DIPYRIDAMOLE (CURANTYL^{R}, ANTISTENOCARDIN^{R}) FAILED TO INDUCE INTERFERON (IFN) IN MAN

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In the recent publication dipyridamole was described as an antiviral agent both *in vitro* (7) and *in vivo* (*5*). Later on it was found that dipyridamole can act also as IFN inducer in vitro, in mice as well as in man (*1*, *2*, *3*). Curantyl (GERMED, G.D.R.) as well as Antistenocardin (Pharmachim, B.P.R.) were administered per os in a single dosage of 100 mg or four times of 25 mg over 2 hrs at one day, respectively. Sera have been collected before the compound was given and 24 and 48 hrs after the treatment. The investigations have been started with 55 persons consulting a policlinic (lapidaty infects) and supplemented by 24 healthy volunteers from the Institute.

The testing of the interferon level was performed in microtiter plates (Lindbro) on human embryonal diploid cells with vesicular-stomatitis virus (VSV) as challenge virus by evaluation of the 50 % inhibition of the cytopathic effect (*6*). After incubation for 20 hrs of diluted sera with the IFN-sensitive cells and their washing before challenge with VSV, it was absolutely impossible to confirm the induction of any kind of interferon by dipyridamole. In few sera from six persons low tires of IFN (10—38 IU/ml) have been found but these were not induced by dipyridamole. When the sera weren't washed before virus challenge then it was possible to detect an antiviral activity in 66 % of the investigated sera. This nonspecific activity has been assayed in dependence on the presence of the (diluted) sera and has been measured as IFN-like units in a level of maximally 1000 U/ml. The appearance of the nonspecific antiviral activities were obviously not in connection with the administration of dipyridamole.

Incubation experiments showed that these sera acted directly against cell-free virus.

So far, we failed to confirm the IFN-inducing activity of dipyridamole in any of cases studied. This seems to be in agreement with results of others recently published (*4*).

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