

**Table I** The body and liver weights for different groups of rats.

Group No. and treatment	No. of rats <sup>a</sup>	Mean initial body weight <sup>b</sup>	Weight at 11 weeks <sup>c</sup>	Final body weight	Total wt. gain	Liver wt.	Relative liver wt. (g/100g weight)
1 DMSO	24 (26)	118.5 ± 5.8	305.8 ± 13.9	483.4 ± 21.3	364.94	18.77 ± 1.95	3.87 ± 0.35
2 DMSO + AFB <sub>1</sub>	25 (26)	117.7 ± 6.7	284.2 ± 21.3	426.7 ± 30.1	309.04	23.90 ± 3.10	5.59 ± 0.71 <sup>f</sup>
3 DMSO + AFB <sub>1</sub> + A10	24 (26)	114.3 ± 7.1	256.5 ± 14.4 <sup>d</sup>	413.2 ± 25.3 <sup>e</sup>	289.89	18.22 ± 1.51	4.34 ± 0.39 <sup>g</sup>

<sup>a</sup> The number in parentheses is the total number of animals at the beginning of the experiment.

Two animals each from Groups 1 and 3, and one animal from Group 2 were excluded, sacrificed, or dead during the course of the experiment for different reasons explained in the text.

<sup>b</sup> Body weight at the time when A10 feeding was started.

<sup>c</sup> Includes AFB<sub>1</sub> dosing period and immediate post-dosing period.

<sup>d,e</sup> The difference in body weight between Groups 2 and 3 is not statistically significant.

<sup>f</sup> The relative liver weight is significantly higher than that of Group 3 ( $p < 0.05$ ).

<sup>g</sup> The relative liver weight in Group 3 is not significantly higher than the DMSO control.

**Table II** The inhibition of hepatocarcinogenesis following Antineoplaston A10 treatment.

Group No. and treatment	Nodules <sup>a</sup>		Tumours <sup>b</sup>		Masses <sup>c</sup>		Total lesions <sup>d</sup>	
	Percentage of rats with lesions	Average No. of lesions per rat	Percentage of rats with lesions	Average No. of lesions per rat	Percentage of rats with lesions	Average No. of lesions per rat	Percentage of rats with lesions	Average No. of lesions per rat
1 DMSO	0	nil	0	nil	0	nil	0	nil
2 AFB <sub>1</sub>	92	3.48	60	1.4	72	0.96	96	5.84 ± 1.35
3 AFB <sub>1</sub> + A10	45	0.54	4.16	0.04	8.32	0.08	54.16	0.67 ± 0.53 <sup>e</sup>

<sup>a</sup> Nodules: macroscopically distinct lesions more than 1 mm but less than 5 mm.

<sup>b</sup> Tumours: lesions larger than 5 mm, but not encroaching more than one-third of a large lobe of the liver or its equivalent.

<sup>c</sup> Masses: several confluent tumours that have encroached greater than one-third of a large lobe of the liver or its equivalent.

<sup>d</sup> Nodules, tumours and masses added together.

<sup>e</sup> The number of neoplastic lesions in Group 3 is significantly less than the control ( $p < 0.001$ ).

large masses, some of them even extending over one-third of one or more major liver lobes (Fig. 3).

Eighty-four percent of the controls had well advanced lesions with nodular or fossilated surface that suggested confluence of the previous discrete lesions. These animals had lost the normal contour of their liver. Hemorrhagic, necrotic, and degenerated cystic areas were present on the cut surfaces of some of the very large masses.

On the other hand, in the A10-fed group, only 54% of the animals had neoplastic lesions in the

liver. Multiple lesions were seen only in 8% and advanced lesions in 12%. In other words, the majority of these tumour-bearing rats had only a single nodule. The inhibitory effect of A10 on hepatocarcinogenesis induced by AFB<sub>1</sub> can be well appreciated by comparing Figs. 3 and 4. In Fig. 4, the liver looks apparently normal and the contour of the liver is well preserved.

The controls given only DMSO (Group 1) had no neoplastic lesions in the liver. Most hepatocellular carcinomas were mixed trabecular and adeno-