AN ABSTRACT OF THE THESIS OF

Jimmie Osborn Cobb, Jr. for the degree of Master of Science

in Forest Ecology presented on September 19, 1977

Title: SOME STRATEGIES FOR CONTROLLING THE DOUGLAS-FIR TUSSOCK MOTH BY REGULATION OF BUDBREAK

Abstract approved: _____
Dr. Michael Newton

The young larvae of the Douglas-fir tussock moth (Orgyia pseudotsugata (McDunnough) (DFTM) are dependent on the new foliage of its hosts (Douglas-fir, grand fir, white fir) for their food supply. The phenology of the DFTM and its hosts are synchronized, with egg hatch and dispersal occurring one to three weeks after first budbreak of the hosts. A method of host management that would upset this synchrony and deny the newly hatched larvae the new foliage necessary for their survival could provide an effective means of control for a DFTM population.

The proposed approach is one of host management utilizing plant growth regulators to delay budbreak of the hosts and thus starve the DFTM. This method would minimize effects on non-target organisms. Growth regulators have been identified with the capability of altering phenology of many agronomic and horticultural plants.

The study involved application of eight different growth regulators, each at three different concentrations applied during three seasons. Study areas were located in western Oregon and in eastern Oregon in an area where the DFTM had previously been a problem. Both Douglas-fir and grand fir were utilized. Growth regulators were asulam, Cycocel®, DPX-3778, ethophon, Krenite®, maleic hydrazide, NAA, and silvex. The applications were made to simulate aerial deposits.

Observations were made on individual trees for date of budbreak, height growth, and foliar damage.

A constant record of air temperature was made during the period of budbreak on both study areas. This record was utilized to compare cumulative budbreak with accumulated heat units.

The effects of the growth regulators on budbreak and height growth were analyzed by various methods of analysis of variance and covariance. On one study area the budbreak history of individual trees was known, and the inherent variation in time of budbreak was removed from the analyses.

A silvex treatment in November provided the longest delay, one of 6 days. Some other treatments, notably Krenite® and maleic hydrazide fall treatments, indicated some delay capabilities, but they also caused foliar damage. The majority of the treatments provided no delay, and the day of budbreak analyses indicated a lack of significant delay attributable to any treatment.

The treatments had a significant effect on height growth response as expressed by a ratio of growth the year after treatment to growth the year before treatment. Ethophon and silvex stimulated growth, and all other treatments reduced growth.

In general, the chemical treatments observed in these experiments did not show potential for de-synchronizing the DFTM and its two important host species. The failure of treatments to cause a major postponement implies that the tendency toward normal phenology is very strong.

Some Strategies for Controlling the Douglas-fir Tussock Moth by Regulation of Budbreak

by

Jimmie Osborn Cobb, Jr.

A THESIS

submitted to

Oregon State University

in partial fulfillment of
the requirements for the
degree of

Master of Science

Completed September 1977

Commencement June 1978

APPROVED:

Professor of Forest Ecology
in charge of major

Head of Department of Forest Science

Dean of Graduate School

Date thesis is presented September 19, 1977

Typed by Susie Avery for Jimmie Osborn Cobb, Jr.

ACKNOWLEDGEMENTS

I would like to sincerely thank Dr. Michael Newton for his assistance and guidance on this project and in my development as a scientist.

My thanks are also extended to Dennis Muscato for his invaluable assistance with data processing. Boise Cascade Co. and Starker Forests kindly cooperated and assisted by providing the land and trees utilized during the experiments.

I would also like to thank my wife for being there when I needed her.

TABLE OF CONTENTS

Table of Contents (Continued)

LIST OF FIGURES

LIST OF TABLES

SOME STRATEGIES FOR CONTROLLING THE DOUGLAS-FIR TUSSOCK MOTH BY REGULATION OF BUDBREAK

INTRODUCTION

Traditional technology for control of the Douglas-fir tussock moth (Orgyia pseudotsugata (McDunnough)) (DFTM) and other forest insect pests has focused on direct chemical control methods. The methods depend primarily on insecticides, and to a lesser extent biotic agents, that are used largely after epidemics have erupted and some stand damage has occurred. Insecticidal control methods have effects on non-target organisms which are either known and considered undesirable, or are viewed with considerable uncertainty. There is a need for a means of control in which side effects do not occur or do not cause extensive mortality of non-target organisms. This thesis examines a DFTM control method designed to alter specific host conditions necessary for survival of the insect.

The proposed approach is one of host management designed to upset the relations that exist between host and insect. The specific technology entails use of plant growth regulators to delay budbreak of the hosts. A synchrony exists between budbreak of host trees and egg hatch and dispersal of the DFTM. The insect is dependent on this synchrony to insure the timely emergence of new foliage necessary for survival of juveniles. The ecological question under investigation is whether a stimulus that affects the host alone will cause a

breakdown in the arrangement that exists between the insect and its host. A physiological question under investigation is whether chemical growth regulators can be found that will delay budbreak of host conifers with minimal non-phenological effects.

This research approaches the question of management of the hosts of the DFTM for the first time, and in doing so may provide an additional option in DFTM management. Perhaps equally important, it considers a fundamental relationship between defoliators and hosts that is not specific to the DFTM, but may carry over to other insects.

Growth regulation would have some advantages over methods using insecticides, and in some instances selection and breeding. Direct effects on animal, bird, or insect populations would not occur as with animal specific toxicants. The trees' phenology would not be permanently altered, and insect adaptation would be unlikely. A short delay in budbreak would still allow the host trees adequate time for completion of growth and development. The only possible non-target effects would occur if the growth regulators utilized had a detrimental effect on flora essential for some associated consumer population.

LITERATURE REVIEW

Ecological Relationship of Insect and Host

The DFTM has three main hosts; Douglas-fir (Pseudotsuga menziesii (Mirb.) Franco), grand fir (Abies grandis (Dougl.) Lindl.), and white fir (Abies concolor (Gord. and Glend.). The DFTM is a univoltine insect which seriously defoliates Douglas-fir and true fir over much of the western United States and Canada (94). In late summer or early fall, each female deposits several hundred eggs in a single mass, most commonly on or near foliage of the host tree. The eggs over-winter, and hatch occurs in late spring or early summer, shortly after budbreak of the host trees. Some shoot elongation takes place before the larvae begin to feed (93).

The phenology of many phytophagous insects and their hosts is delicately synchronized, particularly in host species that exhibit markedly seasonal growth patterns (23). Wickman (92 and 93) has studied the phenology of the DFTM and its hosts in California and N. E. Oregon. His initial work in California in 1971 showed that at various elevations egg hatch occurred consistently several days after first budbreak, with 25 percent of the season's shoot elongation taking place by the time most larvae started feeding. In 1972, he found on three different sites studied that the period between first budbreak and dispersal of all larvae was 17 to 20 days (Table 1).

Table 1. Length of phenological development (in days from first budbreak), Eldorado National Forest, CA. From Wickman (93).

area	first budbreak	50% budbreak	first egg hatch	90% budbreak	peak egg hatch and dispersal	dispersal ends
1	0	4	6	7	8	20
2	0	4	6	11	16	20
3	0	2	3	12	7	17

First egg hatch occurred when budbreak on white fir was 63 to 86% complete, with approximately 25% of shoot elongation taking place while the larvae were still in the second instar. New larvae remained on the egg masses for an average of three days after hatching, with dispersal then beginning and progressing rapidly. Most larvae left the egg mass within the next day or two. Within a few hours after dispersal, many larvae were seen crawling up to the upper crown foliage (six to ten ft. trees) and beginning to feed.

In N. E. Oregon in 1973 (92) slightly different results were found, but strong synchrony was consistent (Figure 1). The period from first budbreak to complete larvae dispersal was slightly longer than that in California, with shoot elongation 90% complete by the second instar. In Oregon, the summer of 1973 was particularly warm and dry, resulting in a rapid rate of shoot growth. First egg hatch coincided with 86 to 100% budbreak on grand fir and 70 to 97% budbreak for Douglas-fir, slightly more than the 63 to 86% budbreak for white fir in California in 1972. Time of egg hatch was the most

variable factor, taking place over a 6 to 14 day period at a given location.

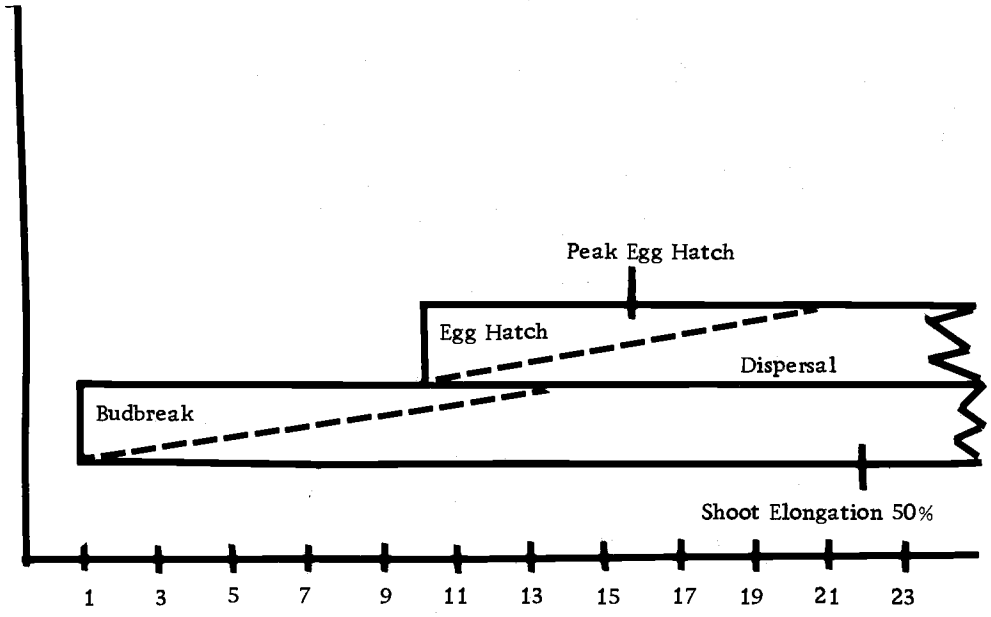

Figure 1. Schematic representation of average budbreak and egg hatch, from Wickman, N. E. Oregon (92).

There consistently appears to be a period of approximately 20 days between first budbreak and larvae dispersal, with shoot elongation well underway when larval feeding begins. Mitchell and Wiggins (56) reported the peak dispersal period immediately follows the beginning of egg hatch, and that the first few days may be the most important period of dispersal, in terms of numbers of larvae.

The synchrony with budbreak gives the juvenile DFTM an initial advantage by assuring a suitable food supply during the first several instars of development. Not all defoliators follow the same phenological pattern. Varley (84) found that the winter moth in England generally emerges two weeks before most of its host oaks

have flushed, resulting in high starvation and mortality. Embree (24) stated that the synchrony of budbreak and egg hatch was a key factor in survival of the winter moth larvae. He concluded that "Numbers of hatching larvae which survive on the foliage are controlled by the degree of synchronism between insect and host phenology."

Hatching DFTM larvae will always find foliage available, since the hosts are evergreens. The year-old foliage of its host, however, is not an acceptable food for the young larvae. The first and second instar larvae are dependent on new foliage for a food supply and cannot effectively utilize the older needles until the third instar (8). Mason and Baxter (55) found when new white fir foliage was killed by a late frost, mortality and starvation of young larvae were high. When acceptable food was not available, the larvae often dispersed from the foliage altogether rather than feeding on older needles. They dispersed by crawling or ballooning on silk threads, with survival dependent on coming into contact with preferred foliage (new growth). But with the lack of new foliage, the larvae are exposed to more physical hazards as they seek food, and quick mortality results from starvation and increased risks of physiological disorders, virus, predators, and parasites.

The synchrony of budbreak and egg hatch normally provides the DFTM with an adequate food supply. But if the synchrony were

upset and new foliage were not available, the dependence of the young larvae on the new foliage would make them vulnerable. This phenological linkage between pest and host could become a major weakness in the moth's life cycle. This suggests a possible strategem for controlling the DFTM; the artificial delay of budbreak as suggested by Eidt and Little (23) for the spruce budworm.

Insect Dynamics

In order to discuss strategies for controlling the DFTM, we must know more about the population dynamics of the insect. Forest defoliators consistently go through three population phases: release, outbreak, and decline (30). These phases may each last several years. In population cycles of the DFTM, all phases commonly occur in three years, with one year for release, one to one and a half years of outbreak, and one half to one year for decline. Larval populations may increase five to ten times or more during the release phase, growing from an insignificant level to outbreak proportions in one year. The outbreak phase, or second year, is when the moth is usually noticed and heavy defoliation takes place. Sometimes the population collapses at the end of the second year from natural causes, but often a large number of eggs will be laid that indicates a large beginning population in the third year. During the decline phase, if past defoliation has been severe, survival during

early instars is low and the population usually collapses during the third summer of the outbreak (94).

The dense population present combined with the already heavily defoliated host creates survival problems for the larvae. Under conditions of dense populations, the degree of acceptance of old foliage is postulated as governing the survival rate of the moth. Beckwith (8) found that the larvae (third instar and later) will consume new foliage instead of old foliage, when both are available. Larvae forced to feed on old foliage took longer to develop from the third instar to pupae stage, weighed less than those fed on new foliage, and laid fewer eggs. He concluded:

> It appears that primarily new growth foliage will be consumed during the release phase of the hypothetical model of an outbreak as proposed by Wickman, et al. (1973). Larvae will be forced to feed upon old growth foliage during the outbreak phase because of high population density. Depletion of current growth in the early instars will bring about nutritional stress, mass starvation of the young larvae, delayed development, increased exposure to biological controls, reduced egg production and a general decline in population quality. These influences are part of the many factors leading to overall population collapse.

Hard (33) has found that on experimental plots treated with Dimillin, it took 21 to 35 days to achieve a population reduction greater than 90%. During this time, primarily new foliage was consumed, with very little damage to old growth foliage. It is apparent the moth's food preference and nutritional need requires new growth foliage.

In order for budbreak delay to provide effective control, there must not be enough new growth to feed the early instar larvae. The probable timing of a budbreak control method would be the outbreak phase, when a high larval population will be present. Peak egg hatch will occur approximately eight to ten days after normal budbreak, with most larvae dispersed after 17 days (92, 93). After dispersal the larvae's life span without food would be approximately two days (Wickman, personal communication). Synthesizing from the various reports of Wickman, Beckwith, Mason, and others, this pattern indicates that budbreak delay of three weeks would reduce a DFTM population drastically, and provide a very definite means of control.

With a high outbreak level population, large amounts of new foliage must be present for the first instars to feed upon until they develop into the late instars capable of feeding on old growth foliage. In a situation with a high larval population and only a small amount of new foliage, the larvae presumably would deplete the new foliage before the population had matured to stages capable of utilizing the older foliage. If budbreak were delayed for a two week period, and egg hatch were average, there would likely exist shortages of food for the hatching larvae. As a result, many larvae would be dispersed and already searching for food before budbreak. New foliage would be available only in small amounts; presumably the food supply could be depleted faster than the postulated course of budbreak and

elongation could replenish it. The heavy larval feeding on the just broken buds would reduce the amount of food available by destroying the new needles before they were fully expanded. This feeding could cause severe foliage damage on early flushing trees, but the depletion of current growth could help create some of the population stresses noted by Beckwith (8) in a decline phase population. The larvae might also disperse from the old foliage in search of food, making them more susceptible to natural biological or insecticidal controls. The critical question of rates of foliage supply and demand cannot be easily answered with present knowledge.

One could surmize that a delay of eight days or less would not provide effective control of population levels.

Phenological Development of Hosts and Insect

Knowledge of phenological development of host trees and insects is necessary in scheduling population level sampling and control operations. The difficulty encountered in monitoring egg hatch of forest insects can be alleviated if a phenological relationship can be found that is simpler to monitor than an insect egg hatch. Studies with many insects have shown that correlations between insect development and host phenology exist and are practical to monitor (6, 7, 11, 18, 23, 34, 57, 64, 88).

The first attempt to relate DFTM egg development and ambient air temperature was made by Perkins and Dolph in the Burns, Oregon, infestation in 1965 (65). They noted, "whenever the daily average cumulative air temperature nears 50°F. (10°C.), DFTM eggs are likely to start hatching." Wickman (92, 93) related his findings on date of budbreak, egg hatch, and dispersal to accumulated degree-days. He arbitrarily used a threshold of 42°F. (5.56°C.) because the developmental threshold for hatching DFTM eggs was unknown. Forty-two degrees F. (5.56°C.) has often been used for a 'base temperature' for the onset of vegetative growth (77). Wagg (88) observed spruce budworm development was related to that of grand fir, and the threshold of development for grand fir was 42°F. On studies of the noctuid moth, Hardwick (34) also used 42°F. as a threshold.

Wickman obtained the daily mean temperature by summing the maximum and minimum temperatures and dividing by two. Heat units were accumulated by subtracting 42°F. from the daily mean temperature, with each degree remaining termed a degree-day. Negative values were considered zero, since growth cannot be reversed, only arrested. Figure 2 and Table 2 show patterns found in Oregon and California. The degree-day x percent budbreak graph clearly shows the relation in egg hatch and budbreak previously defined and also illustrates their close relationship to degree-days.

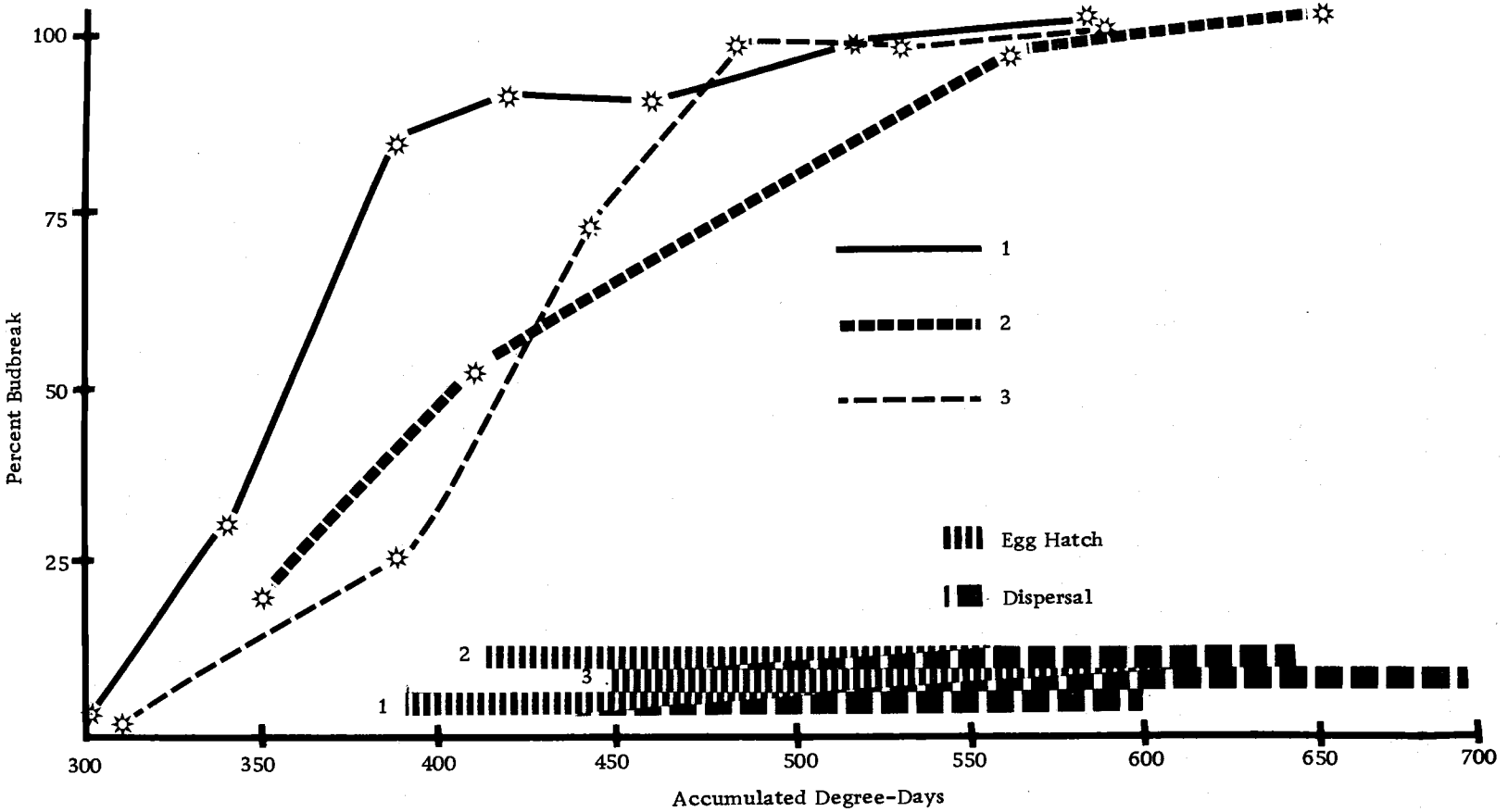

Figure 2. Accumulated degree-days, percent budbreak, and egg hatch and dispersal for each study area, Eldorado National Forest, CA. Wickman (93)

The Oregon results are similar to the phenological relationship found by Wagg (88) in the same area. Wickman concluded that the phenological studies in California and Oregon show that monitoring budbreak is a practical and simple phenological index that can be used effectively to predict DFTM egg hatch. (Fahrenheit is used here and in the rest of this study so that comparison can be made between past results and those of the author.)

Table 2. Progression of phenological events related to accumulated degree-days. Eldorado National Forest, CA., and Blue Mountains, OR. Wickman (92, 93)

	Event	Started	50%	90%
California	Budbreak	290-356	354-414	464-535
			Peak	End
	Egg Hatch	386-446	441-468	520-604
	Dispersal	420-484	520-604	601-696
Oregon	Budbreak	244-268		340-386
	Egg Hatch and Dispersal	380-420		540-600

In using a degree-day index it is important to keep in mind some of its shortcomings. Arnold (1) pointed out that these accumulations assume a linear relationship between temperature and plant development, but the true relationship is probably not linear. Other environmental factors besides temperature are affecting the rate of development. Temperature measured in one microsite is used to

base predictions for many other sites. A few days of abnormal weather could alter the relationship. As long as the degree-day index is used as a guide index and samples of the target are periodically examined, many of the shortcomings can be overcome.

Since the relationship of bud development with temperature is not linear but probably curvilinear, the onset of budbreak increases with the rate of temperature accumulation (93). Wickman found budbreak onset between 244 and 268 degree-days in Oregon and 290 and 356 degree-days in California. California had a cool spring and Oregon a hot spring during the year each was observed, so the Oregon trees had a faster rate of development for the same accumulated temperature.

One of the shortcomings of the index can be illustrated in the process of budbreak in Douglas-fir. During a spring with unseasonally cool weather, the degree-day relationship to budbreak is known to breakdown, with very slow rates of bud development. Temperature effect on budbreak can then become less important than daylength, which could then stimulate processes important in budbreak (Lavender, D. personal communication).

The tree's phenological development does not progress simultaneously in all its parts. In observing trees in various provenance studies, Ching (Ching, K. personal communication) has noted that some trees exhibit a time difference between budbreak of

lateral and terminal buds, with either flushing first, and the other lagging up to five days. Laterals may flush first with budbreak then progressing toward the terminal, the opposite may occur, or buds on all positions of the tree could break at once. Trees from identical seed sources planted in different locations will exhibit different patterns of development from location to location. In a given location as the trees age, the lapse of time in budbreak in various portions of the tree may narrow or reverse order. Ching does not attempt a simple conclusion as to why these changes in phenology take place.

Provenance studies have shown that phenology of various Douglas-fir seed sources changes in various environments (16). Budbreak times change as the seed sources are moved to different areas, and not usually corresponding to the natural seed source. This demonstrates the tree has genetic codes that vary from seed source that govern the trees response to environmental changes. The relation of budbreak or other phenological developments to degree-days cannot be considered valid unless they have been tested in the particular area. The complexity of phenological development and its relation to the environment of a particular location is such that it is hard to predict a tree's reaction to various environmental stimuli. However, the inability to understand different interactions of the whole system should not discourage us from a simplified approach that has shown merit through field correlations (58).

Physiology of Budbreak and Dormancy

Dormancy of most temperate zone perennial plants can be divided into two intergrading physiological stages; summer dormancy or correlated inhibition, and winter dormancy or rest (50). Summer dormancy results from unfavorable conditions for growth such as high temperatures or water stress, and can be reversed by more favorable conditions for growth. If the unfavorable environment is maintained, summer dormancy will gradually convert to winter dormancy. Internal conditions would then prevent growth even if favorable growth conditions were provided. Plants thus limited by internal conditions are said to be in winter dormancy. Samish (70) further divided winter dormancy into three phases; pre-rest, rest, and post rest. Lengthening nights in the fall apparently trigger the transition from summer to winter dormancy in some species. Chilling generally induces post dormancy (post-rest). When ambient temperatures become favorable for growth, post-rest is alleviated and growth resumes.

Implication of hormonal control over dormancy has been discussed since the thirties. Most evidence now available indicates that bud dormancy is regulated by a balance and interaction among endogenous growth promoters and inhibitors and environmental stimuli, but the precise mechanism of regulation is not understood

(4, 5, 20, 89, 90). It is difficult to establish a direct cause-and-effect relationship between changes in plant hormone levels and associated plant responses. Changes in gibberellins, cytokinins, IAA, and inhibitors have been noted in the literature as causes of bud dormancy and budbreak, but there is presently no general approach that will unequivocally link the changes in hormones and dormancy phenomena (50). It is reasonable to assume that connections between the plant's hormonal and nucleic acid components and environmental cues are important in the process of dormancy (90). It is likely that no one factor will ever be singled out as the controlling process of dormancy, but rather a number of processes and changes within the plant will be identified that are keyed to different environmental factors and changes with complex interrelationships and feedback mechanisms. Budbreak in Douglas-fir has been shown to be affected by such diverse factors as water stress (17), root disturbances in the fall (37, 50), fall photoperiod (45), plant maturity (15), and soil temperature (48). The delay in budbreak in Douglas-fir caused by a fall root disturbance indicates that dormancy can be affected by long term processes that are not seemingly related.

Investigations of Douglas-fir showed that only buds of this species have a definite chilling requirement (46). The roots may have periods of growth and rest, but the entire root system is never entirely dormant (69, 75). Changes in cell constituents, including

growth substances, associated with dormancy changes in buds on Populus balsamifera have been recently reported by Bachelard and Wightman (4, 5). The authors presented data pointing toward hormonal control over dormancy, but did not indicate the nature of the substances or how they operate in induction or breaking of dormancy.

A large number of references related to plant dormancy and growth substances are found in reading a selection of review papers (28, 54, 40, 70, 85, 86, 90), but the literature dealing with dormancy in coniferous species is not extensive. Wareing and Saunders fail to mention coniferous species in their review of dormancy and hormones (90). Some specific methods of dormancy induction and release have been proposed for certain species (5), but none of these could be applied closely to Douglas-fir or grand fir on the basis of present knowledge. The work of Lavender, et al (49, 45, 51) has indicated that the physiology of dormancy may be more complex in Douglas-fir than in other species, particularly angiosperms. The differences in response of conifers and the angiosperms that have mainly been used in examination of dormancy control can be confusing. For these reasons the following discussion of dormancy mechanisms will be based on information related to conifers and as closely related to Douglas-fir and grand fir as possible. Most of the work discussed here is from Douglas-fir, but grand fir should behave similarly (Lavender, personal communication).

Relatively dry summers induce a condition of midsummer dormancy or quiescence in Douglas-fir. Douglas-fir apparently forms buds because of moisture stress rather than photoperiod or temperature. Buds generally form no later than mid-July, while photoperiod and temperatures are still favorable for growth (50, 51). This condition may be related to a build up of abscisic acid and/or other inhibitors and possibly changes in the levels of gibberellins, cytokinins, and IAA. ABA levels in Douglas-fir buds increase in the summer (July) to levels approximately ten times those of March (91). As summer progresses, substances responsible for deep dormancy continue to build up until in the fall the tree is converted to a stage of deep or true dormancy. Exposure to long photoperiods, plentiful moisture, or chilling temperatures during this transitional period will reduce substantially the beneficial effects of 'chilling' during the winter months, greatly delaying budbreak and shoot elongation the following spring. Perhaps because most other plants investigated initiate dormancy in response to photoperiod, this requirement for a period of mild, short, relatively dry days between formation of the winter resting bud and the initiation of 'chilling' had not been reported previously (50).

The exact mode of dormancy regulation is not known. Some evidence indicates dormancy may involve a reduction in the capacity of DNA to support the synthesis of RNA, and ABA may be involved

in this effect (89). Owens (63) has concluded from his studies directly on Douglas-fir that inhibition of DNA synthesis is not a cause of dormancy, but is one of the manifestations of the dormant apex. Based on his work and that of others (22, 26), he believes that changes in carbohydrate levels and/or the accumulation of inhibitors and reduction in temperature may account for the marked decreases in both DNA synthesis and mitotic activity.

In November the plant can tolerate freezing temperatures without damage, but it cannot tolerate root disturbance without harmful effects the following spring (reduced survival, delayed budbreak, loss of vigor). Loss of roots or damage in the fall results in insufficient hormones exported to the shoots the following spring (50). Changes in cytokinin levels, which are high in the fall, could be one reason for the effects, and the levels are lower in midwinter when there is little damage from disturbance. ABA levels are also high in the fall (91). Hormone levels in the fall would prevent growth even if a favorable environment is provided. Long days in the fall produce later budbreak than short days (45). This indicates some balance is affected by photoperiod that may or may not be a factor in normal dormancy regulation.

Fall and winter temperatures result in gradual changes in levels of promoters, inhibitors, and carbohydrates. Owens (63) observed that the period of lowest metabolic activity in Douglas-fir

buds extends from about mid-December to mid-March. After chilling requirements have been met, root disturbance has little effect on subsequent changes in dormancy. As spring approaches, ABA levels are down, gibberellin levels start increasing, as do IAA and cytokinins, and starch levels rise in one-year-old shoots.

At this point the tree is capable of breaking its buds when the right environmental conditions are provided and after the time necessary for the tree's physiological responses. There are a host of environmental conditions that can affect budbreak (Ching, personal communication), and a number of internal regulatory processes that undoubtably affect budbreak. Lavender, et al.(48), and Thielges and Beck (78) have provided evidence that once the dormant condition of the vegetative bud is broken and all environmental conditions are favorable for growth, budbreak is stimulated by growth substances synthesized in the roots and exported to the buds. Lavender et al. (48) have demonstrated that budbreak in Douglas-fir is significantly delayed by either chilling the roots (5°C.) or by preventing root export to the shoots. They have also correlated marked increases in gibberellin levels with rising soil temperatures in the spring. They demonstrated that seedlings with chilled roots (5°C.) broke buds two weeks later than seedlings with warm roots (20°C.), and that exogenously applied GA speeded budbreak of the seedlings with chilled roots so that their budbreak coincided with those with warm

roots. Exogenously applied GA did not affect budbreak on the seedlings with warm roots.

Lavender, et al. (48) and Thielges and Beck (78) believe that gibberellin export is a factor responsible for stimulating budbreak. This hypothesis does not preclude budbreak initiated by endogenous compounds in nondormant (chilled, but not open) buds, but attributes a significant stimulatory effect to additional gibberellin synthesized and exported to the buds by actively growing root systems. The correlation of the increases in soil temperatures, GA activity, and stimulated budbreak does not necessarily establish causality. However, a genetic response keyed to the relatively consistent increase of soil temperature with time in the spring would match well with the existing knowledge of the precision of control of Douglas-fir phenology by the climate in which it has evolved (12, 76). It is reasonable to assume a growth regulator that would interrupt processes in the GA synthesis, translocation, activity at receptor sites, or other supporting process necessary for plant response would have a delaying effect on budbreak.

Mode of Action and Use of Synthetic Growth Regulators

Determining the mode of action of synthetic growth regulators can be difficult. Mode of action can vary with the dosage. At some concentrations certain processes are affected, and at increased

concentrations, additional processes may be involved. For example, some chemicals are herbicides at one dose and growth regulators at another. (For the purposes of this paper, an herbicide is defined as a substance that either completely kills a plant or has formative and/or physical effects that reduces the stature of the plant and its ability to compete. A growth regulator is a substance that regulates some plant activity physiologically without having harmful formative effects.) Nearly all chemicals brought into use for growth regulator activity are identified empirically and prescribed according to the symptoms they produce. Determination of mode of action is seldom a major part of early developmental research.

Parts of the background data utilized in the selection of chemicals for this project came from the unpublished material furnished by agricultural chemical development companies. Plant responses other than those specifically sought are often ignored in testing new chemicals, or used as the basis for considering the chemical unsuitable for use. Herbicidal materials that have inadequate effects are often discarded, though they may have interesting non-herbicidal effects on vegetation. Some of the chemicals examined in this study were identified by the manufacturers' screening processes as having growth regulating properties.

Ammonium ethyl carbamoylphosphonate($C_3H_{11}N_2O_4P$) (Krenite®) was screened as a growth regulator by the DuPont company. Their

data showed that Krenite can be a potent growth inhibitor when applied in the spring. The chemical was reclassified as a herbicide when season-dosage interactions showed the product to be a brushkiller when applied in the fall. It is capable of preventing or inhibiting foliage emergence on deciduous species for periods of up to a month without causing apparent serious injury. No data from conifers were available, and no specific mode of action has been reported. Acute mammalian toxicity of the chemical is very low (59).

3-(p-chlorophenyl)-6-methoxy-s-triazine-2, 4-(1H, 3H)-dione, triethanolamine salt (DPX 3778) is a DuPont experimental chemical. The chemical has prevented pollen release in corn, wheat, and rice, and has inhibited pod development in snap beans. The mode of action has not been reported. DPX 3778 exhibits low acute mammalian toxicity (personal communication and non-published data from DuPont research personnel, Wilmington, Del.).

Asulam, as the sodium salt ($C_8H_{10}N_2O_4S$), is a herbicide registered for use for control of several perennial grasses and weeds, including bracken fern and tansy ragwort. It is readily absorbed by foliage and easily translocated. The site of action appears to be the meristematic regions of the plant, and it apparently interferes with the process of cell division and expansion. In some grasses, asulam is translocated to the roots resulting in death of dormant buds on the rhizomes. Acute mammalian toxicity is low (36).

Maleic hydrazide ($C_4H_4N_2O_2$) (MH) is generally used to control suckers on tobacco, for sprout inhibition in stored potatoes and onions, and turf and roadside maintenance. Utilities use it for prevention of elongation of woody species on rights of way (36). It has been used to increase the length of the dormant period in citrus trees (35). MH is absorbed slowly, but once absorbed, translocates freely to active growing points in the plant. MH is considered a uracil antimetabolite (31, 36). The chemical is used to keep potatoes from sprouting, a process that is encouraged by application of gibberellins (98). Suppression of apical dominance and reduction of internode elongation are often caused by maleic hydrazide (13). MH itself is moderately low in toxicity, but one of its metabolites, hydrazine, is a carcinogen (36).

Schwerdtferger (72) and Schütte (71) treated oaks with MH in order to delay budbreak and thus starve the winter moth larvae. They found that MH was phytotoxic at effective doses. Van Lear, et al. (82), studied the influence of MH on growth and survival of Douglas-fir seedlings. They found a one hour top soak of MH at 1000 to 5000 ppm. could delay budbreak for up to a month in 2-0 seedlings, but root growth was inhibited. The results contained wide variations, with some seedlings in a treatment group showing 100 percent budbreak while others in the same treatment had no open buds.

Shibakusa (74) has found MH at a dose of 100 ppm. effective in delaying budbreak of Abies sachalinensis (Masters) for a few days. At higher concentrations budbreak could be delayed more, but phytotoxic effects were noted. One-year-old Douglas-fir seedlings in England were sprayed with 0.05, 0.1, and 0.2 percent solutions of MH during the period of bud swell in the spring. The purpose was to control seedling size and late season flushing, but no significant response in seedling growth was noted (51).

Ethophon ($C_2H_6ClO_3P$) is used to elicit various responses in plants, including fruit ripening, abscission, flower induction, breaking of apical dominance, and many others. The chemical releases ethylene to plant tissues. Ethylene's mode of action, according to van Overbeek (83) has been traced to changes in permeability of lipoprotein membranes. Other suggestions of mode of action include that of Burg and Burg (10) that ethylene becomes attached to some metalloprotein site in the cell and serves in some regulatory manner. That ethylene serves to regulate plant processes through an alteration of RNA and resultant alteration of RNA-directed protein synthesis and resultant enzyme patterns has been suggested (53, 27).

Evidence indicates that ethylene can cause reduced cell elongation but enhanced isodiametric cell expansion in stems, similar to supraoptimal concentrations of auxins (89). Ethylene may cause

some inhibitory effects on growth, such as the inhibition of elongation that is responsible for the geotropic bending of roots (14), and suppression of bud growth in pea seedlings (10). Some experiments have shown that ethylene can break dormancy of seeds under certain conditions (81, 25).

(2-chloroethyl)trimethylammonium chloride (Cycocel)® is effective in dwarfing and reducing internode length of a broad spectrum of plant species without causing apparent injury or necrosis, except at high doses (2, 13, 60, 80). Cycocel has a definite dosage range beyond which it can cause 'burning' and temporary discoloration of foliage. Frequent foliar applications are required to maintain growth control on many plants (13).

The likely mode of action of the chemical is due to a blocking of gibberellin biosynthesis (32, 60). It appears that Cycocel inhibits specifically the cyclization of geranylgeranyl pyrophosphate to (-)-Kaurene, a transformation in the biosynthetic pathway of gibberellins (89).

Reitveld (67) slowed slightly the rate of budbreak in 1-0 Douglas-fir seedlings treated twice with a spray of 200 ppm Cycocel. Seedlings sprayed at bi-weekly intervals with concentration up to 2500 ppm. developed marked chlorosis and greatly shortened crowns.

1-napthaleneacetic acid ($C_{12}H_{10}O_2$), NAA, is generally used to stimulate root formation, thin apple and pear blossoms, and

control apple and pear preharvest drop. The chemical is mobile and accumulates in meristems, with short persistence in plants (36). Although not a natural plant hormone, it has been shown to produce many of the same growth responses of indoleacetic acid, but somewhat less effectively (9).

Early in the history of plant growth regulator use, NAA and related growth regulators had been shown effective in prolonging from one to several weeks the dormancy of tops of deciduous trees and shrubs (73, 38, 79). Ostrom (61) utilized NAA and other chemicals in various concentrations to try to inhibit shoot growth in red, loblolly, and table mountain pines. The sprays were found to be effective in preventing emergence of new needles, but could not prevent the initial surge of leader growth. Duffield (21) found that aqueous sprays of NAA at 125 to 250 ppm applied in August produced early dormancy in Douglas-fir seedlings. Shibakusa (74) found that NAA at 100 ppm could inhibit budbreak of terminal buds of A. sachalinensis for up to 9 days, and inhibit lateral buds for up to 2 days. The experiment utilized three year old seedlings in a nursery bed. Higher concentrations were damaging to seedlings, though more effective in some cases.

2-(2, 4, 5-trichlorophenoxy)propionic acid ($C_9H_7Cl_3O_3$) (silvex) is a registered phenoxy herbicide commonly used for control of certain woody plants, broadleaf herbaceous weeds, and aquatic weeds.

Sublethal doses of silvex are used to hold certain fruit tree species in a certain state of phenology to delay fruit drop, flowering, and other seasonally dependent functions (36). On certain woody vegetation it can delay budbreak and shorten internodal elongation. This effect is observed on conifers at doses greater than or equal to 2.2 kg/ha. in spring (Newton, M., 1958, unpublished data). The chemical is translocated with food materials in the phloem. Its mode of action is similar to 2, 4-D in disturbing the normal processes of cell differentiation. The particular process affected is dependent on dosage and season (36, 3).

Effects of herbicidal treatments with phenoxys applied in late summer or early fall often do not show on shrubs and weed trees until the following year. Delayed response may be due to reduced physiological activity in the plants during late summer and early fall (29). Sufficient herbicide is absorbed and translocated to damage dormant buds or be stored with starches in mature cells of the shrubs. Crafts (19) stated that mature cells involved mainly in starch storage do not respond to the presence of chlorophenoxy compounds. When weather warms and growth begins the following spring, these food reserves would be mobilized and phenoxy herbicides transported with the carbohydrates to regions of active growth in the shrubs (3).

Silvex justifies special consideration as a candidate growth regulator because it is already registered for use in forestry and it has a long record of safe use as an aquatic herbicide at high rates of application. These factors would make it possible to utilize silvex in normal operations with very little time and effort spent on registration and labeling requirements. Silvex is considered moderate in mammalian toxicity (36, 59).

An ideal approach to the selection of growth regulators would have been to choose chemicals that would disrupt a specific process essential for budbreak. Unfortunately, there is not enough known about the biochemical processes occurring during budbreak to identify specific processes essential for budbreak. Also, the knowledge of the effects of many growth regulators is based almost entirely on empirical evidence. A detailed study of the processes of budbreak and the biochemical activity of growth regulators could have helped identify a chemical with the ability to control budbreak, but such a study was beyond the scope of this program. This study examined the best available growth regulators for their potential to control budbreak. Growth regulators were selected based on available knowledge about the chemicals' ability to control phenology in Douglas-fir or other conifers, or in some cases where data was lacking for effects on conifers, data was extrapolated from deciduous species.

PROCEDURES

Research Approach

The initial approach was to identify the unifying principle in the synchronizing of budbreak and egg hatch and then to determine if tree phenology could be managed in a manner that denies the insects conditions necessary for their survival. The control hypothesis is that phenology can be managed by application of growth regulators. This study, therefore, investigated methods by which delays could be induced in budbreak of Douglas-fir and grand fir, with minimal tree effects other than phenological, and minimum non-target effects. Growth regulators were considered in several ways for their ability to postpone growth initiation. Treatment effects were then compared to postulated requirements for de-synchronization with the DFTM to determine the potential for operational tests. Effects on tree growth were also recorded to determine if the treatments had an effect on growth of the trees.

Selection of the treatments (growth regulators, dosage, and season of application) utilized in this study involved a large step from documented knowledge about the treatments efficacy in regulating dormancy in Douglas-fir. Some preliminary screening of the treatments in a greenhouse or nursery beds possibly could have helped identify promising chemicals and narrow dosage and season

ranges, but there are many variables in an artificial environment that could make interpretation difficult.

Time constraints of the DFTM research and Development Program limited the ability to perform preliminary testing. The fact that budbreak occurs only once a year limits the extent of testing in a short time period. An examination of available literature and advice of scientists in various agricultural chemical development companies were utilized to help choose treatments that promised to be the most effective in delaying budbreak.

Study Areas

Two areas were utilized for this study. One plot was located near Blodgett, Oregon, eighteen miles west of Corvallis in the Oregon coast range. The trees utilized were young open grown Douglas-fir and grand fir in an established old field plantation in the Starker Forests research area. The plot elevation is approximately 500 feet. The area is characterized by mild winter and summer temperatures, but frequent frosts in May and early June.

The second plot location was on Gordon Creek, ten miles northwest of Elgin, Oregon. This is in an area that was heavily defoliated by the DFTM in the 1972-73 outbreak. Trees utilized here were natural grand fir and planted Douglas-fir on Boise Cascade land. The trees were randomly spaced over a moderate

east slope with some brushy areas, but they were generally open grown or dominants. The plot elevation is approximately 4, 000 feet. The area receives large amounts of snowfall, with little precipitation in the summer.

The Blodgett plot is located on the 'west side', an area that does not have DFTM epidemics, as far as is known. The west side plots were chosen for proximity and the knowledge of prior history of the plantation. The proximity of the plot allows for ease of treatment application and more complete observation. It was hoped that the tree responses to growth regulators would be the same in Blodgett and Elgin, allowing ready comparison and extrapolation of data. Time and logistic factors did not allow a full range of treatments to be applied in Elgin. If the Blodgett results are consistent with the Elgin results, in general, there will be some basis for extrapolating to expected Elgin results despite the lack of treatment at that site.

Experimental Design and Procedures

Blodgett Study Area

In early spring, 1975, 180 grand fir and 400 Douglas-fir were tagged and height growth over the last four seasons was measured. Prior to the 1975 budbreak, these trees were five years old and averaged 155 cm. tall for the Douglas-fir and 60 cm. tall for the

grand fir. On May 30, 1975, budbreak was observed and each tree ranked according to its bud development (the ranking method is discussed later).

Of the tagged trees, 144 grand fir and 360 Douglas-fir were randomly selected for treatment with growth regulators. The remaining trees were selected for controls. There were seventy-two different treatment combinations, each with two grand fir and five Douglas-fir. Controls were pooled in groups of two and five, respectively. Treatment consisted of application of eight different growth regulators x three concentrations x three seasons of application. Specific treatments are found in Table 3.

Table 3. Growth regulators and concentrations.

Chemical (Company)	Concentration (ppm)		
Asulam (Rhodia)	1, 200	3, 000	12, 000
Cycocel (Cyanamid)	300	800	2, 000
DPX-3778 (DuPont)	600	1, 500	6, 000
Ethophon (Amchem)	300	800	2, 000
Krenite (DuPont)	3, 000	8, 000	12, 000
Maleic hydrazide (Ansul)	1, 200	3, 000	12, 000
NAA (Amchem)	300	800	2, 000
Silvex (Dow)	600	1, 500	6, 000

Applications were made at a rate of 188 liters per hectare, in water, with 1% X-77 surfactant. All applications were done with a hand-held boom sprayer calibrated to simulate treatments made by aerial methods. (A description of the spray equipment is in the Appendix.) Treatment applications were made in the early morning hours under conditions of still air and high humidity to allow maximum chemical retention and absorption. Treatments were applied only when there was no prospect of any precipitation in the next eight hours.

Seasons of application were early September, November, and Spring. The timing of the treatments was designed to coincide with periods of the tree's phenological cycle associated with control of dormancy. In early September, the trees are in a state of quiescense, not truly dormant but not actively elongating. Growth has slowed down, metabolic activity is reduced in some tissues, abscisic acid and/or other inhibitors may build up, changes occur in levels of other hormones, and changes occur in developmental pathways. Lavender (45) has shown that short photoperiods in the fall lead to an earlier budbreak in Douglas-fir than natural or long photoperiods. The changes going on in the tree at this time are apparently 'triggers' that can be manipulated to affect phenology at a later date. Chemical disruption of normal processes in this transitional period was postulated to alter phenology by upsetting normal quantitative

and qualitative changes of regulatory compounds. Some of the growth-regulating brush killers are most active during this season, with activity being manifested at the time of budbreak the following spring (Krenite, glyphosate).

By early November the plant has entered the stage of deep dormancy. The fact that November lifted trees have a lower survival rate and later budbreak than winter lifted trees indicate that a disruption of normal processes at this time can substantially alter the phenology of the tree (37, 46).

Many changes are taking place in the tree as Spring approaches. Levels of growth promoters and metabolic activity are rising as the buds begin to swell. Growth regulators applied at this time have been shown to affect phenology in Douglas-fir (67, 82), and in other species. A chemical that would disrupt any of the many activities leading up to budbreak presumably would slow phenological development through interaction with internal growth regulators or merely by causing a hiatus in regulator activity, hence postponement of elongation.

The September treatments were applied after height growth had ceased and buds had set. November treatments were made as early in November as possible, weather permitting. The Spring treatments were timed to be made at 50 percent bud swell. The actual treatment dates were: September 2 to 15, 1975; November

12 to 18, 1975, and April 22 to 28, 1976.

Elgin Study Area

The Elgin study area was established in early October, 1975. One hundred eighty grand fir and 400 Douglas-fir were tagged and height growth over the past four seasons was measured. Height of trees of both species averaged approximately 150 cm. Of the tagged trees, 108 grand fir and 270 Douglas-fir were finally treated, with weather and scheduling problems affecting treatment numbers. Treatments and sample sizes at Elgin followed the same combinations as on the Blodgett study area, except that the September treatments could not be scheduled. Freezing temperatures and snow prevented complete November treatments from being made. All three doses of Krenite and DPX - 3778 were applied. The remainder of the treatments had to be dropped when the freezing conditions ruptured the spray boom and prevented further treatments after equipment repair. Because of the availability of the tagged trees, the number of trees per treatment was doubled for the Spring treatment, with ten Douglas-fir and four grand fir per treatment.

Growth regulator application was by the same method outlined for the Blodgett study area. Treatment dates were November 2, 1975, and May 10 and 11, 1976. The phenological development of trees at Elgin is later than in Blodgett during the spring, so the

spring treatments were later in Elgin to coincide with bud swelling. The treatments took fewer days to apply because they were made during the whole day rather than being limited to early morning, because of cost and time factors involved in field work in Eastern Oregon. Despite the application during times other than early morning still air and high humidity, care was taken to insure that uniform application was made.

Weather Data Collection

Two (2) thirty-one day recording thermographs were installed on the Elgin plots in standard weather shelters. Air temperature was recorded continuously from April 11, 1976, to June 16, 1976. One (1) seven day recording thermograph was installed on the Blodgett plots in a standard weather shelter. Air temperature was recorded continuously from April 1, 1976, to May 31, 1976. Only one thermograph was utilized in Blodgett because of uniform topography and exposure, the ability to visit the site more often, and the ability to check the thermograph with others in a similar location.

Observations

Observations were made similarly on both plots. The treated trees and controls were observed for date of budbreak, height growth, and foliar injury. Budbreak observations were made using

a numerical rating system. The ratings were:

0 - a tight bud that showed little or no sign of swelling
1 - a closed bud with visible signs of swelling
2 - a bud with foliage showing through the bud scales and up to one and one half inches of foliage elongation
3 - a bud with foliage elongated one and one half to three inches
4 - a bud with foliage elongated over three inches

Observations were made throughout the period of budbreak, at two to five day intervals, depending on the rate of change in the buds. Numerical ratings were started at first signs of significant swelling into the 1 category, and continued until all buds were at least through the 2 category. The terminal and top whorl of lateral buds were the ones examined on all trees, except in cases of unusual tree damage. If the terminal buds were significantly out of phase with the buds on the rest of the tree, both rates of development were noted. First numerical ratings were assigned in Blodgett on May 5, 1976, and last ones on May 31, 1976. Ratings were made in Elgin from May 28, 1976, to June 16, 1976.

Signs of foliar injury or bud damage were noted during budbreak observations and throughout the growing season. At the end of the growing season, total height growth for the season was measured and recorded.

Analysis

Blodgett Study Area

Budbreak response data was collected on seven dates, May 5, 8, 10, 14, 18, 25, and 31. This provided numerous points in a developmental pattern that related budbreak and elongation to time, but was not suitable for statistical analysis. For analysis purposes, effective budbreak was defined as the date the bud reached the 2.5 rating. This was chosen as the point of foliage development when the larvae could begin effective feeding. The progression of budbreak from stage zero to four was considered to approximate an exponential function over time. An exponential equation ($y = Ae^{bx}$, with x = day of budbreak and y = bud rating) was utilized to fit the seven pairs of data points by a least squares fit method. Solving for x ($x = \frac{-(\ln A - \ln y)}{b}$) the equation used the solution of the coefficients a and b to determine the value of x at bud stages 2.5 from this fitted function.

The bud rating of the trees had been determined on one date during budbreak in 1975, pretreatment. In order to correct the day of budbreak post treatment for inherent early or late flushing tendencies in the population, the average day of budbreak (1976) of control trees was determined for each 1975 rating class. (When the trees were rated in 1975, all were either in the 2, 3, or 4 class.) The correction factor for each class was then subtracted from the treated

trees in the respective class. This gave an adjusted day of budbreak with the inherent early or late tendencies of the tree removed. Actual and adjusted day values for budbreak were then analyzed for effects of treatment by a three factor analysis of variance and covariance. The covariate used in the analysis was the height of the tree in 1975, when treated. This was used because of the assumption that height of the tree affected time of budbreak.

The height growth response to treatment was analyzed by using two methods to examine differences in growth. The absolute elongation in 1976 was one expression of growth examined (hereafter referred to as simple growth difference). The growth in the 1976 season divided by the growth in the 1975 season was used to determine a ratio value as an expression of proportional change.

The grand fir analysis for the Blodgett study area was dropped because of many factors that complicated the analysis. The seed source of the grand fir was off site, and the plantation area contained a natural frost pocket. These factors contributed to a low vigor group of trees and repeated frost damage. Deer use of the area was high, with the grand fir being a favorite target for antler rubbing. The deer caused damage to approximately 20 percent of the grand fir. With only two replications per treatment and the high loss to deer, frost, and low vigor trees, the analysis became meaningless.

Elgin Study Area

Budbreak data from Elgin consisted of numerical observations taken on three dates. These three ratings can be considered to form a sequential 'pattern' of development (0 2 2, 0 1 2, 1 2 3, etc.). There were 15 different patterns recorded, with 8 appearing commonly. Knowledge obtained from examining the progression of bud development in the field and comparing this with the numerical ratings indicated that a subjective analysis assigning a date of budbreak to each pattern would provide an adequate estimate of date of budbreak. Each tree was thus assigned a single date of budbreak based on its pattern of bud development. This date was then analyzed for delay by analysis of variance and covariance for both species. Height in 1975 was utilized as the covariate, as in the Blodgett analysis. Because of sample size requirements of various programs, an analysis of variance was done: (1) for all treatments; and (2) for the Spring treatments only. The analysis of covariance was performed for the Spring treatments only.

Height growth response differences for both species were determined by the same two methods outlined for the Blodgett analysis. The two values were analyzed by analysis of variance for all treatments and for May treatments only.

Weather and Phenology Data

Degree-days were accumulated following the procedures used by Wagg (88) and Wickman (92). The daily mean temperature was determined by summing the daily maximum and minimum and dividing by two. From the daily mean 42°F was then subtracted and each degree remaining was termed one degree-day. The cumulative degree-days were recorded and plotted against cumulative budbreak percent.

RESULTS

The results of this study will be reported individually for each study area and species. Results are expressed in terms of main factors of the experiments. Interrelationships will be reported and discussed after the individual factors are presented.

Blodgett Study Area, Douglas-fir

Budbreak Data

The actual day of budbreak, by treatment, can be found in Table 1 of the Appendix. The adjusted day of budbreak is shown in Table 4. Data utilized in determining the correction to adjust day of budbreak for inherent tree differences can be found in Table 2 of the Appendix.

The three factor analysis of variance comparing chemicals, doses, seasons, and their interactions indicated that date of budbreak was not significantly affected by any treatment. This was consistent regardless of whether the observed date of budbreak was used raw or was adjusted to reflect its inate tendency. (Table 5, three factor analysis, adjusted date, Appendix Table 3, three factor analysis of variance, actual day.) The mean adjusted day of budbreak for each chemical, chemical x dose, chemical x season, and chemical x dose

Table 4. Blodgett study area, Douglas-fir. Days budbreak delay and standard deviation. Listed by individual treatment (chemical x dose x season combinations), chemical x dose, chemical x season, and chemical combinations. All values in terms of days. (-) values are days early.

Chemical Season	Low Dose Delay (days)	Low Dose Std. Dev. (days)	Middle Dose Delay (days)	Middle Dose Std. Dev. (days)	High Dose Delay (days)	High Dose Std. Dev. (days)	Chemical x Season Mean Delay (days)
Asulam							
April	-2.1	6.7	2.0	4.5	1.5	7.6	.5
Sept.	1.4	8.3	-2.5	5.4	1.1	3.3	.0
Nov.	-1.7	3.6	.7	7.4	-.7	8.3	-.6
							Chemical Mean
Chem x Dose Mean	-.8		.5		.6		-.03
Cycocel							
April	-.5	2.1	-4.0	4.9	-3.2	4.5	-2.5
Sept.	-.1	3.3	-2.4	4.9	-4.7	7.8	-2.4
Nov.	-.4	3.4	-.4	7.7	3.3	5.1	.8
							Chemical Mean
Chem x Dose Mean	.3		-2.3		-1.8		-1.4
DPX 3778							
April	-1.9	5.8	1.5	3.9	-.8	3.0	-.4
Sept.	-4.9	7.3	3.1	4.0	1.4	3.4	-.1
Nov.	1.7	5.2	-2.0	5.2	-1.7	7.9	-.7
							Chemical Mean
Chem x Dose Mean	-1.7		.9		-.4	.4	
Ethophon							
April	-2.9	5.8	-5.1	8.0	2.8	4.4	-1.7
Sept.	-6.7	7.4	-2.2	4.7	-3.6	4.1	-4.1
Nov.	-.2	5.3	-2.7	5.4	.4	1.7	-.8
							Chemical Mean
Chem x Dose Mean	-3.3		-3.3		-.1		-2.2
Krenite							
April	-4.7	6.6	.1	2.6	-1.3	8.3	-2.0
Sept.	3.5	8.0	.9	7.5	1.6	8.7	2.0
Nov.	-3.7	5.1	-4.6	2.9	-.8	3.8	-3.1
							Chemical Mean
Chem x Dose Mean	-1.6		-1.2		-.2		-1.0

Table 4. (Continued)

Chemical Season	Low Dose Delay (days)	Low Dose Std. Dev. (days)	Middle Dose Delay (days)	Middle Dose Std. Dev. (days)	High Dose Delay (days)	High Dose Std. Dev. (days)	Chemical x Season Mean Delay (days)
Maleic Hydrazide							
April	1.0	7.5	-3.5	4.4	-6.5*	6.0	-3.0
Sept.	3.5	4.9	2.0	5.1	3.1	11.0	2.9
Nov.	-2.2	7.1	-.5	11.4	-1.5	3.6	-1.4
							Chemical Mean
Chem x Dose Mean	.8		-.7		-1.6		-.5
NAA							
April	.2	2.2	1.3	2.1	.8	1.8	.7
Sept.	.2	8.5	-3.3	4.3	-1.2	4.7	-1.4
Nov.	-.8	4.3	-2.2	4.4	-1.1	5.6	-1.4
							Chemical Mean
Chem x Dose Mean	-.1		-1.4		-.5		-.684
Silvex							
April	-2.4	6.0	.1	1.7	-.3	5.6	-.9
Sept.	-.9	6.4	-3.3	7.3	1.2*	5.3	-1.0
Nov.	-1.7	6.7	6.4	13.2	.8	5.8	3.9
							Chemical Mean
Chem x Dose Mean	-1.7		3.2		.6		.7
Controls	-1.5	1.7					
(by groups	-1.6	4.7					
of five)	-.9	1.6					
	1.1	1.4					
	2.4	3.8					
	.1	5.7					

* All values are the mean of five observations, except treatments marked with an (*) contained one missing observation.

Table 5. Three factor analysis of variance for adjusted day of budbreak, Blodgett Douglas-fir.

Source of variation	df	SS	MS	F
Chemical	7	243.9731	34.8533	.709
Dose	2	11.9602	5.9801	.122
Season	2	41.2696	20.6348	.420
Chemical x Dose	14	526.2667	37.5905	.765
Chemical x Season	14	937.7916	66.9851	1.363
Dose x Season	4	33.8527	8.4632	.172
Chemical x Dose x Season	28	1149.7862	41.0638	.836
Error	288	14149.9031	49.1316	
Total	359	17094.8032		

x season interaction can be found in Table 4.

The three factor analysis of covariance of adjusted day of budbreak with height in 1975 as the covariate also indicated a lack of significance for either the treatments or the influence of height on date of budbreak (Appendix, Table 4). The adjusted MS and SS were virtually unchanged by the use of covariance, and the adjustment of budbreak date by the covariate was small. A regression of adjusted day of budbreak in 1976 on height in 1975 for control trees produced a significant correlation coefficient of -.3777, and a R^2 of .14 (Table 6). Although the height of the trees in 1975 (height when treated) had some effect on budbreak, it was not correlated with budbreak to a very large extent.

Table 6. Simple linear regression of adjusted day of budbreak on height in 1975. Blodgett Douglas-fir. Controls only.

$Y = 3.98057 - 0.030231(X)$

$R = -0.3777$ Std error A = 2.17474

Std error B = .01426

$R^2 = .14$

$F = 4.5$ (significant at the .01 level)

None of the analyses identified effects of treatment as responsible for a significant amount of variance regarding day of budbreak. In anticipation that an analysis with a large number of treatment combinations can obscure a biologically significant result, the

residual variation was examined for specific patterns not considered in the analyses. The range of budbreak times was from 6.7 days early to 12.6 days late, although 12.6 is an extreme value. The next two longest delays were two of 3.5 days. The 12.6 days delay was from the silvex medium dose level (1,500 ppm) in November. The low silvex November dose had a delay of -1.7 days, and the high dose .8 days. Upon close examination of the bud rating results of the trees treated with the middle dose, a small computational 'fluke' was found. One tree in this group had six consecutive bud ratings of 1, which caused the exponential equation that was fitted to the observations and used to determine day of budbreak (time to stage 2.5) to flatten out and over-estimate considerably the time of budbreak for this tree. With the budbreak time of the tree corrected, the delay for the treatment group would be approximately six days. This delay is still quite out of the range of the other values for silvex but field observations would indicate it is biologically correct. (Complete data for this treatment is in Appendix Table A20).

The silvex middle dose in November was the longest delaying treatment, but the silvex November results were not consistent over the doses applied. There were only two places where consistent delays over season of application were found. The MH September treatments had delays for the low to high dose of 3.5, 2.0, and 3.1, respectively. The Krenite September doses had delays of 3.5, .9,

and 1.6. These are the only treatments that are at all consistent over a season or dose range.

There was also a lot of variation in delay times within a single treatment. One tree in the group could be many days early, while another would be many days late. Table A2, Appendix, shows that there was up to eight days variation in 1976 budbreak date of the control trees ranked in class three and four in 1975. Table 4 lists standard deviations for all treatments, which were generally high.

One of the pooled control groups had a delay of 2.4 days. With the small delays recorded and the relatively late budbreak date of this control group, it appears that the silvex 1,500 ppm. dose in November is the only treatment with any apparent delay worthy of further analysis.

Height Growth Data

The effect of growth regulator applications on height growth response was analyzed by three factor analysis of variance by the simple growth and growth ratio methods previously discussed. The simple growth analysis method indicated a lack of significance (Appendix, Table A5). The three factor analysis of variance of the growth ratio method was significant at the .05 level for chemical (.10 level for dose, .20 level for season, .25 level for chemical x season) (Table 7).

Table 7. Three factor analysis of variance of ratio growth difference, Blodgett Douglas-fir.

Source of Variation	df	SS	MS	F
Chemical	7	5.1406	.7344	2.631*
Dose	2	1.4898	.7449	2.668
Season	2	1.0399	.5199	1.862
Chemical x Dose	14	3.8083	.2720	.974
Chemical x Season	14	4.8034	.3431	1.229
Dose x Season	4	.6967	.1742	.624
Chemical x Dose x Season	28	5.6133	.2005	.718
Error	288	80.4007	.2792	
Total	359	102.9927		

Means

Chemical	Asulam 1.3235	Cycocel 1.2882	DPX 3778 1.3441	Ethophon 1.4630
	Krenite 1.1488	Maleic Hydrazide 1.1724	NAA 1.1925	Silvex 1.4854
Dose	Low 1.3401	Middle 1.3550	High 1.2117	
Season	April 1.3782	September 1.2615	November 1.2670	

*Significant at the .05 level

The ratio method would have to be considered a better representation of growth differences than the simple growth method because it is not influenced by the range of original height differences. The use of a ratio of two years growth eliminates inherent growth differences attributable to absolute size. The taller trees of the size range utilized in the experiment have a naturally higher growth rate than the smaller trees.

The growth ratio means, by chemical group, ranged from 1.1488 for Krenite to 1.4854 for silvex (Table 7), with the control trees averaging 1.3549. Means for ethophon and silvex were higher than the control mean, all other chemical means were lower than the control mean. The possibility that ethophon and silvex could have increased growth is consistent with the properties of the chemicals. The means for Krenite, MH, and NAA are all low and closely grouped, and the ethophon and silvex means are high and closely grouped, with the other three means bridging the difference.

Although the height analysis was significant at the chemical level only, there appear to be some real growth differences by season of treatment for some chemicals. Growth ratios ranged from a low of .8073 for the Krenite September treatments to a high of 1.5329 for the Ethophon September treatments. (The high dose of Krenite in September had a growth ratio of .466.) Krenite April treatments had a growth ratio of 1.5294, and November treatments a ratio of 1.1907.

The Krenite April treatment had the highest growth ratio, while the September treatments were the lowest. Other chemicals do not exhibit this striking difference, but some differences appear to exist.

Some of the effects of the lower growth means in various treatments were due to the growth regulators actually causing tree damage. September Krenite and MH doses caused extensive tree damage, with Krenite actually killing portions of the tree crown. Both chemicals at times prevented terminal growth completely by killing the terminal buds. These effects were markedly seasonal, with either considerably less or no damage occurring during other seasons of application. (Damage that resulted from other treatments will be discussed later.)

Rankings

With the large number of treatments and the large variation within treatments, there could be some biologically important patterns that were not statistically significant. In order to examine the data closer, the 72 treatment combinations and six control groups were ranked by day of budbreak, with one the earliest and 78 the latest (Appendix Table A7a) (See key to treatment codes, Appendix Table A6). The treatments and controls were also ranked by the ratio growth value, with one the least growth and 78 the most growth (Appendix Table A8a). The ranks were then summed and

ranked by chemical, chemical x season, and chemical x dose combinations (Appendix Tables A7b and A8b). The summed rankings will differ slightly from a listing of the treatments by the numerical mean of budbreak time or growth ratio, but the rankings indicate an ordered relationship of one treatment combination to the others.

Some of the chemicals had a high budbreak delay rank in one season and low ranks in the other seasons. These chemicals included silvex, NAA, MH, Krenite, and Cycocel. Neither asulam nor DPX 3778 had one of the best delay times, but all three chemical x season combinations were in the upper one half of the ranked delay times. All three ethophon chemical x season combinations appear in the first thirteen budbreak ranks (out of 24 combinations). The activity of asulam, DPX 3778, and Ethophon appears most uniform across seasons. MH and Krenite had the best two delays for chemical x season combinations (both in September), while their other two seasons of application ranked low. An examination of the occurrence of the treatments in the upper and lower half of the rankings of chemical x season shows November treatments appeared five times in the upper one half and three in the lower one half, September is split four and four, and April is three and five. These results indicate the fall application may be slightly more effective in delaying budbreak. A bunching of certain treatments can be also seen in the analysis of individual treatment ranks, and these will be considered

later. No trends were evident across chemical x dose ranks.

Growth response rankings by chemical combinations indicate that NAA, MH, and Krenite had the largest depressing effects on growth. Only ethophon and silvex had rankings higher than the controls, agreeing with the differences noted in the mean values. The first ten ranks of the individual treatments consist of two Krenite treatments, two NAA, two Cycocel, three MH, and one DPX 3778 treatment, indicating some of the chemicals with the most effect on growth. When the chemical x season ranking is divided into upper and lower halves, three November, three September, and six April treatments appear in the lower half (most growth), and five November, five September, and two April treatments appear in the upper half (least growth). The chemical x season interaction in the three factor analysis of variance of the ratio growth method was significant at the .25 level, not statistically significant, but suggestive of a chemical x season trend. The November and September treatments appeared to have more effect than April treatments, but the degree of effect varied among chemicals.

Individual Treatment Results

The individual treatments with the greatest effects on budbreak or growth are discussed below. In discussing these trends it is important to remember that the differences in time of mean budbreak

were small, variances were large, and variations could have been introduced by factors other than treatment. Also, the analysis of growth results was only significant at the chemical level, so other factors may be involved here.

Asulam April treatments had budbreak ranks, by dose, of 23, 69, and 65, and an absolute delay of -2.1, 2.0, and 1.5 days. Growth response ranks were 71, 43, and 22. (High budbreak ranks indicate greater delay, high growth ranks indicate greater growth.) The stronger two doses caused chlorotic new foliage on one tree each, and the foliage died before the end of the summer (chlorotic new foliage is a common indication of injury caused by application of asulam to dormant conifers.) Only some of the foliage was affected, that in upper parts of the tree and on the side of the tree that was approached with the sprayer (the parts that would have received the highest dose). On another tree the terminal bud did not open. The September doses of asulam had delay ranks of 63, 17, and 60, and delays of 1.4, -2.5, and 1.1 days. Growth ranks were 17, 61, and 39. Three trees that had received the high dose had chlorotic new foliage in parts of their crowns. Most of the foliage recovered, as opposed to the April treatments. The chlorotic new foliage indicated that the chemical was stored in the tree overwinter and acted as growth was initiated in the spring. On one of the trees the terminal expanded about seven cm. and then died, while the lateral

buds were very late in emerging and then exhibited chlorotic new foliage. While asulam showed signs of potency at higher doses on individual buds, results were inconsistent and it would not provide sufficient whole tree bud control. The ability to cause foliage damage is also a liability.

Cycocel provided no bud control except the strong dose in November. The November rankings of bud delay were 44, 45, and 75, and delays were -.4, -.4, and 3.3 days. Growth ranks were 10, 23, and 32. There was no foliage damage attributed to Cycocel. The growth ranks were low, but all trees appeared to grow normally. Cycocel has been shown to cause bud delay, chlorotic foliage and stunted growth on young Douglas-fir seedlings (67). It is possible that a higher dose of Cycocel could have been utilized.

DPX 3778 September doses had budbreak ranks of 4, 74, and 64, and delays of -4.9, 3.1, and 1.4. Growth ranks were 35, 13, 16. The strong dose killed the terminal bud on one tree, but a lateral bud took over and grew normally. Little is known about this chemical, but stronger doses of it could possibly be tested for bud delay activity.

Ethophon in April had budbreak ranks of 3, 15, and 72, and delays of -5.1, -2.9, and 2.8. Growth ranks were 41, 60, and 50. It would be expected that the low dose could encourage budbreak while the higher dose could cause delay, and that is what happened

in this case. Of all the chemicals, ethophon had the second highest growth ranks (behind silvex), and the overall lowest delay ranks (actually encouraged budbreak).

Krenite September doses had delay ranks of 76, 58, and 67, and delays of 3.5, .9, and 1.6. Growth ranks were 3, 21, and 1. The delay action was fair, but extensive tree damage resulted. On nine of the fifteen trees treated in September, defoliation occurred, the terminal was killed, or other parts of the tree were killed, including the cambium. The damage occurred in all doses. Although the damage was severe, it was usually in the upper parts of the tree, with budbreak and growth occurring normally in the lower parts of the tree, suggesting low mobility in conifers. Some of the cause of the low growth rankings was not just a reduction in growth, but a complete loss of the upper buds on the tree. Some of the variation in response between trees (some grew normally with no sign of damage) might be attributed to uneven dosage applications, but the damage occurred over all dosage ranges. The variation must come from inherent tree response differences at dosages near the damage threshold. No delays and little tree damage was recorded for Krenite in other seasons indicating the markedly seasonal effects of this chemical.

Maleic hydrazide September treatments had delay ranks of 77, 70, and 73, and delays of 3.5, 2.0, and 3.1. Growth ranks

were 37, 55, and 2. This was the best chemical x season combination from a delay standpoint, and the tenth worst from a growth standpoint. The high dose caused foliage damage to some trees. On some trees the new foliage was short and curly, but eventually grew satisfactorily. On one tree the terminal grew only 18 cm., the needles were very short and fine, and the lateral whorl of terminal buds never opened. On two trees the terminal failed to open and a lateral took over. Like Krenite, the chemical did not damage the lower branch buds at all, indicating that the chemical is concentrated in the terminal areas or does not translocate at all. The chemical does not provide effective whole tree bud control, and this coupled with the damage and growth reduction effects would rule MH out of the picture for further investigation in a DFTM control program. The November MH applications had a poor delay, but had a high growth reduction effect.

NAA April treatments had budbreak ranks of 52, 62, and 56, with delays of .2, 1.3, and .7 days. The treatments had growth response rankings of 5, 33, 18. One terminal was damaged by the high dose. The growth effect was fairly large, and NAA when ranked over all seasons and doses had a noticeable growth reduction effect.

Silvex November treatments had delay ranks of 28, 78, and 57, with delays of -1.7, 12.6 (~6 corrected), and .8 days. The growth response ranks were 70, 68, and 25. The long delay for the middle

dose would not be expected intuitively. Silvex should disrupt normal processes of the plant more at higher doses. There is no good explanation of why the higher dose had a smaller delay than the middle dose. All trees appeared to grow normally after treatment. A difference noted between silvex and the other chemicals was that it gave the best whole tree bud control. When the terminal bud was delayed, the rest of the tree was delayed similarly. Since no damage from silvex treatment was observed, it is possible a higher dose could be tested for budbreak control.

Elgin Study Area

The budbreak data for the Elgin study area was analyzed without any knowledge of inherent early or late budbreak tendencies of the trees. The number of control trees utilized was larger than in Blodgett, providing a better base for comparison to treatment means. Since the number of treatments applied was incomplete, analysis was by one factor analysis of variance (ANOVA) only, and by two methods: (1) with all treatments and controls, and (2) with the May treatments and controls only.

Elgin, Douglas-fir

Day of budbreak for all treatments and controls is found in Table 8. The budbreak date ANOVAs for all treatments, and for May only,

Table 8. Elgin Douglas-fir. Budbreak day, delay, and standard deviation by treatment. Budbreak day and delay for chemical x season combination. All values in days. (-) values are days early.

Chemical Season	Low Dose Budbreak Day / Delay (days)	Low Dose Std. Dev. (days)	Middle Dose Budbreak Day / Delay (days)	Middle Dose Std. Dev. (days)	High Dose Budbreak Day / Delay (days)	High Dose Std. Dev. (days)	Chemical x Season Mean Budbreak Day / Delay (days)
Asulam							
May	9.90	4.8	12.8	3.5	11.9	5.3	11.5
	-1.6		1.3		.4		.03
Cycocel							
May	10.6	3.7	12.4	4.2	10.0	4.2	11.0
	-.9		.9		-1.5		-.5
DPX 3778							
May	13.1	4.3	13.2	5.1	11.9	5.6	12.7
	1.6		1.7		.4		1.2
Nov.	11.2	1.8	15.6	3.5	14.0	2.7	13.6
	-.3		4.1		2.5		2.1
Ethophon							
May	13.1	3.9	12.1	4.6	10.8	3.6	12.0
	1.6		.6		-.7		.5
Krenite							
May	10.2	6.2	13.7	4.6	13.5	4.4	12.5
	-1.3		2.2		2.0		1.0
Nov.	9.6	1.7	12.2	3.2	12.8	4.1	11.5
	-1.9		.7		1.3		.06
Maleic Hydrazide							
May	13.6	4.3	12.4	6.7	12.0	7.8	12.7
	2.1		.9		.5		1.2

Table 8. (Continued)

Chemical Season	Low Dose Budbreak Day Delay (days)	Low Dose Std. Dev. (days)	Middle Dose Budbreak Day Delay (days)	Middle Dose Std. Dev. (days)	High Dose Budbreak Day Delay (days)	High Dose Std. Dev. (days)	Chemical x Season Mean Budbreak Day Delay (days)
NAA							
May	10.3	2.1	9.1	5.8	14.2	3.8	11.2
	-1.2		-2.4		2.7		-.3
Silvex							
May	10.7	7.8	12.6	5.5	13.0	3.2	12.1
	-.8		1.1		1.5		.6
Controls	8.1	6.0					
	12.9	3.7					
	10.4	4.9	Control Mean = 11.5				
	13.6	6.3					
	12.3	5.8					
	12.5	3.5					
	10.5	6.2					

indicated a lack of significance of treatment (Appendix, Table A9a, b). The analysis of covariance (for May treatments only) with height in 1975 as a covariate was non-significant (Appendix, Table A10). The analysis reduced error SS and MS slightly and increased treatment SS and MS slightly, but still without significance. A simple linear regression of height and day of budbreak of control trees had a significant correlation coefficient of -.4379 and a R^2 of .19 (Table 9).

Table 9. Simple linear regression of day of budbreak on height in 1975. Elgin Douglas-fir, controls only.

$Y = 21.0411 - 0.0851(X)$	
$R = -0.4379$	Std error A = 2.5116
	Std error B = .0218
$R^2 = .19$	
$F = 15.188$ (significant at the .01 level)	

The height analyses for the simple growth difference and ratio, methods were all non-significant (Appendix, Table A11, a, b, c, d).

The treatment and control means for budbreak were ranked by length of delay, with one (1) the shortest delay and 37 the longest delay (Appendix, Table A12). Control groups ranked 33, 26, 22, 19, 9, 8, and 1. The high ranks of the first control groups indicate that there was very little, if any, budbreak delay provided by the treatments. DPX 3778 May and November treatments, Krenite May,

and MH May treatments provided the best delays for the chemical x season groups. The higher two doses of DPX 3778 in November and Krenite in May provided the most consistent indication of delay (Table 7). Only four individual treatments had delays longer than control group 4001 (2.1 days). The rankings indicate there could be some slight differences between chemicals, but that there is no significant difference between any chemical treatment and the control means. No treatment showed a possibility of providing a delay that would have any effect on a DFTM population.

The growth ratio means were also ranked, with one (1) the least growth and 37 the most growth (Appendix, Table A13). Asulam, Cycocel, and the Krenite November and May treatments had the lowest growth ranks for the chemical x season groups. Control groups ranked 4, 16, 20, 21, 22, 27, and 37, being well spread throughout the rankings. Krenite, Asulam, and Cycocel also had the lowest individual treatment growth rates. Individual treatment ratios ranged from 1.119 to 2.104 and the simple growth difference from 36.2 to 47.1 cm. There were not any serious growth reductions. All individual treatments have a 1976 to 1975 growth ratio of greater than one.

The fall treatments caused the most tree damage and greatest growth reductions in Blodgett, with little damage and growth reduction resulting from the May treatments. There was little tree

damage recorded in Elgin, and the limited number of fall treatments were responsible for much of the damage that did occur. The Krenite November treatments prevented some buds from opening through the entire growing season. The asulam May treatment caused some chlorotic new foliage, some of which recovered. Otherwise, there appeared to be no tree damage on Douglas-fir from the chemical applications.

Elgin, Grand fir

Day of budbreak and standard deviations for all treatments and controls is found in Table 10. The analysis of variance for all treatments and for the May treatments only indicated a lack of significant difference in day of budbreak (Appendix, Table A14a, b). The analysis of covariance (for May treatments only) also produced non-significant results (Appendix, Table A15). Error SS and MS were reduced slightly and treatment SS and MS increased slightly by the analysis, but results were non-significant. A regression of height in 1975 on day of budbreak of control trees produced a non-significant correlation coefficient of .0682 and an R^2 of .004 (Appendix, Table A16).

In examining the data to determine if there was a pattern of action in the chemical treatments that did not show up in the budbreak analysis, two consistent areas were found. Both seasons of Krenite application had consistent delays of 2.7 to 4.7 days, and were the

Table 10. Elgin grand fir. Budbreak day, delay, and standard deviation by treatment. Budbreak day and delay for chemical x season combination. All values in days. (-) values are days early.

Chemical Season	Low Dose Budbreak Day / Delay (days)	Low Dose Std. Dev. (days)	Middle Dose Budbreak Day / Delay (days)	Middle Dose Std. Dev. (days)	High Dose Budbreak Day / Delay (days)	High Dose Std. Dev. (days)	Chemical x Season Mean Budbreak Day / Delay (days)
Asulam							
May	19.5	5.6	14.8	4.5	14.8	4.5	16.3
	5.3		.5		.5		2.1
Cycocel							
May	16.3	3.0	15.3	4.9	14.5	2.9	15.3
	2.0		1.0		.3		1.1
DPX 3778							
May	10.8	7.8	12.8	3.0	18.0	7.9	13.8
	-4.3		-1.5		3.7		-.4
Nov.	9.0	11.3	12.5	6.4	18.0	1.4	13.2
	-5.3		-1.8		3.8		-1.1
Ethophon							
May	15.0	3.6	13.5	5.3	13.5	4.4	14.0
	.8		-.8		-.8		-.3
Krenite							
May	17.0	0.0	17.0	0.0	18.5	2.1	18.2
	2.7		2.7		3.0		2.8
Nov.	17.0	0.0	19.0	0.0	18.5	2.1	18.2
	2.7		4.7		4.2		3.9

Table 10. (Continued)

Chemical Season	Low Dose Budbreak Day Delay (days)	Std. Dev. (days)	Middle Dose Budbreak Day Delay (days)	Std. Dev. (days)	High Dose Budbreak Day Delay (days)	Std. Dev. (days)	Chemical x Season Mean Budbreak Day Delay (days)
Maleic Hydrazide							
May	11.3	7.6	15.0	6.1	14.7	4.0	13.6
	-3.0		.7		.4		-.6
NAA							
May	13.0	4.6	14.8	4.5	14.8	4.5	14.2
	-1.3		.5		.5		-.6
Silvex	11.8	7.5	13.0	4.7	17.1	2.1	14.0
May	-2.5		-1.3		2.9		-.3
Controls	13.5	5.0	16.5	5.3			
	14.8	3.3	11.3	3.1			
	14.3	4.5	17.5	5.3			
	16.5	2.3	11.7	4.5			
	13.5	2.5	15.7	4.3			
	13.0	7.5	10.0	4.3			
	16.5	1.0					
	14.8	4.3	Control Mean = 14.3				

two best treatments over the chemical x season category (Table 10). The treatments caused some slight foliar injury, but only of limited extent. The Krenite applications to the Elgin Douglas-fir did not produce the same consistent pattern, with the treatments ranking third and seventh out of ten, with less consistent results over the doses. The asulam May treatments were the third best treatment on the grand fir, with delays of 5.3, .5, and .5 days, results that do not fit the expected pattern of action. The asulam treatment on the Douglas-fir ranked eighth. The chemical x season treatment with the least bud delay on the grand fir was the DPX 3778 November treatment, which had the best delay time on the Douglas-fir.

The treatment and control means for day of budbreak were ranked by length of delay, with one the least delay and 44 the most delay. The ranks were then summed and ranked by chemical x season combinations (Appendix A17). The two Krenite, asulam, and Cycocel treatments were the only ones with ranks higher than the control group average. The rankings for the other combinations were closely bunched below the control mean. The rankings confirmed the consistency of the performance of the Krenite treatments, although statistical analysis showed these results were not significant. The inconsistencies found in most of the treatments and the lack of comparison in delay effects with the Douglas-fir results seem to indicate that inherent tree differences and/or random effects are the

cause of much of the variation found in the results. With the small delay times found and the inherent variation in the population that could not be removed because of no knowledge of the normal bud-break characteristics of the individual trees, analysis was not very fruitful.

The height growth analysis with all treatments was significant at the .05 level for the simple growth difference (Table 11), while the ratio (.22 level) method was non-significant (Appendix, Table A18). The analysis of variance of the May treatments simple growth difference was significant at the .05 level (Table 12), and the ratio method was significant at the .01 level (Table 13). Growth ratios ranged from .304 to 2.524, with a population mean of 1.519. Only three ratios were less than one, with most of the rest spread through the range of 1.2 to 1.9.

In order to facilitate the height analysis, the treatments were ranked and summed by the ratio growth value, with one the least growth, and 44 the most growth (Appendix Table A19). Cycocel and the two Krenite treatments had the greatest growth reduction effects, and ranked considerably lower than the rest of the treatments. The control group ranked high, indicating that the majority of the chemicals had some effect on reducing height growth. Silvex was associated with a slight growth increase.

Table 11. Analysis of variance, simple growth difference, Elgin grand fir, all treatments.

Source	df	SS	MS	F
Treatment	43	8100.8079	188.3909	1.5116*
Error	122	15205.2222	124.6330	
Total	165	23306.0301		

*Significant at the .05 level

Table 12. Analysis of variance, simple growth difference, Elgin grand fir, May treatments.

Source	df	SS	MS	F
Treatment	24	5609.0042	233.7085	2.0357*
Error	75	8610.4167	114.8055	
Total	99	14219.4209		

*Significant at the .05 level

Table 13. Analysis of variance, ratio growth difference, Elgin grand fir, May treatments.

Source	df	SS	MS	F
Treatment	24	15.3839	.6410	2.1319**
Error	75	22.5501	.3007	
Total	99	37.9340		

**Significant at the .01 level

Foliage damage on the grand fir was very limited. Maleic hydrazide at 12,000 ppm. in May resulted in no terminal elongation on one tree, with some laterals also being inhibited. Both seasons of Krenite application resulted in some curly new foliage that later grew normally. Cycocel treatment at 2,000 ppm. in May resulted in chlorotic new foliage in one tree.

Results of Phenological Studies

Graphs of accumulated degree-days vs. cumulative budbreak percent for each area and species are shown in Figure 3a and c. Graphs of calendar days vs. cumulative budbreak percent for each area and species are shown in Figure 3b and d. The degree-days graph for the Blodgett plot (3a) corresponds to the general level of heat units necessary for budbreak found by Wickman in California and Oregon. Budbreak occurred over relatively the same span of accumulated degree-days, but the degree-days started accumulating at an earlier date than in either of Wickman's studies. This would be expected since the Blodgett area has a warmer climate than either of the areas studied by Wickman. The first 50 percent of budbreak occurred over a much shorter time interval than the last 50 percent, and by Wickman's definition (93), this is because the rate of degree-day accumulation was higher at the onset of budbreak.

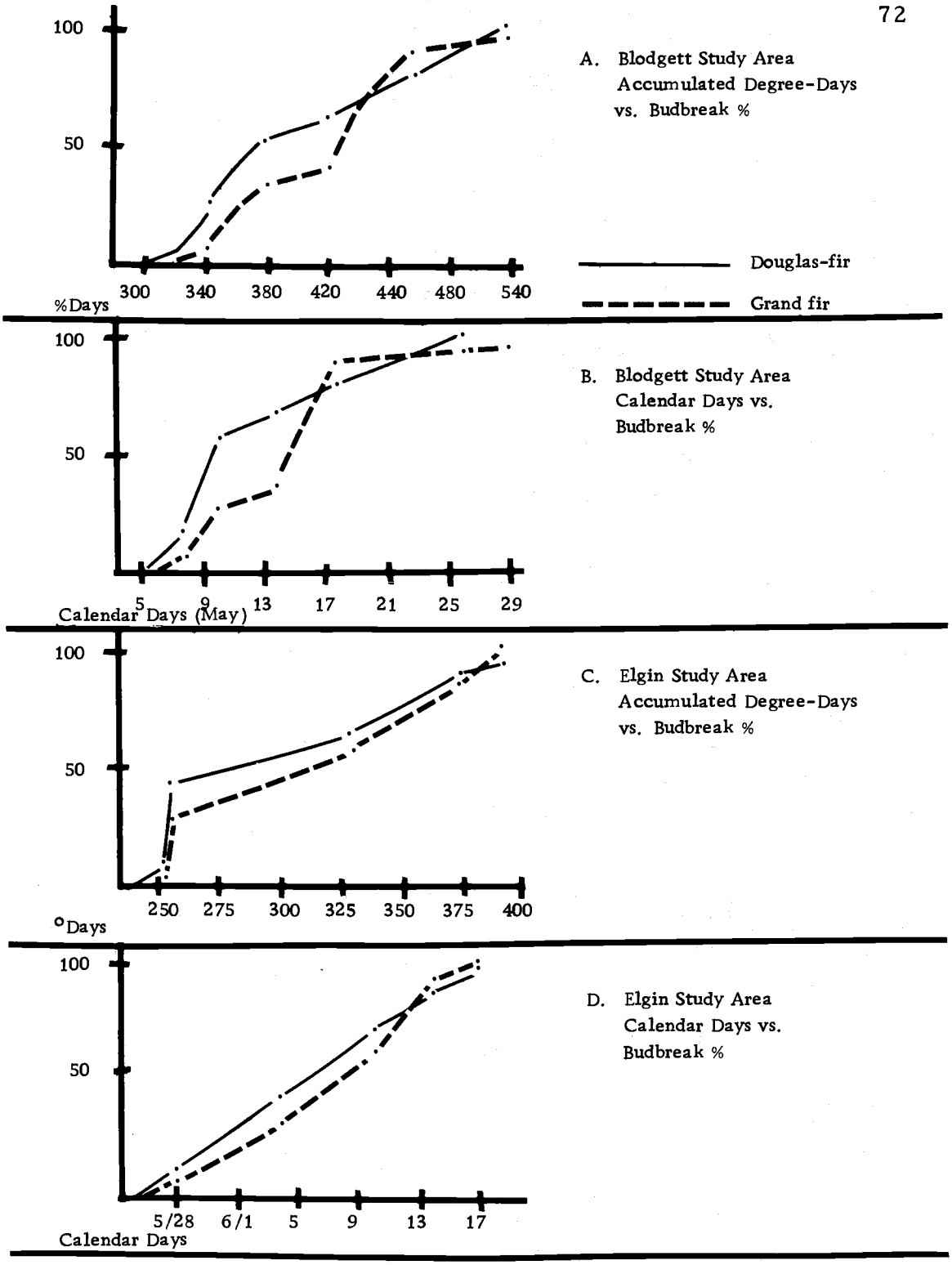

Figure 3. Phenological relationship of budbreak %, degree-days, and calendar days for Douglas-fir and grand fir

The phenology data obtained from the Elgin plots closely parallel results obtained by Wickman in his Eastern Oregon studies (1973) at the same elevation. (Some of his plots were within 25 miles.) The spring he collected his data was warmer than spring 1976, and degree-days accumulated about one week earlier in 1973. Budbreak 1976 occurred over the same span of degree-days as he recorded in 1973.

Results from Elgin point out a fault in the budbreak - temperature accumulation relationship. Between 5/28 and 6/4, only 3.5 degree-days accumulated, but there was approximately a thirty percent increase in cumulative budbreak percent in both species. The trees were responding to the warmer weather of the previous days, even though the period of 5/28 to 6/4 was cold and wet. The relationship between degree-days and budbreak does not function as a direct day for day relation. The accumulated heat units give an indication of when a phenological event can be expected to occur when it has been correlated with heat unit accumulation. The graphs of budbreak vs. calendar days for the Elgin plots gives a smoother graph more typical of a biological event over time. This suggests that a factor independent of degree-days but loosely correlated with it, such as daylength, may override temperature after some phenological point is reached.

DISCUSSION

Several obvious topics for discussion emerged from the foregoing: (1) an interpretation of the differences in results between the two species and the two study areas, (2) a discussion of some procedural and technical factors with possible bearing on the results, (3) a discussion of problems involved in interpretation of previous work with growth regulators and dormancy regulation, and (4) a discussion of budbreak delay results and postulated requirements for control.

Interpretation of Differences in Species and Study Areas

The budbreak delay and height growth response results differed considerably between the two species in Elgin and between the Elgin and Blodgett study areas. In order to facilitate comparison, the ranked results of the chemical by season combinations for Blodgett and Elgin study area were combined in Table 12. (The only treatments included for the Blodgett Douglas-fir are those that correspond to those in Elgin.) Differences in the rankings are as common as consistencies.

Since a comparison of results shows such large variations in supposedly identical treatments, there must be some explanation for the differences. A few differences in the two areas themselves

Table 14. Comparisons of budbreak and growth ratio response results from Blodgett and Elgin study areas.

Blodgett Douglas-fir	Elgin Douglas-fir	Elgin grand fir
A. Budbreak ranks by chemical x season combination, 1 least delay, 10 most delay.		
1. Krenite November	1. Cycocel May	1. DPX November
2. Cycocel April	2. Asulam May	2. Maleic H. May
3. Maleic H. April	3. NAA May	3. DPX May
4. Krenite April	4. Krenite November	4. NAA May
5. Ethophon April	5. Ethophon May	5. Silvex May
6. Silvex April	6. Silvex May	6. Ethophon May
7. DPX November	7. Maleic H. May	7. Cycocel May
8. DPX April	8. Krenite May	8. Asulam May
9. Asulam April	9. DPX May	9. Krenite May
10. NAA April	10. DPX November	10. Krenite November
B. Growth ratio ranks by chemical x season combination, 1 least growth, 10 most growth.		
1. NAA April	1. Asulam May	1. Cycocel May
2. Krenite November	2. Cycocel May	2. Krenite May
3. Maleic H. April	3. Krenite November	3. Krenite November
4. DPX November	4. Krenite May	4. Maleic H. May
5. DPX April	5. Silvex May	5. NAA May
6. Asulam April	6. Ethophon May	6. Asulam May
7. Ethophon April	7. Maleic H. May	7. DPX May
8. Cycocel April	8. DPX May	8. Ethophon May
9. Silvex April	9. DPX November	9. DPX November
10. Krenite April	10. NAA May	10. Silvex May

and the treatment of the areas can be pointed out. The Elgin spring growth regulator applications were made earlier in the bud swell cycle than those in Blodgett. Treatments were applied in Elgin at the period of apparent bud swell in approximately 25 percent of the population, and the Blodgett applications were made at 40 to 50 percent bud swell. (It had been hoped to make both treatments at 50 percent bud swell, but technical problems prevented this.) The trees of the two areas are also of greatly different ecotypes, and there is a large variation in the site conditions in the two study areas. Reitveld (67) has shown that Douglas-fir of different ecotypes differ in their sensitivity to IAA, Cycocel, and B-995. He hypothesized that differences in surface-layer anatomy (cuticle and outer epidermal wall thickness) could contribute to the varying responses found by causing differential uptake of the applied chemicals. After close examination he concluded the differences in responses among the ecotypes could not be attributed to anatomical features affecting penetration, but to several physical and physiological factors in unknown combination.

Since the growth regulators were applied in Elgin during warm and dry weather, while application was made in Blodgett during cool to warm and usually humid conditions, it is possible that environmental factors could have played a role in varying the amount of chemical finally entering the trees in the different areas.

Because the chemicals did not produce comparable budbreak results in the areas tested, and the analyses of their effects on budbreak were not statistically significant, it is possible that the treatments had little or no effect on budbreak and the data is due completely to random or inherent differences. The results would seem to indicate that the chemicals did cause some tree reaction, although it was small. The growth response analyses demonstrated that there was a definite treatment effect on height growth in Blodgett Douglas-fir and Elgin grand fir, and although these results were not strictly comparable, there were some consistencies. The treatments that had the greatest growth effects in Blodgett were not applied in Elgin, and the lack of these treatments affected the comparison. The data collected from these experiments do not provide an adequate basis for concluding why the responses varied as they did.

Procedural and Technical Factors

The adjustment factor utilized to correct inherent variation in budbreak time for the Blodgett Douglas-fir had some shortcomings. There was no overlap in the 1976 budbreak times of control trees classed two and three in 1975, but considerable overlap in classes three and four. There is a large difference in the adjustment factor for class two (27.44) and the adjustment factors for class three and four (14.0, 11.8), and a small difference in the factors for class

three and four. (Complete listing of the factors and their computation is in Appendix Table A2). This is probably a result of: (1) The class two represents a larger biological period than the numerical value represents. The process of budbreak should have been rated by a scale with finer steps. (2) The rankings were assigned too late in 1975. When the 1975 rankings were made, all trees were in the two, three, or four class. An earlier ranking time that would have included all five classes (zero to four) would have given a more accurate indication of inherent budbreak time differences.

Although the method had these problems, the adjustment did reduce inherent variation. All control trees ranked two in 1975 broke buds in 1976 later than all those ranked three and four, indicating a real difference in the two and three, four classes. The variation within the correction terms is large compared with the resultant adjusted day differences, but the magnitude of the possible error due to correction is considerably less than the accuracy gained by it.

The inate time of budbreak was quite variable. With the small sample sizes utilized, random selection of trees for each treatment group often put all early or all late trees into one treatment group. Analysis without some correction would have been impossible unless a treatment had a very large delay effect. With a larger sample of trees, analysis might have been done without correction for inherent differences.

Although both analyses of budbreak time (actual and adjusted values) for Blodgett indicated a lack of significance, the F ratios of the analyses were considerably different. All but two F ratios considered were higher (nearer the significance level) in the actual day analysis. The variation attributable to inherent tree differences that was removed by the adjustment would have reduced the precision of the results if left in the analysis. If the treatments had had a greater delaying effect (with a delay close to the significance level), and the data was analyzed without adjustment, the reduced precision could have led to an invalid conclusion.

Some error could have been introduced into the analysis by the equation utilized to determine days to bud stage 2.5 on the Blodgett Douglas-fir. Observations were taken in the field as 0, 1, 2, 3, and 4, as previously described. Using these observations to exponentionally determine a point at bud stage 2.5 ignored the fact that the '2' recorded for an observation might have already been '2.5' or '2.3' stage. A recorded '3' observation could have actually represented a '2.9' or '3.3', etc. This underlines the difficulty of looking for something in data that was not actually recorded.

Interpretation of Previous Work with Growth Regulators and Dormancy Regulation

A basic problem exists in drawing conclusions from the body of

literature dealing with effects of applied growth regulators. Many reports deal with a small part or function of an organism that has been isolated and does not examine responses of the whole tree. Van Lear (82) reported budbreak delay and shoot control with MH, but also noted that inhibition accompanied the treatments. Interrelationships of such factors can confuse effects and are often ignored. Many experiments are done with potted material or in nursery beds with young seedlings. Extrapolation of these data for field use on plant material with a much larger biomass growing in natural conditions may not be valid. Researchers at Weyerhaeuser Company (Ross, Greenwood, personal communication) have noted that a growth regulator application that is effective in the greenhouse has little or no effect under field conditions, and hypothesize that cuticle thickness, varying absorption, sun, or other environmental factors can cause the variation.

Some experimenters have tested growth regulators by soaking entire seedlings or parts of them in a solution, applied sprays to the drip point with a mist, and repeated these application at varying intervals. This project was aimed at developing a practical method of application for forestry uses. Limiting the approach to treatments that can be applied by aircraft effectively prohibits the use of many of these techniques. Cost would prohibit repeated application.

Other workers have had difficulty in delaying budbreak and growth in coniferous trees. Ostrom (61) identified treatments that could sometimes prevent needle growth in red pine, but could not delay the initial stem elongation. Greenwood (personal communication) has found treatments that will prevent second flush of growth in southern pines, but not the first flush. A tree such as Douglas-fir that does all of its elongation growth in two months has a lot 'invested' in seeing that the growth takes place. Good evidence for this is shown by the results of some of the Krenite September treatments. Up to one half of the tree would have been completely defoliated and parts of the cambium killed by time of budbreak, but the lower buds broke on schedule and grew normally.

Budbreak Delay Results and Postulated Requirements for Control

The budbreak delay capabilities of the growth regulators tested fell short of the postulated delay necessary to adversely affect or control a DFTM population. The silvex November treatment at 1,500 ppm. provided the longest delay, one of approximately one week. A delay of one week or less was postulated as not having any population control abilities. Silvex treatments caused no foliar damage and actually stimulated height growth of treated trees. It is possible that a higher dosage could be tested for its ability to delay budbreak.

A few treatments, notably fall doses of Krenite and MH, demonstrated ability to cause some bud delay (less than a week), but the treatments also caused foliage damage and/or defoliation. Six of the eight growth regulators tested in this study significantly reduced height growth of treated trees, but most of these regulators exhibited no ability to control budbreak under the conditions utilized.

A number of trees that sustained extensive defoliation (some trees lost one-half of their foliage and the cambium in the defoliated areas also died) broke buds and elongated normally, with no indication of budbreak delay.

The tree's tendency toward normal phenology is apparently very strong. The tree either responds normally in spite of growth regulator application, or is damaged severely. The system that controls growth in the tree is apparently well buffered and has the ability to respond independently of the applied growth regulators in a manner that neutralizes or masks their ability to function.

While no growth regulator treatment was identified that can meet the postulated requirements outlined by this study, this does not mean that this approach of host management is invalid. The growth regulators studied were among the most promising candidates for budbreak activity. A comprehensive understanding of the tree's growth regulatory system will probably be a prerequisite to successful manipulation of time of budbreak. Present technology has not

produced this knowledge or a growth regulator capable of regulating this system.

CONCLUSION

No treatment examined by this study demonstrated the ability to delay budbreak of host conifers of the DFTM for a period sufficiently long to cause a breakdown in the synchrony of budbreak and egg hatch necessary for the insect's survival. A silvex November treatment exhibited a delay of approximately one week and the possibility of providing an adequate delay. Further testing utilizing higher concentrations and a larger sample of trees is need to determine if silvex can cause the necessary budbreak delay. While no growth regulator treatment was found that assures delay necessary to de-synchronize the host and insect, the approach of host management must still be considered valid, pending a successful growth regulation system.

Six of the growth regulators tested were associated with a decrease in height growth. A slight increase in growth was attributed to Ethophon and silvex treatments.

The growth regulators studied here were among the most promising candidates for budbreak activity. The failure of treatments to cause a major postponement implies that the tendency toward normal phenology is very strong. A comprehensive understanding of the regulatory system involved is likely to be a prerequisite to successful manipulation.

BIBLIOGRAPHY

1. Arnold, C. Y. 1960. Maximum-minimum temperature as a basis for computing heat units. Proceedings Amer. Soc. Hort. Sci. 76:682-92.

2. Asher, W. C. 1963. Effects of 2-chloroethyltrimethylammonium chloride and 2, 4-dichlorobenzyltributyl phosphonium chloride on growth and transpiration of slash pine. Nature. 200:912.

3. Ashton, F. M. and A. S. Crafts. 1973. Mode of action of herbicides. John Wiley & Sons, New York. 504p.

4. Bachelard, E. P., and F. Wightman. 1973. Biochemical and physiological studies on dormancy release in tree buds. I. Changes in degree of dormancy, respiratory capacity, and major cell constituents in overwintering vegetative buds of Populus balsamifera. Can. J. Bot. 51:2315-2326.

5. Bachelard, E. R. and F. Wightman. 1974. Biochemical and physiological studies on dormancy release in tree buds. III. Changes in endogenous growth substances and a possible mechanism of dormancy release in overwintering vegetative buds of Populus balsamifera. Can. J. Bot. 52:1483-1489.

6. Bean, J. L. 1961. Predicting emergence of second-instar budworm larvae from hibernation under field conditions in Minnesota. Ann. Entomol. Soc. Am. 54:175-177.

7. Bean, J. L. and L. F. Wilson. 1964. Comparing various methods of predicting development of spruce budworm, Choristoneura fumiferana, in northern Minnesota. J. Econ. Entomol. 57: 925-8.

8. Beckwith, R. C. 1976. Influence of host foliage on the Douglas-fir tussock moth. Envirn. Entomol. 5(1):73-77.

9. Bonner, J. 1950. Plant biochemistry. Academic Press, New York, 537p.

10. Burg, S. P. and E. A. Burg. 1968. Ethylene formation in pea seedlings: its relation to the inhibition of bud growth caused by indoleacetic acid. Plant Physiol. 43:1069-1074.

11. Cameron, D. G., G. A. McDougell, and C. W. Bennett. 1968. Relation of spruce budworm development and balsam fir shoot growth to heat units. J. Econ. Entomol. 61:857-8.

12. Campbell, R. K. 1974. Use of phenology for examining provenance transfers in reforestation of Douglas-fir. J. App. Ecology. 11(3):1069-1080.

13. Cathey, H. M. 1964. Physiology of growth-retarding chemicals. Annual Review of Plant Physiol. 15:271-302.

14. Chadwick, A. V. and S. P. Burg. 1970. Regulation of root growth by auxin-ethylene interaction. Plant Physiol. 45:192-200.

15. Ching, K. K. and D. Bever. 1960. Provenance study of Douglas-fir in the Pacific north west region. Silvae Genetica 9, Heft 1, 1-32.

16. Chrispeels, M. and J. E. Varner. 1966. Inhibition of gibberellic acid induced formation of α amylase by abscisin II. Nature. 212:1066-67.

17. Cobb, C. 1977. Water relations and the root regeneration response of Douglas-fir. M. S. thesis, Oregon State U. 86 numbered pages.

18. Cook, W. C. 1945. The relation of spring movements of the beet leafhopper (Eutettix tenellus) in central California to temperature accumulations. Ann. Entomol. Soc. of Am. 38(2):149-162.

19. Crafts, A. S. 1961. The chemistry and mode of action of herbicides. Interscience Publishers, New York. 269p.

20. Domanski, R. and T. T. Kozlowski. 1968. Variations in kinetin-like activity of Betula and Populus during release from dormancy. Can. J. Bot. 46:397-403.

21. Duffield, J. W. 1962. Personal communication quoted by Lavender, D. P. and J. B. Zaerr. 1967. In the role of growth regulatory substances in the physiology of Douglas-fir seedlings. Proc. of the Int. Plant Prop. Soc. Ann. Mg. 146-156.

22. Ebell, L. F. 1971. Girdling: its effect on carbohydrate status and on reproductive bud and cone development of Douglas-fir. Can. J. Bot. 49:944-45.

23. Eidt, D. C. and C. H. A. Little. 1968. Insect control by artificially prolonging plant dormancy - a new approach. The Can. Entomol. 100: 1278-9.

24. Embree, D. G. 1965. The population dynamics of the winter moth in Nova Scotia, 1954-1962. Mem. Entomol. Soc. Can. 46.

25. Esashi, Y. and A. C. Leopold. 1969. Dormancy regulation in subterranean clover seeds fy ethylene. Plant. Physiol. 44: 1470-73.

26. Fernandez, A. G., G. Gimenez-Martin, and C. De La Torre. 1971. The duration of the interphase periods at different temperatures in root tip cells. Cytobiologie, 3:367-371.

27. Gahagan, H. E., R. E. Holm, and F. B. Abeles. 1968. Effects of ethylene on peroxidase activity. Physiol. Plant. 21:1270-79.

28. Galston, A. W. and P. J. Davies. 1969. Hormonal regulation in higher plants. Science. 163:1288.

29. Gratkowski, H. J. 1977. Seasonal effects of phenoxy herbicides on ponderosa pine and associated species. Forest Science. 23: 2-12.

30. Greenbank, D. O. 1963. Climate and the spruce budworm. p. 174-180 In The dynamics of epidemic spruce budworm populations. R. F. Morris, Ed. Entomol. Soc. Can. Mem. 31: 332p.

31. Greulach, V. A. and J. G. Haesloop. 1954. Some effects of maleic hydraside on internode elongation, cell enlargement, and stem anatomy. Amer. Jor. Bot. 41:44-50.

32. Harada, H. and A. Lang. 1965. Effect of some (2-chloroethyl) trimethylammonium chloride analogs and other growth retardants on gibberellin biosynthesis in Fusarium moniliforme. Plant Physiol. 40:176-183.

33. Hard, J. 1976. Unpublished progress reports given at USDA DFTM R & D Program working group meeting, Sacramento, Ca.

34. Hardwick, D. F. 1971. The 'phenological date' as an indicator of the flight period of noctuid moths. Can. Entomol. 103:1207-16.

35. Hendershott, C.H. 1962. Influence of maleic hydrazide on citrus trees and fruits. Am. Coc. Hort. Sci. Proc. 80:241-46.

36. Herbicide handbook of the Weed Science Society of America. 1973. W.F. Humphrey Press, Inc. Geneva, N.Y. Third edition.

37. Hermann, R.K. 1967. Seasonal variation in sensitivity of Douglas-fir seedlings to exposure of roots. For. Sci. 13(2):140-49.

38. Hitchcock, A.E. and P.W. Zimmerman. 1943. Summer sprays with potassium α-naphthaleneacetate retard opening of buds on fruit trees. Proc. Amer. Soc. Hort. Sci. 42:141-145.

39. Jones, R.L. 1973. Gibberellins: their physiological role. Ann. Rev. Plant. Physiol. 24:571-598.

40. Kozlowski, T.T. 1964. Shoot growth in woody plants. Bot. Rev. 30:334.

41. Kreuger, K.W. 1967. Nitrogen, phosphorus, and carbohydrate in expanding and year-old Douglas-fir shoots. For. Sci. 13(4): 352-56.

42. Kurashi, S. and R.M. Muir. 1963. Mode of action of growth retarding chemicals. Plant. Physiol. 38:19-24.

43. Kurashi, S. and R.M. Muir. 1963. Mode of action of growth retarding chemicals. Plant. Physiol. 37(sup.): xxiii.

44. Larson, P.R. 1960. Gibberellic acid induced growth of dormant hardwood cuttings. For. Sci. 5:389-95.

45. Lavender, D.P. and B.D. Cleary. 1975. Coniferous seedling production techniques to improve seedling establishment. North Amer. Containerized Forest Seedling Symp. Denver. Aug. 26-29, 1974.

46. Lavender, D.P., R.K. Hermann, and J.B. Zaerr. 1970. Growth potential of Douglas-fir seedlings during dormancy. In: Physiology of tree crops, ed. by L.C. Luckwill and C.V. Cullins. Academic Press, London. 382p.

47. Lavender, D.P. and W.S. Overton. 1972. Thermoperiods and soil temperatures as they affect growth and dormancy of Douglas-fir seedlings of different geographic origin. Research Paper 13. Forest Research Lab., Corvallis, Or. 26pp.

48. Lavender, D.P., G.B. Sweet, J.B. Zaerr, and R.K. Hermann. 1973. Spring shoot growth in Douglas-fir may be initiated by gibberellins exported from the roots. Science. 182:838-839.

49. Lavender, D.P. and P.F. Wareing. 1972. Some investigations of the root growth of dormant Douglas-fir (Pseudotsuga menziesii (Mirb.) Franco) seedlings. New Phytologist. 71:1055-67.

50. Lavender, D.P. and J.B. Zaerr. 1975. Growth regulators in the xylem sap and dormancy in Douglas-fir. Research proposal submitted to NSF, not funded. On file, Oregon State Univ., Forest Research Lab.

51. Lavender, D.P. and J.B. Zaerr. 1967. The role of growth regulatory substances in the physiology of Douglas-fir (Pseudotsuga menziesii (Mirb) Franco) seedlings. Proc. of the Int. Plant Prop. Soc. Ann. Mtg. p. 146-156.

52. Leopold, A.C. and W.K. Klein. 1952. Maleic hydrazide as an anti-auxin. Physiol. Plant. 5:91-99.

53. Leopold, A.C. and P.E. Kriedemann. 1975. Plant growth and development. McGraw Hill. New York. 545p.

54. Levitt, J. 1956. Hardiness in Plants. J. Levitt, Ed. Academic Press, New York.

55. Mason, R.R. and J.W. Baxter. 1970. Food preference in a natural population of the Douglas-fir tussock moth. J. Econ. Entomol. 63(4):1257-59.

56. Mitchell, R.G. and R.E. Wiggins. 1974. Field dispersion of early instar Douglas-fir tussock moth larvae. unpublished draft. Forestry Sciences Lab., Corvallis, Or.

57. Morris, R.F., F.E. Webb, and C.W. Bennett. 1956. A method of phenological survey for use in forest insect studies. Can. J. Zool. 34:533-40.

58. Newton, M. 1971. Resiliency of ecosystems. In Pesticides, pest control, and safety on forest lands. J. Capizzi and J. Witt, Ed. Office of Continuing Education, OSU, Corvallis, Or.

59. Newton, M. and J. Norgren. 1977. Silvicultural chemicals and protection of water quality. U.S. Environmental Protection Agency, Seattle. Report # EPA-910/9-77-036.

60. Ninnemann, H., J.A.D. Zeevart, H. Kende, and A. Lang. 1964. The plant growth retardant CCC as inhibitor of gibberellin biosynthesis in Fusarium moniliforme. Planta 61:229-35.

61. Ostrom, C.E. 1945. Effects of plant growth regulators on shoot development and field survival of forest tree seedlings. Bot. Gazette. 107:139-183.

62. Owens, J.N. 1968. Initiation and development of leaves in Douglas-fir. Can. J. Bot. 46:271-278.

63. Owens, J.N. and M. Molder. 1973. A study of DNA and mitotic activity in the vegetative apex of Douglas-fir during the annual growth cycle. Can. J. Bot. 51:1395-1409.

64. Pearis, L.M. 1927. Some phases of the relation of temperature to the development of insects. W. Virginia U. Agr. Exp. Sta. Bull. 208. 62p.

65. Perkins, R.F. and R.E. Dolph, Jr. 1967. Operational and entomological report, 1965 Burns project, Douglas-fir tussock moth control. USDA For. Ser. Forestry Sciences Lab, Corvallis, Or. p. 26-28.

66. Perry, T.O. 1971. Dormancy of trees in winter. Sci. 171: 29-36.

67. Reitveld, W.J. 1966. Effects of three plant growth regulators upon growth patterns in seedlings of three races of Douglas-fir, Pseudotsuga menziesii (Mirb) Franco. M.S. thesis. Oregon State U. 62 numb. leaves.

68. Robinson, P.M. and P.F. Wareing. 1964. Chemical nature and biological properties of the inhibitor vary with photoperiod in sycamore maple. Physiol. Plant. 17:314-323.

69. Romberger, J.A. 1963. Meristems, growth, and development in woody plants. Tech. Bull. No. 1293. USDA For. Ser. 214 p.

70. Samish, R.M. 1954. Dormancy in woody plants. Ann. Rev. Plant Physiol. 5:183-204.

71. Schutte, F. 1963. Bekampfung des eichenwicklers (Tortrix viridana L.) durch verzagerung des knospenaustriebs. Proc. 4th Int. Congr. Crop Prot. 2:1329-1332.

72. Schwerdtferger, F. 1962. Das eichenwickler problem. Forsch. u. Berat. Landesaussch. Landw. Forsch. Landes Nordhein-Westfalen. (Ser. C), No. 1.

73. Sell, H.M., W. Reuther, E.G. Fisher, and F. Langasse. 1942. Effect of chemical treatments in prolonging dormancy of tung buds. Bot. Gaz. 103:788-793.

74. Shibakusa, R. 1973. Experiments on inhibiting the budbreak and lammas shoot growth of seedlings of Abies sachalinensis (Masters) by spraying growth substances. Res. Bull. of the College Forests, Hokkaido U. 30(1):33-41.

75. Sutton, R.F. 1969. Form and development of conifer root systems. Commonwealth Forestry Bureau Tech. Comm. #7. 131p.

76. Sweet, G.B. 1965. Provenance. Seed and seedling characteristic differences in Pacific West Douglas-fir. Silvae Genetica. 14:46.

77. Taylor, J.A. 1967. Growing season as affected by land aspect and soil texture. p. 15-36 In, Weather and agriculture, J.A. Taylor, Ed. Pergamon Press, Oxford.

78. Thielges, B.A. and R.C. Beck. 1976. Control of budbreak and its inheritance in Populus deltoides. In, Tree physiology and yield improvement, M.G.R. Cannel and F.T. Last, Ed. Academic Press, Inc. NY.

79. Thimann, K.V. and F. Skoog. 1933. Studies on the growth hormone of plants. III. The inhibiting action of the growth substances on bud development. Proc. Nat. Acad. Sci. Wash. 19: 714-716.

80. Tolbert, N.E. 1961. Structural relationship among chemicals which act like anti-gibberellins. Advanc. Ghem. Sci. 28:145-151.

81. Toole, V.K., W.K. Bailey, and E.H. Toole. 1964. Factors influencing dormancy of peanut seeds. Plant. Physiol. 39:822-32.

82. Van Lear, D., H. Lowenstein, and F.H. Pilkin. 1970. The influence of maleic hydrazide and certain auxins on growth and survival of Douglas-fir seedlings. U. of Idaho. Forest, Range, and Wildlife Exp. Sta., Moscow, Sta. paper #6. 11p.

83. Van Overbeek, J. 1966. Plant hormones and regulators. Science. 152:721-31.

84. Varley, G.C. 1969. The concept of energy flow applied to a woodland community. p. 389-404 in Animal populations in relation to food resources, A. Watson, Ed. Blackwell Scientific Publications, Oxford.

85. Vasil-yev, I.M. 1961. Wintering in plants. Amer. Inst. of Bio. Sci., Wash., D.C.

86. Vegis, A. 1964. Dormancy in higher plants. Ann. Rev. Plant Physiol. 15:185-224.

87. Villiers, T.A. 1975. Dormancy and the survival of plants. Edward Arnold Limited, London. 68p.

88. Wagg, J.W.B. 1958. Enviornmental factors affecting spruce budworm growth. Forest Lands Research Center, Res. Bull # 11. 27p.

89. Wareing, P.F. and I.D.J. Phillips. 1970. The control of growth and differentiation in plants. Pergamon Press, Oxford. 303p.

90. Wareing, P.F. and P.F. Saunders. 1971. Hormones and dormancy. Ann. Rev. of Plant Physiol. 22:261-288.

91. Webber, J.E., J.B. Zaerr, and M. Laver. 1972. The occurrence of abscisic acid in the shoots of dormant Douglas-fir. Abst in: Amer. Soc. Plant Physiol., West, Section meeting, U. Oregon, Eugene. June, 1972. p. 47.

92. Wickman, B.E. 1976. Douglas-fir tussock moth egg hatch and larval development in relation to phenology of grand fir and Douglas-fir in northeastern Oregon. USDA For. Serv. Res. Pap. PNW-206, 13p., illus. Pacific Northwest Forest and Range Exp. Sta., Portland, Or.

93. Wickman, B. E. 1976. Pheonology of white fir and Douglas-fir tussock moth egg hatch and larval development in California. Env. Entom. 5(2):316-322.

94. Wickman, B. E., R. R. Mason, and C. G. Thompson. 1963. Major outbreaks of the Douglas-fir tussock moth in Oregon and California. USDA For. Serv. General Tech. Rep. PNW-5, 18p., illus. Pacific Northwest Forest and Range Exp. Sta., Portland, Or.

95. Wickman, B. E., C. G. Trostle, and P. E. Buffam. 1973. Douglas-fir tussock moth. USDA For. Serv., Forest Pest Leaflet No. 86.

96. Williams, C. B. 1953. Graphical and statistical methods in the study of insect phenology. Trans. IXth Int. Congr. Entomol. 11:174-89.

97. Zaerr, J. B., D. P. Lavender, and G. B. Sweet. 1973. Growth regulators in Douglas-fir during dormancy. From, Dormancy in Trees, Int. Symp., Polish Acad. of Sci. 4p.

98. Zimmerman, M. H. and C. L. Brown. 1974. Trees, structure and function. Springer-Verlang. New York. 336p.

APPENDIX

DESCRIPTION OF SPRAY APPLICATION EQUIPMENT

A Solo Knopsack Sprayer #425 was the basis of the application equipment. A custom designed boom was made that covers a swath 152 cm. wide from a 1.2 meter height. The sprayer applied 188 liters/hectare at 20 psi. Nozzle bodies used were tee-jet diaphragm type, with 06015 tips. An Ashcraft 1850 pressure gauge and a hand powered pump were used to regulate pressure.

Table A1. Actual day of budbreak and standard deviation, Blodgett Study Area, Douglas-fir.

Chemical Season	Low Dose Budbreak Day	Low Dose Std. Dev. (days)	Middle Dose Budbreak Day	Middle Dose Std. Dev. (days)	High Dose Budbreak Day	High Dose Std. Dev. (days)
Asulam						
April	16.4	6.8	18.6	10.5	19.5	11.2
Sept.	23.1	12.5	17.2	5.38	17.2	6.2
Nov.	15.3	9.3	20.4	10.2	18.5	8.0
Cycocel						
April	16.6	6.6	20.2	6.0	13.4	2.4
Sept.	15.9	8.1	14.8	3.4	20.1	4.5
Nov.	19.9	10.7	19.3	5.9	21.9	9.8
DPX 3778						
April	14.6	31.0	18.1	5.9	13.7	3.0
Sept.	14.8	3.2	22.3	10.2	18.0	5.6
Nov.	16.3	1.5	16.6	5.6	17.4	7.0
Ethophon						
April	16.8	2.9	16.5	3.2	20.0	9.8
Sept.	15.1	3.3	18.9	6.5	18.1	7.2
Nov.	15.8	3.4	22.1	7.7	17.0	6.3
Krenite						
April	14.4	3.8	19.2	7.5	17.9	5.8
Sept.	22.1	5.4	20.0	11.2	23.3	11.5
Nov.	14.8	7.4	13.9	7.6	18.5	8.4
Maleic Hydrazide						
April	19.6	11.0	16.2	5.7	17.7 *	5.9
Sept.	22.6	10.9	17.4	19.6	21.6	11.2
Nov.	19.5	7.6	21.2	21.6	20.7	5.5
NAA						
April	16.2	7.1	14.7	3.3	17.9	6.4
Sept.	22.5	9.9	15.8	3.8	15.9	2.1
Nov.	17.8	6.8	14.9	2.8	18.5	6.9
Silvex						
April	14.2	1.9	17.3	6.4	18.3	6.9
Sept.	16.2	3.1	18.4	10.1	22.9 *	3.9
Nov.	23.1	8.1	25.4	12.2	22.5	10.4
Controls	14.5	6.5	22.1	8.7		
	18.1	3.7	20.3	12.7		
	15.6	7.4			Control Mean, 17.638	
	14.9	6.2				

*All values are the mean of five observations except treatments marked with an (*) contained one missing observation.

Table A2. Blodgett study area, Douglas-fir. Determination of correction factor for day of budbreak.

tree #	1975 Bud Rank	Actual day of budbreak	tree #	1975 Bud Rank	Actual day of Budbreak
0161	2	28.465	0179	3	18.000
0361	2	35.718	0187	3	18.347
0178	2	26.028	0367	3	13.887
0273	2	21.361	2154	3	17.326
0582	2	26.028	2162	3	13.887
2791	2	35.622	2172	3	15.096
3021	2	21.361	2182	3	12.200
3082	2	26.028	2492	3	9.826
3102	2	26.028	2	3	12.952
			2701	3	12.952
Mean for rank 2		27.404	2702	3	10.288
standard deviation		5.225	2730	3	11.868
			2943	3	12.952
2154	4	17.326	3012	3	17.326
2232	4	12.952			
2254	4	9.002	Mean for rank 3		14.065
2731	4	9.826	standard deviation		2.780
2892	4	10.919			
2983	4	10.667			
Mean for rank 4		11.782			
standard deviation		3.021			

Table A3. Blodgett study area, Douglas-fir. Three factor analysis of variance, actual day of budbreak.

	DF	SS	MS	F
Chemical	7	541.6972	77.3853	1.150
Dose	2	89.1444	44.5722	.662
Season	2	344.1096	172.0548	2.556
Chemical x dose	14	555.4454	39.6747	.589
Chemical x season	14	1082.7283	77.3377	1.149
Dose x season	4	135.7967	33.9492	.504
Chemical x dose x season	28	1104.6823	99.4529	.586
error	288	19383.1055	67.3024	
Total	359	23236.7093		

Table A4. Blodgett study area, Douglas-fir. Three factor analysis of covariance, main factor adjusted day of budbreak, covariable height in 1975.

Source	df	X x X	X x Y	Y x Y	df	Adjusted SS	MS
Total	359	564906.4889	-4302.5791	17094.8032			
Chemical	7	4541.0667	-0.7813	243.9731			
Dose	2	43.0721	-21.5291	11.9602			
Season	2	324.8390	85.8682	41.2696			
Chemical x dose	14	27119.6833	-1540.2311	526.2667			
Chemical x season	14	34807.3832	119.4445	937.7916			
Dose x Season	4	4726.0778	54.1665	33.8527			
Chemical x dose x season	28	42680.7667	912.7148	1149.7862			
Error	288	450663.6001	-3912.2314	14149.9031	287	14115.9408	49.1845
Chemical + error	295	455204.6667	-3913.0127	14393.8762	294	14360.2393	48.8444
Dose + error	290	450706.6722	-3933.7605	14161.8633	289	14127.5295	48.8842
Season + error	290	450988.4391	-3826.3632	14191.1726	289	14158.7083	48.9921
Chemical x dose + error	302	477783.2834	-5452.4625	14676.1698	301	14613.9463	48.5513
Chemical x season + error	032	485470.9833	-3792.7869	15087.6947	301	15059.0632	50.0268
Dose x season + error	292	455389.6779	-3858.0649	14183.7557	291	14151.0702	48.6291
Interaction + error	136	493344.3668	-2999.5168	15299.6893	315	15281.4523	48.5125
Regression on error					1	33.9623	33.9623
Chemical adjusted for average error regression					7	244.2985	34.8998
Dose adjusted for average error regression					2	11.5887	5.7944
Season adjusted for average error regression					2	42.7674	21.3837
Chemical x dose adjusted for average error regression					14	498.0055	35.5718
Chemical x season adjusted for average error regression					14	942.1224	67.2945
Dose x season adjusted for average error regression					4	35.1294	8.7823
Chemical x dose x season adjusted for average error regression					28	1165.5115	41.5254

Table A5. Three factor analysis of variance, simple growth difference, Blodgett Douglas-fir.

Source	df	SS	MS	F
Chemical	7	7385.1000	1055.0143	1.554
Dose	2	2037.0389	1018.5194	1.500
Season	2	1001.7556	500.8778	.738
Chemical x dose	14	7679.4500	548.5321	.808
Chemical x season	14	8910.0667	636.4333	.937
Dose x season	4	1610.8278	402.7069	.593
Chemical x dose x season	28	14973.0833	534.7530	.787
Error	288	195570.0000	679.0625	
Total	359	239167.3222		

Table A6. Key to chemical, dose, and season designation codes for following tables A7, A8, A14, A15, A19, and A22. *

chemical	dose	season
1 - Asulam	1 - light dose	1 - April (Blodgett)
2 - Cycocel		- May (Elgin)
3 - DPX 3778	2 - middle dose	
4 - Ethophon		2 - September
5 - Krenite	3 - high dose	
6 - Maleic hydrazide		3 - November
7 - NAA		
8 - silvex		
Control TRTS are labeled X001		
X = 1 or 2 digit number		
Key applies to both study areas.		

* Each treatment is identified by three numbers, i.e. 111. The first number indicates the chemical, the second indicates the dose, and the third the season, according to the table above.

Table A7a. Budbreak rank by treatment, Blodgett, Douglas-fir. Summary ranks by chemical and chemical x season. 1 least delay, 78 most delay.

Rank/Treatment			
1. 412	21. 422	41. 133	61. 832
2. 631	22. 613	42. 211	62. 721
3. 421	23. 111	43. 623	63. 112
4. 312	24. 323	44. 223	64. 332
5. 411	25. 311	45. 233	65. 131
6. 232	26. 113	46. 831	66. 321
7. 523	27. 333	47. 413	67. 532
8. 221	28. 813	48. 212	68. 313
9. 513	29. 4001	49. 3001	69. 121
10. 432	30. 633	50. 521	70. 622
11. 621	31. 2001	51. 821	71. 6001
12. 822	32. 531	52. 711	72. 431
13. 722	33. 732	53. 712	73. 632
14. 231	34. 5001	54. 433	74. 322
15. 411	35. 733	55. 123	75. 213
16. 423	36. 812	56. 731	76. 512
17. 122	37. 1001	57. 833	77. 612
18. 811	38. 331	58. 522	78. 823
19. 222	39. 713	59. 611	
20. 723	40. 533	60. 132	

b. Summary rank by chemical

chemical	Σ ranks	chemical	Σ ranks
Ethophon	239 (least	Maleic hydrazide	387
Cycocel	301 delay)	silvex	387
Krenite	344	DPX 3778	390
NAA	363	Asulam	419 (most delay)

Summary ranks by chemical x season combination

1.	Ethophon September	32	13.	Ethophon November	117
2.	Krenite November	56	14.	DPX 3778 November	119
3.	Cycocel April	64	15.	Asulam November	122
4.	Maleic hydrazide April	72	16.	DPX 3778 April	129
5.	Cycocel September	73	17.	Asulam September	140
6.	Krenite April	87	18.	DPX 3778 September	142
7.	Ethophon April	90	19.	Asulam April	157
8.	NAA November	94	20.	silvex November	163
9.	Maleic hydrazide Nov.	95	21.	Cycocel November	164
10.	NAA September	99	22.	NAA April	170
11.	silvex September	109	23.	Krenite September	201
12.	silvex April	115	24.	Maleic hydrazide Sept.	220

Table A8. Treatment rank by ratio growth difference. 1 least growth, 78 most growth. Summary ranks by chemical and chemical x season.

Rank	Trt.	ratio	Rank	Trt.	ratio
1.	532	.466	40.	221	1.347
2.	632	.562	41.	411	1.348
3.	512	.731	42.	611	1.360
4.	433	.891	43.	121	1.367
5.	711	.937	44.	712	1.372
6.	713	1.043	45.	523	1.372
7.	513	1.066	46.	133	1.376
8.	323	1.074	47.	722	1.377
9.	623	1.096	48.	821	1.384
10.	213	1.120	49.	821	1.384
11.	222	1.121	50.	431	1.398
12.	633	1.132	51.	1001	1.398
13.	322	1.156	52.	113	1.400
14.	232	1.160	53.	212	1.405
15.	723	1.173	54.	822	1.408
16.	332	1.186	55.	622	1.414
17.	112	1.188	56.	531	1.427
18.	731	1.188	57.	423	1.432
19.	331	1.191	58.	613	1.436
20.	3001	1.224	59.	413	1.441
21.	522	1.225	60.	421	1.452
22.	131	1.226	61.	122	1.464
23.	223	1.231	62.	832	1.465
24.	621	1.231	63.	6001	1.469
25.	833	1.241	64.	433	1.496
26.	123	1.247	65.	811	1.501
27.	631	1.248	66.	4001	1.510
28.	5001	1.255	67.	231	1.524
29.	432	1.258	68.	823	1.525
30.	733	1.272	69.	511	1.563
31.	2001	1.285	70.	813	1.576
32.	233	1.299	71.	111	1.579
33.	721	1.282	72.	521	1.598
34.	333	1.312	73.	831	1.598
35.	312	1.318	74.	211	1.656
36.	311	1.322	75.	812	1.670
37.	612	1.322	76.	313	1.690
38.	732	1.325	77.	321	1.848
39.	132	1.344	78.	422	1.944

A8b
Summary ranks

chemical	Σ rank
NAA	236 (least growth)
Maleic H.	266
Krenite	278
DPX 3778	314
Cycocel	324
Asulam	377
Control	388
Ethophon	487
Silvex	540 (most growth)

chemical x season	rank
Krenite September	25
NAA November	51
NAA April	56
Krenite November	56
DPX 3778 September	64
Cycocel November	65
Cycocel September	78
Maleic H. November	79
Maleic H. April	93
Maleic H. September	94
Asulam September	117
DPX 3778 November	118
Asulam November	124
NAA September	129
DPX 3778 April	132
Asulam April	136
Ethophon April	151
Ethophon September	156
Silvex November	163
Ethophon November	180
Cycocel April	181
Silvex April	186
Silvex September	191
Krenite April	197

Table A9a. Elgin Douglas-fir. Analysis of variance for day of budbreak, all treatments.

Source	df	SS	MS	F
Treatment	36	789.8339	21.9398	.8962
Error	290	7099.8175	24.4821	
Total	326	7889.6514		

Table A9b. Elgin Douglas-fir. Analysis of variance for day of budbreak, May treatments.

Source	df	SS	MS	F
Treatment	21	427.2139	20.3435	.7775
Error	198	5180.8889	26.1661	
Total	219	5608.1028		

Table A10. Elgin Douglas-fir. Analysis of covariance for day of budbreak, main factor day of budbreak, height in 75 covariable.

Source	df	X x X	X x Y	Y x Y
Total	219	463914.1820	-14996.1260	5608.1028
Treatment	21	50210.9818	551.1994	427.2139
Error	198	413703.2000	-15547.3254	5180.8889
		SS	MS	F
Total	218	5123.3497		
Error	197	4596.6069	23.3330	
Treatment	21	526.7426	25.0830	1.0750

Table A11. Height growth analyses, Elgin Douglas-fir.

Source	df	SS	MS	F
a. Analysis of variance, simple growth difference, May treatments only.				
Treatment	21	3862.5852	183.9326	.9555
Error	198	38113.6000	192.4929	
Total	219	41976.1852		
b. Analysis of variance, ratio growth difference, May treatments only.				
Treatment	21	5.2676	.2508	.9615
Error	198	51.6557	.2609	
Total	219	56.9233		
c. Analysis of variance, simple growth difference, all treatments.				
Treatment	36	6489.9567	180.2766	1.1193
Error	290	46707.8476	161.0615	
Total	326	53197.8043		
d. Analysis of variance, ratio growth difference, all treatments.				
Treatment	36	12.9839	.3607	.8478
Error	290	123.3688	.4254	
Total	326	136.3526		

Table A12. Treatments by budbreak rank, Elgin Douglas-fir. Summary ranks by chemical x season. 37 most delay, 1 least delay.

Rank/treatment						Summary Ranks	Sum of 3 Ranks
37.	323	24.	533	11.	811	DPX Nov.	85
36.	731	23.	821	10.	211	DPX May	73
35.	333	22.	6001	9.	7001	Krenite May	71
34.	521	21.	221	8.	3001	Maleic H. May	68
33.	4001	20.	621	7.	711	silvex May	61
32.	611	19.	5001	6.	511	Ethophon May	58
31.	531	18.	523	5.	231	Control	51
30.	321	17.	421	4.	111	Krenite November	45
29.	411	16.	631	3.	513	NAA May	45
28.	311	15.	331	2.	721	Asulam May	43
27.	831	14.	131	1.	1001	Cycocel May	36
26.	2001	13.	313				
25.	121	12.	431				

Table A13. Treatments by ratio growth rank, Elgin Douglas-fir. Summary ranks by chemical x season. 1 least growth, 37 most growth.

Rank/treatment						Summary Ranks	Sum of 3 Ranks
1.	513	13.	521	25.	321	Asulam May	25
2.	523	14.	131	26.	831	Cycocel May	33
3.	111	15.	231	27.	7001	Krenite November	35
4.	3001	16.	6001	28.	611	Krenite May	48
5.	531	17.	431	29.	731	silvex May	55
6.	211	18.	121	30.	511	Ethophon May	59
7.	311	19.	811	31.	721	Maleic H. May	59
8.	621	20.	1001	32.	533	Control	63
9.	421	21.	4001	33.	411	DPX May	66
10.	821	22.	2001	34.	331	DPX November	71
11.	323	23.	631	35.	711	NAA May	95
12.	221	24.	333	36.	313		
				37.	5001		

Table A14a. Elgin grand fir, analysis of variance for day of budbreak, all treatments.

Source	df	SS	MS	F
Treatment	43	886.3872	20.6137	.9750
Error	122	2579.3056	21.1418	
Total	165	3465.6928		

Table A14b. Elgin grand fir, analysis of variance for day of budbreak, May treatments.

Source	df	SS	MS	F
Treatment	24	460.1598	19.1733	.8900
Error	75	1615.6667	21.5422	
Total	99	2075.8265		

Table A15. Elgin grand fir, analysis of covariance, main factor day of budbreak, height in 1975 covariable. May treatments.

Source	df	X x X	X x Y	Y x Y
Total	99	186746.9900	-2843.1603	2075.8265
Treatment	24	38353.2400	912.2862	460.1598
Error	75	148393.7500	-3755.4463	1615.6667

Source	df	SS	MS	F
Total	98	2032.5404		
Error	74	1520.6264	20.5490	
Treatment	24	511.9139	21.3297	1.0380

Table A16. Simple linear regression of day of budbreak on height in 1975. Elgin grand fir, controls only.

$Y = 13.2380 + .0091 (X)$

$R = .0683$ std. error A = 2.0956

$R^2 = .005$ std. error B = .0181

$F = .2529$

Table A17. Treatments by budbreak rank, Elgin grand fir. Summary ranks (sum of three doses over season) by chemical x season. 44 most delay, 1 least delay. See treatment key, Appendix Table A6.

Rank/treatment			Summary ranks	Sum of 3 Ranks
44. 111	29. 1002	14. 6002	Krenite November	121
43. 523	28. 221	13. 431	Krenite May	107
42. 533	27. 621	12. 711	Asulam May	89
41. 333	26. 411	11. 8002	Cycocel May	76
40. 7002	25. 10002	10. 821	Control	60
39. 333	26. 731	9. 321	NAA May	57
38. 531	23. 121	8. 323	Ethophon May	54
37. 831	22. 131	7. 11002	Silvex May	53
36. 513	21. 721	6. 811	DPX May	52
35. 521	20. 3002	5. 611	Maleic H. May	51
34. 511	19. 631	4. 12002	DPX November	49
33. 13002	18. 231	3. 14002		
32. 2002	17. 5002	2. 311		
31. 9002	16. 4002	1. 313		
30. 211	15. 421			

Table A18. Analysis of variance, ratio growth method, Elgin grand fir, all treatments.

Source				
Treatment	43	23.5577	.5479	1.1966
Error	122	55.8562	.4578	
Total	165	79.4139		

Table A19. Treatments by ratio growth rank, Elgin grand fir. Summary ranks by chemical x season. 1 least growth, 44 most growth.

Rank/treatment			Summary ranks	Sum of 3 Ranks
1. 531	16. 2002	31. 13002	Cycocel May	35
2. 523	17. 221	32. 731	Krenite Nov.	36
3. 721	18. 421	33. 831	Krenite May	36
4. 211	19. 711	34. 321	Maleic H. May	52
5. 611	20. 4002	35. 621	NAA May	54
6. 521	21. 533	36. 11002	Asulam May	58
7. 333	22. 431	37. 6002	DPX May	60
8. 14002	23. 411	38. 323	Ethophon May	63
9. 131	24. 7002	39. 121	DPX November	73
10. 111	25. 1002	40. 8002	Control	89
11. 311	26. 5002	41. 10002	Silvex May	107
12. 631	27. 3002	42. 9002		
13. 513	28. 313	43. 12002		
14. 231	29. 511	44. 821		
15. 331	30. 811			

Table A20. Budbreak data for silvex 1,500 ppm treatment in November, Blodgett study area.

Tree No.	Bud ratings							Actual day of budbreak	Bud rank 1975	Adjusted day of budbreak
	day 5	8	10	14	18	25	31			
D0472	0	1	1	1	1	2	3	27.9	2	.6
D0566	1	1	1	1	1	2	3	26.0	2	-1.4
D2091	0	1	1	1	1	1	1*	44.0	3	29.9
D2311	1	1	1	2	3	3	4	16.0	3	1.9
D2841	1	1	2	3	3	4	4	12.9	4	1.2

*Data collected from this tree could not be accurately analyzed by the method utilized for other trees. The data of budbreak was subjectively determined based on field observations.