is 105 days. Between the tropics of Capricorn and Cancer, there is a zone in which the sun passes through the zenith twice each year at 260- and 105-day intervals. Near the old Maya city of Copán, in Honduras, the fall and spring passages of the sun through zenith take place on August 13 and April 30, respectively. Soon after the sun passes the zenith on its northern passage the rainy season starts. Then there is a lapse of 105 days until the sun again passes the zenith on its way south. Thus the year is divided into a planting and growing period of 105 days and a harvesting and devotional period of 260 days, which may be the origin of the Tonalpohualli.

Regarding Apenes' nomination of Copán as a logical site for the origin of the 260-day calendar, it may be noted that, prior to the early 1960's, the significance of the Izapa, El Baúl, Miraflores, and Esperanza horizons as vehicles for cultural transferral between Olmec and Maya had yet to be realized, and therefore Copán (already the subject of considerable investigation) probably amounted to an "only choice." No doubt we may expect further enlightenment on what surely is one on the world's more intriguing cultures.

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The hypothesis that I advanced for the origin of the 260-day Mesoamerican calendar (*1*) was predicated on two geographic (that is, locational) arguments: (i) that the length of the calendar represents the time interval between zenithal sun positions near the 15th parallel of latitude and (ii) that the choice of faunal symbols used on the calendar strongly suggests a tropical lowland place of origin. I was led to conclude that both of these conditions could only be met by the Late Preclassic site of Izapa in southeastern Mexico.

It is now abundantly clear (from the comments of Henderson and Fitchett) that I was "anticipated" in the first of my arguments by at least four other researchers, beginning with Nuttall in 1928 (*2*). However, it is just as clear that several Mesoamerican scholars [including Thompson (*3*) and Coe (*4*), as well as Henderson] have remained unconvinced of the validity of that argument; for them, the questions of how and where the 260-day calendar originated continue to be—in Coe's words—"an enigma" (*4*, p. 55). Henderson contends that an "argument for a correspondence with some natural phenomenon must be not merely plausible but compelling" (*5*). Yet, nowhere in his own argument does he make any attempt to explain two "coincidences" which lend great support to the astronomical origin of the calendar. The first coincidence is that the zero starting point of the Mayan calendar as calculated by the Goodman-Martinez-Thompson correlation is 12–13 August —the very date on which the 260-day interval between zenithal sun positions begins near the 15th parallel of latitude. The second coincidence is that, of all the places the Mayas could have erected their principal center of astronomical studies, they chose Copán near the 15th parallel of latitude, despite the fact that it lay more than 300 km away from the center of their civilization in Petén. What more compelling arguments does one need to demonstrate the importance of the zenithal sun to Mayan calendrics?

The second of my arguments regarding the faunal symbols used on the calendar is based on an observation of Gadow (*6*), not of Thompson. The fact that Thompson has "recently reversed himself" (*5*) is hardly a cause for discrediting Gadow; it merely suggests that Thompson is now willing to ignore the faunal "evidence" as well as the astronomical and geographic coincidences I mentioned above. Fitchett seems to imply that, if only Apenes had known what we now know about the cultural significance of such places as Izapa, he would probably have "anticipated" me in this argument as well (*7*). However, this misses the point, for the thrust of my argument is that lowland Izapa is situated in an ecological niche that is quite distinct from all the other (highland) sites located along the 15th parallel—a clue to which Apenes presumably was as much privy as I.

Finally, Henderson's plea for greater precision in the use of terminology in Mesoamerican calendrical studies will be seconded by all researchers in the field, providing they can agree on the list of definitions he has provided to start them off.

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Possible Noninhibition of Cellular-Mediated Immunity in Marihuana Smokers

Nahas *et al.* (*1*) report that the T (thymus derived) cell immunity of chronic marihuana smokers is impaired, a finding that would represent a heretofore unrecognized effect of *Cannabis* in humans. However, their results should be interpreted with caution for the following reasons.

It is unfortunate that the authors did not define in precise terms the "eighty-one healthy volunteers . . . used as controls." While it is implicit that these controls were not marihuana smokers, it is essential to know if the controls were subjects who (i) smoked tobacco cigarettes, (ii) did not smoke tobacco cigarettes, or (iii) were a mixed population of (i) and (ii) for the following reason. In an earlier study Vos-Brat and Rümke compared 60 heavy tobacco cigarette smokers with 31 nonsmokers (*2*). They found that the responsiveness of lymphocytes to phytohemagglutinin (PHA) was significantly lower in the smokers than in the nonsmokers. Hence, in the Nahas *et al.* study, if all the normal controls were in fact tobacco cigarette smokers, then the results shown [table 1 in (*1*)] may be considered unequivocal. However, in the absence of such data for the controls, it is not clear whether or not the reduced blastogenic response of the lymphocytes derived from the marihuana smokers was the exclusive result of smoking marihuana as Nahas *et al.* suggest. On the basis of the Vos-Brat and Rümke report (*2*), it would appear that smoking of tobacco as well as of marihuana decreases, in some manner, the response

of lymphocytes to PHA-induced blastogenesis. Moreover, Nahas *et al.* mention the fact that certain abnormalities were found in cultures of human lung explants exposed to marihuana smoke (*3*). Although this was indeed the case, it should also be pointed out that similar lung explants exposed to smoke from ordinary tobacco (Kentucky Standard) displayed essentially the same abnormalities found in the explants exposed to marihuana smoke (*3*).

Nahas *et al.* (*1*) further describe an in vitro inhibitory effect of Δ^9-tetrahydrocannabinol (THC) on the PHA-induced blastogenesis of normal human lymphocytes. This observation should not be taken as final evidence that THC inhibits blastogenesis for the following reasons. It is well known that THC binds to human plasma proteins (*4*). Similarly, it is not unreasonable to assume that THC can bind to protein sites on PHA. If this were to occur, the resulting THC-PHA complex might not be capable of inducing blastogenesis of lymphocytes, thereby leading to false-negative test results. However, no experiments were carried out to detect possible interactions between THC and PHA. If THC does indeed inhibit blastogenesis, this effect should have been demonstrable in the mixed lymphocyte culture (MLC) assay. However, it appears that THC was not tested in the MLC system.

On the basis of the foregoing remarks, further experimental data is needed to demonstrate unequivocally whether or not either or both THC and marihuana smoking inhibit blastogenesis of normal human T lymphocytes.

Note added in proof: In a recent report, Thomas *et al.* (*5*) discuss the phenomenon of impaired immunity observed in humans who smoked tobacco cigarettes as well as in animals that were continuously exposed to tobacco smoke.

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To some of the questions raised by Segelman and Segelman (*1*) we have the following answers:

1) In our study the percentage of tobacco smokers was the same in the control group as in the group smoking marihuana: 30 percent of each group. In addition, the greatest depression in lymphocyte transformation was observed in a 16-year-old high school student who had smoked marihuana, but not tobacco, daily for 2 years. After 2 months of abstaining from marihuana use, the blastogenic response of this subject was close to that of the control group.

2) As we were also aware of the possible interaction of tobacco and marihuana smoking on cellular immune responsiveness, the functional state of peripheral T lymphocytes in 41 healthy staff members 22 to 46 years of age was studied in the same laboratory, 20 of whom were tobacco smokers (average: 20 cigarettes a day) and 21 were nonsmokers. There were no significant differences in the percentages of E-rosette forming cells, mixed lymphocyte culture (MLC), and phytohemagglutinin (PHA) reactivity between smokers and nonsmokers (*2*).

3) Experiments have been carried out to detect possible interaction between Δ^9-tetrahydrocannabinol (THC) and PHA. These two compounds were first incubated together for 5 hours and then added to the cultures. The resulting blastogenic response was similar to that observed when similar doses of PHA and THC were added without prior incubation to the cultures.

4) THC was tested in the MLC system. A dose-related depression of blastogenic response of normal human lymphocytes was observed. This test appeared to be even more sensitive than the PHA test in showing the inhibition of thymidine uptake induced by THC.

5) THC is not the only natural cannabinoid that inhibits in vitro the blastogenesis of lymphocytes: we have now observed that cannabinol and cannabidiol, which were considered inactive, also produce the same dose-related inhibition of lymphocyte transformation as does THC.

6) Our observations indicating that cannabinoids impair nucleic acid synthesis of PHA- or MLC-stimulated human lymphocytes were preceded by three other reports that described in different models of eukaryote cells this basic property of the natural cannabinoids. Besides the study of Leuchtenberger mentioned by the Segelmans one must add the paper by Jakubovic and McGeer (*4*) reporting the inhibition of rat brain protein and nucleic acid synthesis by natural cannabinoids in vitro, and the data of Zimmerman and McClean indicating that THC in 3 to 9 μM concentration inhibits the growth of tetrahymena by reducing RNA and DNA synthesis (*5*).

7) We completely agree with the last paragraph of Segelman and Segelman. These authors, however, fail to present in their critique any direct experimental evidence which would help to answer the questions they raise.

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Possible Noninhibition of Cellular-Mediated Immunity in Marihuana Smokers

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