PPR (1) 14 APR, 1991

(1) Chapter 12 from ~~Infections~~ Infectious Tropical Diseases & Domestic Animals, by G.J. Losos — Longman

Clinical signs & lesions of respiratory & alimentary systems involvement

Fourth memember of genus Morbillivirus of family Paramyxoviridae.

Human measles, canine distemper & rinderpest. (RNA) [PPRV same as Kata]

Cytopathic effect ~~6 = 15~~ between ~~16~~ 6 and 15 days after inoculation in ovine & Caprine kidney cells.

Has an Envelope with projections. Virion enveloped in helical particles about 500 nm long.

Pathogenecity retained in cultures at 40°C but not at 37°.

[why?] Virus grown at lower temperatures could be used to immunize animals.

Nigeria, ~~20~~ upto 20% of sheep & goats had sera which neutralized rinderpest virus. However, there had NOT been in contact with rinderpest infected cattle. (1966)

Another survey, from abattoir, 57% of sheep & 44% goats had serological evidence of previous exposure to PPR.

PPRV

Proportion of positive animals increased with age.
In goats and sheep, 25% animals were positive.
Large variation from region to region.

Infection transmitted by close contact, mainly by inhalation.

Virus excreted by all routes once clinical signs appear after a short incubation period of upto 5 days.
Does not persist in the tissues of recovered animals.

Carriers not ~~no~~known. But -
White tailed deer (Odocoileus virginianus) at times develops infections without clinical signs.

Disease highly contagious. Quickly spreads within a flock.
With onset of fever & diarrhea, excretion of PPRV is ~~&~~ in feces & nasal, ocular, buccal discharge.
Cattle → ~~no~~PPR not pathogenic. Neither replication nor excretion demonstrated, but antibodies are produced.

~~Acute~~ Peracute, acute & subacute syndromes.
Morbidity often upto 100%. Mortality can be upto ~~80~~ 90%.
Fever, nasal catarrh, stomatitis, diarrhea & death.

PPRV

Acute: Incubation 5 days.
course of 5-6 days terminating in death.
First clinical sign is fever,
Lasts for 5 days in animals that die
3 days in those which survive.
Fever followed by anorexia, inflammation of nasal & bucal mucosa.
Exudates profuse.
American goats 25% mortality.

Legions are confined to upper alimentary and respiratory tracts.
Exudates initially serous & later mucopurulent.
Complications by secondary bacterial infections. (Secondary pneumonia in 25% cases)
Legions extend to pharynx & esophagus.
Inflayement, hemorrhage & edema of the lymph nodes.

PPR protects cattle against rinderpest. conversely,
Rinderpest protects sheep & goats against PPR.
Antigenic homology between PPR & CD, MV & RV not greater than the known homogeneity between these three.

Bovine tissue culture rinderpest can be used as vaccine.
Only partial protection can be induced with MV or CDV.

(4)

Goats vaccinated with Kakeleo Rinderpest protected against PPR challeye for at least 12 months.

Neutralising antibody against RV was present before challeye, and higher titer against both developed after challeye with PPR

Rinderpest vaccine precipitated the development of PPR in apparently health goats which were incubating the disease.

---

challeye PPR was found to multiply in animals immunized with RV, CD or MV; but NOT in animals recovered from PPR.

Gibbs et.al. 1979 Interwirology 11 : 268-74

(5)

PPRV 5

Distribution & Epidemiology of PPR
W.P. Taylor Prev. Vet. Med. 2, 157–166 (1984)

Epidemiology of PPR & RV is completely seperate, and neither depends on the other for a continued existence in nature.

First reported in Ivory coast 1936
May be Senegal, 1871

How far back in the past ~~could~~ did RV & PPRV have a common ancestor?

same question for CDV, MV etc.

RV infects sheep & goat from time to time.
India, a small ruminant adapted strain of RV appears to have evolved (1974)

Incubation period 4 – 5 days.
Broken by ~~Pyrexia~~, onset of pyrexia (peak 40–41°)
on second or third day of illness.
Temp. remains high for another few days before slowly returns to normal. While febrile period complete in 6–8 days from onset

PPRV

Death ~~usu~~ Animals less interested in food, & become depressed. Death towards the end of the period & acute disease.

serous nasal discharge commences with ~~on~~ onset of pyrexia. This may progress to give profuse catarrhal discharge. much sneezing to clear the nose.

Progression varies between animals & may depend on secondary bacteria.

~~Ne~~ Small areas of necrosis in the mouth 2-3 days after onset of pyrexia. These may heal ~~in~~ within 48 hours. Else, they increase & eventually diarrhoea develops.

Less severe in sheep than goats, but not necessarily.

Virus when injected in British livestock shows severe disease in goats, mild in sheep & none in cattle. But cattle are resistant to subsequent challenge with virulent RV.

Cross protection is seen, but no cross-neutralization Hence PPRV & RV are distinct.

PPRV

PPR spreads by CONTACT both among & between species.

Substantial quantities found in ocular, nasal & oral secretions & of sick goats & in faeces late in the disease.

Infected bedding may contribute.

Mainly through inhalation of the infectious aerosol, produced by combination of sneezing & coughing at the height of the disease.

Cattle → act as dead-end hosts.

[Calves may ~~be~~ show pyrexia & oral erosion]

High morbidity & mortality in flocks.

Morbidity 77%; case mortality 82% Nigeria North

527 goats (five field outbreaks)

62% morbidity, 54.8% case mortality. South Nigeria

90% morbidity 60% " Saudi Arabia.

Sheep have innate resistance, but occasional field strain can originate, which can overcome it.

PPR

Disease throughout the year; a peak in ~~August~~:

Peak of kidding in February & maternal immunity of 3-4 months, so susceptible state attained in July.

Outbreaks occur after acquisition of sheep from market, return of ~~to~~ unsold lambs, heavy trading in festival season etc.

PPR → waves of infection at 3-5 yearly intervals.

But antibodies occured in a proportion of goats of all ages from 4 months to 2 years.

In outbreak, majority of affected animal are 1-2 year old. (Those over 2 years may have already been infected).

Heterologous antibody present, but low, so that 10^{-1} serum will neutralise only the homologous one.

PPR (Goat plague) Virus (9)

PPRV

- G.R. Scott. in

Z. Dinter & B. Morein Ed.

Virus Infections & Vertebrates vol. 3: Virus Infections & Ruminants.

Elsevier Scince Pub. B.V. Amsterdam 1990.

Half life of hydrated virus ≈ 2 hrs at 37°C

Infectivity destroyed in 30 min. at 50°C.

molar sdn. & $MgSO_4$ slows heat inactivation — Why?

Also sensitive to lipid solvents & low pH.

~~Re~~Stable in lymph nodes. Recoverable from carcasses held for 8 days at 4°C.

PPR does not hemagglutinate; but antibodies to PPR inhibit hemagglutination of monkey erythrocytes by MV.

More older goats recover than younger goats.

African goats mostly die; not so for European goats

Sheep - subclinical most often, & animals recover.

Epizootics mostly seen in village flocks.

similar incidence of infection in sheep & goats. antibodies rose with age to 74% in animals over 3 year old (Serological study)

Affected animals mortality - 70-80%

In rainy season in humid tropics, goats are kept indoors & huddle together. Virus spreads by aerosol route. Explosive epizootic,

PPRN

All ~~do~~ adolescent are affected. Whole herds are lost.
Source - New animals or return from Market unsold.

PPR through aerosol route, invades body through tissues lining the upper respiratory tract.
Disseminate before the onset of clinical signs.
Shed by all secretions. Quantity increases as disease progresses.

(i) Incubation ~ 6 days, after which sheep and less commonly, goats, develope subacute reactions. Low fever, nasal catarrh etc.
Recovery after 10 - 14 days.

(ii) Acute - incubation 3-4 days. Sudden fever. Erosion in upper alimentary & respiratory tracts. 1-2 days after the onset of fever. These coalesce, many lesions in further 2-3 days
Most die within 10 days.

(iii) Peracute incubation 2 days. Sudden onset of fever.
Diarrhea sets in within 3-4 days. Death 4-6 days after the onset of illness.

---

within 24 hours of fever, LEUKOPENIA is evident, & persists till death, or for 10 days in animals which recover.

PPRV

Pasteurella Pneumonia often sets in in acute cases.
Wide range of secondary bacteria invade.
Mycoplasma capri & PPR together have far more severe effect.
Enteric & hemoprotozoal infection, exacerbated by latent viral infection (Adenovirus) activated.
Multinucleate giant cells with intranuclear inclusions are formed.
Immunity lifelong.
Attenuated rinderpest protects. Vaccinated animals develope RV antibody but not PPR antibod.
When challenged with PPR, ~~[illegible]~~ homologous antibodies appeared anamnestically.
Colostral immunity protects kids & lambs until weaning.
Artificial passive immunity successful, by injection of hyperimmune serum.
Virus samples to be obtained from febrile animals, killed with 5 days of onset of fever.
PPR has not been detected in India, where rinderpest in sheep & goat is not uncommon.
Chloroform ~~[illegible]~~ inactivated vaccine effective for 1 year at 4°C.
Hyperimmune serum useful to limit spread, but no effect on diseased animals.
Biannual vaccination of adolescent kids recommended.

(12)

PPRN

The Epidemiology of PPR in the Sultanate of Oman

- W.P. Taylor, S. Al Busaidy & T. Barrett

Veterinary Microbiology 22 341-352 (1990)

- [Omani strains evolved independently of African strain, by adaptation from RV?]

Sick animals invariably belonged to small holder farmers, who could not prevent contact between animals (own or neighbours). [~~Towns~~ Town is a focus of disease; many free rangy animals present.]

Incubation 4-7 days, pyrexia daily 4-8 days.

Of 568 small ruminant sera, ~~none show~~ at 1:10 dilution NONE neutralised RPV, while 167 ~~were~~ +ve against PPRV.

PPR +ve					TOT
Sheep	14/43 (33%)	22/79 (28%)	1/12 (8%)	0/22	37/156 24%
Goat	30/123 (24%)	45/125 (36%)	22/82 (27%)	5/82 (6%)	102/412 24%
Tot	44/166 (26%)	67/204 (33)	23/94 (24)	5/104 5%	

Age	1-2	1.5-3	2.5-4	3.5-7
household with disease	11/15 (73)	–	2/2 (100)	10/11 (91%)
without	0/11	2/2 (100%)	–	8/11 (73%)
farm	0/5	–	–	0/5

when free rangin, mixing groups + nomadism is practised, infections are seen.

54/249 (21.7%) yearlys had been infected, 77/183 (42%) in 3-4 year olds. This implies endemism.

Enclosed farms, large villages free of disease

Enclosed farms 42/55 (75%) vs enclosed (less than 10%)

4 Feb 1992

Rhesus — 17 animals, 27 behav.

Hanuman Langur 17 animals, 27 behav

(i) PCA + plotting in first two comp. space for Rhesus

(ii) " for Hanuman Langur

(iii) " for pooled (34) sample.

The names need not be keyed in.

(iv) Single linkage clust for Rhesus.

dist ~~similarity~~ $S_{ik} = \sum_{j=1}^{27} (x_{ij} - x_{kj})^2$

(v) → H. l.

(vi) — both.

120ct 92

Let T_n = No. of traits shown by n communities.

As n increases, T_n increases, but sublinearly.

Three possible functions are (many more may be imagined!)

i) $T_n = T_1 \cdot n^z$

ii) $T_n = T_\infty (1 - e^{-an})$

iii) $T_n = \dfrac{T_\infty n}{b + n}$

Here T_1 and T_∞ correspond to traits by 1, and by infinite no. of communities.

The proportion of unshared traits by a pair of communities will be

$d = \dfrac{T_2 - T_1}{T_1}$, where d denotes dissimilarity.

(ii)

For the three functions described above, the dissimilarity turns out to be

i) $\frac{T_1 \cdot 2^z - T_1}{T_1} = 2^z - 1 = d_1$ or $z = \frac{\ln(d+1)}{\ln 2}$

ii) $\frac{T_\infty(1-e^{-2a}) - T_\infty(1-e^{-a})}{T_\infty(1-e^{-a})} = e^{-a} = d_2$

or $a = -\ln d_2$

iii) $\frac{\frac{T_\infty \cdot 2}{b+2} - \frac{T_\infty}{b+1}}{\frac{T_\infty}{b+1}} = \frac{2(b+1)}{b+2} - 1 = \frac{b}{b+2} = d_3$

or $b = \frac{2d_3}{1-d_3}$

Thus, knowing d_3, the pairwise dissimilarity, one can estimate

(i) $z = \ln(d+1)/\ln 2$

(ii) $a = -\ln d$

(iii) $b = \frac{2d_3}{1-d_3}$

(iii)

Similarly, average no. of traits per community gives us T_1.

Thus, using the observed values T_1 and d, we can write down the ~~expressions~~ three expressions as

i) $T_n = T_1 \, n^{\left[\frac{\ln(d+1)}{\ln 2}\right]}$

---

ii) Since $T_1 = T_\infty (1 - e^{-a})$, and $e^{-a} = d$

$$T_\infty = \frac{T_1}{1-d}$$

$$\therefore T_n = \frac{T_1}{1-d}\left(1 - d^n\right)$$

(which is the expression we discussed yesterday!)

iv

(iii) $$T_1 = \frac{T_\infty}{1 + \frac{2d}{1-d}} = \frac{T_\infty (1-d)}{(1+d)}$$

$$\therefore \quad T_\infty = \frac{T_1 (1+d)}{1-d}$$

and $$T_n = \frac{T_1 \frac{(1+d)}{1-d} \cdot n}{\frac{2d}{1-d} + n}$$

$$= \frac{T_1 (1+d) \cdot n}{2d + n(1-d)}$$

145 24
" "
2.63
200 40 4000
55 " 1.1
363

Typically, $T_1 = 200$, $d = 0.45$

i) $T_{\cancel{4625}\,100} = 2000$ ~~$~~

ii) $T_\infty = 364$

iii) $T_\infty = 526$

(since $Z \simeq 0.5$)

So the third model seems to be the least unsatisfactory of the lot! Also validated by curve fitting!

17 Jan 93		18 Jan 93		19 Jan 93		24 Jan 93	
P	N	N	P	P	N	P	N
14 14	10 10	14 14	3 3	28 28	22 22	18 18	11 11
7 21	28 38	26 40	12 15	17 45	17 39	14 32	14 25
33 54	19 57	7 47	20 35	- 45	17 56	13 45	13 38
4 58	32 89	15 62	- 35	- 45	20 76	11 56	31 69
71 129	- 89	13 75	10 45	16 61	33 109	9 65	- 69
9 138	21 110	15 90	85 130	21 82	~~7~~ 116	16 81	8 77
- 138	10 120	10 100	14 144	13 95	14 130	- 81	- 83
26 164	18 138	7 107	- 144	36 131	~~4~~ 134	10 91	31 114
45 209	13 151	11 118	- 144	20 151	~~12~~ 146	38 129	22 136
20 229	27 178	4 122	- 147	20 ~~17~~ 171	71 217	21 150	9 145
14 243	14 192	48 170	14 161	40 211	36 253	22 172	18 163
14 257	13 205	20 190	13 174	8 219	5 ~~258~~ 258	24 196	18 181
15 272	13 218	20 210	2 176	18 237	26 284	12 208	16 197
11 283	10 228	51 261	81 257	6 243	31 315	14 222	9 206
17 300	73 301	18 279	13 270	7 250	18 333	12 234	8 214
24 324	22 323	33 312	30 300	23 273	15 348	10 244	19 233
25 349	7 330	14 326	16 316	20 293		26 270	20 253
15 364	2 332	18 344	4 340	+4 297	-5 343	12 282	18 271
3 367	6 338	13 357	9 349			24 306	17 288
8 375	6 344	7 14	- 349	20 ~~317~~ 315	- 385	24 330	4 292
+3 372		4 368	6 355	10 325	8 393	-9 321	+9 301
		8 376	-4 351	-5 320	+5 398		
		+2 380					

P	N	20 Jan 93
- 0	28 28	
7 7	20 48	
- 7	22 70	
~~12~~ 13	28 98	
2 15	20 118	
71 86	15 133	
19 105	10 143	
9 114	13 156	
3 117	10 166	
23 140	13 179	
~~22~~ 168	18 197	
42 204	23 220	
16 220	69 289	
30 250	33 322	
12 262	33 355	
~~34~~ 293	30 385	

| 24 Jan 93 | | | | |

26 Jan 93		30 Jan 93	
P	N	P	N
88	10 10	88	6 6
715	4 14	1422	11 17
8196	36 50	224	10 27
33129	10 60	-24	24 51
-129	14 74	1539	31 82
131421	95	1857	30 112
-142	20 115	3 60	17 129
48190	9 124	5 65	19 148
1201	16 140	1479	6 154
213	23 163	685	20 174
217	19 182	39174	28 202
-217	7 189	18192	10 212
7791	420 209	9201	9 221
16310	29 238	77278	54275
16326	17 255	20298	24 299
18344	14 269	17315	13 312
10345	5 274	25340	16328
28382	16 290	24361	13341
153977	7 297	11372	12 353
-397	10 307	6378	-1352
7404		+379	
6 410	-10		
24124	297		
T10422			

30 Jan 93	
N	P
32 32	2 2
11 43	91 93
5 48	26 119
16 64	21 140
24 88	6 140
22 110	8 148
9 119	3 151
4 123	26177
33	32 209
4	12 221
16 160	24 245
29 189	15 260
3201	-260
18219	11 271
20239	12 293
30 269	-293
23292	18311
16308	36347
12320	15362
12 332	6 368
4 336	3 371
3 333	+3374

and Crick made it clear that since the two chains are intertwined, "it is essential for them to untwist if they are to separate." Noting that "a considerable amount of uncoiling would be necessary" they admitted that "it is difficult at the moment to see how these processes occur without everything getting tangled." Yet they did not feel that this objection will be insuperable" (1953 b, 964). One important consequence of the exploration of the genetical implications was that the two salient aspects of the double-helical model, the physical structure and the biological hypothesis of replication became two distinct domains of investigation. Of the two, the physical structure was established as correct much more quickly than the biological hypothesis. Wilkins (1956, 75), for example, observed: "It seems necessary to stress that correctness of the structure does not imply correctness of the biological hypothesis."

Watson and Crick (1953 c) presented a more detailed consideration of the unwinding problem which the

1 Aug 1993

The papers on Rinderpest epidemic modelling by James & Rositter:

The papers describe a realistic, rigourous, ingeneous and simple model to describe the spread of rinderpest in a ~~given~~ single herd.

It predicts the number of infected animals on a day to day basis, from the information on the number of susceptible, infected & immune animals, time course of the disease, and the contact rate.

The model can be used to answer questions such as:

(i) will the disease ~~reach an~~ lead to an epidemic? If so, what will be the mortality and survival?

(ii) what herd sizes, strain virulence, contact rates etc. determine endemic ~~or~~ epidemic persistence or non-persistence?

(iii) What vaccination strategies (levels, frequency ~~of~~ of vaccination) are needed to ~~cont~~ prevent epidemic?

The authors seem very thorough, have gone out of their way to emphasize the limitations, have ~~clear~~ taken pains to describe the model and parameter estimation with great detail and clarity. More impressive is the fact that they already have some field success with their model.

—x—

What can be done next?

(i) Thanks to the details given by the authors, it is relatively straightforward to set up the model; it can be done in a weeks time, and with ~~lucky~~ luck, much less.

(ii) If one just goes by these two papers, there is a lot of scope to play with the model (of course, the authors may have done a great deal in the last five years!).

(a) The entire range of parameter values e.g. contact rate, initial immunity level, etc. can be scanned, and the combinations of these which lead to ~~persistence vs~~ epidemic vs endemic persistence can be estimated.

(b) While theirs is a stochastic model, one can explore the underlying, less realistic, deterministic model perhaps analytically & computationally, (i.e., without simulation). A comparison of these results with ~~the~~ those from stochastic models will give an idea of the ~~relative~~ importance of chance factors.

(c) Simply by running the simulations a very large number of times, statements about probability of persistence for a much longer duration (e.g. several years) can be made, while the authors, (at least in this m/s) have stopped at, (perhaps as an illustration) one year.

(iii) Since PPRV is probably similar to rinderpest in biology, but perhaps different in disease parameters (e.g., incubation period, mortality rate, virus excretion duration etc.), the model may be run for PPRV to answer more or less similar questions as done for rinderpest.

(iv) One of the limitations of the model, as repeatedly underlined by the authors, is the assumption of complete mixing ~~(homogeneous)~~ of the herd; each susceptible animal is equally likely to be infected. This can be modified in various ways.

(a) A typical village composed of many small herds which spend nights in the same stable, but ~~one all mixed when taken~~ animals from different herds mix when taken out for grazing during the day.

(b) At the next level, herds from various villages mix at the weekly market.

(c) Many weekly markets within a region (district), and a village may participate in one of the several markets, changing markets from week to week; ~~All animals~~, and simple modifications of this schedule.

(v) If vaccination is to be undertaken, it has to be at a sufficiently high level if it is for the

control of epidemics. Vaccinating only a few animals may not serve any purpose.

The model may be used to estimate the minimum level of vaccination necessary for preventing epidemics.

(vi) Modification of the model to include population of wild animals may be attempted. This could include the possibility of the spread of infection from domestic cattle on the periphery of a sanctuary. Effect of vaccinating the cattle in these areas (as ~~happend~~ happened in Africa) could be modelled.

{ from here onwards, things are more and more nebulous, & imprecise & speculative.

(vii) One of the intriguing points made by the authors is about the evolution of milder strains, which can persist much longer. They suggest that the reduced severity of outbreaks may be more likely due to the evolution of such strains than due to the developement of disease resistance in hosts.

It may be interesting to set up population genetic models to explore the evolution of different levels of virulence, perhaps under the scenarios of different levels of mixing, different levels of vaccination etc.

(viii) While modelling wildlife is practised extensively, and perhaps including the impact of diseases, ~~it may~~ the main focus is on mortality. For diseases such as mild rinderpest, ~~reduced~~ a reduced level of well-being (fitness!) is more likely. It may be interesting to quantify how the effects of this reduced vitality translate into ~~long~~ long term survival rates.

(ix) What (at the molecular level) are the similarities and differences between the viruses which affect similar species? What may be the changes brought about by processes such as murinization, lupinization etc.? What may be the data needed to answer such questions (sequences? antibody affinities?)

Sequence info is needed.

(1)

16AUG93

1. ~~For~~ For two sites, data is at weekly interval for 3 years.
Does it mean at each site, there are $52 \times 3 = 156$ values?
At the other two sites, are there $12 \times 3 = 36$ values?

2. What does each data collection involve?
Is it a fixed no. of net sweeps (5 – 10 – 15?)
Is the 50 m^2 area subdivided into say
two areas of 25 m^2, or 5 areas of 10 m^2 etc.
All these would be valuable to determine within site within
a day variability.

3. What do the three abiotic factors mean?
e.g. Temp at the time of net sweep? Max – min – mean day day!
Rainfall of the week, of the day, or at that instant?

4. I have no idea of what grasshoppers are, but if on
the names I guess they would like grass. Do you have
data on ammount of grass or some similar

5. Are there any known seasonal patterns? If the
population depends on phenology, which in turn depend on
seasons (day length etc.), then it may be difficult
to obtain influence of abiotic factors

6. If I have understood your question correctly,
I'd like
$$N_t = f(T_t, R_t, H_t)$$, i.e. the grasshopper

(ii)

Population at time t as a function of the temp, ~~Rel~~ Rainfall, Rel. humidity.

It does not seem very reasonable.

7. The data may be entered in lotus 123 as 150 Rows, 3 col. for ~~abiotic~~ and my 3 for T, R, H & 1 (or more) for grasshopper population N

8. You can then just ~~excel~~ look at graphs

(i) N vs T, (ii) N vs R (iii) N vs H

(iv) may be N_{t+1} vs N_t

You may have done this already.

9. If seasonality is not known, you may ask questions such as

(i) Does population vary from month to month?

(ii) ~~Do~~ Is there a variation from year to year?

You can by t test, analysis of variance i.e., which you may have done already.

CNRR. Colloquium. | GP — There are many Rao's but only one CNR Rao
Commonly benefit with heroes. so CNR is a role model
5 Oct 93

b 1934. M.Sc. 1951 | Benares by Res.

B Sc in Physics, central college. → Physics Prof. said "Res is very difficult".

Some work in IPC (IISc); then Kharagpur IIT 1951–53.
Ph.D. in Purdue – electron diffraction in gas phase.
Worked on X-ray as well. with many others.
Repeated Pauling's work on Al–Mg alloys by better instruments
Results same -- Intuition is very important. – Reference in Pauling's book.

Physical Chemistry officially was born in 1934.
Ostwald – Goltzen as physical chemist, half salary of Org. Chem.
even after Nobel prize.

Worked on oxides/metal oxides from 1960 ~ 1984. Wrote major review.
(Afterwards, 1986, superconductivity was discovered)!
Bhatnagar award in 1968 – (Paid heavy price i.e. got the award)

Worked on phase transition, defects in solids etc.
Always wanted to study solid state – structure, synthesis, reactivity.

One must not work in the same field. Always exciting things happen in many fields, so one should change.
Fullerenes – worked in it because everyone in country asked him to.

Young people don't want to work in new areas. He won't wait for them in new areas – metal clusters, diamonds etc. but will work with students. Inorganic ~~ca~~ carbonates ⟶ hydrocarbons.
By ~~#~~2050 no hydrocarbons – when his grandson will be 60 years old
but being a Hindu, he beliefs he will be reborn, so work for next generation.

Physicists work (scratch their head) only on systems far removed from reality. He works (like Rutherford) to keep theorist near, but not so near as to influence the experiments

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(a) computational veterniary medicine

(b) computational Disease modelling

(c) computational demographic ~~epidemiology~~

epidemiology and statistics / Biostatistics.

Dedicated Comparative Sequence Editor

11 Dec 93

DCSE 2.60

uiam3.uia.ac.be

143.169.8.1

ftp uiam3.uia.ac.be

cd [.dcse]

dcsepc.zip

manuals

examples

bitmap options

24 Feb 94

1. Repeat
Jones / Rossiter / Pastret model
with parameters for PPRV

2. Repeat RV models with
mixing studies for goats / sheep.

3. Dose & previous exposure
dependent probability of infection
— TJRP models.

(I)

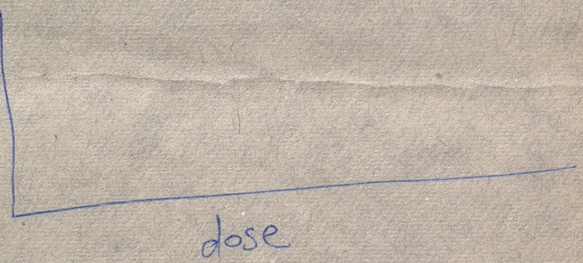
24 Feb 1994

1. Would a repeatation of the same exercise for PPRV / sheep-goat system be of interest to anyone? ✓

2. Are the parameters for PPRV available (the rinderpest values on p 449)?

3. What would be the consequence of contacts between infected – susceptible which are NOT intense enough to lead to infection?
Would it lead to immunity? (Prof. R. Nayak feels so).

(a) What happens to the animal when it is infected by a single dose of virus?
Qualitative shape?



(b) How would this alter if instead of one large dose, several small doses are given?

4. If the above point about low dose leading to immunity is true; would it be of interest to try this out for rinderpest itself?

P.T.O.

5. What would be the questions of interest for the ~~zine~~ RV/PPRV competition?

(i) How is the probability of ~~epide~~ outbreak of PPRV affected in a population which ~~have~~ has once been exposed to RV?

(ii) Will PPRV/RV stay in equilibrium, or will one outcompete the other?

Will ~~one~~ there be cycles of PPRV-RPV-PPRV-RPV epidemics?

(iii) Can a simultaneous outbreak occur?

It is known that animals immunized against RV are also able to resist challege by PPRV and vice versa. Against this backdrop, it would be interesting to investigate the competition between the RV and PPRV, in the arena of natural livestock ecosystem — cattle, sheep, goats etc., both from an epidemiological as well as evolutionary point of view.

Recent years have seen the initiation by the modern molecular biological techniques of recombinant DNA, vaccines against RV, and also PPRV are the research on designing vaccine against RV and the PPRV, using recombinant DNA technology. The models developed epidemiological mathematical models would also be used to investigate the effects of vaccination. In particular, these may be useful in devising optimal vaccination strategies, minimizing the which would lay down the risk of PPRV outbreak by i.e. the patent an which is to minimize the risk of outbreak in a cost-effective manner.

Peste des petits ruminants in India 1 March 94

S. Ramachandran and R. Nagaic

Curr. Sci. 63 55 1992 (25 July 92)

(1)

15 APR 94 RV

Rinderpest

G.R. Scott Adv. in Veterinary Science 9, 113-24 (1964)

Known since 4th centy AD
Killed 2 M cattle in 1949

ALL epizootics traced to imported livestock
Goat attenuated virus as vaccine successful

£40000 outbreaks, 1 million deaths in 10 years.

Virus contained in leucocytes on attacked by them.

Dia 1200-1300 Å or 1500-3000 Å.

Outer membrane, fringed with projections.

Tightly coiled component 180 Å in dia., with serrations along the length at periodicity ~~4~~ 50-60 Å

Heat inactivation, half life 5 min at 55°, 905 min at 37°, 6.4 hrs at 25°C & 2.3 days at 7°C.

Activation energy was 24 Kcal mol^{-1}, entropy 82 cal deg^{-1} $mole^{-1}$

Leucocytes may contain more than one virus.

RV used as vaccine in ferrets against canine distemper

All die in 4 days when challenged, 19/24 die after 5-10 days but all survive after 11 days.

Calves with hyperimmune serum & CDV + virulent RV survive.

Those without serum die.

CD & MV show hemagglutination, but RV did not; may be technical problems.

RV

RV infected animals looked like struck with pox, due to cutaneous eruptions in the past. Currently, these are not seen.

Order Artiodactyla are natural hosts.
[Article has a list of animals]

Repeated passage of virus through goats attenuates it for cattle. 50 passage kills in 7-9 days, 69-80 in 12-16 days. First survive after 83 days. After 250 passages, it is free from virus for use as vaccine.

~~Foot~~ Goats infected with ox strain of virus
Incubation 4-5 days, followed by sudden fever.
After 3 days, discharge nasal etc.
6-10 days after fever, congested gums.
Death after 2-3 weeks.
Mortality 2% to 75%.
Some strains 15%, others 50%, other 95-100%.

Kabete 'O' strain was adapted to cell culture.

Lymph nodes, spleen rich in virus. In blood, attached to leucocytes.
Virus injected into actively immunized cattle disappears completely.
Virus seen in all secretions & excretions during fever.

RV

Nasal discharge - prime route & excretion.
It ~~is~~ is infectious 2 days before the onset of fever and remained so 2-3 days after complete remission of fever.

Urine ~~&~~ 3 days after fever, feces 6 days after fever & non-infectious 3-5 days after fall in temperature.

☆ Idnani J.A. Transmission & virulence by expired air.
Ind. Jour. of Vet. Sci. 14 : 216-220 (1944)

Minimal ~~distance~~ exposure between sick & health cattle was 6 hrs at a maximal distance of 6 feet.

Nasal exudates detected in some animals 2 days before the onset of fevers (6 days before clinical signs); by 4th day of illness, 88% cattle were nasal excretors. Excretion ceased after 9th day.

BLOOD

$\log_{10}$ ID_{50} goes from 0 at -1.5 days after onset of fever to 4.5 at 2.5 days after onset of fever.
Then reduces to 0 ~~after~~ at 10.5 days after fever.

Goat adapted; same titre, man at same time, but to 3.5, & 0 at 9.5 days. (exponential growth & decay)

RV

In lymphoid tissue, start at −2.5, peak at ~6 at 1 and fall to 0 by 14.5 day.

[Plowright W., 1962, Ann. N.Y. Acad. Sc. 101: 327-382]

Intracellular increase exponential from 4–48th hours.
Peak plateau persists till 6th day, followed by slow decline.
Mean # per cell ~ 3.7 to 0.5 when all cells were affected.
Small particles develope within or contiguous to mitochondria, and preceded the formation & the large complete virus particles.

Complement fixing antigen start −1.5, peak-plato 3–5, 0 at 10.5

precipitiingen +1.5, " 5–6 − 0 at 9.5

Goat adapted virus may interfere with virulent RV.
Cattle injected with vir. RV before, along, and after upto 33 hrs. showed rinderpest symptoms; but not after 48–57 hours.

Rabbit virus — 88 hrs.
Avianized 96 hrs; but antibodies only after 8–12 days
Cell culture. 3–5 days " " 6th day.

Dutch cattle resistant. Indian hill zebu non-resistant, but plain zebu highly resistant
Breeds derived from crosses tend to be non-resistant.

(5)

Nellae bulls are more resistant.

Resistant races evolved by natural selection.

RU

Pigs can be infected by drenchy with blood; or fed with infected tissue.

[Scott G.R. & Ramplan C.S. 1962
J. Comp. Pathol. 72 : 299: 302
~~also~~ 65 : 236-238 1955

Incubation period - 3 — 15 days.

☆ P. 163 diagram to be removed

~~Also~~ Calves resistant to infection 8 — 12 months if born to recently ~~infected~~ immunized mothers; else 4 months.

P. 163 → From onset of fever

Leucocytosis - 3 to 2 ; Leucopenia 3 — 14 (1 month)

Fever - 1 to 5-6
Constipation - diarrhea 1 — 10 ~~Also~~
Nasal discharge 2 — 11
Mouth lesion 2 — 9
Death 5 — 13
Dehydration 7 — 14

(6) RV

Rinderpest

Gordon Ramsay Scott.

Infectious Diseases & Wild Mammals. ed. Davis J.W. et al

Iowa ~~Un~~ State Univ. Press 1970 (p 21-35)

Many current anomalies in the distribution of wildlife in Africa can be traced to Rinderpest panzootic.

Uganda & Kenya — 60% Eland, ~~Kund~~ Kudu, warthogs, 50% buffalo, bushbuck & giraffe, 40% of impala ~~perished~~ died.

Does not hemagglutinate.

Virus particles enveloped in helical particles of dia 1250 Å.

Tables for host species.

Infected carcasses in tropics become harmless within a few hours.

Infections are airborne.

Infected animals infection before the end of incub. period (~~but~~ when apparently healthy) and continue till start of convalescence.

Natural incubation in cattle & buffalo 3 – 15 days.

Onset of peracute & acute is sudden high fever for 2-3 days.

Most die within 3 – 4 days. (~~with a~~ about a week after illness is evident.

RV

R. virus destroyes lymphoid tissue. It enters the body through mucosa of upper respiratory tract. Then disappears for several hours. Primary multiplication in pharyngeal lymph nodes, tonsils. Onset of viremia precedes that of fever. Disseminated by polymorphonuclear neutrophils. Multiplication in epithelial cells follows. Excreted from lungs.

Two weeks after onset of fever, animal is free of detectable virus.

(8)

RV

Rinderpest

Chap. 15 in Infections ~~diseases~~ Tropical diseases & Domestic animals by G.J. Lasos - Longman (1981 or so)

Virions are enveloped helical particles; pleomorphic; majority are spherical, but some are filamentous.

Fragile, and unlikely to survive for many hours outside the host.

HeLa cells - titer within cells (intracellular) 10—100 times greater than that of free virus.

Bovine kidney cells; cytopathic effects prominant on 5th passage, and peak titres 6-8 days after infection.

Virus is adsorbed to the cell within 8 hours

~~eclipse~~ eclipse phase 14-20 hours, which ends with the progeny of input virus.

Growth phase — peaks after 48-72 hours.

Enzootic areas:

Calves have passive immunity: Adults are actively immune due to natural infection. ∴ Immature & young animals are most at risk.

Mortality 30%.

Rinderpest epidemic 'burns itself out'. Animal with low resistance important in dissemination, but not in maintainance.

RV

Sudan; sheep & goat, mortality 60-70% in young stock & 20-25% in adults. ← PPRV → (not rinderpest!)

Nigeria - 75% cattle (& 10 millim) immune to Rinderpest.
~~18%~~ 18.8% sheep & 15.2% goat had antibody to RV
[Due to PPR infection]

Fields & contaminated stables infective upto 4 days.
Virus present in feces upto 8 months after recovery.
~~Milk~~ present in milk upto 45 days after recovery.

Mortality in experimentally infected buffalo - 25-30%.
overall susceptibility, 90%.

Course of infection Incubation Day 1 — 4
prodromal 5 — 7 — onset of fever to appearance of lesions.
mucosal 8 — 12
early convalescent 13 — 16

Low level viremia began on 2nd or 3rd day after infection, generalized by the end of incub. period.
By 5th day after infection, high titre in lungs. By 9th day onwards low titre (& false antibodies presence was seen)
Virus disappeared during early convalescent period.

AV

Lack of pathogenicity when the virus fails to proliferate in gastro-intestinal or respiratory tracts.

Peracute – death with 1-2 days after clinical sign appear
acute – symptoms last 4-7 days
mild – animals may live upto 3 weeks or longer.
Low virulence - 10% develope asymptomatic infection.
Newly infected - 100% morbidity, 90% mortality.
Incubation - 3-9 days. longer in enzootic areas.
Followed by Fever, followed by 1-2 days by diarrhoea.
Temp. peaks between 3-5 days. 8 days as diarrhoea begins
Death with 6-12 days.

~~Post~~ Infection → fever, the interval is incubation.
Artificial - 3-9 days, Natural 8-15 days.
Long duration, less severity.
Fever starts when virus has spread over the body.
Mucosal lesions commenced 2-5 days after onset of fever.
at this stage, high conc. of virus in tissues.
Followed by diarrhea after 1-2 days, and other sign which last 3-5 days
This terminates with death, or begins of convalescence
Virus disappears from tissue by 9-10 day after last clinical sign.

(11)

RV

RV produces nine types of polypeptides.
RV suppresed humoral & cell-mediated immunity in rabbits.

Surveillance & Monitory Programme in the control of Rinderpest: A Review
P. B. Rossiter & H. M. Wamwayi
Trop. Anim. ~~Hlth~~ Hlth Prod. 21, 89-99 (1989)

Disease transmitted by close contact, though limited airborne spread may occur.

---

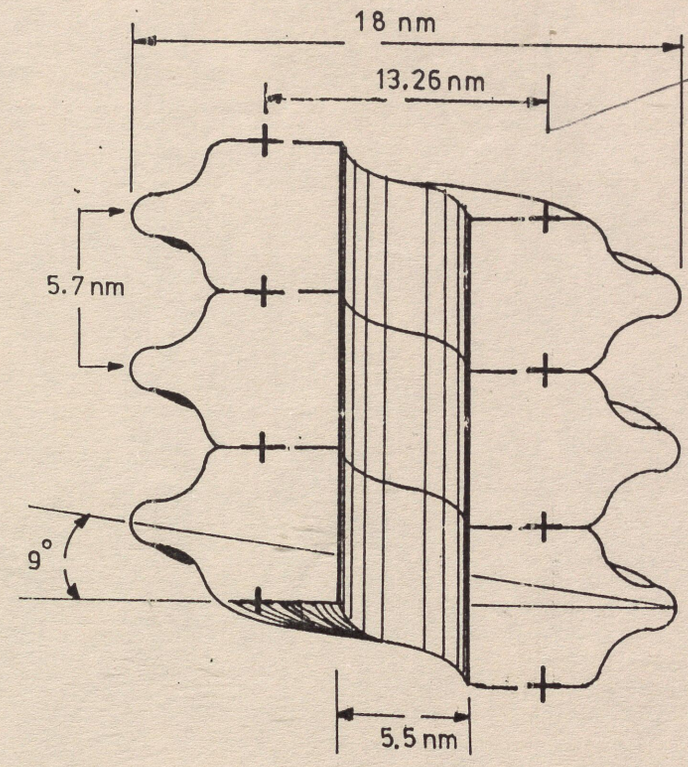
13 Nov 1994

Parameters	Normal nucleo Capsids	Fu - Nc
length	1144 ± 62 nm	195 ± 26 nm
No. of helical turns	197 ± 19	31 ± 12
Pitch	5.7 ± 0.17	6.2 ± 0.11
external diameter	18 ± 0.28 nm	8.7 ± 0.22 nm
Diameter of inner hollowcore	5.5 ± 0.12	-
Subunit angle	60^0	40^0
Helix angle	9^0	Cannot calculate (0^0)

No. of N Protein molecules / Genome

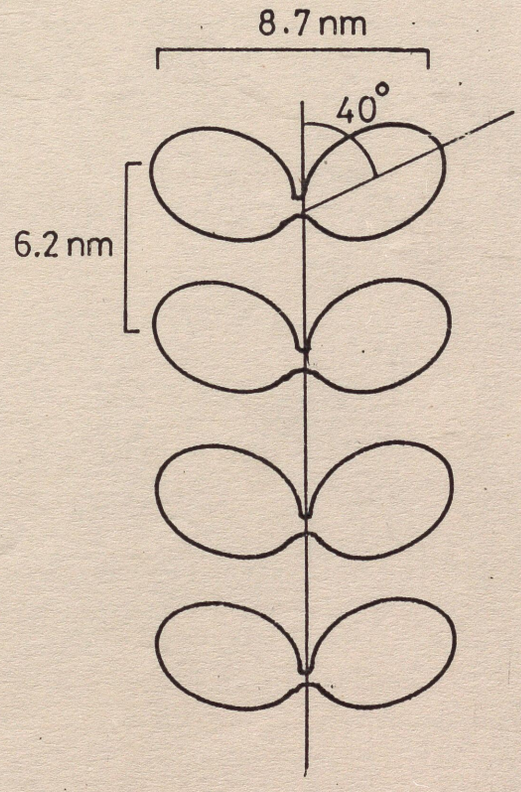
= 2400

2.09 mol / nm



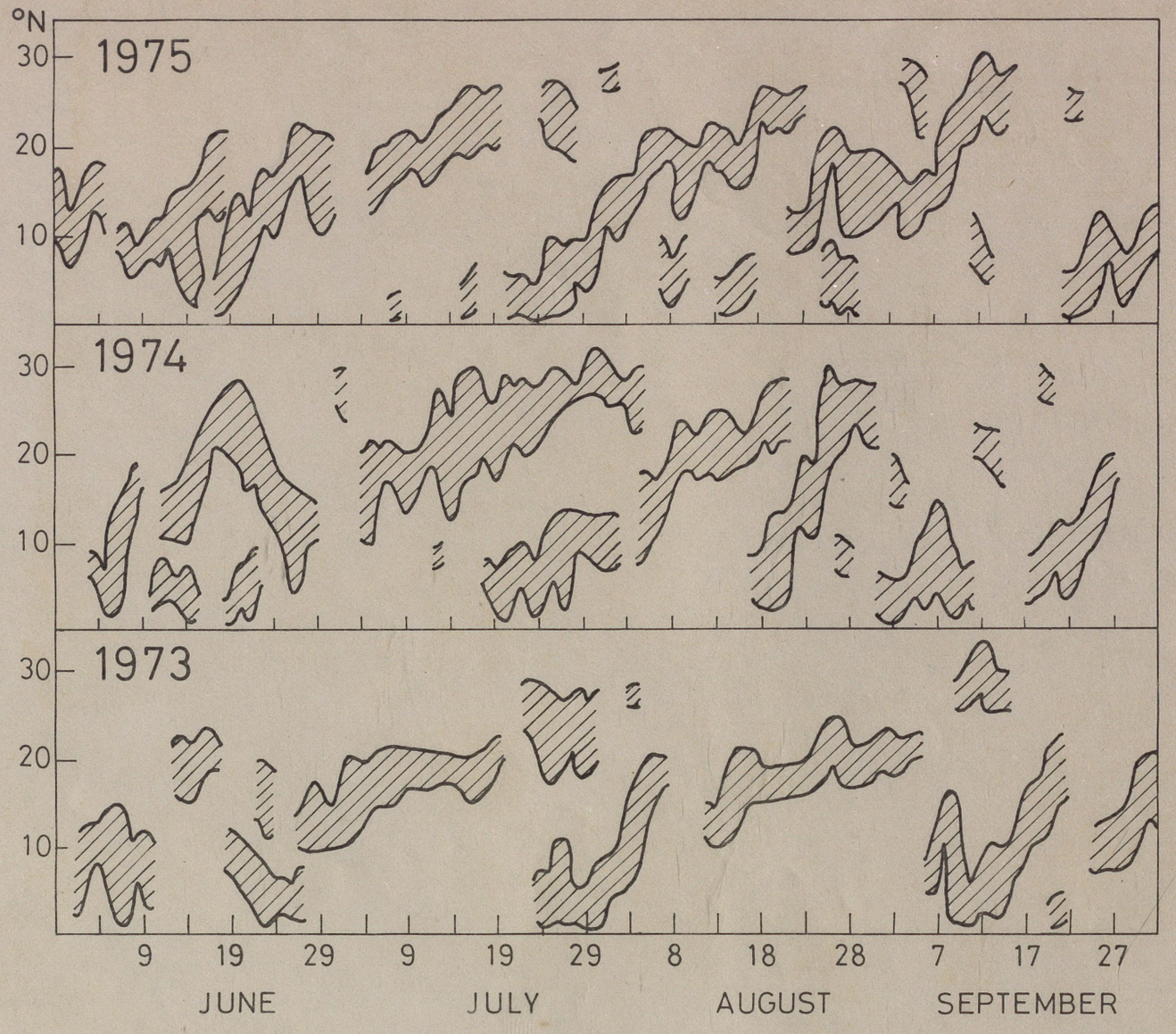
diameter of RNA helix
(calculated using a formula ref Lund et al Paper)

Normal NC



Fu – NC


RV13.TGA
RV15M.TGA
RV16.TGA
RV5FU01M.TGA
RV5FU05M.TGA
RV5FU06M.TGA


°N
30
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1975
1974
1973
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7
17
27
JUNE
JULY
AUGUST
SEPTEMBER

~~Fu:~~

1. View from top both C & Fu ✓

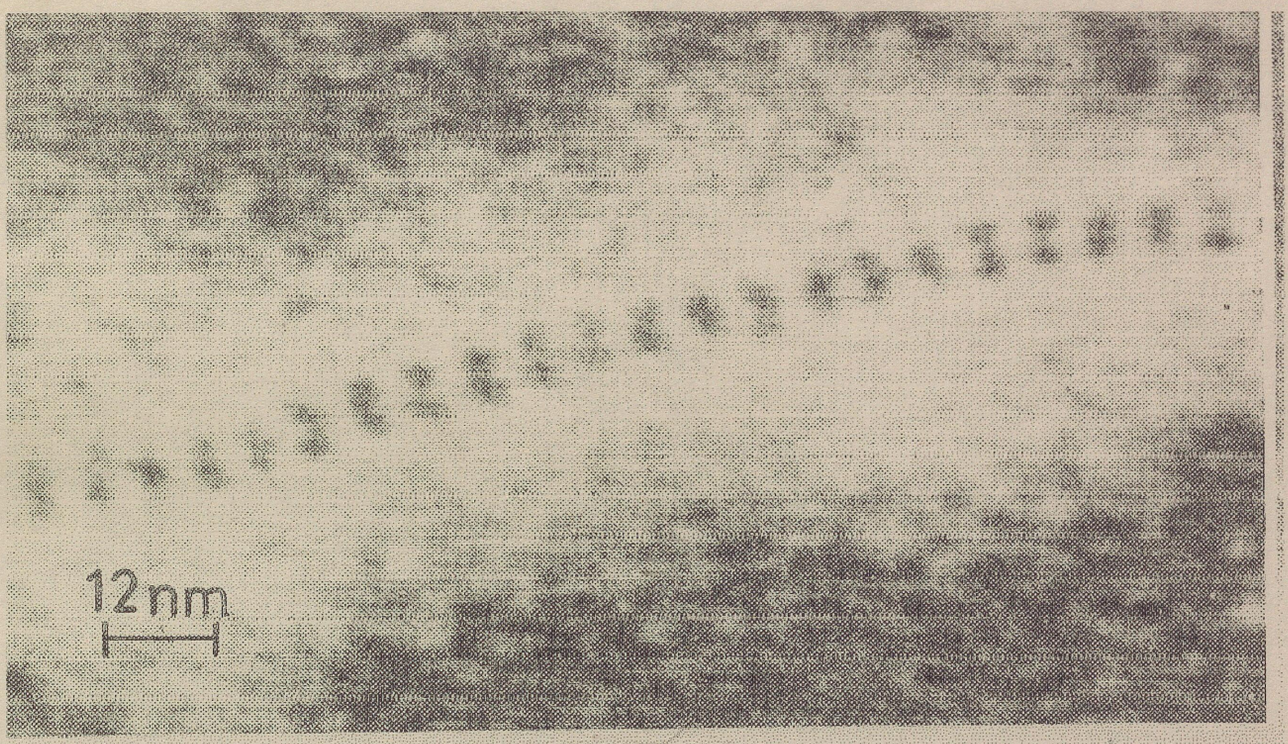
2. Fu NC : unchanged Prot. mol. size
3. Fu NC : Changed Prot. mol. size ✓

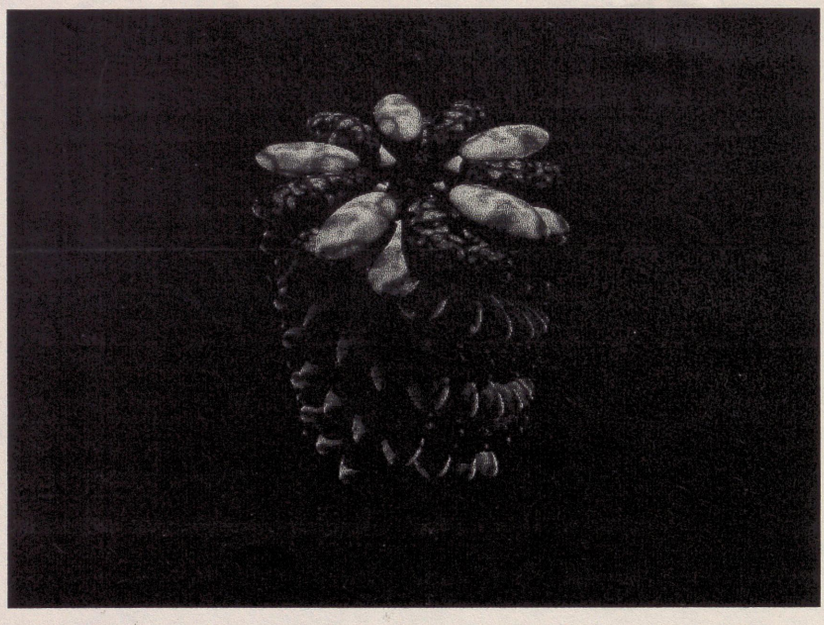
4. Normal & Fu NC together ? {can be seen}

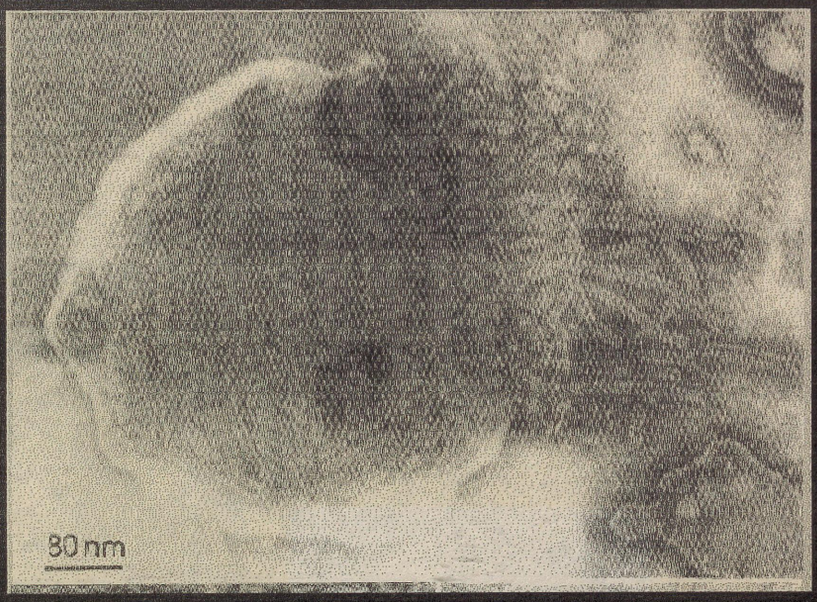
5. Side view ✓

Colours

(1) Gold & black background ✓
(2) Silver " ✓


12nm




80 nm

15 Nov 1994

Rinderpest nucleocapsid.

$L = 1144$ nm $\qquad \frac{1144}{197} = 5.8$

turns = 197

pitch = 5.7 $\qquad (5.8)$?

particles = 2400 $\qquad$ size (length) $\simeq$ 6 nm

$\therefore$ If helically arranged, each particle

moves $\frac{1144}{2400} = 0.4767$ nm $= 4.7$ Å

along the helix axis w.r.t. the previous one.

and moves $\frac{360 \times 197}{2400} = 29.55^\circ$ around

the helix axis.

If disks of 12 particles are formed, the # disks $\simeq$ 200

and they are spaced apart at 5.7 nm

C:\MSSNOV94\ RV01.POV ... etc.

RV07.POV — Top view

plane {<0,0,1>, 20

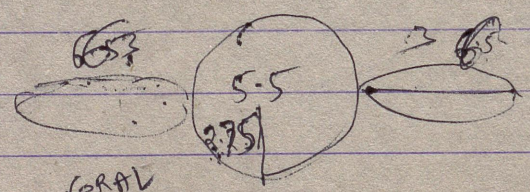
pigment {}}

RV10.POV

↓ RV11.POV

RV12.POV

RV13.POV



CORAL

LightBlue

18
− 5.5
12.5
6.25 $\simeq$ 6

C:\ANSS Nov94\ RV5fuol.pov 16 Nov 94

Angle = 40°, Translate = half the size = 3

Length = ~~18 KB~~ 16 - 18 KB RNA

base-base dist in DNA ~ 3.5 Å

Length of a helix, Radius = R, unit ht. (pentum) = h !

Length = h, $2\pi R \cdot \frac{L}{2\pi R} = 2\pi R$, $R = \frac{L}{2\pi}$ if h=0

$dL = R\,d\theta$ ~~$2\pi R \cdot \frac{L}{2\pi R}$~~ $= L$

$2\pi \equiv h$ $\therefore dh \Rightarrow \frac{d\theta}{2\pi} \times h$ or $d\theta = \frac{2\pi}{h}\,dh$

$$\therefore (dh)^2 = (R\,d\theta)^2 + (dh)^2 = \left(\frac{2\pi R}{h}\,dh\right)^2 + (dh)^2$$

$$= \left(\frac{4\pi^2 R^2}{h^2} + 1\right) dh^2$$

$$dL = \sqrt{\frac{4\pi^2 R^2 + 1}{h^2}}\,dh$$

$$L = \int_0^H \sqrt{\frac{4\pi^2 R^2 + h^2}{h^2} + 1}\,dh$$

$$\approx \sqrt{\frac{4\pi^2 R^2 + h^2}{h^2}} = \int_0^H \frac{1}{h}\sqrt{4\pi^2 R^2 + h^2}\,dh$$

Length of one complete turn of a helix, Radius R, unit ht. H 17 Nov

Initial point, ~~0, 0, R~~ $R, 0, 0$

when it moves through a height δ,

the angle is $\frac{2\pi}{H} \times \delta$

$\therefore$ * $R \cos \frac{2\pi}{H}\delta, \; R \sin \frac{2\pi}{H}\delta, \; \delta$

$\delta \simeq 0 \Rightarrow 0, \; R\frac{2\pi\delta}{H}, \; \delta$

$\therefore \text{dist}^2 = \frac{4\pi^2 R^2 \delta^2}{H^2} + \delta^2 = \left(\frac{4\pi^2 R^2}{H^2} + 1\right)\delta^2$

$dL = \sqrt{\frac{4\pi^2 R^2}{H^2} + 1} \cdot dH$

$\therefore L = \sqrt{\frac{4\pi^2 R^2 + H^2}{H^2}} \cdot H = \sqrt{4\pi^2 R^2 + H^2}$

Check: $R = 0 \Rightarrow L = H$ & $H = 0$ $L = 2\pi$ o.k.

Normal,

$H = 5.7$, $R = 2.25$ $R^2 = 5$, $4\pi^2 R^2 = 200$, $H^2 = 32.5$

$L = \sqrt{232.5} \simeq 15$ nm

$\frac{57}{399}$
$\frac{285}{32.49}$

$\therefore$ 200 turns $\simeq$ 3000 nm 30,000 Å

(I)

CES annual report on PPR 8 March 1995

~~simplest model → No births~~ Herd of sheep & goats.

(i) simplest model – No births. Day to day updaty.
Dependence of the fate of the herd (% survy) at the end of a 120 day period, ~~given~~

Deterministic model; herd sizes 100, 1000, 5000 } Initially infected animals 1, 2, ...

(ii) Stochastic model – probability of a specific mortality

~~Need~~ ~~C~~ Catgm. Susceptible, Infected ← catgomen, Cured / Immune, ~~Dead~~ | State index = 1 susceptible

susc → inf. 4 contact. Each infective (spread) a certain # contacts

Even a sylle contact is adequate for sc. — inf.

Av. No. of contact per indi. can be obtained; ~~which~~ Z

is ~~same for~~ ~~1~~ $1 - e^{-Z}$, assuming poisson, of not getting a sylle on

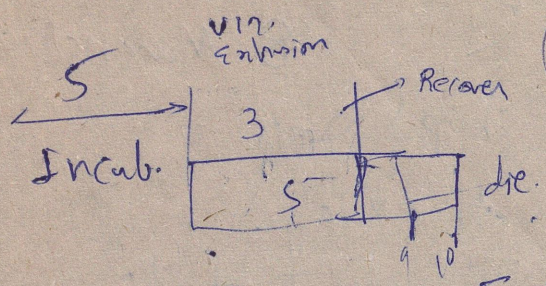
$1 - e^{-Z}$ = prob. of being infected.

PPR — incubation period 5, 6; 3-4

course of infection		~~5~~ 5 days, 6-8 days			10-14	10
mortality	25%	82	62	55		
morbidity	100%	77	90	60		

Inc 6 3-4 2
Dn 10 10 4-6
25 50 80

II



State 1 2 3 4 5 6 7 8 9 10 11 12 immune

1 → 2 infection

2 → 9 all of them contr

from 9, 75% go to state 12

25% go to 10 → 11 → death.

(12) remove (12) . # new infected is computed as

$$Z = \frac{(N_7 + N_8 + N_9 + N_{10} + N_{11}) \times \text{No. contact}}{\text{All}}$$

Prop. escaping

$\therefore$ ~~Prop. without~~ $= e^{-Z}$

Prop inf. $= 1 - e^{-Z}$

Can be run for infinite population — del.

Dimension $X(13)$

$X(1) = 1 - PPR$

$X(2) = PPR$

PPRI

$XX(3) = X(2)$

$\vdots$

$XX(9) = X(8)$

$XX(12) = X(12) + 0.75 * X(9)$

$XX(10) = 0.25 * X(9)$

$XX(11) = X(10)$

$XX(13) = X(13) + X(11)$

$$Z = \frac{X(7) + \cdots X(11)}{1 - X(13)}$$

$Q1 = \exp(-Z)$

$Q2 = 1.0 - Q1$

$XX(2) = X(1) * Q2$

$XX(1) = X(1) * Q1$

& $X(i) = XX(i)$

plot $X(1)$, $X(12)$, $X(13)$ & 1 - then

a fn of time.

(III)

Question 1 – ~~How does the fi~~ what is the equillibrium situation

{ Simplification Death ~~begins on day~~ on days 90 & 11

~~mortality~~ ~~susc~~ survival = $S^2 \simeq 0.75$ ∴ $S = 0.\cancel{88}$ 0.90

∴ mortality = $1 - S^2 = 0.25$ — give ray

$XX(10) = S\,X(9)$

$XX(11) = S(10)$

$XX(12) = \cancel{(1-S)(X9 + X10)}\ S\,X(11)$

$XX(13) = (1-S)(X9 + X10 + X11)$

Variable param = contact rate C

& survival S

Initial prop. of infection P

C:\ ANURPT95\ EPIMOD RV 9 March 95

EPIMOD01.FOR ... EPIMOD03.FOR

EPIMOD03.WK1 10 March 95

(1) Prop. immune, prop. of death ~~log~~ vs prop. of initial infected gives a st. line at log log. (Min. R^2 0.7859), slope increases with mortality

(2) For low contact rate, higher mort → lower # susc.

high contact rate, opposite trend.

EPIMOD03.OUT, #Gen I7 { infec. pop. 7 of 10.6 | cont. rate | mort. ~~Survi~~ | susc. -ve | imm. +ve | dead

9 val. ..

(9)

9 values ~~0.1 to~~

10 value

0.1 – 1.9

step 0.2

contact (mort (inf)

PTO

NXY, CSLP YINT / RNSIM X, N AV SD MX MN

(16)

11 March 95

More simplified model —
Death only on the last day
EPIMODOTOR

Replication rate earlier = $c\left(1 + (1-m)^m + m(1-m)^2\right)$
If m = mortality $c\left(1 \cdot m + 2m(1-m) + 3\right)$
Replicat rate in current model $3c$

Repl. rate in simple model = $3c$
In earlier model = Prob. of die exactly after 1 day = m
Prob. of die exactly at 2 days = $m(1-m) = m - m^2$
Prob. of surv beyond 3 on month = $1 - 2m + m^2$

$\therefore$ Rate = $3 - 6m + 3m^2$
$+ \; 2m - 2m^2$
$+ \; m$
$= 3 - 3m + m^2$

$m = 0 \rightarrow 3$
$m = 1 \rightarrow 1$

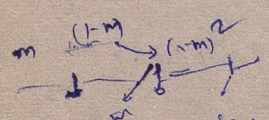
Earlier model — rate goes down from $3c$ to c with increasing mortality

---

Earlier $3 \cdot m + 4(m - m^2) + 5(1 - 2m + m^2)$

$5 - 10m + 5m^2$
$+ 4m - 4m^2$
$+ 3m$
$= 5 - 3m + m^2$

$m = 0$ $m = 1$
$c(5 \rightarrow 3)$



New $5c$

Old $5c$ to $3c$
New $5c$

logg $\propto c$
e^{-c}

11 March 95

V

(i) Prop. of susceptibles decreases exponentially with contact rate

(ii) Of dead, increases & saturates at mortality value.

(iii) Of immune, " 1-mort. value.

EPIMOD10 ⟶ Peak size & time of peak

0.001 0.05

P	C	M
0.001,	0.25	0.1
"	0.50	0.9

PPR01.FOR ⟷ EPIMOD09.FOR Taken in C, mort, ini. inf.

Prob. That one animal infect = P

∴ That does not = 1-P ∴ If K inf. animal,

C	M			
0.5	0.1	0.01	peak ≃ 0.3,	at ≃ 40
		0.05	≃ 0.3	" ≃ 30
		0.001	0.30	≃ 60

Peak usually

PPR02 ⟵ EPIMOD10.FOR

For 3 vals of mort 0.1, 0.5, 0.9 ⟶ 2I5, 8F8.5

~~2I5~~

~~EP~~PPR03.FOR ⟵ EPIMOD08.FOR

Figure 1 ~~C~~ C dth P

A	0.10	0.10	0.05
B	0.4	0.4	~~0.0015~~ 0.01

Proportion vs time.

Fig. 2 Suc. v. contact rate 13 March 95

Fig. 3 Time for peak vs contact rate

Fig 4 Peak vs contact rate

(vi)

PPR04.FOR → old model, monthly at ~~age~~ Sl. 8, 9, 10.

~~PPRTBL1~~ PPRFIG3M → generate a table.
PPRFIG3M.PRN — Duration & epidem epizootic.

PPRFIG5 → old model.
daily mortality

PPR01VAC → prop. initially vaccinated
~~0.3 0.1 0.001 0.10~~

PPR01 0.50 0.10 0.001
PPR01VAC 0.10

---

14 March 95

C:\ANURPT95\EPIMODRU\RPT\

RPT03.

FIG1.WK1 (1A)	FIG2.WK1 (2B)	FIG3.WK1 (2)
↓ FIG4.WK1	3→5	5→6 FIG6.WK1

(2)

11 March 1995

Abstract:

A deterministic mathematical model has been developed to investigate the propagation & ~~persistence of~~ a PPRV epizootic in a nonreproducing herd & small terminal & arbitrary size. The parameters corresponding to the incubation and virus excretion periods & the disease ~~were~~ were obtained from ~~the~~ published literature. ~~The~~ The mortality rate was varied over a range, since different strains of PPRV and different geographical areas report considerable differences in the mortality rate, the ~~immunity~~ rate was varied over a wide range. The contact rate / transmission efficiency was also ~~also allowed to vary in the model for a~~ treated as a variable parameter. For every combination of the parameters and for various levels of initial infection, ~~the fate of the epizootic was investigated till~~ ~~simulated till~~ the changes in the ~~levels~~ populations of infected, immune susceptible, infected, immune, and dead animal were simulated till they reached ~~a constant~~ near constant values.

It is seen that more or less independently of the initial infection level, depends on the threshold value of the contact rate, the infection either peters off, or builds up into an epidemic with a large proportion of animal contacts the disease ~~and~~, ~~with subsequent~~ followed by death or ~~status~~ recovery into an immune state. A set of simulations was carried out to determine the dependence of the onset, duration, and ~~peak~~ magnitude of the epizootic on the virulence level, and contact rate of the virus.

(3) * combine of parameters and initial effect sec, for any number of days.

Myhlyth – In conformity with other epidemiological models, a threshold effect is observed ~~where such~~ for the contact rate parameter, such that a ~~lower~~ high value results in ~~a~~ high levels of infection by bull/fungi, ~~what~~ whereas for lower values, infection dies out. Inherently the peak level is ~~higher for~~ of infection is higher for ~~higher~~ ~~virulent~~ more virulent strains (higher mortality rates).

---

46. ~~The population can be~~ Any individual in the population can be characterized as being in one of the ~~four~~ five states;

Susceptible, infected/non infectious, infected/infectious, recovered (immune) and dead.

Initially, all animals were assumed to be susceptible, except for ~~the~~ a small ~~pre~~proportion of introduced infected animals. The infected animals ~~were~~ go thru an incubation period of five days, when they are not infectious, followed by a period of 5 days, when they are infectious. At the end of 10th day of observing, ~~the animals either~~ ~~recovers with a~~ proportion d of the ~~d~~ observed animals die, while a proportion 1-d recovers into an immune stage.

Each infection animal is assumed to make C contacts with other animals ~~(~~ a single contact is ~~assumed~~ to be adequate for successful transmission of infection). If ~~PI~~ ~~PI density the prop~~ PS, ~~PI~~ PINF and PIM denote proportions of susceptible, infection and immune animals, and if the number of effective contacts per animal is $Z = C \cdot PINF / (PINF + PS + PIM)$. ~~There are assumed to be poisson stage~~ Assume poisson distribution, the probability that a susceptible animal will receive at least one contact is $ts = 1 - \exp(-Z)$. Hence a proportion ts of the susceptible animal is assumed to have become infected on ~~that~~ day t+1 ~~using the~~ A FORTRAN program was developed to simulate the above situation for any state

4c Result

4.d Discuss & Analy

4.e (i) # susc., infl, dead & imm as a funct of time ✓

(ii) Prop. susc vs contact rate ✓

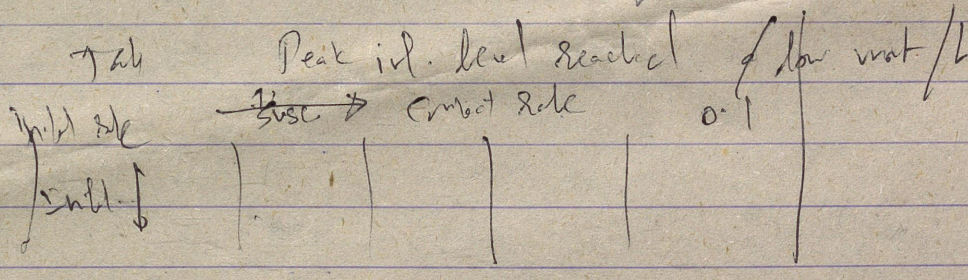
(iii) Prop. ~~dead~~ infect vs contact rate all models ✓

(iv) ~~Prop. dead, immune~~ ,,

(v) Peak infl level vs contact rate, all models

(vi) Peak inf dur vs ~~initial inf. rate~~ ,, initial conc.

4.e



4.g. The work is at a preliminary stage, ~~not~~ more models.

detail data on attributes, population structure and mobility rates at the local state and urban area level, is not yet available, so it is too early ~~F~~ to make the type predictions to give. A impact

(iii) 1 Recommendations: The ~~main~~ project ~~objectives~~ at this stage investigations envisioned in the proposal are yet to reach a level where recommendations can be made.

2. Nil. Collaborations

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PLEASE CO-OPERATE. THANKS

N.V. Joshi

संस्कृतसङ्घः
भारतीयविज्ञानसंस्था, बेङ्गलूरु - ५६० ०१२

SAMSKRITA SANGHA
Indian Institute of Science, Bangalore-560 012

क्रमाङ्कः 66 दिनाङ्कः 7-9-95

SPOKEN SAMSKRIT WORKSHOP
VALEDICTORY FUNCTION

Chief Guests :

Prof. K. J. Rao
Chairman, Division of Chemical Sciences,
Indian Institute of Science, Bangalore.

Shri C. V. Venkatesh
Workshop Instructor,
Hindu Seva Pratisthna, Bangalore.

VENUE: **L1 Lecture Hall**
DATE: **Saturday, 9th September 1995**
TIME: **5:30pm**

Programme Schedule

5:30pm	**Coffee**
5:45pm	**Invocation**
5:50pm	**Welcome Address**
6:00pm	**Chief Guests' Addresses**
6:45pm	**Vote of Thanks**
6:50pm	**Cultural programme by the participants of the workshop.**

All are welcome

D:\ANURPT96\P12\ (1) 5 May 96

P129601.FOR (PRR01.FOR + modified) →

9602.FOR (02) can may comb. of pan.

convergence time, ~~max~~ time for peak, init inf, contact Rate, death, survi, susc, immm dead, max size.

CMPROUT.270 = old res PPR02 + new res P129602.OUT

9 init inf × 10 × 3 = 270

cont rate 0.05 100.50 — surv rates

0.001 0.2 0.9
0.002 0.2 0.9

thresh cont > 0.25, rate death > 0.9, epi over much faster, & ~~max~~ peak reache earlier.

lower value of susc. always in old ok

High death rate → lower ~~survi~~ susc. eqn pop.

higher contact rate, lower discr.

---

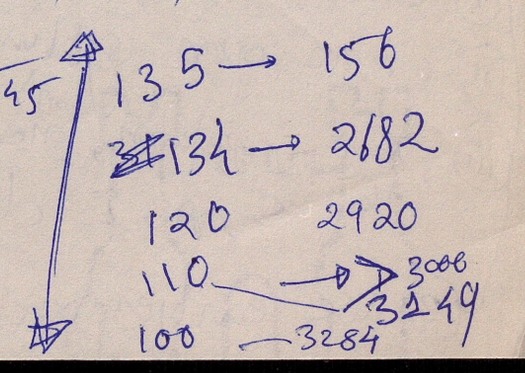
model 2 → ~~A small fraction (1%~~
The proportion who were immune, lose immunity after n days (n ~ 100, 200)

~~P129604.FOR~~
P129605.FOR → MEMOR

c=0.4, dth=0.4, p=0.01, IMU=300, 200, 150,

~~145~~	Res 156 gen,	0.116668	0.529998	145
	156	0.12266	0.51	
140	161	0.1344		
135	156	0.13		
		0..		
2888				

135 → 156
~~31~~134 → 2682
120 2920
110 → 3000
3149
100 — 3284



Epi mod.

~~Simplifie~~ Original model (2) 5 May 1996

susc, infected non infective, infected & infective, immune, dead

$x(1)$ $(x2) \to x(6)$ $x(7) \to x(11)$ | $x(12)$ $d\,x(11)$

S ~~S~~ A ~~A~~ ~~B~~ A ~~C~~ V $(1-d)\,x(11)$ I D

modified form; 5×5 matrix

$x1 \to x2$ depends on infection

$x2 \to x7 - x11$; $x(6)$

$x7 - x11 \to x12^{13}$ $\to x13$

$S \to A$

$A \to V$, $1/5^{th}$

$V \to r \times 1/5$ I (recovered)

$\to r(1-r) \cdot 1/5$ D (dead)

$$\begin{pmatrix} S \\ A \\ V \\ I \\ D \end{pmatrix} = \overset{T}{\begin{pmatrix} \cdot & 0 & 0 & 0 & 0 \\ \cdot & 0.8 & 0 & 0 & 0 \\ 0 & 0.2 & 0.8 & 0 & 0 \\ 0 & 0 & \frac{r}{5} & 1 & 0 \\ 0 & 0 & \frac{1-r}{5} & 0 & 1 \end{pmatrix}} \begin{pmatrix} S \\ A \\ V \\ I \\ D \end{pmatrix}$$

Tot. infecty contacts $= cV$

tot. per animal

~~Av. No contacts =~~

$= \dfrac{cV}{1-D}$

∴ prop. with no contacts.

$F = \exp\left(-\dfrac{cV}{1-D}\right)$

$T_{11} =$ # remaining susceptible $=$

$= \exp - \left(\dfrac{c \cdot V}{1-D}\right)$

$T_{21} = 1 - T_{11}$

(0.5, 0.8, 0.001)

c death, initial.

Parameters: Duration of infected; noninfective stage

Duration of infected, infective stage

Recovery rate r

contacts. c

Δ ~~in~~ Initial conditions

(i) check correspondence with earlier results

(ii) make open system; a proportion 'new', to be added

(iii) Two population; only one initially infected; a fraction 'c' of the S, A, I classes exchanged between the two.

(0) Use earlier model itself, making it 'open' const pop. model by replacing 'dead' with susceptibles daily, weekly, monthly

(3)

5 May 96

		peak	dip		
OUT 134	P1	259	, 417	, 564	305
120		236	377	507	271
110		220	348	468	248

OUT 134 period (peak to peak) ~ 303 ~~First~~

		Max val.	First max	period
OUT134	2683	0.2244	51	303
120	2921	✓	✓	268
110	3150			244
100				220
75				160

Period does not depend on init inf.

0.3 0.4 0.01 100 period ~~226~~ 206

peak sum

200 ~ 425 eliminated.

500, saved.

P129606-TOR (immunity loss + input of new animals)

with 0.4 0.4 0.01 150, continuous cycling

200 cont cycle, peak 0.25

Check, (0.5 conta, 0.1 mort, 0.5 init,) equil at 0.1

300, ~~wi equil~~ equil at 0.28

100, period 184, damped oscillation

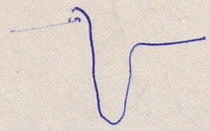
0.4 0.4 0.01 50 ? → equilibrium at 0.5 (endemic occurrence!)

(4)

6 May 96

Memo1S → imm. recovery, without stopping.

0.4, 0.4, 0.01, 300



500 → same.

900

same

REPO1S → replacement, no stopping.

REPO1D → replacement, no stop, double precision

0.4 0.4 0.01 0.269 sure, rest immune.

0.5 0.4 0.01 0.21

0.40 0.50 0.01 0.315

0.50 0.50 0.01 0.240

MEMO1D.FOR — Peak 4 55

0.4, 0.4, 0.01, 300 Reach 0.6466 sheep

600 ; no immune left

period 293 130 ; pop. wiped out in oscillating manner

period 220 100

99

p c dth

0.50 0.15 0.10 0.35521 0.64472

next

0.001, 0.15, 0.9

REPO2S.FOR — make table

REPMEMD1.FOR

Both elect, 0.40 0.40, 0.01, 500 → cycles ; Peak 0.25

period 898

100 → period 184 sust cycles 900, 1320

0.3 to p-0

old PPRO1

double prec.

0.1167 0.5300

c=0.50, cycle, 107

→ mem 60 — cycle 113

(tang) osc. convergence

5 9 300

Equilib between infected & susc. & immune.

with a 3x0 more complicated behav

5 June 97

C:\POONA971\

BIRD212.132
BIRDS132.LSE
BIRDS132.LOC

2 I5, A3, A4, A25
2 I5, 4F9.0 → Lat alt RF Dry

9 habitats, ~~24~~ → 3 – 24 transects in each.

For each sp, compute variance
then μ6 eb.6 variances
3.704 11.3947 98.8771 0.0076 0.2847
median

Testanv1
Nmsa, vmsw, fval, vara, pera, vno
ANOVA02.FOR
no = 14.2121
9 groups, (lse types) 132 transect

0212
339204

∴ df among = 9 – 1 = 8
df with = 132 – 9 = 123
$F_{0.05}(8,123)$ = 3.55 (0.001)
2.02 (0.05) 2.66 – 0.01

Above 2.02 ≈ 70 species
2.66 ≈ 53 spece
3.55 ~ 34 spece

HAB

~~LOC~~

MS with =	3.75	11.86	102	0.0054	med 0.41
var among =	0.38	2.15	26.0	–3.78 ?	med 0.28
F	2.13	2.05	24.38	0.32	med 1.15
% among	5.7%	10.6%	62%	~0	Med 1%

Anova02.wk1

PTO

(2)

When pop → prevance/absence

NS with	0·17	0·26	1·55	0·0023	
amoy	0·0595	0·0588	0·2517	0·0054	
F	2·56	3·07	24·66	0·37	1·44
pt amr	7·68%	12·2%	62%	—	mod 3%

41 sp > 3·55
62 > 2·66
81 > 2·02

Check: Use only the 76 species found in at least 8 transects

With pop → F = 2·67, % = 8%
pop 50, 17 found = 2·5 & 8·2%.

FOR pop, ANOVA02.wb1. 125 with +ve var A
sum var with = 491 & amoy = 87·95, %amy = 16·3%
126—212 = 344·6 — 6·67

Prevence/absence	144 > 0	
sum	9·81	1·798
	2·807	−0·0469

% = 14·8%

Pooled, 12·9%. pop LOC

5 Jne 97

$x_1 \ldots x_n$

$$n\sigma^2 = \sum (x_i - \bar{x})^2 = \sum x_i^2 - 2\bar{x}\sum x_i + \sum \bar{x}^2$$

$$= \sum \left(x_i - \frac{x_1 + x_2 + \cdots + x_n}{n} \right)^2 = \sum x_i^2 - n\bar{x}^2$$

$$= n\overline{x^2} - n\bar{x}^2$$

$$= \sum \frac{1}{n} (n x_i - x_1 \ldots$$

$$\sum_i \sum_j (x_i - x_j)^2 = \sum_i \left(\sum_j x_i^2 - 2 x_i \sum_j \right.$$

$$= \sum_i \sum_j (x_i^2 - 2 x_i x_j + x_j^2)$$

$$= \sum_i (n x_i^2 - 2 x_i \sum x_j + \sum x_j^2)$$

$$= \sum_i (n x_i^2 - 2 x_i n \bar{x} + \sum x_j^2)$$

$$= n \sum x_i^2 + n \sum x_j^2 - 2n^2 \bar{x}^2$$

$$= 2n \sum x_j^2 - 2n^2 \bar{x}^2 = 2n^2 \overline{x^2} - 2n^2 \bar{x}^2$$

∴ Avg. dist.^{2} = divide by n^2

$$\overline{d^2} = 2\overline{x^2} - 2\bar{x}^2$$

$$\therefore \frac{n\overline{d^2}}{2} = \overline{x^2} - \bar{x}^2 = \frac{n-1}{n}\sigma^2$$

$$\therefore SSQ = \sum (x_i - \bar{x})^2 = \frac{n^2 \overline{d^2}}{2} = \frac{n}{2} \overline{d^2}$$

$$= \frac{n}{2} \frac{\sum \sum (x_i - x_j)^2}{n^2} = \frac{1}{2n} \sum \sum d_{ij}^2$$

∴ If n points, sum of all pairwise distances divided by $2n$ (or all non-repeated pairwise dist. divided by n.

Mahesh Ronade (1)
City Comp 5597600

Ext. 350, 351

Marketing group

MS ~~VAR~~ amog (Jaccard) = 0.8456 df 9
~~between~~ within = 0.4088 df 123

F = 2.068 signl at 5% just about

var amog = 0.03 & ~~%~~ = ~~0.0699~~

percent amog ≈ 7%

(1 − sin²) − (1 − sin²) sin² − sin²

with chord dist. 0.90, 0.42, F = 2.13, % = 7.4%

6 June 97

with localities

ANOVA2LO no = 7.3016 ; 17 classes & 210 species
+ sp. # 95, with var = 0 : omitted, 2

~~VMS amog~~ ~~Var wit~~	7.43	22.3	162.9	0.0037
~~Var~~ VMS with ~~Amog~~	3.72	12.0	98	0.0073
F	1.88	1.86	16.37	0.40
Var amog	0.507	1.58	11.4	−0.419
% Exp	7.9%	13.5	67.8	−0.8%

F 16, 115 — 1.75 68 sp; 2.19 51; 2.78 39

Med ~~5~~ 4%

149 sp, vara > 0

Med 8% for +ve val

(5)

6 June 97

<I POONA971>

DISTANV2. FOR → dist. cal. with Localities

Jacard, LOC M Among 0.826 DF 17
Within 0.3811 df 115
F. = 2.167, highly sgnl.
var among = 0.03 ≈ 7.6%

Chord dist F = 2.10, % among = 7.2%

1	2	5	8	9	10	11	12	13	≈ 9
1	24	13	23	17	3	13	23	5	
SH	EVG	SEM.	DC	DIG	GR	MONO	HAB	PADDY	

BHB01. FOR → Make 212 Rows X 9 Hab file
B212HAB9

ANOVA03T. FOR
Trees398.108 TREES108.LSE

15.12 = no, 7 types reg. 17% pooled
~~9%~~ 12% mean

DISTANV3. FOR → Mean
7b101 Jackard F = 2.7, 10%
Chord F = 2.56 9.9%
(opt.) 5%

(1)

14 June 97

: Quantitative Techniques :

How quantitative approach, and computer simulations can be used in the analysis of ecological data.

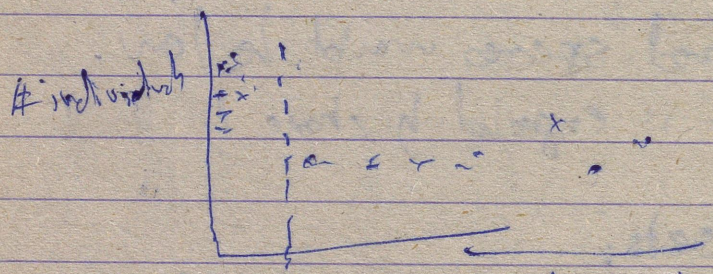
Would like to describe a series of questions, and indicate how they can be answered.

Most of what I talk on would be based on work carried out in ~~connection~~ collaboration with ~~Prof.~~ Dr. R.J.R. Daniels, and Prof. Madhav Gadgil.

Most of the data was collected by Dr. Ranjit Daniels in the evergreen and degraded evergreen forests of Uttara Kannada dist.

30 transects, ~~20~~ 600 x 4m.

Record all woody plants above 1 meter height, and diameter, identify down upto species level.



Proportion of decided individuals.

① Two categories: Evergreen and Disturbed Evergreen

20	10
13886 indiv.	3790 indiv.
172 sp.	116 sp.

(2)

(A1) Question: Are the two sets really distinct in terms of ~~influence & dism~~ evidence of agents of influence

fire	5	5
grazy	3	3
trails	7	6
	out of 20	out of 10

Simulations → ~~confidence interval~~ significance tests.

(A2) Are the two sets different in terms of composition?

200 species: 84 excl. to low 28 excl. to high 88 shared.

If composition were different; shared species would be low. Is this lower than what is expected by chance?

Model 1. Make random sets:
shared ranged from 109 to 137, $\mu = 125$

Model 2. make random assignment.

$\frac{1}{3790}$ $\frac{3790+13886}{17676}$ $\frac{3600}{18000}$ $1/5 = 4/5$

141 — 163 $\mu = 153$.

A3. Have the number of species decreased as a result of disturbance?

How many species expected from 3790 individual out of 13886?

(i) Pick up transects

(ii) Pick up individual.

111 ± 6.

∴ No reduction in the level of α diversity.

A4 What are the levels of β diversity, proportion of species shared.

~~0.73~~ 73% ~~+-7%~~ for EVG, 65% of DEV.

Is there evidence for low β div.?

Try rarefaction. Per transect EVG 694 ind. 48 sr.
DEC 379 36 sr.

~~β~~ : Difference is seen to be about 70%.

∴ Not enough evidence

A5 What are the attributes which make these species characteristic of ~~DE~~ EVG – DEV.

Pioneer, ~~Helip~~ Heliophilic, Thorny, ~~No~~ Coppicy, used by plywood. Restricted to EVG No copp. & use ply

Not ~~exclusive to EVG – withstand~~. Seen in DEV thorny, coppic.

Tree mortality in 50 H plot 14/Nov97

(i) Table :: ~~sigle~~ all sigle stemmed omit multiple cases

~~# trees~~ SP # trees # dead #MF #ME ~~#EM~~ Oth. | 4 Proportions

(ii) Table ... same ~~but~~ but ~~girth~~ dia classes.

dia range from 10 mm. in steps of 5 mm

max = 1405 →

(iii) Same two tables; seperately for fire affected & fire unaffected.

Extra colums for # and proportion of fire affected.

{ can this be used to predict multiple stem deaths ?!

(iv) Maps - 10x5, 50x25, 200x100, indiv. trees

Green → living Red - Fire, Blue → Elephant Yellow - other.

Seperate maps for each cause.

(v) Spatial autocorrelations / clumping for mortality cause.

How many distinct patches of fire affected / Elef affected trees ?

Are 'other' Random ?

(vi) Correlation (ha, Q, SQ) (50, 1250, 5000 ph)

between % mortality due to F vs E etc.

(vii) Mortaprob vs girth for species seperately for elephant affected mortality.

(viii) In how many ha(Q, SQ) there has been at least one instance of $\varepsilon(F, O)$ mortality:

i.e. table like (i) & (ii) for Ha(Q, SQ?)

A. Do elephants prefer specific size classes?

B. ~~Do~~ Is this affected by proximity

i.e. Do similar sized plants away from the elephants escape?

Do different sized plants nearer to elephants get affected?

C. ~~Is~~ Is the pattern from one 'patch' same as from others?

(ix) ~~Are patches in~~

Statistics 3 Feb 98

Q. Why statistics → Referee.

- Concise description
- Estimation & precision
- Decision making under uncertainty

1. Description of data - M, σ, box plots.
2. Distributions
3. Hypothesis testing
4. Probability
5. Tests & significance. Parametric
6. Test & significance - Nonparametric
7. Parameter estimation, curve fitting etc.
8. Use & simulations

Sample Theory
Design & Experiments
Anova
~~Regression~~ Multivariate Analysis.

Examples:

Sample vs Population

(i) Do thermophilic bacteria show higher gc content?

(ii) Do tropical forests contain more tree species than temperate forests?

(iii) ~~Do Are polar~~ Do ~~sm~~ Are birds on smaller islands heavier than those on large ones?

(iv) Do RNA viruses replicate faster than DNA viruses?

(v) Is a genetically engineered plant more resistant to pest attacks?

7 Oct 98

~~Kattat~~ ~~Kalakkad~~ - Mund.

~~Kalakad - Mudanth~~

KALAKAD - MUNDANTHURAI TIGER RESERVE

⑥ Dr. Ajith Kumar

① Prof. Rauf Ali — Enclaves / Bonnet

② Prof. AJTJS →

③ Mahesh Sankaran → grazy & fine, herbivors

④ Jayashree Ratnam → Bonnet macaq

⑤ Madhusudan Katti → ~~Leaf~~ Migrants

⑥ Kaberi Kar-gupta → slender Loris

⑦ N.M. & Ishwar. → Reptiles

⑧ V. Karthikeyan → Amphibians

⑨ Divya Mudappa → small mammals

⑩ T.R. Shankar Rama — Birds

⑪ Johnson → Fish

⑫ T. Ganesh — ? seed predation } Pondichery.

⑬ R. Ganesan — plant communities

(13) Saubhadra Devy — Pondich — Pollination Ecology

(14) Ronald — } Biodiversity Survey,

(15) Jayanthi }

(16) Forest Dept. — Eco-development Management

Dr. V. K. Melkani — Field director

Mr. Venkatesh — Ecodevelopment officer.

(17) Wesley Sundarraj — Nilgiri Langur

(18) Justus Joshua — ~~Avifana~~ Avifauna

(19) Uma Ramakrishnan — Bonnet Macaque.

(20) Surendra Varman, — elephants.

(21) Thirunelelli — St Xaviers, St. Joseph's Allg. ferns etc.

(22) Prof. K. S. Bawa — (~~Sta~~ Satchidanand Jha) — Biodiversity & human impact.

16 oct 99

1. How many fish in the pond?
2. How many species & birds in the region?
3. How big should a seed be?
4. ~~How much grass should be cut?~~

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1. ~~F~~ = # fish in the pond. ~~M = marked fish~~

C_1 = # fish ~~be~~ caught = marked

next time, C_2 fish are caught out of which M are marked.

$$\frac{C_1}{F} = \frac{M}{C_2} \qquad F = \frac{C_1 C_2}{M}$$

~~M = 1000~~
$C_1 = 100$
$M = 10 \rightarrow \frac{100 \times 100}{10} = 1000$

Same method to estimate # rats in a shola patch.
errors in a printed page
errors in a program
internet connections etc.

Assumptions: complete mixing
no difference between marked and ~~un~~ unmarked.

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2. B = # species of birds
S = skill of the observer which can be seen ~~# spe~~ proportion of species.

$O_1 = S_1 \cdot B$
$O_2 = S_2 \cdot B$
$O_{12} = S_1 \cdot S_2 \cdot B$

$$\frac{O_1 \times O_2}{O_{12}} = \frac{S_1 \cdot B \times S_2 \cdot B}{S_1 S_2 B} = B.$$

Alternatively, P = probability that a bird is observed by one watcher.
∴ Population

2. Let B = total # birds.

p = probability that a bird is seen by an observer

$1-p$ = " that it is not seen.

$\therefore$ p^2 = both

$2p(1-p)$ = only 1

$(1-p)^2$ = neither $\therefore$ invisible birds.

$B = 400$
$p = 0.5$

$p^2 = 50$
150
150

Similarly 3 observers

$B \cdot p^3 \quad 3p^2(1-p) \quad 3(1-p)^2 p \quad (1-p)^3$

invisible = 50

$0 < p < 1$ $\therefore$ vary p & obtain best fit

How many words did Shakespeare know?

what if ~~observers are not~~ all birds are not equally sitable?

3. Total resource available = R

if S seeds are made, each will have $\frac{R}{S}$

Survival probability





~~Different~~ Estimate 'a' and test the prediction.

Use other functions

$\therefore$ $(w - w_0)^a$ $\quad 0 < a < 1$

$\therefore$ # surviving seed $= S \times \left(\frac{R}{S} - w_0\right)^a$

maximize $S\left(\frac{R}{S} - w_0\right)^a$ by d/ds " " complicated.

Let w = wt. of seed

$\frac{d}{dw} = \frac{R}{w}(w-1)^a \qquad -\frac{1}{w^2}(w-1)^a + \frac{a}{w}(w-1)^{a-1} = 0$

$\therefore \frac{w-1}{w^2} = \frac{a}{w}$ or $\frac{w-1}{w} = a$

$1 - \frac{1}{w} = a \quad \therefore 1 - a = \frac{1}{w} \quad \therefore w = \frac{1}{1-a}$ comment on 'a'

Application of mathematics and statistics to some ~~problem~~ simple problems in biology. 16 Oct 1999

(botany and ecology).

1. How big should a seed be?
2. How many fish are there in a pond?
3. How many species of birds are there in a region?

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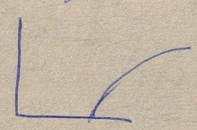
1. The plant has an ammount of resource 'R'

It can make 1 seed of wt R, 2 of R/2 ..., 5 of R/n.

what is the best choice?

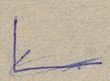
what is meant by best? ~~Maximum~~ Look for what happens with each choice — in terms of survival and germination.

For each seed, ~~how~~ we have prob. of ~~ge~~ successful germination, as a function of w, wt. of seed.





$R/w \times$ prob. of succ. germin.



$(w-w_0)^a$ is one such function

$\frac{x^{100}}{1+x^{100}}$

$$\frac{d}{dw}\left(\frac{R}{w}(w-w_0)^a\right) = 0$$

$$\frac{(w-w_0)^a}{w^2} = a(w-w_0)^{a-1}$$

∴ How big a seed should be does not depend on the total resource