A new algorithm for predicting risk of hip fracture in postmenopausal women

Identifying the factors that might lead to bone fractures can help physicians to suggest preventive lifestyle changes for at-risk patients. To create a predictive algorithm, Robbins *et al.* evaluated the clinical risk factors for hip fracture within 5 years in postmenopausal women.

In an observational, multiethnic cohort of the Women's Health Initiative (WHI) consisting of 93,676 women, risk of hip fracture was assessed by questionnaires over a mean of 7.6 years. A prediction model based on findings from the observational cohort was created, and validated in 68,132 women enrolled in the clinical trial of the WHI, with a mean follow-up of 8.0 years. The authors developed a simplified point score to identify the probability of hip fracture.

Eleven factors selected from the observational study predicted hip fracture within 5 years: age, self-reported health, weight, height, race/ethnicity, self-reported physical activity, history of fracture after the age of 55 years, parental hip fracture, current smoking, current corticosteroid use and diabetes treated with medications. During the observational study 1,132 hip fractures occurred, and 791 hip fractures were reported during the clinical trial. In the clinical trial group, most hip fractures occurred in women predicted to be at low risk by the algorithm.

Age alone is the best predictor of hip fracture; however, this study demonstrated that prediction can be enhanced with the addition of other factors.

Original article Robbins J *et al.* (2007) Factors associated with 5-year risk of hip fracture in postmenopausal women. *JAMA* **298:**2389–2398

The CD40/CD40L system might have a role in inducing skin lesions in patients with SCLE

Subacute cutaneous lupus erythematosus (SCLE), an autoimmune-mediated disease that is characterized by widespread hardening of the skin, is triggered by various environmental factors, including ultraviolet light. The CD40/CD40 ligand (CD40L) costimulatory system, which amplifies immune responses and can induce inflammation, is believed to have a role in skin manifestations of other autoimmune diseases, for example discoid lupus erythematosus (DLE). Caproni and colleagues, therefore, studied the expression of CD40 and CD40L in skin lesions of patients with SCLE.

Immunohistochemical staining for CD4, CD40 and CD40L was performed on lesional and healthy, sun-protected skin biopsy samples obtained from six female patients with SCLE. Biopsy samples were also obtained from patients with DLE ($n=5$), dermatomyositis ($n=5$), lichen planus ($n=3$), erythema multiforme ($n=2$), and five healthy volunteers.

$CD40^+$ and $CD40L^+$ cells infiltrated the perivascular, periadnexal and interstitial dermis in all lesional biopsy samples; considerably fewer $CD40^+$ and $CD40L^+$ cells were detected in sun-protected skin, with $CD40L^+$ cells absent from the biopsy material from healthy volunteers. Skin from patients with DLE showed the most intense CD40 and CD40L staining, and staining in SCLE skin was comparable with that observed in patients with other diseases.

The authors conclude that the CD40/CD40L system might have a substantial role in inducing SCLE skin lesions. The finding of increased CD40 and CD40L expression in sun-protected skin, albeit at lower rates than in lesional skin, might suggest that CD40/CD40L activation is, in part, independent of UV light in patients with SCLE.

Original article Caproni M *et al.* (2007) The CD40/C40 ligand system in the skin of patients with subacute cutaneous lupus erythematosus. *J Rheumatol* **34:** 2412–2416

The cellular interplay that underlies the synovial inflammatory process in RA

Results from animal models of rheumatoid arthritis (RA) suggest that T-helper 17 (T_H17) cells, $CD4^+$ T cells that secrete interleukin 17, have a key role in the progression of the disease. To understand the process more fully, Hirota *et al*. searched for cell-surface molecules specifically expressed by T_H17 cells to identify which molecules might be responsible for their migration to joint tissue.

Using the SKG mouse strain, which spontaneously develops a T-cell-mediated autoimmune arthritis similar to human RA, the researchers showed that the CC chemokine