



Fig. 2. Relation of Q-factor and frequency calculated from the curves of Fig. 1.

When one plots the data Békésy obtained in human cochlear specimen (2, Fig. 11-49) in a normalized manner with respect to frequency (4), then indeed it might appear as though Johnstone and Boyle were correct. Although there is some nonsystematic spread of individual data, the Q-factor, for the frequency range studied, does not appear to vary with frequency and it has a value of approximately 1.7.

However, Fig. 1 presents the data Békésy obtained in guinea pigs (2, Fig. 12-23) in a normalized manner with respect to frequency and the curve of Johnstone and Boyle (1), also obtained for guinea pigs, is appropriately entered. There is a systematic change of the Q-factor with frequency. For better illustration, the latter change is presented in Fig. 2. The relation follows, in first approximation, a straight-line function, and the value of Johnstone and Boyle, also included in this figure, represents a reasonable extension of Békésy's low-frequency data. [There is a numerical discrepancy between the Q-factors as computed by Johnstone and Boyle from their data (2.5) and that calculated here (3.56). Since we had to take the data from the graph in (1), we are not sure whether the discrepancy is genuine.]

It is gratifying that the new data of Johnstone and Boyle (1) fit those of Békésy (2) rather well. In fact, we find the agreement remarkable.

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Tonndorf and Khanna's method of plotting the tuning curve is interesting, as is their graph of the relationship between Q and frequency. We had also noticed the trend in Q (1). As to the question of absolute values, our statement was that no absolute values had been given for the tuning curves, and this statement is correct as far as we know. We acknowledged that Békésy did present some absolute values, and we quoted them (400 Å at 90 db SPL); however, the relationship of this figure to the tuning curves is obscure.

Our results are in some aspects different from Békésy's, almost certainly because they were measured at a different place and with different frequencies; however, our measurements are in no way inconsistent with his observations.

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Cancerostatic Action of Methylglyoxal

Several years ago, experiments on a cancerostatic agent found in normal tissues (1) indicated that it might be methylglyoxal, a keto-aldehyde, or a derivative thereof (2). Several α -keto-aldehydes were therefore synthesized and studied (3), and were found to have a specific inhibitory effect on cell proliferation by inhibiting protein synthesis (4, 5). Bacteria, germinating seeds, flagellates, fertilized eggs of sea urchins, and cells in tissue culture were used as test material (5). Some of our findings were corroborated by others (6). We found that cancer cells in tissue cultures were more sensitive to methylglyoxal than normal ones were, a finding of possible significance for cancer therapy (3, 7).

Swiss albino mice were injected intraperitoneally with 20 million ascites sarcoma 180 cells, and were then treated with an intraperitoneal injection of methylglyoxal (8) for 9 consecutive days. The mice were divided into four groups of 20 animals each. In the first group, treatment began 1 hour after inoculation; in the second, 4 hours; in the third, 24 hours; and in the fourth,

48 hours after inoculation. Each animal received 18 injections (four 2-mg injections followed by fourteen 1-mg injections) twice daily, 12 hours apart. Control animals received the same volume of physiological saline. The mice were observed for 10 months. In the first group, 15 animals remained free from ascites; in the second group there were 13; in the third group, 7; and in the fourth group, 4. The rest showed varied lengths of survival. All the control animals died in the first 26 to 34 days of the experiment. The cured animals had normal-sized, healthy litters.

Our experiments show that mice inoculated intraperitoneally with sarcoma 180 can be cured by intraperitoneal injections of methylglyoxal.

These experiments, performed in the winter of 1966 to 1967, were not published because we doubted the value of local treatment of cancer, and no therapeutic effect was obtained with solid tumors of sarcoma 180 on intraperitoneal or subcutaneous injection of methylglyoxal. Our experiments were preliminary, and we planned to extend and improve them. We report them here in response to the paper in which Apple and Greenberg (9) described similar results. These authors seem not to have been fully acquainted with our work on methylglyoxal, whose derivative, Kethoxal (Upjohn), is used as a cancerostatic agent.

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8. Commercial methylglyoxal was purified on resin treatment (3), diluted with water to a concentration of 2 mg/0.4 ml. The pH of the solution was adjusted to 6.6. The solution was made isotonic with NaCl and sterilized by Millipore filtration. The possible loss of ascites fluid through the needle wound during treatment was prevented by application of Aero-plast[®] surgical dressing.
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