Experimental contact sensitivity: a model for both antigen (hapten)-specific and innate immune mechanisms

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ABSTRACT

In the present study, experimental model of contact sensitivity to dinitrochlorobenzene (DNCB) in inbred AO rats was employed to determine both antigen (hapten)-specific as well as parameters of antigen-non-specific aspects of contact hypersensitivity (CHS) response. Following local epicutaneous application of 2% DNCB, increased spontaneous, hapten-stimulated and interleukin (IL)-2-driven draining lymph node cell (DLNC) proliferation was detected, reflecting the antigen (hapten)-specific aspect of CHS. Changes in the ratio of lymphocyte subsets were noted in DLNC in the sensitization phase of CHS. At the dose of DNCB employed for the elicitation of CHS in sensitized animals, increased activity of peripheral blood granulocytes (including activation, adhesion and cell survival) was detected. Collectively, these data demonstrate the activation of both antigen (hapten)-specific and innate immunity in contact hypersensitivity. The importance of studying both mechanisms in this same model has been discussed.

KEY WORDS

contact hypersensitivity, DNCB, lymphocytes, granulocytes, rat

Introduction

Skin responds to toxic agents by various inflammatory/immunologic cascades of events leading to the induction of allergic or irritant contact dermatitis or hyperkeratosis (1). A commonly used model in testing the animal's or human's ability to mount a cutaneous immune response is contact hypersensitivity (CHS) reaction to skin reactive chemicals, haptens. The afferent phase of the CHS response, the sensitization phase, is initiated by epicutaneous application of the hapten to the dorsal or abdominal skin, and it is characterized by the activation and division of hapten-specific T lymphocytes in the regional lymph nodes and the appearance of effector cells in the lymph nodes and spleen (2). In the efferent phase, which follows a subsequent challenge with hapten, e.g. epicutaneous application of the sensitizer to the skin of the ear, primed T lymphocytes are recruited to the site of challenge where they produce a variety of inflammatory mediators, amplifying a background inflammatory response into a more vigorous process. It is the classical manifestation of con-