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Review

What is known about the mechanisms of dietary influences in Crohn's disease?

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ABSTRACT

Much has been written about the role of diet and risk for Crohn's disease (CD). However, the evidence is contradictory. Recent evidence has pointed to fiber playing an important role along with the possibility that dietary fat and overnutrition also have a role. Diet has a clearer place in disease modification, with some diets used in the treatment of CD. The lack of clarity stems from a poor understanding of the mechanisms underlying the relationship between diet and CD. Gut permeability is likely to play a key role in the risk for CD. Mechanisms whereby diet can affect gut permeability, including the effects of the gut microbiota, are reviewed. Modification of disease behavior is likely to be influenced by additional mechanisms, including recognition of complex food antigens. As with many other chronic diseases, a surrogate marker of CD risk would greatly aid evaluation of the dietary factors involved. Formal measures of gut permeability are too cumbersome for large-scale use, but fecal calprotectin may be a convenient measure of this. There are only preliminary data on the effect of diet and microbiota composition on fecal calprotectin and these require further investigation.

Introduction

More than 250,000 individuals in the United Kingdom are affected by inflammatory bowel disease (IBD) and approximately 10,000 new cases are diagnosed every year. IBD comprises predominately two conditions: ulcerative colitis (UC) and Crohn's disease (CD). CD is a chronic inflammatory disease of the gastrointestinal (GI) tract that can affect anywhere from the mouth to the anus. Individuals with this condition often experience periods of symptomatic relapse and remission.

Although the etiology of IBD is not completely understood, both UC and CD are thought to occur through a combination of genetic, environmental, and immunologic factors. The current thinking is that the intestinal flora drives an unmitigated intestinal immune response and inflammation in a genetically susceptible host, although the precise nature of this remains to be elucidated [1,2].

These diseases were traditionally limited to the Western industrialized world, with a high incidence and prevelance in western Europe and North America [3]. Over the past decade, however, they are becoming more common in countries that have adopted a Western lifestyle [4] (Fig. 1). Additionally increasing numbers of first- and second-generation migrants to these Western countries are being diagnosed [5,6]. The reason for this is uncertain, however, diet is believed to play a key role in the pathogenesis.

Increasing evidence supports the role of diet in the pathogenesis of CD, particularly with regard to dietary fiber intake, support for which has come from powerful prospective studies. Also, emerging studies suggest a role of overnutrition or obesity in the pathognesis of certain forms of CD.

Genetic studies have identified more than 160 risk loci associated with IBD, most of which are asscoiated with both UC and CD [7]. Many of these are involved in gut epithelial barrier function and immune responses to foreign microbes and antigens. We will review the evidence to date that diet and, in particular the particular components of diet, play a role in CD pathogenesis and disease modification. Then we will explore the

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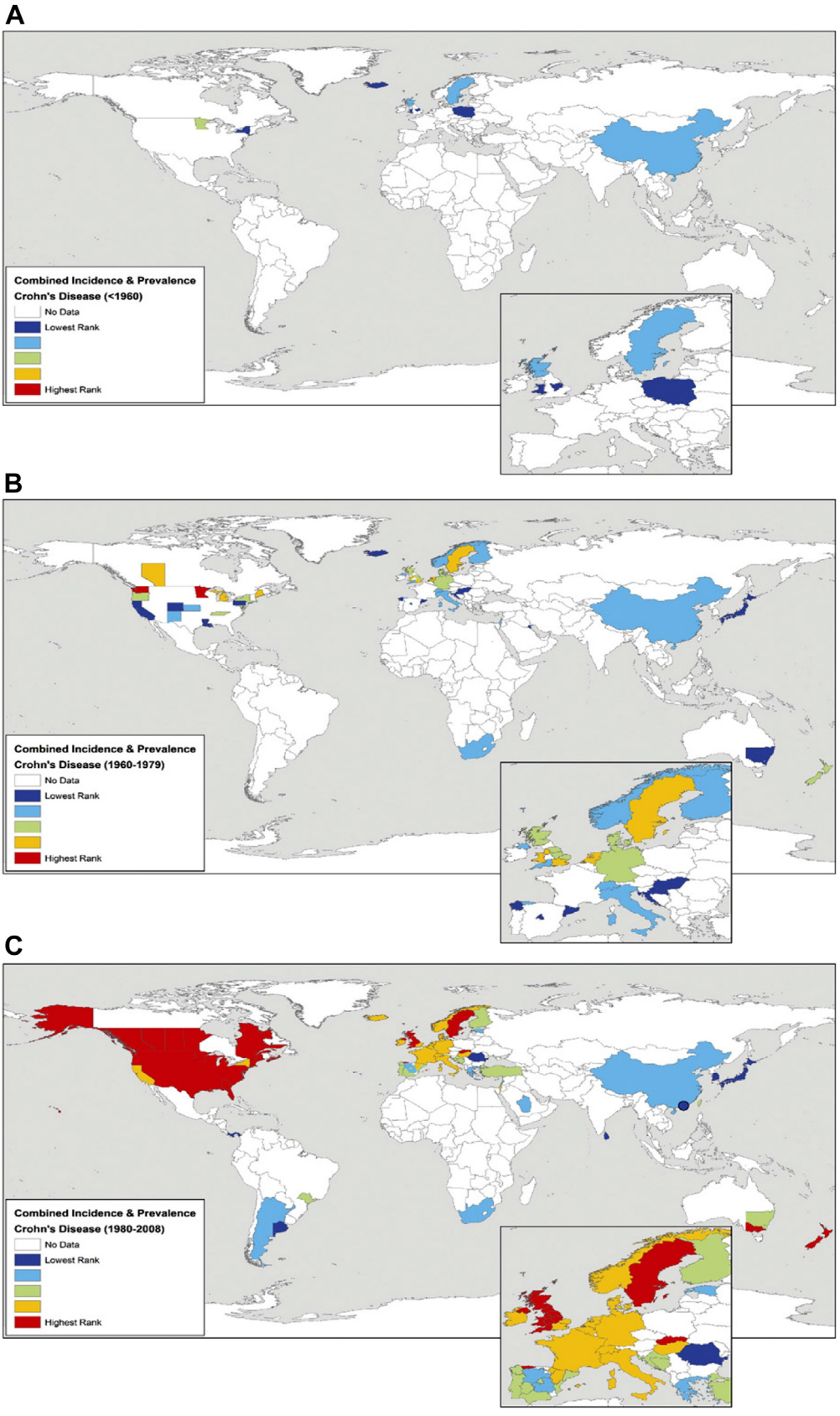

Fig. 1. Worldwide Crohn's disease incidence rates and/or prevalence for countries reporting data (A) before 1960, (B) from 1960 to 1979, and (C) after 1980. Incidence and prevalence values were ranked into quintiles representing low (dark and light blue) to intermediate (green) to high (yellow and red) occurrence of disease. Reprinted reference 3; Copyright 2012 by Elsevier. Reprinted with permission. (The color version of this figure is available online at www.nutritionjrnl.com.)