small number of studies. These results show that aside from the report on accelerated development of spontaneous mammary tumors in mice [Szmigielski et al.1982], exposure to 915 and 2450 MHz promotes accelerated tumor growth in animals only if tumor is first initiated by other means.

Several *in vitro* studies of carcinogenesis at the cellular level, using mouse fibroblast cultures, showed a similar trend of response [Balcer-Kubiczek and Harrison, 1985, 1989, 1991]. It is noteworthy that this series of experiments was originally designed to extend the findings of Szmigielski et al. [1982] and Szudzinski et al., [1982]. They discovered that simultaneous exposure to benzopyrene and modulated microwave radiation did not affect fibroblast transformation. Instead, neoplastic transformation occurred only following combined treatment of microwave and the cancer promoter, TPA. The frequency of neoplastic transformation was SAR dependent. Other reports have shown modest changes in proliferation of human glioma cells following 2-hr of exposure to 2450 MHz at 5 W/kg [Cleary et al., 1990]. Ornithine decarboxylase activity in cultured cells was shown to increase following exposure to lower levels of modulated microwave fields and phorbol-ester tumor promoters [Byus et al., 1988]. Over expression of ODC can lead to neoplastic transformation.

Acute (2-hr) whole body exposure to 2450 MHz microwaves has been reported to produce DNA damage in brain cells of rats [Lai and Singh, 1995,1996]. An increase in both single and double strand DNA breaks was observed 4 hours after exposure to either pulsed (2 microsecond wide, 500 pulse/s) or CW radiation. The average power density and SAR were 2 mW/cm^2 and 1-2 W/kg, respectively. Local SAR in the brain was estimated to be 1-4 W/kg. Brains removed immediately after microwave exposure did not show any increase in DNA strand breaks. CW and pulse operation, likewise, did not reveal any difference. The authors indicated that a significant change in body temperature was not expected, but not measured. That no difference was seen for CW or pulsed radiation seems to suggest an averaging effect such as heating, albeit at a low level over an extended time period (2-hr). Nevertheless, the authors speculated that the findings could result from a direct effect of microwave field on DNA molecule and/or impairment of DNA damage repair mechanisms in brain cells. Since DNA damages, in particular, strand breaks play a role in carcinogenesis, these observations could become important clues to the carcinogenic potential of low level RF and microwave radiation.

It should be noted that the studies by Szmigielski et al.,[1982], Szudzinski et al., [1982], and Wu et al., [1994] used relatively high SARs (6-8 and 10-12 W/kg). SARs at these magnitude are known to induce appreciable temperature increases in the animal body. Since chemical action is facilitated by thermal energy, microwave induced heating could have influenced the action of such chemical agents as benzopyrene and tetradecanoylphorbol-13-acetate. However, the thermal enhancement apparently did not have any influence on the action of dimethylhydrazine (DMH).

The investigation by Chou et al. [1992] was designed to study effects of pulsed microwave exposure on a large number of animals throughout their life-spans and with special emphasis on general health status and longevity. Beginning at eight weeks of age, Sprague-Dawley rats were irradiated by pulsed microwaves (10-microsecond square pulses modulated at 8 Hz and pulsed at 800 pps) for 25 months. A statistically significant increase was observed in primary malignancies (Cortical, c-cell, basal cell, auditory, squamous cell, and pituitary carcinoma, hemangiosarcoma, malignant and lymphocytic lymphoma, myelomonocytic leukemia, liposarcoma, and fibrosarcoma.) at death in irradiated rats (18) vs. sham-irradiated controls (5). It is noteworthy that the life-long, continuous exposure did not reveal any