Introduction

William J. Cunliffe, Leeds

There are many oral, topical and physical treatments for acne, and therefore most patients should respond well, but some treatments have side-effects. These include a primary irritant dermatitis from many topical therapies [1] or other unpleasant significant adverse effects [2, 3], e.g. common problems such as abdominal discomfort, vaginal candidiasis and the occasional drug rash. More rarely, for example with minocycline, serious systemic side-effects can be a major problem including lupus erythematosus and benign intracranial hypertension [4, 5].

There are several other reasons for poor response [6]. It has been shown that with some topical therapies compliance is only of the order of 45% over a 4-month period [6]. Some acne types also respond badly to the more conventional therapies. For example, patients with sandpaper acne and macrocomedones are a group of poor responders, as is significant inflammatory disease, for example patients with sinus tract disease [7, 8]. There are also increasing problems with *Propionibacterium acnes*

resistance, which can be detected in 60% of patients by laboratory testing but is of likely clinical significance in about 20% [9].

Therefore there still remains a need for the development of new therapies. These are likely to be most beneficial if they have a mechanism of action against 2 or more of the 4 aetiological factors of acne: androgen-driven seborrhoea, comedone formation, colonisation of a duct with *P. acnes* and the production of inflammation [10–13].

This publication discusses the development, mechanism of action and clinical trials on Diacnéal®, which is a combination of a topical retinoid retinaldehyde and glycolic acid (RALGA). The drug could well be expected, based on its known mechanism of action, to reduce comedones, reduce *P. acnes* without the risk of *P. acnes* resistance, and also reduce inflammation. It may also be expected to have some effect on reducing post-inflammatory pigmentation and scarring. Obviously much work is required before the true outcomes are known.

References

- 1 Stern RS: Acne therapy: Medication use and sources of care in office-based practice. *Arch Dermatol* 1996;132:776–780.
- 2 Minkin W: Photo-onycholysis due to tetracycline hydrochloride and doxycycline. *Arch Dermatol* 1971;103:520–521.
- 3 Okada N, Moriya K, Nishida K, et al: Skin pigmentation associated with minocycline therapy. *Br J Dermatol* 1989;121:247–254.
- 4 Meacock DJ, Hewer RL: Tetracycline and benign intracranial hypertension (letter). *Br Med J* 1981;282:1240.
- 5 Gough A, Chapman S, Wagstaff K, et al: Minocycline induced autoimmune hepatitis and systemic lupus erythematosus-like syndrome. *Br Med J* 1996;312:169–172.
- 6 Zaghloul SS, Cunliffe WJ, Goodfield MJD: Compliance in acne is highly correlated to psychological well-being and self preservation. *Br J Dermatol* 2002;147:1084–1091.
- 7 Cunliffe WJ, Holland DB, Clark SM, Stables GI: Comedogenesis: Some new aetiological, clinical and therapeutic strategies. *Dermatology* 2003;206:11–16.
- 8 Jansen T, Lindner A, Plewig G: Draining sinus in acne and rosacea: A clinical, histopathological and experimental study. *Hautarzt* 1995;46:417–420.
- 9 Coates P, Vyakarnam S, Eady EA, et al: Prevalence of antibiotic resistant propionibacteria on the skin of acne patients: A ten-year surveillance data and snapshot distribution and study. *Br J Dermatol* 2002;146:840–848.
- 10 Burton JL, Shuster S: The relationship between seborrhoea and acne vulgaris. *Br J Dermatol* 1971;84:600–601.
- 11 Knaggs HE, Holland DB, Morris C, et al: Quantification of cellular proliferation in acne using the monoclonal antibody Ki-67. *J Soc Invest Dermatol* 1994;102:89–92.
- 12 Leyden JJ: The evolving role of *Propionibacterium acnes* in acne. *Semin Cutan Med Surg* 2001;20:139–143.
- 13 Norris JF, Cunliffe WJ: A histological and immunocytochemical study of early acne lesions. *Br J Dermatol* 1988;118:651–659.