combine models sequentially*
- must
  - *predictive model validity*
  - *overall model validity*
  - *that variability is represented*
- must
  - *model biological reality*
  - *preserve mathematical logic*

Modeling microbial ecology of foods

- *can it grow?*
- *when does it start to grow (lag time)?*
- *how fast does it grow?*
- *how much does it grow (i.e. MPD)?*
- *will it die?*
- *how quickly does it die?*
- *what are the kinetics of death?*

Integrating growth/death rates over time

- assume constant conditions, model many scenarios
- effect of averaging fluctuating temperature?
- incorporating lag time
- have to maintain realistic sets of conditions
  - *fast growth rates means shorter shelf life*
  - *effect of other microbiota, e.g. lactic acid bacteria in vac packed ready to eat foods*

Assumption of constant temperature

- growth not directly proportional to temperature
- averaging T always* leads to an underestimate of growth rate
- not significant in most cases
- greater if temperature range is wide
- effect greatest near limits for growth

(* as long as temperature is below the temperature for maximum growth rate)

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Lag time

- relative lag time
  - *lag time/generation time*
- lag time responds to environment similarly to generation time (within limits)
- lag time can contribute a 1 to 2 log decrease in predicted growth (and predicted risk)

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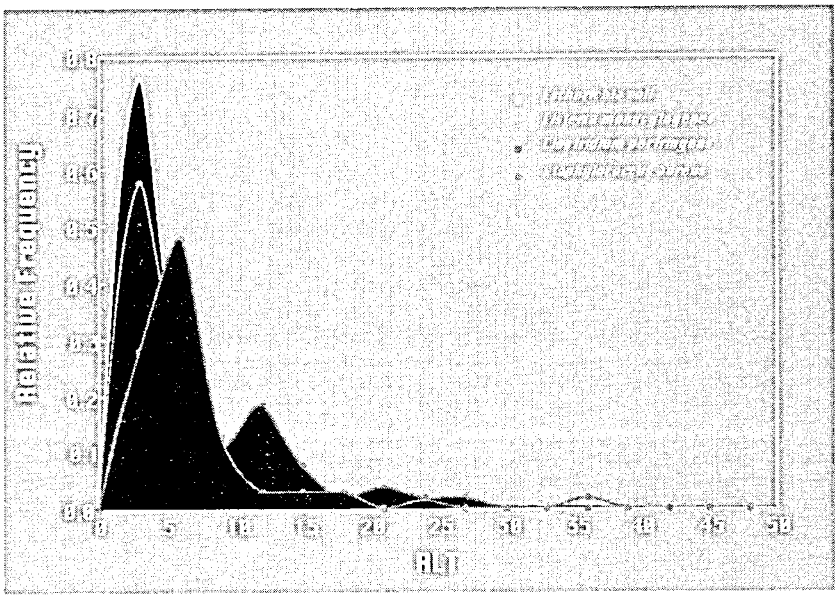
Relative lag time distributions



Example 1:
the effect of strain variability on risk estimates

Example 2: the importance of variability in exponentially appearing terms

Conclusions

- predictive microbiology is strongly 'science-based'
- resources are well developed and accepted in risk management
- it can provide a powerful tool for inferring doses at *consumption*, and hence risk of illness
- microbial populations change exponentially over time, meaning that risk of illness also changes exponentially over time
- uncertainty/variability in microbial responses can lead to large confidence intervals in risk estimates, thus reliable *absolute* risk estimates are difficult
- reliable *relative* risk estimates are achievable

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