EVIDENCE OF SYNCHRONIZED CYCLES IN OUTBREAK PATTERNS OF DOUGLAS-FIR TUSSOCK MOTH, *ORGYIA PSEUDOTSUGATA* (McDUNNOUGH) (LEPIDOPTERA: LYMANTRIIDAE)

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Abstract

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Outbreak patterns of Douglas-fir tussock moth, *Orgyia pseudotsugata* (McDunnough), over western North America historically appear to be synchronous, particularly in British Columbia, Washington, Oregon, and northern Idaho. Populations of the insect increase to outbreak and collapse in a variable cycle, averaging 9 years between peaks. A review of all outbreaks suggests repeated, widespread, nucleopolyhedrosis viral epizootics are responsible for the collapse of the population and, hence, the cycle. The virus appears to survive in the soil between outbreaks and to be carried incidentally to foliage where it is occasionally consumed by larvae. Ingestion of a single particle is probably sufficient to cause infection. Populations of the moth increase until density reaches the point where larvae to larvae infection is established. The viral inoculum builds rapidly following that point and spreads widely so that distant populations at all densities become infected, and collapse in the same year. The epizootic continues for another year. Then foliage contamination disappears, and populations reach their lowest densities before starting the cycle again.

Résumé

Les infestations de la chenille à houppes du douglas *Orgyia pseudotsugata* (McDunnough), dans l'ouest de l'Amerique du Nord suivaient, historiquement, un cycle de 9 ans. L'effondrement des infestations était synchrone sur une grande étendue, plus particulièrement en Colombie-Britannique et dans le Washington, l'Oregon et le nord de l'Idaho. On émet l'hypothèse qu'un virus provoquant une polyédrose nucléaire soit responsable : le virus, qui survit dans le sol entre chaque infestation, se retrouve parfois sur le feuillage où il est consommé par des larves. L'ingestion d'une seule particule de virus suffit probablement à causer l'infection. Les faibles populations endémiques augmentent pendant quelques générations sans qu'il y ait régulation par le virus. Lorsque leur densité atteint un certain point, il se produit une réinfection de larves à larves; l'inoculum viral augmente rapidement et est dispersé sur une grande distance, de sorte que des populations éloignées de toutes densités sont infectées et s'effondrent la même année. L'épizootie se poursuit pendant une autre année avant que la contamination ne disparaisse du feuillage, et les populations atteignent alors leurs densités les plus faibles, puis le cycle recommence.

Introduction

Populations of Douglas-fir tussock moth, *Orgyia pseudotsugata* (McDunnough), can fluctuate widely over time. They rapidly increase from a few individuals to large numbers that defoliate and kill trees. Damage by this pest has been reported from New Mexico to British Columbia, often recurring in the same areas every 7–10 years (Mason and Luck 1978). Outbreaks often end with an epizootic of nucleopolyhedrosis virus (NPV) (Torgersen and Dahlsten 1978). In this paper we compare patterns of all recorded outbreaks

in western North America to determine spatial and temporal relationships and thus obtain a better understanding of the dynamics of this pest and the NPV.

The tussock moth overwinters in the egg stage, hatching as host shoots expand in the spring. Larvae feed through June and early July, consuming first the new foliage and, later, if insect densities are high, the older foliage. Cocoons are found in the tree crowns in July, and moths emerge and lay eggs in August. At high elevations, these events may occur 1 or 2 months later. After emergence, the flightless females remain *in situ*, attracting and mating with males and laying eggs on their own cocoons. Favourite hosts are Douglas-fir [*Pseudotsuga menziesii* var. *glauca* (Beissn.) Franco], grand fir [*Abies grandis* (Dougl.) Lindl.], and white fir [*A. concolor* (Gord. and Glend.) Lindl.]. Occasionally, subalpine fir (*A. lasiocarpa* Hook.) and Engelmann spruce (*Picea engelmannii* Parry) act as hosts. Further details on biology, host relationships and population dynamics can be found in Brookes *et al.* (1978).

Methods

Records of outbreaks of tussock moth were collated for all areas where the insect has caused defoliation in western North America (Harris *et al.* 1985; Tunnock *et al.* 1985). We noted the date an outbreak was detected, the years of noticeable defoliation, the primary hosts involved, and the control measures as well as estimating size and severity of outbreaks. An outbreak was considered to have occurred whenever defoliation had been detectable from an aircraft. Severe defoliation (class 2) was arbitrarily defined as occurring when 50% or more of the trees had all the new and possibly some of the older foliage removed. Less severe defoliation was defined as light (class 1). Size was indicated by one of four classes: I, <40 ha; II, 40–399 ha; III, 400–3999 ha; and IV, ≥4000 ha.

Outbreaks were grouped into regions when they were within the same forest type, had a similar climate and exhibited approximately the same historical outbreak pattern. Regions were mapped and labelled 1–53 (Table 1, Fig. 1).

Host species was used as the main criterion for a second level of grouping in which regions were grouped into zones. A full description of the forests and habitat types has been given by Wellner (1978). A brief summary of host distributions is worthwhile here as background. Douglas-fir is abundant and readily attacked in southern British Columbia, eastern Washington, northeastern Oregon, Idaho, Montana, Arizona and New Mexico. It is present but not a prime host in California and Nevada. Grand fir and white fir are prime hosts that have complementary ranges; grand fir occurs north of the California and Utah borders, and white fir occurs south of that boundary. There is, however, an area in southern Oregon where these closely related species overlap and interbreed. In California and Nevada, white fir is the only prime host. Both these *Abies* species are absent or are only a minor component in the infested areas of British Columbia and adjacent north central Washington and in southwestern Idaho and Montana. In these cases, Douglas-fir is the only prime host. Other areas support *Abies* and Douglas-fir, and both are readily attacked.

Subalpine fir covers much of the same outbreak range as Douglas-fir but at a higher elevation. It is absent or only a minor component in California and Nevada but is attacked occasionally at other scattered locations, mainly in Idaho, eastern Washington and northeastern Oregon. Engelmann spruce is sometimes mixed with other species at high elevations and may be defoliated. When planted at lower elevations, various spruce species are the main hosts of "single-tree infestations" common in suburban gardens and parks. Northern Idaho and New Mexico, in particular, have supported many such local infestations. These instances were not included in the comparisons.

Outbreak regions were grouped by these prime host species and locations into seven zones (Fig. 1): the Douglas-fir regions of British Columbia and north–central Washington (Df, BC); the Douglas-fir plus true fir regions of eastern Washington (Df + Tf, W); the

Table 1. History of outbreaks of Douglas-fir tussock moth in 53 regions representing seven zones (Fig. 1)

Region	Place	Host*	Size†	Highest class of defoliation‡	Outbreak year		Start of Observation
					Start	End	
Douglas-fir, British Columbia and north–central Washington (Df, BC zone)							
1	Lillooet, B.C.	Df	?	1	1957	1958	1916
			I	1	1983	1983	
2	Ashcroft, B.C.	Df	IV	2	1947	1949	1916
			IV	2	1982	1983	
	Cache Creek, B.C.	Df	III	2	1948	1949	1916
			IV	2	1982	1984	
3	Savona, B.C.	Df	III	2	1949	1949	1916
			III	2	1973	1975	
			III	2	1982	1983	
	North Thompson, B.C.	Df	II	2	1917	1921	1916
			III	2	1930	1931	
			III	2	1948	1949	
			I	2	1964	1964	
			IV	2	1973	1976§	
			IV	2	1983	1984	
	Kamloops, B.C.	Df	III	2	1919	1921	1916
			III	2	1928	1931	
			I	1	1939	1939	
			I	1	1947	1949	
			I	1	1973	1976§	
			IV	2	1982	1984	
4	Chase, B.C.	Df	III	2	1916	1921	1916
			III	2	1929	1931	
			III	2	1946	1949	
			III	2	1982	1983	
	Monte Ck., B.C.	Df	IV	2	1946	1949	1916
			IV	2	1981	1983	
5	Armstrong, B.C.	Df	III	2	1918	1921	1916
			III	2	1930	1931	
			IV	1	1936	1939	
			IV	2	1947	1949	
			III	2	1961	1964	
			III	1	1972	1972	
			IV	2	1981	1983	
	Vernon, B.C.	Df	?	2	1919	1921	1916
			III	2	1928	1931	
			III	1	1937	1939	
			I	2	1945	1949	
			III	2	1961	1964	
			II	1	1972	1974	
			II	2	1983	1983	
6	Kelowna, B.C.	Df	IV	2	1920	1921	1916
			II	2	1947	1948	
			II	2	1971	1974	
			I	1	1982	1983	
	Westbank, B.C.	Df	II	2	1971	1973	1916
			III	1	1982	1983	
7	Hedley, B.C.	Df	IV	2	1917	1919	1916
			IV	1	1945	1947	
			IV	1	1962	1963	
			III	2	1981	1982§	
8	Olalla, B.C.	Df	II	1	1947	1948	1916
			II	1	1955	1956	
			II	2	1971	1973	
			III	1	1983	1983	

Table 1. (*Continued*)

Region	Place	Host*	Size†	Highest class of defoliation‡	Outbreak year		Start of Observation
					Start	End	
9	Twisp, WA	Df	III	2	1946	1947	1945
	Okanogan, WA	Df	III	1	1970	1973§	1947
10	Wenatchee, WA	Df	III	1	1970	1973§	1947
	Douglas-fir & true fir, eastern Washington (Df & Tf, W zone)						
11	Curlew, WA	DF + Tf	II	1	1963	1965	1928
			III	1	1980	1983§	
12	Colville, WA	Df + Tf	IV	2	1928	1930	1947
			III	1	1972	1974§	
13	Colville, I.R., WA	Df + Tf	IV	2	1972	1974§	1947
14	Spokane, WA	Df	III	1	1953	1956	1947
		Df	II	1	1962	1964	
		Df + Tf	III	2	1973	1974§	
	Douglas-fir, Montana (Df, M zone)						
15	Kalispell, MT	Df	II	1	1963	1964	1963
	Hungry Horse, MT	Df	I	1	1963	1964	1963
16	Flathead Lake, MT	Df	II	1	1956	1956	1956
			I	1	1965	1965	
			III	1	1974	1975	
17	St. Ignatius, MT	Df	I	1	1974	1976§	1956
	Ravalli, MT	Df	I	1	1974	1975	1956
18	Frenchtown, MT	Df	II	1	1973	1975§	1956
	Lolo, MT	Df	II	2	1973	1974	1956
19	Bonita, MT	Df	I	1	1964	1964	1956
20	Clearwater Junction, MT	Df	I	1	1983	1983	1956
	Douglas-fir & true fir, northern Idaho and northeastern Oregon (Df & Tf, IO zone)						
21	Mica, ID	Tf	I	1	1963	1963	1946
	Plummer, ID	Df + Tf	II	1	1956	1956	1946
			I	1	1982	1982	
	Viola, ID	Df + Tf	II	1	1945	1947§	1945
	Benewah Co., ID	Df + Tf	IV	2	1944	1946§	1944
			II	1	1956	1956	
			IV	1	1963	1965§	
			IV	1	1972	1974§	
	Latah Co., ID	Df + Tf	IV	2	1945	1947§	1945
			IV	1	1963	1965§	
			IV	1	1972	1974§	
	Moscow Mt., ID	Df + Tf	II	1	1944	1947§	1944
	Orofino, ID	Df + Tf	IV	1	1947	1947	1946
			IV	1	1955	1955	
			II	1	1973	1974	
22	Craig Mts., ID	Df + Tf	III	1	1973	1974	1973
			I	1	1982	1983	
23	Troy, OR	Df + Tf	IV	2	1944	1948§	1944
			IV	2	1972	1974§	
	Blues, OR	Df + Tf	IV	2	1972	1974§	1947
	Chesnimus, OR	Df + Tf	IV	2	1972	1974§	1947
24	Wallowa, OR	Df + Tf	II	1	1928	1929	1928
			IV	2	1972	1974§	
	Nezperce N.F., ID	Df + Tf	IV	2	1973	1974	1973
25	Rudio/Spray, OR	Df + Tf	IV	2	1937	1940	1937
26	Gold Hill, OR	Df + Tf	II	1	1928	1929	1928
			?	?	1946	1947	
			IV	2	1963	1965§	
	Malheur, OR	Df + Tf	IV	2	1963	1965§	1947
	Snow Mt., OR	Df + Tf	II	1	1947	1948	1947
			IV	2	1963	1965§	
27	New Meadows, ID	Df	II	2	1928	1929	1928

Table 1. (*Concluded*)

Region	Place	Host*	Size†	Highest class of defoliation‡	Outbreak year Start	Outbreak year End	Start of Observation
		Douglas-fir, southwestern Idaho (Df, I zone)					
28	Middle Fork, ID	Df	IV	2	1961	1964§	1955
29	Bounds Ck., ID	?	II	?	1928	1928	1928
30	Fairfield, ID	Df	IV	2	1973	1974§	1955
31	Wood River, ID	Df	?	2	1935	1939	1935
32	Owyhee Mts., ID	Df	IV	2	1949	1951	1949
			IV	2	1956	1959	
			IV	2	1963	1966	
			III	2	1970	1972	
			III	1	1976	1976	
			IV	2	1981	1983	
33	Jarbidge, NV	Tf	IV	?	1927	1929	1927
			?	?	1938	1938	
			IV	2	1960	1962	
		White fir, California and Nevada (Wf, CN zone)					
34	Wheeler Pk., NV	Wf	III	2	1955	1959	1955
35	Pioche, NV	Wf	II	?	1956	1960	1955
36	Mesquite, NV	Wf	I	?	1972	1972	1955
37	Toiyabe N.F., NV	Wf	II	2	1973	1974	1955
38	Freemont, OR	Wf	II	1	1965	1965	1947
	Bryant, OR	Wf	II	1	1965	1965	1947
			II	1	1978	1978	
39	Knox Mt., CA	Wf	IV	2	1963	1965	1955
40	Burney Mt., CA	Wf	II	1	1969	1970	1955
41	Diamond Mt./Fredomyer Pk., CA	Wf	IV	1	1965	1966	1955
42	Eldorado N.F., CA	Wf	II	2	1964	1965	1955
			III	2	1971	1972	
43	Stanislous N.F., CA	Wf	IV	2	1954	1955	1954
			III	2	1970	1972	
44	Mariposa Grove, CA	Wf	II	2	1970	1972	1955
	Park Ridge, CA	Wf	II	1	1971	1971	1955
45	Mammoth Lakes, CA	Wf	IV	2	1935	1938	1949
		White fir & Douglas-fir, Arizona and New Mexico (Wf + Df, ANM zone)					
46	Nambe Cr., NM	Wf + Df	II	2	1977	1979	1955
47	Los Alamos, NM	Wf + Df	III	1	1976	1979§	1955
48	Cochiti Mesa, NM	Wf + Df	II	2	1975	1978	1955
			II	2	1976	1979	
49	Sandia Mts., NM	Wf + Df	IV	?	1957	1960§	1955
			?	?	1967	1967	
			III	2	1974	1980	
50	Manzano Mt., NM	Wf + Df	III	2	1974	1980	1955
51	San Mateo, NM	Wf + Df	III	?	1961	1961§	1955
52	Capitan Mts., NM	Wf + Df	III	2	1958	1960§	1955
53	Signal Pk., AZ	Wf + Df	III	?	1957	1959§	1955
			II	?	1967	1971	
	Baker Mt., AZ	Wf + Df	III	?	1958	1959§	1955
			II	?	1967	1968	

*Df = Douglas-fir; Tf = true fir; Wf = white fir.
†I = <40 ha; II = 40–399 ha; III = 400–3999 ha; IV = ⩾4000 ha.
‡1 = defoliation detectable by aircraft; 2 = >50% defoliation.
§A control operation was initiated and may have caused or at least helped bring an end to the outbreak.
? = no information is available for this characteristic during the outbreak.

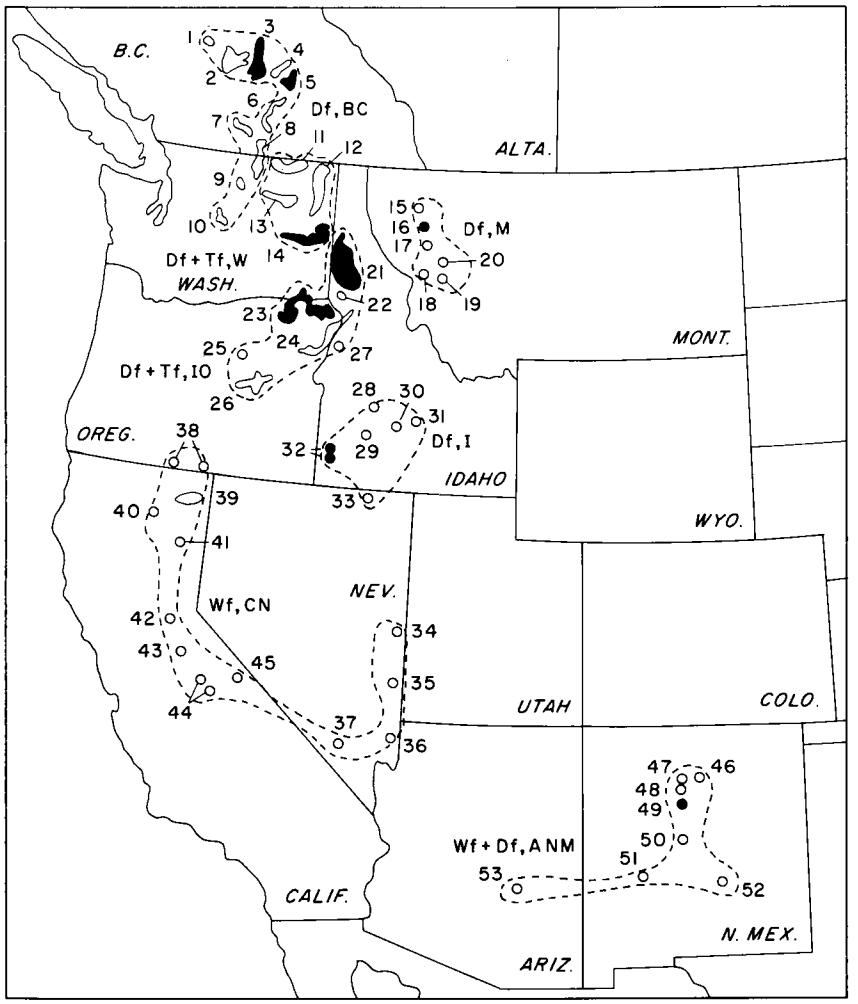

FIG. 1. Location of outbreaks of Douglas-fir tussock moth in western North America up to 1984 grouped by regions (numbered, Table 1) and zones (surrounded by dashed lines). Black regions are those where outbreaks have occurred in more than 50% of potential outbreak periods.

Douglas-fir plus true fir regions of northern Idaho and northeastern Oregon (Df + Tf, IO); the Douglas-fir regions of southwestern Idaho (Df, I); the Douglas-fir regions of Montana (Df, M); the white fir regions of California and Nevada (Wf, CN); and the white fir plus Douglas-fir regions of Arizona and New Mexico (Wf + Df, ANM).

Outbreak characteristics were compared between zones, and chi-square tests or tests of proportions were used where appropriate.

Results

Host species. There does not appear to be any relationship between the various species of host trees and outbreak characteristics (Table 2). Douglas-fir is the major component among forests suffering the most severe and also the least severe attacks. Similarly the presence of white fir or other true firs does not appear to affect outbreak characteristics.

Table 2. Outbreak characteristics by zone (zones correspond to map locations, Fig. 1)

Zone†	Number of outbreaks	Size (proportion in III or IV)‡	Severity (proportion in class 2)§	Duration (mean number of years)	Frequency (proportion of years with outbreaks)	Frequency (proportion of periods with outbreaks)
Df, I	11	0.91	0.90a	2.8ab	0.15abc	0.34b*
Wf + Df, ANM	13	0.69	0.86ab	3.6a	0.18a	
Df, BC	56	0.71	0.69a	2.8ab	0.15ac	0.46a
Df + Tf, W	8	0.75	0.38ab	3.1ac	0.15abc	0.36ab
Wf, CN	17	0.41	0.60ab	2.3bc	0.09b	
Df + Tf, IO	31	0.63	0.42ab	2.6ab	0.12bc	0.38ab
Df, M	11	0.09	0.08b	1.8b	0.10b	

*Numbers followed by the same letter in columns are not significantly different ($P < 0.05$).
†Df, I = Douglas-fir regions of southwestern Idaho; Wf + Df, ANM = white fir + Douglas-fir regions of Arizona and New Mexico; Df, BC = Douglas-fir regions of British Columbia and north–central Washington; Df + Tf, W = Douglas-fir + true fir regions of eastern Washington; Wf, CN = white fir regions of California and Nevada; Df + Tf, IO = Douglas-fir + true fir regions of northern Idaho and northeastern Oregon; Df, M = Douglas-fir regions of Montana.
‡III = 400–3999 ha; IV = ≥4000 ha.
§2 = >50% defoliation.

Size of outbreak. The frequency of outbreaks by size classes was tallied, and the distribution between classes for two zones: Df, BC and Df + Tf, IO, was compared with the total distribution of all zones using a chi-square test (Table 3). Other zones did not have samples large enough for the tests to be statistically useful.

The results for the two zones tested were significant: the Df, BC zone had class III outbreaks more often than average ($P < 0.03$), and the Df + Tf, IO zone had a higher proportion of class IV outbreaks than average ($P < 0.001$). An index of the proportion falling in classes III and IV is included in Table 2 for comparison with other population characteristics.

Severity. The Df, M zone was significantly less severely defoliated than either the Df, I or the Df, BC zone (chi-square, $P < 0.05$) (Table 2); cases in the other zones were too few to be statistically tested with any confidence.

There is a bias in the severity mean of the Df + Tf, IO zone caused by grouping. The Idaho portion of this zone (regions 21 and 22) had an average proportion in the severe class of 0.13, whereas the Oregon portion (regions 23–26 inclusive) had an average severity of 0.77. The average of all these regions, as given in Table 2, was 0.42.

Table 3. Frequency of outbreaks by size class for each zone

Zone†	Size class‡ I	II	III	IV
Df, I	0	1	2	8
Wf + Df, ANM	0	4	8	1
Df, BC	7	9	25*	15
Df + Tf, W	0	2	4	2
Wf, CN	1	9	3	4
Df + Tf, IO	3	9	1	18*
Df, M	6	4	1	0

*Significantly different ($P<0.03$ and 0.001 respectively) from overall distribution.
†Df, I = Douglas-fir regions of southwestern Idaho; Wf + Df, ANM = white fir + Douglas-fir regions of Arizona and New Mexico; Df, BC = Douglas-fir regions of British Columbia and north–central Washington; Df + Tf, W = Douglas-fir + true fir regions of eastern Washington; Wf, CN = white fir regions of California and Nevada; Df + Tf, IO = Douglas-fir + true fir regions of northern Idaho and northeastern Oregon; Df, M = Douglas-fir regions of Montana.
‡I = <40 ha; II = 40–399 ha; III = 400–3999 ha; IV = ≥4000 ha.

Duration of outbreaks. A historical diagram was constructed indicating the times of outbreaks for each region within each zone (Fig. 2). The average duration of an outbreak for all regions except Wf + Df, ANM and Df, M was 2.7 years but varied widely with a frequency of 24, 33, 44, 23, 6 and 1 outbreaks for durations of 1, 2, 3, 4, 5 and 6 years, respectively.

Outbreaks in the Wf + Df, ANM zone were significantly longer on average (Duncan's multiple range test, $P < 0.01$) than those for most other zones. In addition, the DF, M zone had significantly shorter outbreaks than the Df + Tf, W zone ($P < 0.01$) (Table 2). The analysis was done twice, once including all outbreaks and once excluding treated outbreaks on the premise that treatment would shorten outbreaks and bias the data. However, when treated outbreaks were excluded, the average duration was less than the average of all data. Obviously only the longest and severest outbreaks had been treated. Deleting them caused more bias than retaining them; therefore, averages were compiled for all data.

Periodicity and synchrony of outbreaks. Outbreaks occurred at regular intervals (Fig. 2), particularly in the Df, BC; Df + Tf, W; and Df + Tf, IO zones. Sugden (1957) had previously reviewed the chronology of outbreaks for British Columbia and noted the regular pattern. We studied the pattern further by tallying the frequency of onsets and ends of outbreaks by calendar year (Fig. 3). The latter figure confirmed that the outbreaks are periodic, occurring about every 9 years (Fig. 2). In fact, when divided into 9-year periods, all outbreaks fell within the periods; none fell on the boundaries.

The insect population may fail to reach oubreak proportions for two or three periods, but when they do so, they still retain their synchronization with surrounding regions. As an example, a compilation over a large area that extends about 800 km in a north–south direction indicates that outbreaks have remained synchronized over this whole range (Fig. 3).

Within the Df, I zone, only the Owyhee Mountains region of southwest Idaho has sufficient data to show any outbreak pattern (region 32, Fig. 2). The populations here seem to be fluctuating in a periodic manner but are on a shorter cycle than other zones. Populations within the Df, M and Wf, CN and Wf + Df, ANM zones do not have sufficient historical data to determine their pattern.

Data suggest (Figs. 2 and 3) that the variability in periodicity between regions is less with the ends of outbreaks than with the onsets of outbreaks. To test this, we compiled a frequency distribution for the calendar year of starts and of ends of all outbreaks. A chi-square test of the two distributions indicated a significant difference ($P = 0.004$, $n = 97$). When each distribution was compared with a Poisson distribution calculated around the sample mean, there was, again, a significant difference in each case ($P < 0.001$ and $P = 0.004$ for start and end distributions respectively, $n = 97$). The distribution of end dates was much narrower than either the distribution of start dates or of the Poisson distribution (S.D. = 0.84, 1.22 and 2.71 respectively) indicating that the ends of outbreaks were much more synchronized than the starts of outbreaks and that neither occurred on a random basis.

Proportion of years infested. Since surveillance began, the proportion of years when infestations took place was determined for each zone (Table 2). The Wf + Df, ANM zone had a slightly greater proportion of infested years than any other zone and the Wf, CN and Df, M zones had significantly lower proportions than did the Df, BC zone (test of proportions $P < 0.05$). The high proportion of infested years in the Wf + Df, ANM zone probably reflected, in part, the longer outbreaks.

Proportion of periods infested. A better measure of susceptibility to outbreaks than the proportion of infested years is the proportion of outbreak periods in which an outbreak occurred for each zone (Table 2). Df, BC had the highest proportion of periods with

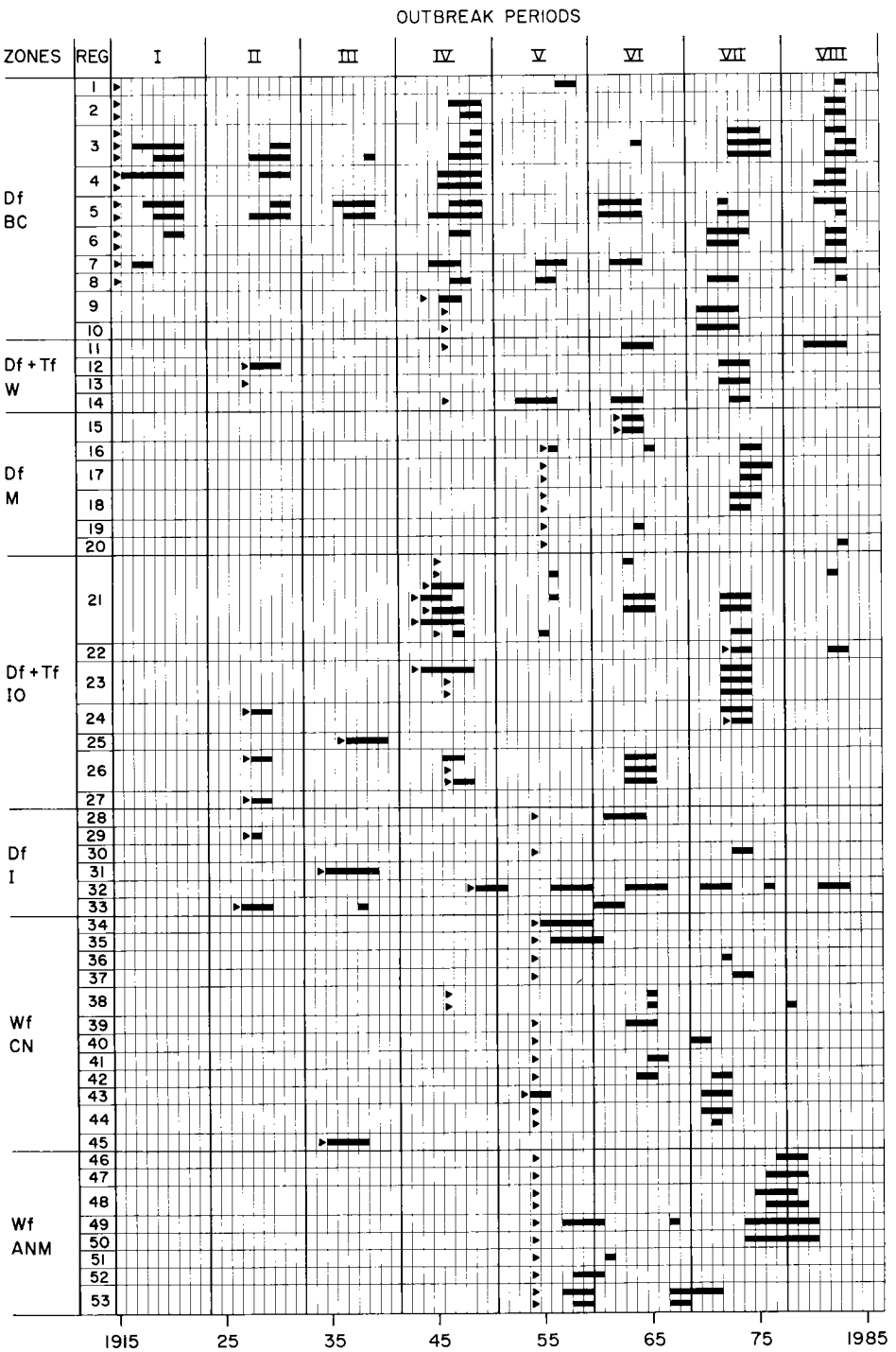

FIG. 2. Historical diagram of outbreaks of Douglas-fir tussock moth to show temporal relationships within zones and regions. Year before the beginning of adequate outbreak detection surveys for each region is indicated by a triangle.

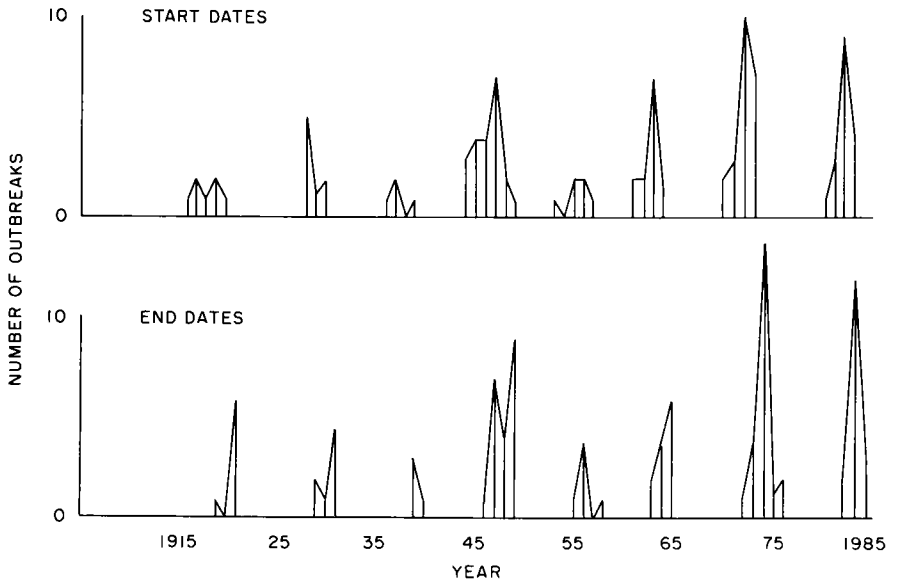

FIG. 3. Frequency of occurrence of the starts and ends of outbreaks by calendar year for zones Df, BC; Df + Tf, W; and Df + Tf, IO.

outbreaks, but the difference was not statistically significant. The Wf + Df, ANM; Wf, CN; and Df, M zones could not easily be divided into periods and, therefore, were not included in this analysis.

Within zones the frequency of outbreaks ranged widely so we calculated an index of outbreak hazard (Stark 1987), based on the proportion of periods with an outbreak. Regions with outbreaks occurring in more than half the potential periods were 3, 5, 14, 16, 21, 23 and 32 (Fig. 1), but only regions where surveillance has been present for at least four periods were considered. The exception was region 49 of WF + DF, ANM zone, which was added to the list because of the frequency of outbreaks even though the periodicity is not comparable to other districts.

Discussion

Mason and Luck (1978) and Thompson (1978) have provided a synthesis of the population dynamics of tussock moth and epizootics of NPV. Some of the main ideas put forward by them and others apply to this discussion.

They report that females are flightless and that dispersal is by wind-blown early-instar larvae. Even within favourable stands, spread is slow and outbreaks initially appear in a pattern of small patches (Shepherd 1980). In subsequent years defoliation spreads out from the initial patches, sometimes coalescing with each other (Shepherd 1977). New patches appear each year as populations in adjacent stands rise to outbreak levels. After 1–5 years, the populations in all stands collapse together, regardless of population density, and rapidly decrease to low numbers (Mason 1974, 1978; Mason and Luck 1978). Data from traps baited with pheromone indicate that 2 years later insect density begins to increase and the cycle starts again (Shepherd *et al.* 1985). Numbers may differ by 10 000–100 000 times between peaks and troughs (Mason and Luck 1978). Trees and stands are often killed after 1–2 years of defoliation (Shepherd 1980). Within a region, outbreaks often appear during every cycle within stands a few km apart. A number of factors affect survival: dispersal,

establishment of early instars, food quality, predation and parasitism are implicated but, undoubtedly, virus is the most important cause of population decline (Torgersen and Dahlsten 1978). Survival of larvae varies with the phase of the outbreak; NPV is the most common cause of death during the delcine phase (Mason and Luck 1978).

There are two morphotypes of NPV: unicapsid with a single rod enclosed in each protein envelope, and multicapsid with a bundle of rods enclosed in each envelope. The difference in virulence between the two morphotypes is only slight (Hughes 1978), but the distribution in western North America is quite different (Hughes 1976). Both morphotypes have been found in British Columbia, Washington, Idaho, Montana and northeastern Oregon. Only the unicapsid morphotype has been found in the other populations to the south.

Between outbreaks, NPV remains viable in the soil (Thompson and Scott 1979; Thompson *et al.* 1981). Particles on foliage are killed when exposed to sunlight but can remain viable in the crown when protected within cadavers stuck to the branches or in shaded niches. Within 1–2 years after the larvae disappear, the NPV also disappears from the feeding zone. For the next 6–8 years, the insect population builds; the distribution of patches of defoliation probably depends on the number of residual healthy insects left after the last population collapse and the rate of increase during the buildup.

NPV is incidentally transferred to the crown with blowing dust or by invertebrates, animals, or birds, etc. (Thompson 1978) and is consumed by the larvae. Infected larvae die and disintegrate, allowing the NPV to be released and spread over the foliage by wind, rain and biological agents. Fifth- and sixth-instar larvae feed more than the early instars so their risk of becoming infected from contaminated foliage is greater. When they die, the large larvae release larger quantities of NPV than do small larvae. Often at this stage, the incidence of infection increases dramatically.

There is no trans-ovum transmission of the disease (Thompson 1978), but egg masses are often surface contaminated with NPV and the newly hatched larvae become infected as they chew their way out of the egg masses. The wind-borne and crawling behaviour of the larvae aids in the distribution of NPV throughout the stand. Infected larvae move to new shoots, die, rupture and spread inoculum over the foliage to be consumed later by larvae that escaped the initial infection (Thompson 1978).

At constant 25°C, the virus usually develops 14 days before the larvae die (Anonymous 1979; Martignoni 1978). In the field, when foliage is sprayed with NPV, epizootics take about 28–49 days to appear, depending on temperatures (Thompson 1978; Shepherd *et al.* 1984). As larval development to pupation usually takes 60–70 days, at least two cycles of infection usually occur for each generation of host, thus permitting a rapid increase of NPV once it is established in the population.

Field populations vary in their susceptibility to NPV, but no stock has been found completely resistant. No increase in resistance has been detected after exposure to an epizootic; at the next outbreak, larvae are just as susceptible as the last (Thompson 1978).

This comparative study of outbreak patterns adds to the theory on population dynamics of this pest. It showed that the Df, BC zone has the greatest frequency of outbreaks; the Df, I zone has the highest proportion of large, severe outbreaks; and the Wf + Df, ANM zone has the longest outbreaks. Although frequency of occurrence is much the same in the Df, M zone as in other zones, outbreaks are consistently less extensive, less severe and shorter than those elsewhere.

Outbreaks in British Columbia, Washington, Idaho and Oregon appear to be not only periodic but synchronous over large areas (Fig. 3). To confirm periodicity, we would need to carry out serial correlations on quantitative data (Moran 1954). This type of data is not available but the tests we were able to conduct indicated that the pattern is nonrandom and is synchronous in at least some of the populations (Mason 1978). Also, the ends of outbreaks were more closely synchronized than onsets; that is, the dates at which outbreaks

begin vary between regions but most outbreaks end in the same year. We conclude from this that some factor or group of factors is causing the sudden collapse of the population in the same year over a large area, and this factor is keeping the population in synchronous cycles. The most logical agent that can cause such an effect is the NPV, which has repeatedly been associated with collapsing populations (Brookes *et al.* 1978). We have known for many years that NPV can destroy populations, but the evidence provided here indicates that it happens over a wide area in a coordinated manner.

With such widespread synchronized epizootics, the dispersal, and therefore the dilution, of inoculum must be great. But, this particular NPV is so virulent (Burges and Thompson 1971) that a single particle may be sufficient to infect the host, rapidly reproduce and kill it (Huber and Hughes 1984). The work of Thompson and Scott (1979) supports this theory; their dilution studies indicate ready infection at less than 10 polyhedra per larva. Therefore, a threshold density of inoculum is not necessary before infection takes place and percent infection is related to virus distribution, not concentration. Increasing concentration of the inoculum will shorten the time to death because the NPV will need fewer generations before reaching lethal levels. However, the proportion of individuals killed in the first wave of mortality will not increase with greater concentration unless there is also an increase in distribution over the foliage.

Disease prevalence is proportional to the number of encounters between feeding, uninfected hosts and virus-contaminated foliage (Fuxa 1987). Virus inoculum can be transmitted from the soil or from previously infected larvae. At endemic densities, inoculum from the soil is probably the major source of infection, and its occurrence and distribution are independent of insect density. But, as insect density increases from generation to generation, a threshold would be reached when insect density is high enough for the probability of infection from larvae to larvae to exceed that from soil to larvae. At this point the amount of virus in the environment begins to increase rapidly, particularly as the virus can usually go through at least two cycles of infection in one generation of the insect. Rate of infection and subsequent rate of virus production would then be expected to be dependent on larval and viral density and distribution. There is a dispersal of NPV from the dead infected larvae until the distribution of NPV is sufficient to cause infection of most larvae. Dispersal and mixing of inoculum must be widespread to maintain synchronization over such wide areas. At this point NPV polyhedra are more widely distributed than the insect and the rate of infection becomes independent of host density (Thompson 1978); an epizootic occurs in all stands shortly thereafter.

The frequency of tussock moth outbreaks varies, and areas where risk of outbreak is high were identified (Fig. 1). Within the high-risk regions there must be a unique set of favourable environmental factors that enable the insect populations to increase rapidly after a crash. In other regions, tussock moth populations rise and fall in synchrony with those in high-risk regions but do not reach outbreak densities before collapsing (Mason *et al.* 1983). Outbreaks occur only every second or third period when conditions are particularly favourable. The factors controlling the susceptibility of a region to tussock moth outbreaks are unknown; perhaps weather has a direct or indirect effect through the buildup period.

Fluctuations in forest pest densities are often attributed to weather, but the regularity in outbreak collapse of tussock moth populations precludes it as being a main causal factor. However, it may be related to the regional differences in rates of population increase. Previous studies resulted in statistically significant correlations (Watt 1968; Clendenen 1975), but no cause–effect relationships were proposed.

There have been outbreaks in isolated stands of New Mexico where virus was not a factor in the collapse; rather, the host trees were quickly killed and the population starved. There are also reports of three suburban infestations on spruce trees where virus did not appear and the outbreaks lasted an extended time (9, 12 and 12 years). These anomalies may be related to the continuity of the forest. In the north there is ample opportunity for

virus to disperse from population to population, whereas, in New Mexico and Arizona, forests are small islands separated by plains of desert or agricultural crops (Harris 1984), and virus cannot spread adequately from population to population to maintain the synchrony of the epizootics. In addition, the high amount of solar radiation experienced in this zone could reduce residual virus concentrations and perhaps even result in local extinctions. The probability of infection of building populations would thus be reduced. This could affect control strategies in the southern zones.

Wellington (1962) discussed the spread of NPV in western tent caterpillar, *Malacosoma pluviale* (Dyar), populations via foliar contamination, trans-ovum transmission, larva-to-larva contact, and even the appearance of latent virus after starvation stress. He did not mention the possibility of wind dispersal but the disease of western tent caterpillar is not as virulent as that of tussock moth, and consumption of higher concentrations may be necessary before infection will take place.

There are documented instances where populations have collapsed without a virus being identified as the primary cause. In 1973–74 the virus did not appear until late in an outbreak in northeastern Oregon. Considerable defoliation occurred with accompanying larval starvation, mortality and reduced fecundity (Mason 1981). Population densities began to drop before the virus was effective. In 1978–79, a suboutbreak population in the Eldorado National Forest, California, dropped in density dramatically with most of the disappearance occurring in the early instars (Mason *et al.* 1983). Investigators could find no evidence of disease in field-collected or reared larvae. Obviously not all outbreaks collapse because of a viral epizootic, but no other factor or set of factors has been identified which can result in causing synchronous, periodic outbreaks over a wide area.

Berryman (1978) pointed out a simple mathematical model based on density-dependent mortality factors with a time delay of one generation. This theoretical formula resulted in a general pattern similar to that exhibited by tussock moth outbreaks but did not identify the specific mechanisms involved.

Anderson and May (1980) suggested that populations of forest insects can exhibit cycles when pathogens are the main driving factors, and they included tussock moth as an example. They proposed differential equations to model the system based on density dependence. They then used a density threshold to trigger epizootics in the model but did not consider rate of spread as being the driving variable. Our data indicate that when epizootics occur, all populations succumb, regardless of density. This fact has been observed repeatedly and verified by experimental application of NPV (Shepherd *et al.* 1984).

Vezina and Peterman (1985) tested the Anderson and May (1980, 1981) models, and variants thereof, but could not generate an outbreak pattern similar to that of tussock moth. They added components for density-dependent mortality, and vertical transmission and an incubation period for the virus but still could not obtain a close relationship. The failure may have been caused by their trying to predict dynamics over time without regard to spatial relationships. Inclusion of factors for the distribution of stands, insects and disease and rates of spread of the organisms may improve the fit of their model.

In the 27 regions of the northern zones, the dates of outbreak have fallen into distinct 9-year periods with no exceptions. This is surprisingly constant in comparison with the dynamics of most forest insect populations; it shows a relatively stable time relationship for NPV epizootics. The critical factors for any model appear to be the timing between population increases of the host and the time of introduction, rate of increase, and, particularly, the rate of dispersal of NPV inoculum over the crowns. Knowledge of the functional relationships of introduction and spread of NPV could be useful when timing NPV applications to prevent outbreaks (Shepherd and Otvos 1986). The most feasible way to obtain this information is to undertake comparative population ecology studies between

the synchronous, cyclic populations of Oregon and Washington with the isolated populations of New Mexico, the short-cycling populations of Montana, and the fast-cycling populations in southwestern Idaho. Special attention would have to be given to the NPV–tussock moth interactions throughout a population cycle, considering both NPV morphotypes. Specifically, the rate of increase and rate of infection of suboutbreak populations of the moth, the carry-over compatibilities of virus in the soil, the effectiveness of transfer mechanisms between the soil and foliage and the distribution and rate of speed of virus through the forest should be investigated.

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