from the 3 strains were observed. However, females of all the 3 strains had consistently higher protein contents.
Differences in the protein contents of diapausing and non-diapausing larvae have been reported within the same strain of pink bollworm[6,11]. Lower protein contents in diapausing larvae was explained by its probable convertion to fat[11]. Since the pink bollworm larvae do not show a uniform diapause intensity[7,8], the present comparison of a diapause and a non-diapause strain yields more meaningful results than diapause and non-diapause larvae from the same strain. The comparison of the IS and NAS strains shows that reduction of the protein concentration is not necessary for the induction of diapause.

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Evidence that the prostaglandin-like substances from *Propionibacterium acnes* are not identical with PGE_2

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Summary. The prostaglandin-like substances (PLS) isolated from *P. acnes* were investigated by reversed phase chromatography and gas chromatography-mass spectrometry. These analyses demonstrated that PLS were not identical with PGE_2, which supports a concept of PLS as a potential mediator of the inflammatory process in acne vulgaris.

The involvement of *Propionibacterium acnes* metabolites as mediators of inflammation has received increased attention during recent years. Our studies on *P. acnes* lipids, were to characterize the properties of the prostaglandin-like substances (PLS) found, as potent biologically active compound(s), in the lipid fraction of these bacteria[1]. Bioassays on gerbil colon[2] and isolated human vessels[3] revealed that PLS mimic the effects of prostaglandins of the E-type. Similarly, in vivo (hamster cheek pouch), these compounds induced a PGE-like microvascular response[4]. In addition, PLS raised cyclic AMP levels (rat ovary) approximately 2-fold[5]. On silicic acid chromatography PLS is eluted with the same solvent mixture as E-prostaglandins (i.e. with ethyl acetate:toluene 6:4, v/v). Therefore, we decided to investigate whether PGE_2 could be a constituent of PLS.
P. acnes were cultured anaerobically or aerobically for 1 week on a solid, artificial substrate. The recovery of the bacterial mass as well as the extraction procedure was reported earlier[1]. About 1 µg of (3,3,4,4-$^2H_4$)–(17,18$^3H_2$)-labelled PGE_2 was added to the resulting lipid extract. (These carrier and tracer molecules will co-chromatograph with a possible PGE_2 generated by the bacteria.) The mixture was further purified and analyzed by gas chromatography-mass spectrometry using the accelerating voltage alternator technique[6,7]. This analysis demonstrated that less than 1 ng of PGE_2 (= lower limit of safe detection) could be present in the bacterial extract. However, the biological activity of this sample corresponded to about 250 ng of PGE_2 equivalent in a gerbil colon bioassay.
In another experiment a sample (biological activity approximately 640 ng PGE_2 equivalent, gerbil colon bioassay) was subjected to reversed phase partition chromatography together with 50,000 dpm of 17,18-$^3H_2$-PGE_2 (50 mCi/µmole) as described earlier[8]. No biological activity could be detected (gerbil colon bioassay) in those fractions where tritium-labelled PGE_2 appeared.

Thus, the present work demonstrates that PLS from *P. acnes,* despite their biological resemblance to E-prostaglandins, do not contain PGE_2. This corresponds with a recent bioassay experiment on the human utero-tubal junction, where the effect of PLS was similar, although not identical with that of PGE_2[9]. Moreover, PLS possess potent chemotactic properties, which was not the case for PGE_2[10]. According to a recent suggestion[11], mediator functions may be associated with other prostaglandin-type compounds (e.g. endoperoxides), while modulator functions are associated with the end-products, i.e. prostaglandins themselves. This idea supports the concept of PLS in *P. acnes* as a potential inflammatory mediator, involved in the pathophysiology of acne vulgaris.

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