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Summary Flyer to Alert Physicians to the Current Herbicide Orange Debate

As a result of recent television and newspaper publicity on Herbicide Orange, Air Force physicians may be asked by their patients to respond to statements made by the media and to investigate symptoms described in the media publicity. The programs and articles resulted from a number of claims filed by veterans with the Veterans Administration alleging that present physical disorders are related to exposure to Herbicide Orange. The following information is intended to aid the physician in managing an active duty patient should the individual present questions or allege symptoms.

Herbicide Orange (frequently referred to by the media as Agent Orange) was an equal mixture of two herbicides; 2, 4, 5-Trichlorophenoxyacetic acid (2, 4, 5-T) and 2, 4-Dichlorophenoxyacetic acid (2, 4-D). They have been used worldwide in agriculture since 1947. Human health hazards associated with the use of these herbicides have been minimal and primarily related to self-ingestion or gross misuse. Herbicide Orange was used extensively in Southeast Asia from 1962 to 1969 to defoliate jungle vegetation and deprive the enemy of a refuge. In most instances, military personnel did not enter treated areas until 4-6 weeks after treatment, thus greatly minimizing their chance of contracting or inhaling the herbicide. Air Force personnel most likely to have been exposed were those involved in actual dedrumming and spraying operations.

A contaminant in the mixture was a substance called 2, 3, 7, 8-Tetrachlorodibenzo-p-dioxin (TCDD, Dioxin). This highly toxic contaminant was present in parts per million amounts in most batches of the herbicide. Symptoms described by the media have been documented only in industrial

accidents or gross overexposure. Immediate symptoms such as headache, eye and skin irritation are the result of the herbicide per se and not dioxin (TCDD). These symptons clear upon removal from exposure. Central and peripheral neuropathy, along with skeletal muscle damage have been described in very high exposure to 2, 4-D alone. Other symptoms are believed to be related to the dioxin (TCDD). Included are an acneiform rash with hyperpigmentation, muscular pain, hepatotoxicity, nephrotoxicity, porphyria cutanea tarda, hirsutism, peripheral neuropathy, hyperlipidemia and constitutional symptoms such as anorexia, fatigue, lassitude, mental depression, and loss of libido. There is no substantiated evidence for carcinogencity, teratogenicity, mutagenicity or fetotoxicity in man.

It is emphasized, however, that in all cases displaying the symptoms mentioned above, the estimated doses of TCDD received were much greater than would be expected in personnel routinely working with Herbicide Orange. With the exception of a few persistent cases of acne, there is no evidence for sustained illness except where permanent organ damage had occurred. Furthermore, there is no documentation of the development of adverse effects more than a few months after expsoure had ceased.

Personnel describing any of the above symptoms should be reassured that it is unlikely that their present symptoms are related to Herbicide Orange. They should be thoroughly evaluated to identify the etiology of the symptoms and treated accordingly.

Further information, if needed, may be obtained by contacting Lt Col John Calcagni, USAF, MC, at the USAF Occupational and Environmental Health Laboratory, Brooks AFB, Texas. AUTOVON 240-2001, Commercial 512, 536-2001.