

Case Studies in Mathematical Modeling

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Contents

List of contributors

Preface

1 A mathematical model relating to herbicide resistance 1

Lee A. Segel

1.1 Introduction 1

1.2 Formulation of a mathematical model 3

1.3 Solution 9

1.4 Discussion 11

Acknowledgement 15

Problems 15

References 16

2 Mathematical modeling of elevator systems 18

Bruce A. Powell

Summary 18

2.1 Introduction 19

2.2 General information about elevators 20

2.3 Some simple models of elevator travel time 21

2.4 Round-trip time and interval 23

2.5 Filling time for a bank of elevators 29

2.6 Simple elevator banking optimization 30

2.7 Generalized optimum elevator banking 35

2.8 Numerical example of generalized banking 38

2.9 Conclusions about banking optimization 40

2.10 Computer simulation of elevator systems 42

2.11 Passenger traffic generator 45

2.12 A simple (but efficient) dispatching strategy 49

Reference 52

Bibliography 53

3 Models of traffic flow 54

Donald A. Drew

3.1 Queue length at a traffic light *via* flow theory 54

Problems 59

3.2 Traffic flow theory 61

3.2.1 Basic equations 61

3.2.2 Propagation of a disturbance 64

Problems 69

- 3.3 Car following theory 73
 - 3.3.1 Introduction 73
 - 3.3.2 California Code 74
 - 3.3.3 The flow-concentration diagram 75
 - 3.3.4 A typical solution 76
 - 3.3.5 Car following with delay 77
 - 3.3.6 Stability of car following models 79
 - 3.3.7 Nonlinear car following model 84

Problems 88

- 3.4 Equilibrium speed distributions 89

Problems 97

- 3.5 A Boltzmann-like approach to traffic flow 99
- 3.6 Gap distributions 106

Problems 109

Bibliography 109

4 Semiconductor crystal growth 111

Lynn O. Wilson

- 4.1 Introduction 111
- 4.2 Background of the problem 112
 - 4.2.1 Semiconductors 113
 - 4.2.2 The impurity 113
 - 4.2.3 Getting the impurity into the crystal 114
 - 4.2.4 Czochralski growth 115
 - 4.2.5 Segregation 115
 - 4.2.6 Diffusion 116
 - 4.2.7 Convection 117
 - 4.2.8 Control 117
 - 4.2.9 Experimental results 118
 - 4.2.10 Conjecture 118
- 4.3 Formulating a mathematical model 118
- 4.4 The Burton-Prim-Slichter model 120
 - 4.4.1 Conservation of impurity atoms 121
 - 4.4.2 Conservation of mass 122
 - 4.4.3 The axial velocity 122
 - 4.4.4 Boundary conditions 123
 - 4.4.5 Solution of the differential equation 124
 - 4.4.6 A cruder model 125
- 4.5 The crystal growth model 126

4.5.1	Preliminary thoughts	126
4.5.2	The model	127
4.5.3	Basic conservation principles	128
4.5.4	Boundary and initial conditions	128
4.5.5	Dimensionless conservation equations	129
4.5.6	A major breakthrough	130
4.5.7	The boundary conditions	132
4.5.8	Initial conditions	135
4.6	A hierarchy of problems	135
4.7	The partial differential equation solving routine	138
4.7.1	POST (partial and ordinary differential equation solver in space and time)	138
4.7.2	Special features	139
4.8	Values of physical parameters	141
4.9	The fluid flow in the melt	142
4.9.1	History	143
4.9.2	Equilibrium results: no suction ($a = 0$)	146
4.9.3	Equilibrium results: suction ($a > 0$)	148
4.9.4	Comparisons	151
4.10	Steady-state crystal growth	154
4.10.1	Dependence on R	155
4.10.2	Dependence on k_0	155
4.10.3	Dependence on Sc and a	157
4.10.4	Getting things started	157
4.10.5	A computational difficulty	158
4.10.6	Results: the effective distribution coefficient	159
4.10.7	Results: concentration in the melt	160
4.10.8	Results: thickness of the diffusion boundary layer	161
4.11	Time-dependent crystal growth	164
	Postscript	164
	References	169

5 Shortest paths in networks 171

**Christoph Witzgall, Judith F. Gilsinn
and Douglas R. Shier**

Introduction 171

Part I: Basic Methodology of Shortest Paths

5.1 Networks 171

5.2	Paths in networks	173
5.3	Shortest paths	176
5.4	Finding shortest paths	177
5.5	Matrix methods	178
5.6	The method of Dantzig	179
5.7	The method of Floyd	181
5.8	Labeling methods	182
5.9	Label correcting	184
5.10	Label setting	184
5.11	Network representation	186
5.12	Sequencing	187
5.13	Dijkstra's method	187
5.14	Dantzig's label setting method	189
5.15	Sorting	190
5.16	Dial's method	191
5.17	Pape's method	192
5.18	Back-up storage	195
5.19	Tree processing	196
	Problems	197
	References	199

Part II: Two Case Studies

5.20	Introduction	200
5.21	Railroad shortline distances	201
5.21.1	Coding the network	204
5.21.2	Checking the network	206
5.21.3	Partitioning the network	208
5.22	Transit information systems	211
5.22.1	Acyclic networks	214
5.22.2	Time-expanded network algorithm	215
5.22.3	Bipartite route/stop algorithms	218
5.22.4	Algorithm comparisons	220
	Problems	221

Part III: Extensions of Shortest Path Methods

5.23	Introduction	222
5.24	Shortest paths revisited	225
5.25	Other network problems	234
5.26	Nearly optimal paths	240
	Problems	252

6 Mathematical models for computer data communication 256

Alan G. Konheim

6.1 What is data communications? 256

6.2 Preliminaries 259

6.2.1 Counting processes 259

6.2.2 Generating functions 266

Problems 269

6.3 Exponential systems 270

Problems 282

6.4 The buffer process 282

Problems 290

6.5 Priority service 290

Problems 294

6.6 Stars and loops 295

6.7 Loops with priority 302

6.8 Polling 309

References 325

7 Operating system security verification 335

Jonathan K. Millen

7.1 Introduction 335

7.1.1 Security problems in computers 335

7.1.2 Security technology areas 335

7.1.3 The confinement problem 336

7.1.4 Access control 336

7.1.5 Indirect channels 336

7.1.6 Theory (a brief survey) 337

7.1.7 Preview 338

7.2 A computer architecture 339

7.2.1 System overview 339

7.2.2 Registers and memory locations 339

7.2.3 The instruction set 340

7.2.4 The microprogram 341

7.2.5 Privileged mode 342

7.2.6 Conclusions 342

7.3 Nonprocedural transition specifications 342

7.3.1 Transition rules 342

7.3.2 Inputs 343

7.3.3 Conditional expressions 343

7.3.4 Operations 343

- 7.3.5 Parameters 344
- 7.3.6 Indices 344
- 7.3.7 Terminology 344
- 7.3.8 Parnas convention 345
- Problem 345
- 7.4 A supervisory program design 346
 - 7.4.1 Processes 346
 - 7.4.2 Supervisor services 346
 - 7.4.3 Discussion of supervisor design 348
- 7.5 Access control verification 349
 - 7.5.1 Access control policy 349
 - 7.5.2 Invariants 351
 - 7.5.3 Proof example 352
 - 7.5.4 New invariants 353
- 7.6 Information flow analysis 354
 - 7.6.1 Insufficiency of access control 354
 - 7.6.2 Information flow concepts 356
 - 7.6.3 Authorization levels 356
 - 7.6.4 Flow policy 357
 - 7.6.5 Prevention of storage channels 358
 - 7.6.6 Explicit flow 358
 - 7.6.7 Conditional flow 359
 - 7.6.8 Implicit flow 359
 - 7.6.9 Indices 361
- Problems 362
- 7.7 Flow analysis in practice 362
 - 7.7.1 Objectives 362
 - 7.7.2 Effects of specification conventions 363
 - 7.7.3 Operations and parameters: the computer 363
 - 7.7.4 Operations and parameters: the supervisor 364
 - 7.7.5 Standard form of supervisor operations 364
 - 7.7.6 Mechanization 365
 - 7.7.7 Origins 366
 - 7.7.8 Constant authorization levels 366
- 7.8 Case study results 366
 - 7.8.1 Authorization level assignment 366
 - 7.8.2 Use of access policy 367
 - 7.8.3 Flow form operation choice variable 368
 - 7.8.4 A formal flow violation 369
 - 7.8.5 A real flow violation 370
- 7.9 Constraints 372

7.9.1	Drawbacks of information flow analysis	372
7.9.2	Deduction and compromise	373
7.9.3	More general deductions	374
7.9.4	Definitions	375
7.9.5	Covers	376
7.9.6	Consensus	377
7.9.7	An infinity of prime constraints	378
7.9.8	The prime constraint graph theorem	379
7.9.9	The construction of the prime constraint graph	381
7.9.10	Consequences for flow analysis	383
References		384
Appendix: Solution of elevator problem		385

1 A mathematical model relating to herbicide resistance

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1.1 Introduction

Since correct attitudes are almost as important as professional skill in successful industrial applications of mathematics, I will depart somewhat from the usual impersonal scientific style in these notes, to insert appropriate advice and comments where it seems relevant. A conventional account of the research described here, less detailed in several respects but containing many more biological comments and some further mathematical modeling, can be found in [1].

My involvement with this project began when I was approached by a colleague at the Weizmann Institute, Dr Jonathan Gressel of the Plant Physiology Department. Dr Gressel asked if I could provide a mathematical model to underpin some of his ideas on why plants seem not to develop resistance to herbicides.

(*Comment:* Perhaps the single most important attribute of a successful *academic* researcher is the ability to discern important problems and to pursue them until a successful conclusion is reached, almost regardless of how long this takes. By contrast, an *industrial* researcher will usually operate as a 'problem solver'. He should love the challenge of an office door that is open to a diverse set of people who can describe, with varying degrees of comprehensibility, problems from many different fields on which they would like a mathematician's help, usually with temporal and economic constraints that mandate the goal of *better* understanding a phenomenon, not *fully* understanding it.)

Conversations with Dr Gressel resulted in the following general outline of the problem.

Repeated use of various antibiotics has resulted in the appearance of bacteria that are resistant to attack by these agents, and an analogous phenomenon has arisen with respect to insect resistance to insecticides. It appears, however, that little weed resistance to herbicides has been observed by farmers and agricultural specialists. The question is, why not?

Under normal conditions, without herbicide spraying, one expects a cycle of seed *germination* (some seeds pass from a dormant to a growing condition), *establishment* (tiny young plants or *shoots* appear), growth to maturity, and scattering of seeds from each mature plant at the end of the growing season.

(*Comment:* One or more of the italicized words may be new to the reader, although there is nothing difficult in their definitions. In general, mastering the jargon of a new field is fundamentally a trivial task, but one which must be undertaken at once. I have found it helpful on occasion to compile a little word-list, just like a vocabulary list that one prepares when studying a foreign language. But here the task is relatively easy, for mastery of just a dozen or two new terms is often enough to break the terminology barrier.)

Two facets of typical weed growth bear special mention. One is that whatever the vicissitudes suffered by the weeds during the year, there is approximately a uniform number of germinating seeds per unit area at the beginning of the next season. Gressel calls this the *Parkinson effect*, for it is reminiscent of ‘Parkinson’s law’ that the amount of work expands to fill the available work-time.

A second special facet of weed growth, related to the first, is that far more dormant seeds can be found in the ground than will germinate in a given year, and that seeds retain their ability to germinate for a number of years. A typical finding concerning the slowly decaying viability of seeds is illustrated in Figure 1.1. Gressel felt that this ‘seed bank’ effect was an important factor in explaining the nonappearance of resistant strains. His reasoning began with the expectation that, in a large population of weed seeds or plants, there would be a few mutants that would be resistant to herbicide killing. Herbicide treatments are usually applied at the beginning of the growing season, when the weeds have just become established. These treatments should considerably increase the proportion of *resistant* shoots, compared to the *susceptibles* that are largely killed by the herbicide. (Typically, 10 percent or at best 1 percent, of the susceptibles remain after a spraying, and we can probably assume that the resistants are virtually unaffected.) But the effect of the resulting increase of resistant seeds will be markedly diminished by the presence in the ground of an overwhelming number of susceptible seeds in the seed bank.

A certain very small fraction of seeds each season is expected

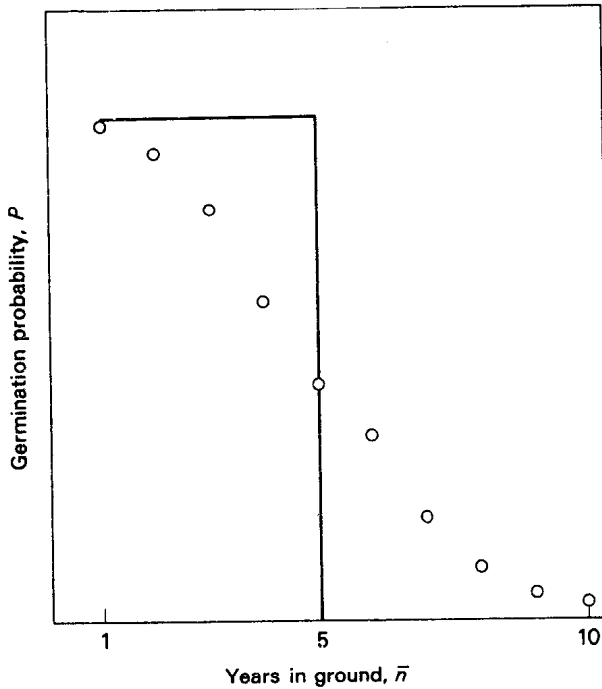


Fig. 1.1 Probability P that a seed will germinate (under optimal conditions) as a function of \bar{n} , its years in the ground. \circ , conjectured 'typical' experimental points; —, an approximation to the experimental results.

to mutate from susceptible to resistant or vice versa. This fraction could be as low as one in 10^{10} . Usually the mutants, while resistant to herbicides, will be 'less fit' in other respects, for example in the number of seeds produced per plant.

1.2 Formulation of a mathematical model

(*Pedagogical note:* At this point in the lectures I stopped and asked the class to tell me what to do next. I requested that they begin by telling me any possibly relevant mathematical or semi-mathematical statement to write on the blackboard.

It is my experience that this 'Socratic' approach to modeling is very successful. (a) With guidance of a greater or lesser extent, depending on how much time can be devoted to the exercise, students can collectively do most of the work necessary to formulate the desired model. Naturally, this is easier in problems like

the present one where little background is required and the situation is close to everyday experience. (b) Next to a genuine model-making experience, this approach provides the best training in model-building. Less good but still effective is very careful and responsible examination of classical models, with the same equation being derived by a number of different approaches (see Lin and Segel [4], Section 14.1). (c) Students, or subject matter experts who have come to the applied mathematician for assistance, are often unimpressed on being presented with a simple mathematical model. They are far more appreciative when they have struggled themselves with the formulation.)

Let us first attempt to model the ‘normal’ situation, in the absence of herbicide treatment. We will take into consideration both susceptible and resistant strains, however, with the latter less fit than the former (anticipating a later modification of the model to take account of the effect of herbicide). It seems natural to take a discrete approach to the problem, when one keeps track of the various effects during the n th year and thus sees what will emerge during the $(n + 1)$ th.

The Parkinson effect can be modeled simply by assuming that the same number N seeds per unit area will germinate. Of these, in the n th year a percentage σ_n will be susceptibles and a percentage ρ_n will be resistants. These percentages will depend on the relative numbers of susceptible and resistant seeds in the seedbank, and on the relative viabilities of these seeds. In a first model, which should normally be the simplest reasonable description of the situation, let us assume that seeds in the bank retain their initial viability for \bar{n} years and then cease altogether to be viable. This amounts to replacing the actual declining viability by a step function (see Figure 1.1).

We shall also assume (which is often the case) that only a relatively small number of the seeds in the bank ever actually germinate, so that in tallying the seeds in the bank one need only keep track of ‘deposits’; withdrawals can be neglected in calculating σ_n and ρ_n .

Let $N_i^{(S)}$ and $N_i^{(R)}$ be the number of susceptible and resistant seeds deposited in the ground at the end of the i th growing season. To determine the percentage of the N annually germinating seeds that are of the two categories, we shall merely count the total seeds in the ground for the past \bar{n} years and weight the count by a

relative germination factor χ . This gives

$$\sigma_n = \frac{\sum_{i=n-\bar{n}}^{n-1} N_i^{(S)}}{\sum_{i=n-\bar{n}}^{n-1} [N_i^{(S)} + \chi N_i^{(R)}]}, \quad \rho_n = \frac{\chi \sum_{i=n-\bar{n}}^{n-1} N_i^{(R)}}{\sum_{i=n-\bar{n}}^{n-1} [N_i^{(S)} + \chi N_i^{(R)}]}. \quad (1.1a,b)$$

The factor χ , $\chi > 0$, has been introduced here in such a way as to preserve the relationship

$$\sigma_n + \rho_n = 1, \quad (1.2)$$

necessitated by the fact that σ_n and ρ_n are essentially probabilities of mutually exclusive events. In most circumstances the number of resistant seeds will be very low, so that the term $\chi N_i^{(R)}$ can be neglected in the denominators of the expressions in (1.1). Then the meaning of χ is clear; it is the relative probability that a resistant seed will germinate. We shall assume that $\chi < 1$ reflects one aspect of the expected poor relative performance of the resistants in all respects except tolerance of herbicide.

Of the seeds that germinate, let β_S (β_R) be the proportion of susceptibles (resistants) that become established, ψ_S (ψ_R) the proportion of established plants that survive to the end of the season, and ν_S (ν_R) the number of seeds per survivor. Finally, let a fraction μ of each type of seed mutate to another type. With all this, the number of seeds deposited in the ground at the end of the n th growing season will be given by

$$\begin{aligned} N_n^{(S)} &= \sigma_n N \phi_S (1 - \mu) + \mu \rho_n N \phi_R, \\ N_n^{(R)} &= \rho_n N \phi_R (1 - \mu) + \mu \sigma_n N \phi_S, \end{aligned} \quad (1.3a,b)$$

where

$$\phi_S = \beta_S \psi_S \nu_S, \quad \phi_R = \beta_R \psi_R \nu_R. \quad (1.4a,b)$$

If we substitute into the above equations the expressions (1.1a) and (1.1b) for the probabilities σ_n and ρ_n , we find that the difference equations thus generated will describe the evolution of the weed population, providing that we prescribe an 'initial situation' of \bar{n} years duration.

The boxed equations (1.1) and (1.3) provide what might be termed a 'basic mathematical model of the phenomenon under

investigation. True, we have yet to incorporate the effect of herbicide spraying, but this only requires introduction of a factor α_s (α_R) to describe the proportion of newly established susceptibles (resistants) that survive the spray treatment.

Formulation of the basic model is often the most difficult step in the analysis. Once such a model is available, however, one begins to feel a measure of optimism that the analysis will eventually prove fruitful. The basic model may be rather intractable, so that simplifications will be mandated; additions and corrections may have to be made, but at least one has translated the essence of the given situation into a meaningful mathematical problem.

Before proceeding to consider the effect of herbicide, let us examine the normal situation. In so doing we shall take advantage of some intuition to simplify the equations considerably, so that calculations become very simple.

Under normal conditions we expect the resistants to be at a disadvantage, to be *less fit* in biological parlance. This we express by the inequalities

$$\chi < 1, \quad \phi_R < \phi_S. \quad (1.5)$$

The first of these we have already postulated; it refers to seed viability. The second inequality establishes overall susceptible superiority in the combined areas of germination, establishment, and reproductivity.

In the absence of mutation it is fairly clear that the inferior fitness of the resistant will lead eventually to its extinction. In mathematical terms, one expects that, given (1.5), the solution of equations (1.1) and (1.3), with $\mu = 0$, regardless of initial conditions, will be such that

$$\lim_{n \rightarrow \infty} N_n^{(R)} = 0.$$

With mutations, a small number of resistants should remain in the population, for every year a few susceptible seeds mutate to resistants. The 'back mutation' of resistants to susceptibles should be negligible, for this is a very small fraction of a relatively tiny number. Thus for realistic values of the various variables and parameters we should be able to approximate (1.1) by $\sigma_n \approx 1$, $\rho_n \ll 1$ (nearly all germinating seeds will be resistant) and (1.3a) by

$$N_n^{(S)} \approx N\phi_S \equiv N_C^{(S)}. \quad (1.6)$$

To find the small number of resistants we have from (1.1b) and (1.3b) the approximate equation

$$N_n^{(R)} \approx \frac{f}{\bar{n}} \sum_{i=n-\bar{n}}^{n-1} N_i^{(R)} + \mu N \phi_s. \quad (1.7)$$

Here we have employed the parameter

$$f = \chi \phi_R / \phi_s, \quad f < 1, \quad (1.8)$$

which we term the *preselection fitness factor*. Equation (1.7) has a steady-state solution that we shall denote by $N_C^{(R)}$; the subscript C refers to the control situation, in the absence of herbicide. We find from (1.7) that

$$N_C^{(R)} = \frac{\mu N \phi_s}{1 - f}. \quad (1.9)$$

As anticipated, the steady-state level is proportional to the mutation frequency. It is possible to show (*see below*) that the solution to (1.7) always approaches this steady-state value as $n \rightarrow \infty$.

We have assumed that the solution of our governing equations would tend to a state where the number of resistant plants is far less than the number of susceptibles. After simplifying our equations accordingly, we found a solution of the anticipated character. This consistency gives us confidence that our answers are a good approximation to the truth. Lin and Segel [4] (Section 6.1) discuss instances wherein consistent approximations are nonetheless inaccurate, but it appears that here we can be fairly certain that there are no hidden ill-conditionings that can give rise to the 'wretched consistent approximations' illustrated by Lin and Segel [4].

Let us now suppose that application of herbicide began in year zero, after a long sequence of normal years in which susceptible and resistant numbers are given by the control steady-state levels of (1.6) and (1.9). It is quite possible that the herbicide will lead to an increase in the numbers of resistants, but the ratio of resistants to susceptibles should nevertheless remain low for some years. Thus the general spirit of our approximate equations can be retained in analyzing the growth of the resistant population, although we must now modify (1.7) to take account of the percentage α_s (α_R) of newly established susceptibles (resistants) that survive spraying. This is done by multiplying ϕ_s (ϕ_R) by α_s (α_R). Thus, in the presence of herbicide, the behavior of a relatively

small resistant population is governed by

$$N_n^{(R)} \approx \frac{f\alpha}{\bar{n}} \sum_{i=n-\bar{n}}^{n-1} N_i^{(R)} + \mu N \phi_S \alpha_S, \quad n = 0, 1, 2, \dots \quad (1.10)$$

Here the *selection coefficient* α is defined by

$$\alpha \equiv \alpha_R / \alpha_S. \quad (1.11)$$

To analyze (1.10) we first look for a steady-state solution

$$N_n^{(R)} = N_{SS}^{(R)}$$

for all n . We find that

$$N_{SS}^{(R)} = \mu N \phi_S / [1 - \alpha f]. \quad (1.12)$$

This solution is positive and thus makes biological sense when

$$\alpha f \equiv \frac{\chi \phi_R \alpha_R}{\phi_S \alpha_S} < 1. \quad (1.13)$$

Indeed, in this situation the resistant has an ‘overall fitness’ (including herbicide resistance) that is less than the susceptible. Thus it is no surprise that a new steady-state level of the resistant can exist. We expect that the resistant level tends to the new value from arbitrary initial conditions; evidence in favor of this view will be presented shortly. When $\alpha f > 1$, the solution (1.12) has no biological meaning, but it is still a particular solution of the governing difference equation (1.10).

A little familiarity with difference equations (for example from Levy and Lessman [3]) reveals many analogies with differential equations. Here we are faced with a linear inhomogeneous difference equation, so we expect (and can easily show) that the general solution will be the sum of a particular solution of the inhomogeneous equation plus the general solution of the homogeneous. Indeed, if we write

$$N_n^{(R)} = N_{SS}^{(R)} + R_n, \quad (1.14)$$

we find that R_n satisfies the homogeneous equation

$$R_n = \frac{\alpha f}{\bar{n}} \sum_{i=n-\bar{n}}^{n-1} R_i, \quad i = 0, 1, 2, \dots \quad (1.15)$$

As mentioned, the initial situation will be taken to be the steady