DISEASE NOTE

FIRST REPORT OF *GYMNOSPORANGIUM SABINAE* ON CULTIVATED PEAR IN LEBANON

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Gymnosporangium sabinae (Dickson) G. Winter (syn. *G. fuscum* DC.), the causal agent of European pear rust disease, can severely reduce yield of cultivated pear (*Pyrus communis*). This fungus, an obligate parasite that alternates between species of *Juniperus* and *Pyrus* to complete its lifecycle, is heteroecious and causes perennial infections on juniper and annual infections on pear (Laundon, 1975). In October 2011, leaves of cultivated pear trees of cv. Coscia, heavily attacked by a rust were collected in two orchards located, respectively, in Baskinta [Mount Lebanon (33°56'39" N, 35°47'15" E)] and in Basloukite [north Lebanon (34°18'28" N, 35°58'02" E)]. Based on morphologic features (Parmelee, 1971; Laundon, 1975), the pathogen was identified as *Gymnosporangium sabinae*. On the upper side of the leaf, there were irregular, bright orange-red spots, which contained compact conglomerations of spermogonia. These structures were dark-brown to black, hemispherical, protuberant, 3-4 mm in diameter, had many receptive hyphae and produced at maturity a large number of oblong hyaline spores (spermatia) measuring 5.1-7.2×1.2-1.8 µm. On the lower side of the leaf, there were loose conglomerations of horn-like aecia located on protruding tissue outgrowths. From these aecia, pale-brown pseudo-peridia arose as small groups of 2-4 mm-long filaments, composed of irregularly prolate peridial cells with inner and lateral walls extremely thickened, ornamented with cylindrical warts, which produced 24-30×23-28 µm subgloboid, ellipsoid aeciospores, with 4-5.5 mm thick orange-brown walls. Symptoms on surrounding *Juniperus* spp. were not observed. To our knowledge, this is the first report of *G. sabinae* on cultivated pear in Lebanon.

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FIG TREE VIRUSES IN NEW ZEALAND

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In late March 2012, cuttings were collected from 11 fig trees growing in Anaura Beach and in a private garden in the vicinity of Gisborne (Bay of Poverty, New Zealand). Each tree apparently belonged to a different cultivar, whose name was unknown to the owners. Due to the late season, symptoms on the leaves were not outstanding. When visible, they consisted of various patterns of mottling resembling very much the symptoms characterizing fig mosaic (Martelli, 2011). Total RNA was extracted from cortical scrapings and subjected to RT-PCR, which was conducted according to the protocols and using the virus-specific primers routinely utilized in our laboratory (Martelli, 2011 and references therein) for the detection of the following viruses: *Fig mosaic virus* (FMV), Fig leaf mottle-associated virus 1 (FLMaV-1), Fig leaf mottle-associated virus 2 (FLMaV-2), Fig mild mottle virus (FMMV), and Fig latent virus 1 (FLV-1). PCR was also carried out for the presence of Fig badnavirus 1 (FBaV-1), using the primers P1s: 5'-GCT GAT CAC AAG AGG CAT GA-3' and P1as: 5'-TCC TTG TTT CCA CGT TCC TT-3' designed on the sequence of the 5' terminal portion of the polyprotein encoded by the viral ORF3 (GenBank accession No. JF411989). Amplification products were obtained from all samples, most of which proved to be infected by two (e.g. FMV, FBaV-1) or more (e.g. FMV, FBaV-1, FLMaV-1, FMMV) different viruses. FLV-1 was not detected, whereas the most widespread viruses were FMV and FBaV-1. Interestingly, the latter virus was detected in the totality of the samples. Fig mosaic is known to occur in New Zealand (Li and Procter, 1944; Cairns, 2006) but, to the best of our knowledge, there are no records of viruses associated with the disease.

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