Development of Guillain-Barré Syndrome following *Campylobacter* Infection

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Guillain-Barré syndrome (GBS) is a disorder of peripheral nerves and is characterized by ascending paralysis. Since the decline in the number of polio cases, GBS is the most common cause of acute neuromuscular paralysis, affecting 1 to 2 persons/100,000 population each year in many parts of the world. Summertime outbreaks of GBS with primarily axonal involvement (acute motor axonal neuropathy) are well described in northern China and also have been reported in Mexico, Spain, and Korea. Although it affects both sexes of any age, GBS is more common among males, and the incidence increases with age. Symptoms often begin with weakness and dysesthesias in the legs, which subsequently spread to the arms and upper body. While most patients (~70%) recover completely, severe irreversible neurologic damage, the need for ventilatory assistance, and death can result. Thus, GBS is a clinically important disorder in both developed and developing countries, but until recently, little was known about its etiology.

In about two-thirds of the cases, GBS is preceded by several days or weeks by an acute respiratory or intestinal infection. Although upper respiratory illnesses are the most commonly described preceding symptoms, the microorganism most frequently identified in patients with GBS is *Campylobacter jejuni*. *C. jejuni* is the leading recognized cause of bacterial gastroenteritis in the developed world and often is acquired by ingestion of contaminated poultry products. In developing countries, *C. jejuni* infection is an important cause of mortality due to diarrhea in young children. It has been hypothesized that GBS may arise as a result of the production of antibodies to *C. jejuni* lipopolysaccharides that, due to molecular mimicry, cross-react with gangliosides or other structures present in peripheral nerves. The ganglioside GM1 may be one target for these antibodies, although other gangliosides also may be involved. Antibody or cell-mediated immune responses (or both) are believed to produce degeneration of the nerve or interruption of neurotransmission. Research concerning the etiopathogenesis of GBS is currently a highly active area.

The purpose of the workshop was to explore the relationship between *C. jejuni* infection and the development of GBS. Scientists studying this problem from around the world were invited to share their latest findings. The disciplines of microbiology, neurology, cell biology, epidemiology, economics, immunology, and genetics were represented. Topics for discussion included the identification of the surface antigens of *C. jejuni* linked to GBS, the pathology of GBS, the identification of the target molecules on nerves attacked by anti-bacterial antibodies, animal models for GBS, possible interventions to prevent the development of GBS or its serious sequelae, the economic impact of GBS, and the implications of these findings for the development of a vaccine against *C. jejuni*.

The broad base of organizational and financial support for this workshop reflects both the importance of the clinical problem and the opportunities for advancing our understanding of the biomedical implications of the relationships between *C. jejuni* infections, immune responses, and neural tissue injury.