The white and golden potato cyst nematodes (PCN) are the most economically important pests of potato in the UK [3]. Potato cyst nematodes originated in the Andes, where they coevolved with the potato. The earliest reports of PCN in the UK were in 1913. PCN were reported increasingly in the UK from 1920 onwards. In 1946, 20% of the potato acreage was estimated to be infected while surveys in 1996 and 2002 determined that 42% and 64% of the potato fields were infested, indicating a significant recent increase in the problem [2]. It is probable that a substantial portion of the remainder are “incubating” infestations too small to detect [4].

Nematodes are round microscopic (.004-.04 inches in length) unsegmented worms (not true worms). Roots of infected plants contain minute, white bodies of female nematodes that have erupted through the roots. When a female dies, its cuticle forms a protective cyst containing 200 to 500 eggs [1]. Cysts containing viable eggs can persist in the soil for up to twenty years. When potatoes are planted, root exudates stimulate 60 to 80% of the juveniles to emerge from eggs. The juveniles locate and enter roots. They cut through cell walls and feed. After a final molt, the adult males emerge through the root. The females rupture the root so that their bodies protrude. They release a substance that attracts males, which mate with them repeatedly [1]. Embryos develop inside eggs into juveniles; the females detach from the roots. Infection reduces root biomass, which can lead to stunting of plants, yellowing and wilting of foliage and small tubers. Heavy infestations often results in total crop loss [1].

In the UK, farmers traditionally follow an integrated approach to control PCN, which involves the use of nematicides, crop rotation and resistant cultivars. The first British potato cultivar with resistance to the golden nematode was released in 1966 and has been widely-planted [2]. Repeated use of cultivars with golden nematode resistance has led to selection for the white nematode, for which potato varieties have only partial resistance and are not varieties in wide demand from the market [2]. In principle, PCN can be effectively managed by only growing potatoes on infested land in very long rotations. PCN populations can be held at levels that do not require nematicide treatment on rotations of at least 1 in 12 [3]. This is not an economically viable option as it would only permit 3 crops of potatoes on the same land in a 25 year period [3].

Prior to planting potato, UK farmers frequently sample fields to determine the need for a nematicide. Between 1996 and 2002, the number of samples processed by the main laboratories in England increased by 67%, indicating that farmer awareness of PCN has greatly increased [4]. The UK potato area treated with nematicides and fumigants increased from 6800 ha in 1976 to 21000 ha in 1982, increased by 67%, indicating that farmer awareness of PCN has greatly increased [4]. Between 1996 and 2002 determined that 42% and 64% of the potato fields were infested, indicating a significant recent increase in the problem [2]. It is probable that a substantial portion of the remainder are “incubating” infestations too small to detect [4].

Two of the products used for PCN control in the 1990s (aldicarb and the fumigant 1,3-D) have not been listed on Annex 1 and are no longer available to UK potato growers. The hazard trigger criteria adopted by the European Parliament is likely to have the effect of removing from the market the three remaining nematicides currently used in the UK for PCN control [3]. Without the use of nematicides, the typical potato grower with a PCN infested field would have a 60% decline in yield within 10 years [3].

References