arousal response in this behavioral preparation. The reaction is initiated in the presence of heightened sensory thresholds normally associated with sleep (*9*). It may be inferred from the behavior data and the heart-rate data that the degree of neural activation underlying the response is related to the intensity of radiation. The arousal response is not dependent upon direct visual stimulation by x-rays. The arousal reactions which arise after or continue beyond the first few seconds very likely involve reflex activation of the adrenal medulla (*4*).

Recent studies with mammals have shown that within the first minute of moderate intensity exposure gastric retention occurs (*10*), oxygen consumption increases (*11*), and electroencephalographic activity is altered (*12*). Although these responses might be related to the arousal response, arising as a consequence of central activation, they might also be primary responses to nervous stimulation with radiation. Reflex-like reactions to ionizing radiations have been described for invertebrates; the most sensitive reaction was found to be tentacle retraction in the snail (*13*). The arousal response in the rat would appear to be of comparable sensitivity.

The nervous mechanisms which are affected by radiation exposure in the production of behavioral arousal and central activation are obscure. Aside from photoreceptors, no sensory receptors have been demonstrated to be directly sensitive to radiation stimulation. Although the visual system is not essential for the reaction, it cannot be ruled out that stimulation through other receptor systems may initiate the central activation. Direct ganglionic sensitivity to ionizing radiation is also possible. This was proposed years ago by Toyama (*14*). More recently, Hug (*13*) suggested that ionizing radiation may act like visible light in activating certain photosensitive processes in ganglionic structures. It is also possible that penetrating ionizing radiation is a particularly efficient means for stimulating large masses of nervous tissue directly since the energy transfer would occur relatively uniformly with minimum spatial or temporal loss.

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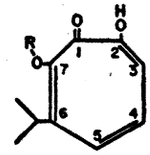
On the Decay Retardant Properties of Some Tropolones

Abstract. The heartwoods of tree species containing tropolone compounds in their extractive components are classed among the more durable woods. Two new tropolones, namely, α-thujaplicinol and pygmaein from the heartwood of *Cupressus pygmaea,* were also found to exhibit strong fungitoxic properties active against a number of wood-destroying fungi. The hydroxyl group appears to be the functional grouping which is largely responsible for the fungicidal action exhibited by this type of compound.

Three tropolones were found to be present in incense cedar heartwood (*Libocedrus decurrens* Torrey), namely, γ-thujaplicin (0.07 percent in wood), β-thujaplicin (trace), and β-thujaplicinol (trace) (*1*). It was previously reported that γ-thujaplicin was among the most potent fungicidal extractive components found in this heartwood (*2*). These three particular tropolones, as well as others, are also present in western red cedar heartwood (*Thuja plicata* Donn) and are largely responsible for the well-known decay resistance of this cedar (*3*).

In recent studies on the occurrence of tropolones in various tree species, two new tropolones were found—α-thujaplicinol (7-hydroxy-6-isopropyl tropolone) (I) (0.4 percent in wood) and its monomethyl ether, pygmaein (7-methoxy-6-isopropyl tropolone) (II) (0.4 percent in wood) in the heartwood of *Cupressus pygmaea* Lemm (*4*). The fungicidal properties of each of these compounds together with γ-thujaplicin (5-isopropyl tropolone) and methylated thujaplicins (laboratory preparation) were determined.



I R = H (α-thujaplicinol)
II R = CH_3 (pygmaein)

The method used, except for minor deviations, was the standard soil-block bioassay procedure (*5*). Test blocks of ponderosa pine sapwood were impregnated with each of the aforementioned compounds and subjected to pure-culture decay. Loss in block weight during the test constituted the measure of decay. The wood-destroying fungi used were: (i) *Lentinus lepideus* (Madison 534), (ii) *Lenzites trabea* (Madison 617), and (iii) *Poria monticola* (Madison 698). The results are summarized in Table 1.

It is quite apparent from these results that α-thujaplicinol is among the most potent of the compounds which we tested. It also will be observed that when one of the hydroxyl groups is replaced by a methoxy group, such as pygmaein, the fungicidal activity decreases. This would suggest that the tropolone hydroxy group is the functional grouping largely responsible for the fungitoxic properties exhibited by this type of compound. This appears to be borne out further by the result with the prepared methylated thujaplicins; these compounds had virtually no decay-inhibiting capacity against the fungi tested, while the naturally occurring γ-thujaplicin was strongly fungicidal. Since each of the two hydroxyl groups in α-thujaplicinol is immediately

Table 1. Decay retardant bioassay. The test blocks contained 1.2 percent (by weight) of chemical; test duration was 6 weeks.

Weight loss caused by decay fungus (%)		
Lentinus lepideus	*Lenzites trabea*	*Poria monticola*
	γ-Thujaplicin	
1	0	18
	α-Thujaplicinol	
1	1	1
	Pygmaein	
4	29	14
	Methylated thujaplicins	
23	51	43
	Control	
33	52	45

adjacent to the carbonyl group, each of these is a "tropolone"—OH. Therefore, each of these hydroxyl groups may be involved in the tropolone function. On the other hand, if either of these groups were in positions 3, 4, or 5, these would be ring hydroxyls—pseudo-aromatic hydroxyls having perhaps a different function than the hydroxyls present in positions 2 and 7, as in α-thujaplicinol. Further, it is of interest to note that, while γ-thujaplicin and pygmaein are intermediate in effectiveness toward *Poria monticola*, α-thujaplicinol, in particular, was very potent. This, again, reflects differences in tolerance of the fungi to different chemicals (*6*).

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Neural Timing of Ovulation in Immature Rats Treated with Gonadotrophin

Abstract. Thirty-two-day-old female rats, injected two days previously with gonadotrophin from pregnant mares' serum, were given barbiturate at different times during the afternoon to determine whether the ovulation that would occur the next day could be blocked. The results indicate that ovulation depends upon release of pituitary ovulating hormone during a "critical period" between 2 and 4 P.M.

Everett *et al.* demonstrated a neural activation of the release of pituitary ovulating hormone (*1*) occurring within a 2-hour period during the afternoon preceding ovulation in adult female rats (*2*). The release of hormone was concluded to be coextensive with activation of the pituitary (*3*). The release of ovulating hormone could be prevented if nervous system blocking agents, including barbiturates, were administered prior to the onset of this "critical period" (*2*, *4*).

Cole induced ovulation consistently in immature female rats with a single injection of gonadotrophin from pregnant mares' serum (*5*). For several years we have been producing ovulation in more than 90 percent of 33-day-old female rats with a single injection of 0.4 Cartland-Nelson unit of this serum gonadotrophin. Nontreated rats seldom ovulate by this age. The treated animals mate, conceive, parturate, lactate, and raise a litter of young in an essentially normal manner.

It was of interest to know whether the ovulation in our rats depended on the release of endogenous pituitary gonadotrophin. Hypophysectomy at various times prior to ovulation indicated that the pituitary was essential up to, but not after, the late afternoon before ovulation. Experiments were then performed with barbital sodium (sodium 5,5-diethylbarbiturate) as a long-acting neural blocking agent.

Immature female albino rats, 26 days old, weighing 65 to 70 g, were obtained from the Holtzman Company. They were caged six to seven per cage with 14 hours of light and 10 hours of darkness at a room temperature of 26±1°C. When the animals were 30 days old they were given, between 9 and 10 A.M. (*6*), a single subcutaneous injection of 0.4 Cartland-Nelson unit of gonadotrophin from pregnant mares' serum (Gonadogen) in 1.0 ml of saline (*7*). Approximately 3 to 5 percent of the rats had open vaginas at 6 to 7 P.M. of the following day and they were discarded.

When the rats were 32 days old and weighed 90 to 110 g, some were injected intraperitoneally with 30 mg of barbital sodium in 0.5 ml of water at 1:55 P.M. ± 10 minutes, and others were given the same treatment at 4, 5, or 6 P.M. The controls received no barbital. Unconsciousness usually occurred within 20 to 30 minutes, and a supplemental intraperitoneal injection of no more than 1.2 mg of sodium pentobarbital (Veterinary Nembutal sodium, Abbott) was given to any individuals in which sedation was slow to develop. After 2 hours or more of unconsciousness, some rats required artificial respiration or aspiration of the pharynx, or both. The animals were autopsied during the afternoon and evening of the next day, and ovulation was determined by counting ovulated follicles and oviducal eggs.

Table 1. Blockage of ovulation by barbital treatment 2 days after administration of gonadotrophin from pregnant mares' serum. Standard errors are given in column 5.

Time of barbital injection (P.M.)	No. of rats	Rats ovulating		No. of eggs per ovulating rat*
		No.	%	
Control	15	15	100	9.60±0.43
2	19	0	0	
4	11	9	82	8.11±0.73
5	7	7	100	9.57±0.72
6	15	12	80	8.33±0.36

* Control vs. 6 P.M.: $p < .05$ (t-test). Other groups: $p > .05$.

The results are presented in Table 1. Ovulation occurred in all of the controls. Barbital, when given at 2 P.M., blocked ovulation, but when it was given at 4 P.M. or later, ovulation occurred in at least 80 percent of the animals. The number of eggs per ovulating rat was slightly smaller than that in the controls; the difference is statistically significant only for the group treated with barbital at 6 P.M.

We interpret our results to mean that there is a release of ovulating hormone from the pituitary during the period between 2 and 4 P.M. This interpretation is supported by experiments in which ovulation was prevented by hypophysectomy just prior to 2 P.M. on the 32nd day of age but was not prevented by hypophysectomy at 5 P.M. or later. The time of the critical period in our immature rats is the same as that found by Everett *et al.* in adult rats (*2*).

The partial reduction in percentage of rats ovulating in the groups receiving barbital at 4 P.M. or later may have been due to the prolonged depression produced by the high dose of barbital. A lower dose of barbital (20 mg per rat), when given at 2 P.M. to a smaller series of animals, produced consistent blockage. Nembutal (3.6 mg per rat) was also effective in blocking ovulation when injected at 2 P.M. if the individual rats remained unconscious for 2 to 2.5 hours; when given at 4 P.M. the percentage of animals ovulating was similar to that of the controls.

Recently McCormack and Meyer in our laboratory have confirmed, by the use of barbital blockage and hypophysectomy, that a similar critical period exists in serum-gonadotrophin-treated rats 8 days younger than those reported here (*8*).

The mechanism by which gonadotrophin from pregnant mares' serum brings about the release of ovulating hormone is not understood but pre-