Prostaglandins are not essential in experimental fever of rats

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Milton & Wendlandt (1970) hypothesised that prostaglandins (PG's) may act as mediators in the development of fever. It is difficult to evaluate this hypothesis, because not only PG's but also other biologically active metabolites from essential fatty acids (EFA) may be involved. To assess the importance of PG's and other EFA derivatives in the fever reaction, EFA deficient rats were treated with yeast and their response compared to normal controls of the same strain. Bonta, Bult, Vincent & Zijlstra (1977) described the characteristics of these EFA deficient rats. They were markedly deficient in bishomo-γ-lineoleic and arachidonic acids. Fever was produced by subcutaneous injection of activated yeast (Niemegeers, Leneaerts & Janssen, 1975) and body temperature was measured every half hour for 6 hours, at an ambient temperature of 27 ± 1°C.

EFA deficient rats as well as controls showed a complex temperature response to yeast (Fig. 1), but control and EFA deficient rats responded identically. The changes in body temperature 2 and 6 h after injection were different from saline controls ($P < 0.005$). It seems that a marked deficiency of prostaglandins or their precursors does not impair the fever reaction. The results described need corroboration from results in other animal species. The rat had to be used as it is the only animal species at present that has been made fatty acid deficient. However in order to confirm this hypothesis it will be necessary to demonstrate that prostaglandins do not occur in the CNS of the EFA deficient rats.

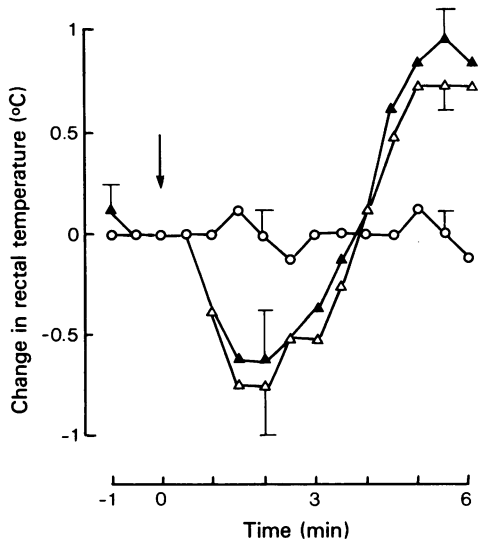

Figure 1 Effect of s.c. injection of saline or yeast on rectal temperature. (○) saline in EFA deficient rats (n = 6), (△) yeast in EFA deficient rats (n = 14). (▲) yeast in normal rats (n = 13). Vertical lines are the standard error.

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Effects of prostaglandin synthetase inhibition on natriuresis induced by diuretics and sodium loading in the rat

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Olsen (1975) suggested that prostaglandins have a role in the natriuretic response to high efficacy diuretics since indomethacin reduced the effect of bumetanide in dogs. Indomethacin pretreatment also reduced frusemide-induced natriuresis in anaesthetised rabbits (Oliw, Kövér, Larsson & Änggård, 1976) and in normal and hypertensive man (Patak, Mookerjee, Bentzel, Hysert, Babej & Lee, 1975). We have extended these investigations by studying the effects of oral pretreatment with indomethacin on the responses to four types of diuretic and to oral sodium loads in female conscious rats.