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Role of dimethyl sulfoxide in prostaglandin-thromboxane and platelet systems after cerebral ischemia

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Abstract

Direct and empirical evidence indicates that intravenous administration of DMSO can arrest or reverse cerebral and extracerebral ischemia following experimental or clinical injury. When the delivery of oxygen and nutrients to the tissue is deficient or unavailable (as in ischemia), cell damage or death with all its attending pathological consequences becomes an end-point. In the brain, this equates to build-up of intracranial pressure, impairment of neural transmission to vital centers, and loss of function or death. We have reviewed a number of studies that show the usefulness of DMSO in preventing significant pathology from developing in various experimental injury models and in clinical subjects. We have proposed that the action of DMSO in biochemical, morphological, and functional subsystems is not specific but rather interactive. Figure 5 illustrates this point. Any one effect by DMSO on these subsystems that does not affect the others seems highly unlikely. How DMSO or similar drugs affect these systems, could provide important clues in clarifying the pathogenesis of ischemia and in reducing its severity. We conclude from the available evidence that ischemic injury is a dynamic process constantly promoting biochemical, vascular, and morphological changes and that DMSO is able to intervene at various levels of this pathochemical cascade. **DMSO may do this by its ability to normalize tissue perfusion when this is lacking or impaired. We speculate that this effect by DMSO is predominantly on the PG-TX and platelet systems, since these appear to be the most important candidates implicated in vessel occlusion and spasm.** It is further concluded from the available and theoretical evidence presented here, that clinical trials using DMSO in cerebral and extracerebral organ ischemia should be designed in order to evaluate the efficacy of this compound to other antithrombogenic therapies. It is reasonable to assume that DMSO may provide a primary approach to the treatment of cerebral, myocardial, renal, and platelet-induced ischemic disorders.

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