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Taro (*Colocasia esculenta*) Transformed with a Wheat Oxalate Oxidase Gene for Improved Resistance to Taro Pathogen *Phytophthora colocasiae*

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***Abstract*. Production of taro [*Colocasia esculenta* (L.) Schott], a tropical root crop, is declining in many areas of the world as a result of the spread of diseases such as Taro leaf blight (TLB). Taro cv. Bun Long was transformed through *Agrobacterium tumefaciens* with the oxalate oxidase (OxO) gene *gf2.8* from wheat (*Triticum aestivum*). Insertion of this gene was confirmed by polymerase chain reaction (PCR) and Southern blot analysis. One independent transformed line contained one gene insertion (g5), whereas a second independent line contained four copies of the gene. Reverse transcriptase PCR (RT-PCR) confirmed the expression of this gene in line g5. Histochemical analysis of the enzyme oxalate oxidase confirmed its activity increased in the leaves of line g5. A bioassay for resistance to TLB used zoospores of *Phytophthora colocasiae* to inoculate tissue-cultured plantlets. Transgenic line g5 showed the complete arrest of this disease; in contrast, the pathogen killed non-transformed plants by 12 days after inoculation. A second bioassay, in which spores of *P. colocasiae* were inoculated onto disks of leaves of one-year-old potted plants, confirmed that transgenic line g5 had greatly increased resistance to this pathogen. This is the first report to demonstrate that genetic transformation of a crop species with an OxO gene could confer increased resistance to a pathogen (*P. colocasiae*) that does not secrete oxalic acid (OA).**

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Taro is a tropical root crop that is grown widely throughout the Pacific, Africa, Asia, the West Indies, and South America (Plucknett et al., 1970). It was the fifth most harvested root crop in the world with production estimated at 9.0 billion kg (Food and Agriculture Organization of the United Nations, 2010). Traditionally, taro is propagated vegetatively from corms or cormels and not from seed as a result of infrequent seed production in commercial cultivars.

Taro corms and cormels are good sources of carbohydrates, and leaves can be eaten as vegetables. They are known to contain abundant levels of both soluble oxalates and insoluble calcium oxalates (Sakai et al., 1984; Sefa-Dedeh et al., 2004). Oxalate is known to function as an antinutrient in insects, animals, and humans, partly as a result of its ability to form complexes with calcium or magnesium, making these minerals unavailable for absorption (Bohn et al., 2004; Franceschi and Nakata, 2005; Palgi et al., 2005). In addition, insoluble calcium oxalate crystals could serve as herbivore feeding deterrents (Korth et al., 2006; Sakai et al., 1984). To reduce its toxicity, taro corms and leaves are well cooked before consumption by humans.

TLB, caused by the oomycete pathogen *Phytophthora colocasiae*, is a major disease that threatens the sustainability of taro worldwide. It reached the Hawaiian Islands during the 1920s and causes yield losses of up to 50% (Trujillo, 1967). During the 1990s, this disease spread to the Samoan Islands and resulted in 95% losses in traditional, TLB-susceptible taro cultivars (Brooks, 2000; Trujillo and Menezes, 1995). In 2004, it invaded the Dominican Republic, infecting 70% to 95% of commercial taro plantings (J. Cho, personal communications, 2009).

OxO was first isolated and characterized from wheat (*Triticum aestivum*) (Lane et al., 1993). It catalyzes the oxidation of oxalic acid by molecular oxygen to form carbon dioxide and hydrogen peroxide (H_2O_2). Researchers have found that increased OxO activity is associated with plant defense systems against a broad range of pathogens including viruses, bacteria, fungi, oomycetes, and nematodes (Dunwell et al., 2008). For example, Schweizer et al. (1999) found that transient expression of the pathogen-induced wheat germin *gf2.8* gene was associated with reduced penetration of the fungus *Blumeria graminis*.

There are several hypotheses to explain the mechanism of OxO in fungal resistance (Lane, 2002). First, H_2O_2 is generated by OxO-mediated breakdown of oxalate, resulting in hypersensitive cell death and antimicrobial activity (Peng and Kuc, 1992). Second, H_2O_2-mediated lignification of cell walls forms effective barriers against fungal penetration (Schweizer et al., 1999). Third, H_2O_2 generated by OxO could have a role in signal transduction cascades that coordinate various defense responses such as the synthesis of pathogenesis-related (PR) proteins and phytoalexins (Hammond-Kosack and Jones, 1996). Fourth, OA-generating fungi such as *Sclerotinia sclerotiorum* secrete high concentrations of OA as a toxin, and OxO is able to break down OA (Donaldson at al., 2001; Liang et al., 2001).

Genetic transformation with OxO genes has been reported in several plant species to improve resistance to pathogenic fungi that secrete OA (Donaldson at al., 2001; Dong et al., 2008). For example, Cober et al. (2003) found that a transgenic soybean line with an inserted wheat OxO gene *gf-2.8* exhibited partial resistance to the white mold disease caused by *S. sclerotiorum*. Peanut (*Arachis hypogaea*) transformed with a barley oxalate oxidase gene showed enhanced resistance to *Sclerotinia minor*, the pathogen that causes Sclerotinia blight of peanut (Livingstone et al., 2005). Hybrid poplar (*Populus* ×*euramericana*) transformed with a wheat OxO gene exhibited enhanced disease resistance against the fungus *Septoria musiva* (Liang et al., 2001). Because OA is a toxin secreted by these pathogenic

fungi, it is not surprising that transgenic plants with an inserted OxO gene exhibited greater disease resistance.

In this study, we report on the *Agrobacterium*-mediated transformation of taro cv. Bun Long with a wheat OxO gene, *gf2.8*. Also, we demonstrated greatly enhanced resistance of transgenic taro plants to the oomycete *Phytophthora colocasiae*. This is the first report of a plant species transformed with an OxO gene that exhibited greater resistance against an oomycete pathogen, *P. colocasiae,* that does not secrete OA.

Materials and Methods

Plant materials. Taro cv. Bun Long was chosen as the plant transformation material, because it is an important TLB-susceptible commercial cultivar in Hawaii. Earlier, we had developed an efficient regeneration and *Agrobacterium*-mediated transformation system for this cultivar (He, 2006; He et al., 2008).

Agrobacterium-mediated transformation. The plasmid pEMBL18:*gf2.8* was provided by Dr. Francois Bernier at I.B.M.P. (Institut de Biologie Moleculaire des Plants) in France. Enzyme digestion followed by PCR analysis and partial sequencing verified that this plasmid contained the intact 2.8-kb *gf2.8* gene including its own promoter and terminator. The promoter for this gene is stimulated by both abiotic stresses (e.g., heavy metals, wounding, and plant growth regulators) and by biotic stresses (e.g., virus, fungus) (Berna and Bernier, 1999).

The intact 2.8kb OxO *gf2.8* gene fragment was extracted from the plasmid pEMBL18:*gf2.8* with the enzyme *Eco*RI and ligated into the *Eco*RI site of the vector pBI121 using standard methods (Sambrook and Russell, 2001). The plasmid pBI121:*gf2.8* contained the *npt*II selection gene, the *gus* reporter gene, and the intact *gf2.8* gene with promoter and terminator (Fig. 1). The plasmid pBI121:*gf2.8* was transformed into the *Agrobacterium tumefaciens* strain EHA105 using the freezing and thawing method described earlier by He et al. (2008). *Agrobacterium*-mediated transformation of taro cv. Bun Long with the OxO *gf2.8* gene was conducted using the method of He et al. (2008).

Verification of presence of the OxO gene using polymerase chain reaction analysis. Genomic DNA was extracted from shoot tissues using the method described by He et al. (2008). The PCR primers specific for *gf2.8* were designed using Clone Manager software (Cary, NC) and based on the *gf2.8* gene sequence in GenBank (Lane et al., 1991). A pair of primers specific for amplifying the 755-bp fragment of the coding region of *gf2.8* was designed: gfF (20 mer) 5′-GCTTA GCAGCAGCAACAACC-3′ and gfR (20 mer) 5′-GCGGCAAACTTGGACTTGAG-3′. The PCR analysis was conducted according to He et al. (2008).

Verification of expression of the OxO gene using reverse transcription–polymerase chain reaction analysis. Total RNA was isolated from fresh shoots and RT using the method described by He et al. (2008). As a control, PCR analysis was also conducted using total RNA without RT to confirm that the PCR product was derived from mRNA and not from the contaminating DNA. The plasmid pBI121:*gf2.8* DNA was used as a positive control.

Southern blot analysis. Genomic DNA was extracted from shoot tissues using the sodium dodecyl sulfate method as modified by He et al. (2008). The DNA blotting, probing, and hybridization methods were described earlier by He et al. (2008).

Verification of OxO activity through the histochemical staining test. Leaves and shoots from one-year-old plants of the transformed g5 taro line or non-transformed cv. Bun Long were analyzed for OxO activity based on a histochemical assay (Dumas et al., 1995). Briefly, leaves and shoots were sliced and immersed in 10 mg·L^{-1} 1-naphthaleneacetic acid (NAA) solution for 2 h to induce gene expression because the promoter of *gf2.8* is induced by this hormone. Then, sliced leaves and shoots were placed in a solution containing oxalic acid (2.5 mM) and 4-chloro-1-naphthol (0.6 g·L^{-1}) as the staining reagent. The incubation was conducted at 25 °C in the dark for 24 h. The OxO converts oxalic acid to H_2O_2 that endogenous peroxidases use to oxidize 4-chloro-1-naphthol, producing a dark blue precipitate (Dumas et al., 1995).

Bioassay of transgenic plants challenged by Phytophthora colocasiae. Six-month-old transgenic plantlets (≈4 cm in height) of the transgenic line g5 or non-transformed plantlets were multiplied using tissue culture methods described in He et al. (2010). The line g5 was selected initially for all bioassays, because it contained a single transgene based on Southern analysis. In addition, propagated plantlets from this line appeared healthy and similar to non-transformed plantlets. Unfortunately, other transgenic lines were lost as a result of contamination after Southern analysis and could not be challenged with the pathogen.

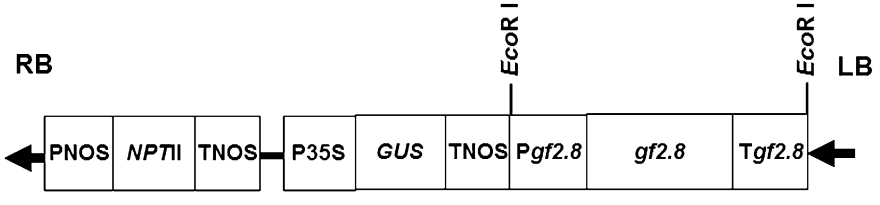


Fig. 1. Diagram of the transformation plasmid pBI121/*gf2.8* consisting of the *npt*II selection gene, the *gus* reporter gene, and a wheat oxalate oxidase gene *gf2.8* driven by its own promoter.

Plantlets were inoculated with the oomycete pathogen *P. colocasiae* according to He et al. (2008). Briefly, a plug of V8 agar (Miller, 1955) bearing active spores (≈0.2 cm^3) was placed spore side down on the cut shoot base of each plantlet. Each inoculated plantlet was placed on moistened filter paper and sealed in a petri dish to maintain 100% humidity. Three non-transformed plantlets and three transgenic plantlets of the line g5 were used in each trial, and each trial was repeated three times using a randomized complete block design. After inoculation, plantlets were observed daily for lesion initiation for 30 d. At 12 d, the lesion diameters were measured, averaged across three plantlets, and analyzed statistically by analysis of variance (ANOVA). The general linear model (GLM) program of SAS software (SAS Institute, Inc., Cary, NC) was used to conduct the ANOVA.

A second leaf-disk bioassay was performed with one-year-old potted plants, modifying the method of Brooks (2000). The youngest fully expanded mature leaves were selected from potted plants of one-year-old transgenic line g5 or non-transformed 'Bun Long' control plants. Leaf-disks (20 mm in diameter) were excised with a cork borer. The leaf-disks were immediately placed in water agar plates. Twenty microliters of spore suspension (1×10^4 spores/mL) were pipetted onto the center of each leaf-disk, and the plates were placed in a growth chamber maintained at 24 °C, 12 h light, and 100% relative humidity. Leaf-disks were observed daily and necrotic lesions were measured at 3 d after inoculation. Six leaf-disks from three individual plants of the g5 line and six leaf-disks from three individual plants of the non-transformed control were inoculated with the pathogen. Results of each leaf disk were averaged per treatment. Each experiment was repeated three times and data analyzed statistically by ANOVA using the GLM program of SAS software (Statistical Analysis System).

To compare the TLB resistance of the transgenic line g5 to naturally occurring TLB-susceptible and TLB-resistant cultivars, a third leaf-disk bioassay was performed using the Hawaiian commercial cultivar Maui Lehua (susceptible to TLB), the transgenic line g5, and three putative TLB-resistant cultivars, Pa'akala, BC99-6, and Ngesuas (P1). Cultivar Ngesuas was introduced from Palau based on observed field resistance to TLB; cv. Pa'akala was bred conventionally by Trujillo for increased TLB resistance (Trujillo et al., 2002); and cv. BC99-6 was bred conventionally by Cho for greater TLB resistance Cho et al. (2007). The bioassay was conducted as described earlier, except for placing five leaf-disks on each plate.

Results

Transformation of taro with the OxO gene. Using methods described earlier (He, 2006; He et al., 2008, 2010), calli were produced based on manipulation of plant hormones in tissue culture. In total, 200 pieces of calli (≈2 g) were transformed. Fifty calluses

survived after 90 d of selection on 50 mg·L^{-1} G418 and were transferred to the shoot-inducing medium containing the 50 mg·L^{-1} G418 with subculturing every 30 d. After 90 d, 30 independent shoot lines were initiated on the selection media. After another 60 d, only 10 lines survived the selection. PCR analysis was performed to screen transgenic lines. The expected 755-bp PCR product specific for the *gf2.8* gene fragment was found in eight lines (Fig. 2A), indicating that the *gf2.8* gene had been successfully transformed into these lines. No PCR product was obtained from the DNA extracted from non-transformed taro.

In addition, RNA was isolated from these eight lines and RT-PCR analysis conducted. The expected 755-bp RT product specific for the *gf2.8* gene fragment was found in all eight lines (Fig. 2B), indicating expression of the inserted gene. There was no RT product specific for the *gf2.8* gene fragment from RNA of non-transformed taro.

Restriction digests of genomic DNA extracted from the eight transgenic lines, using the enzyme *Bam*HI, yielded various bands (Fig. 2C). Transgenic lines 1 to 6 (g1–g6) appeared to have the same pattern, indicating one independent transformation event. Transgenic lines g7 and g8 appeared to have the same integration pattern, indicating a second independent transformation event. A single band was found in g1–g6 and four bands were found in g7–g8 (Fig. 2C), indicating a single site insertion and a multiple (i.e., four) site insertion, respectively, because *Bam*HI only has one digestion site in the T-DNA. This result confirmed the presence and intactness of the transgene *gf2.8* in the transformed taro genome. In the histochemical analysis of OxO activity, a dark blue precipitate was found in leaves and shoots of the NAA-induced transformed g5 taro line but not in the non-transformed control (Fig. 2D). This result confirmed the increased level of this protein in transgenic line g5.

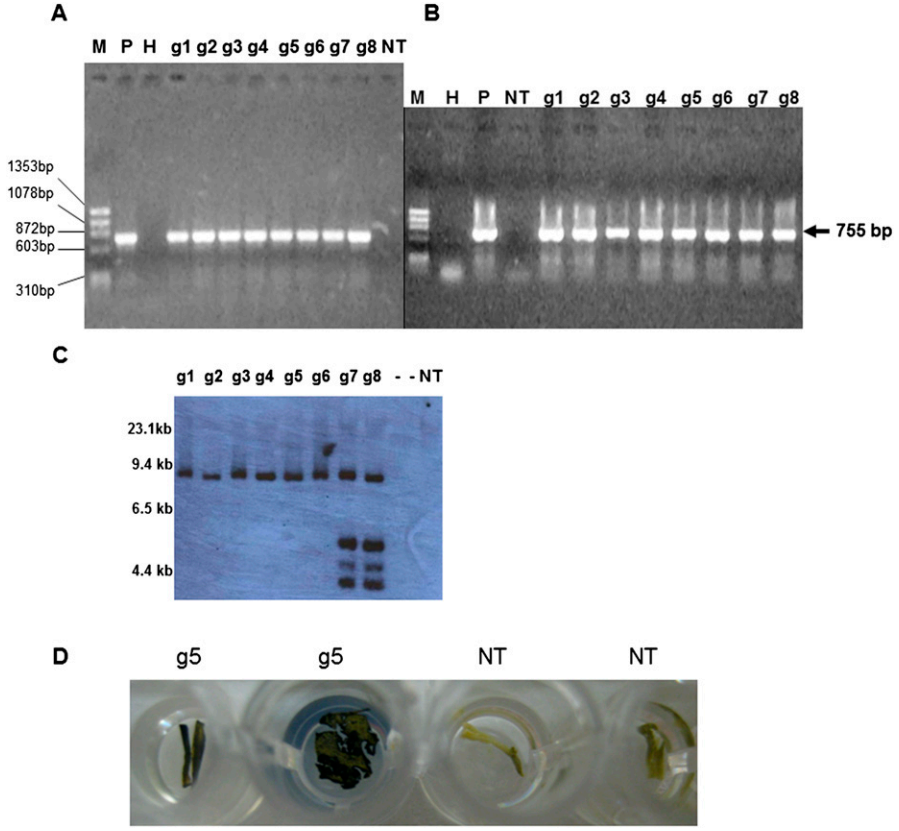


Fig. 2. (**A**) Polymerase chain reaction (PCR) analysis of taro cv. Bun Long lines transformed with EHA105:pBI121/*gf2.8*. M = molecular weight marker ΦX174/HaeIII; P = plasmid pBI121/*gf2.8* control; H = water control; g1–g8 = transgenic lines 1–8; NT = non-transformed plant control. (**B**) Reverse transcription–PCR analysis of taro lines transformed with EHA105:pBI121/*gf2.8*. M = molecular marker ΦX174/HaeIII; H = water control; P = plasmid pBI121/*gf2.8* DNA control; NT = non-transformed plant control; g1–g8 = transgenic lines 1–8. The expected 755-bp PCR product specific for the *gf2.8* gene fragment was found in these eight transgenic lines, indicating both integration into genomic DNA and expression. (**C**) Southern blot analysis of eight lines (g1–g8) and a non-transformed control (NT). The enzyme *Bam*HI cuts only once within the T-DNA region so that one band indicates one insertion of the transgene and four bands indicates four insertions of the transgene. (**D**) Histochemical assay of OxO activity in the transgenic line g5 (g5) or the non-transformed plantlet control (NT) after induction with NAA. Dark coloration indicates the presence of OxO activity in sliced g5 shoots (in Well 1, from left) and leaves (in Well 2, from left) that were incubated in 4-chloro-1-naphthol staining buffer with oxalic acid. Shoots (Well 3) and leaves (Well 4) of NT plantlets were unstained. OxO = oxalate oxidase; NAA = 1-naphthaleneacetic acid.

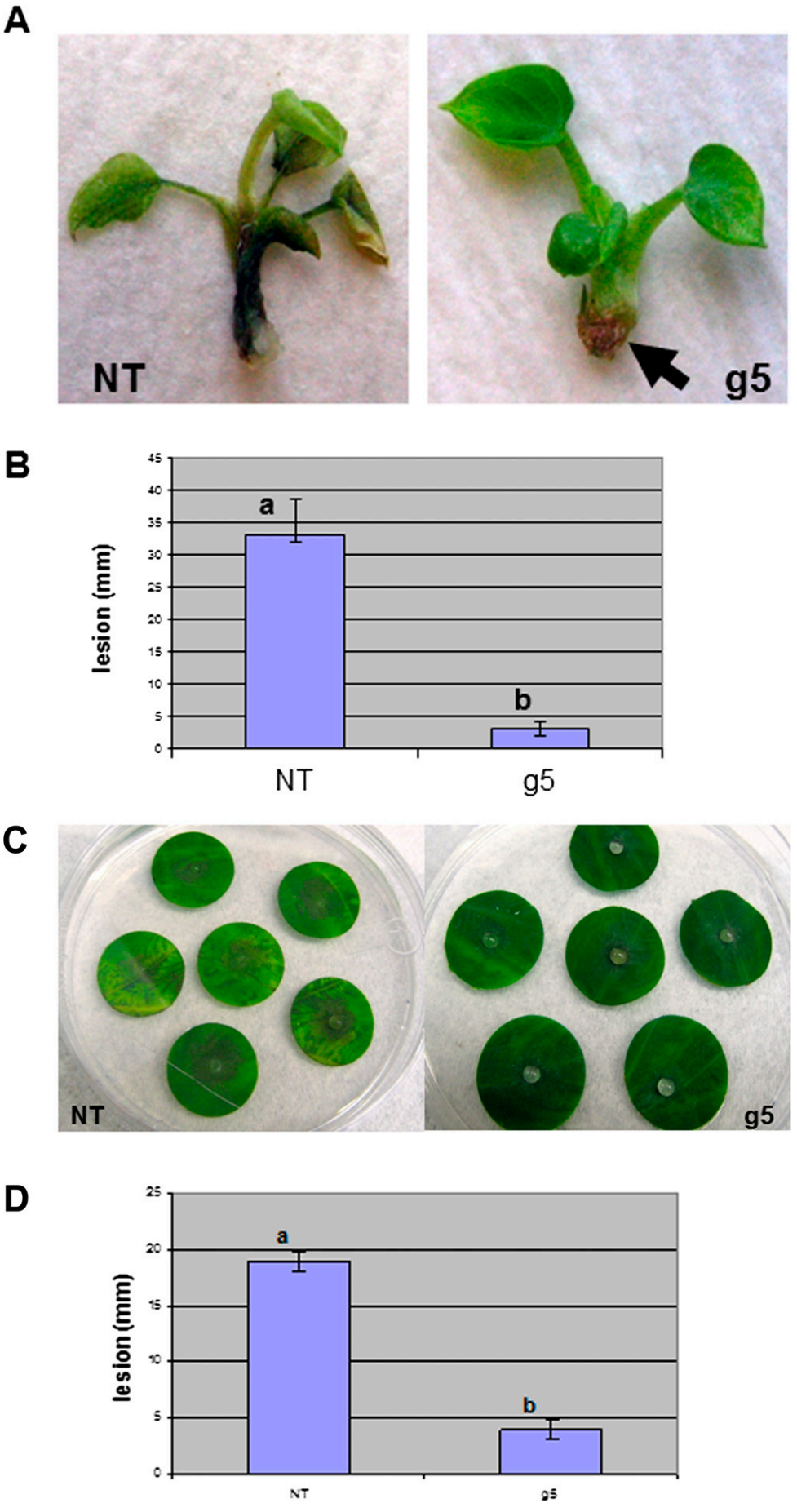
Pathogenicity of the oomycete Phytophthora colocasiae. In a bioassay that inoculated tissue-cultured plantlets with *P. colocasiae*, chlorotic and necrotic, water-soaked lesions of non-transformed plantlets were visible at 3 d after inoculation, and these lesions increased in size over time until the plantlets were dead on the 12th day (Fig. 3A, left). In contrast, in each transgenic plantlet of line g5, small, reddish brown lesions occurred at the inoculation area within 3 d after inoculation, and they did not extend much further by 12 d after inoculation (Fig. 3A, right). All transgenic plantlets of the line g5 were alive and appeared healthy with green leaves and shoots even at 30 d after inoculation (Fig. 3A, right), indicating complete cessation of the movement of the pathogen *P. colocasiae* in transgenic plantlets of line g5. The lengths of lesions measured at 12 d after inoculation were significantly shorter ($P < 0.05$) in transgenic plantlets of line g5 with the wheat oxalate oxidase gene gf2.8 compared with those of the non-transformed plantlets (Fig. 3B).

In a bioassay of one-year-old plants, inoculation of leaf disks from non-transformed taro with zoospores of *P. colocasiae* resulted in chlorotic and necrotic, water-soaked lesions within 24 h after inoculation. This lesion area increased over time, resulting in necrosis of the whole leaf disk of control plants within 3 d after inoculation (Fig. 3C, left). In contrast, leaf disks of transgenic line g5 did not exhibit water-soaked lesions within 24 h after inoculation, and only small lesions occurred at the inoculation area within 3 d after inoculation (Fig. 3C, right). The average lesion diameters measured at 3 d after inoculation were significantly smaller ($P < 0.05$) in the transformed g5 line than in the non-transformed control (Figs. 3D).

In the third bioassay, necrotic lesions were observed on all leaf disks of susceptible cultivar Maui Lehua within 24 h after inoculation. This lesion area increased over time, resulting in necrosis of the whole leaf disk of cv. Maui Lehua within 3 d after inoculation (Figs. 3E, left). In contrast, much smaller lesions were evident on several leaf disks of TLB-resistant cultivar Pa'akala (Figs. 3E, middle) and all leaf disks of transgenic line g5 (Figs. 3E, right) within 24 h after inoculation. The white mycelium of *P. colocasiae* could be observed on several leaf disk surfaces of line g5, but there were either no lesions or only a few lesions of limited diameter that developed (Fig. 3E, right). Both cv. Pa'akala and transgenic line g5 showed significantly smaller lesions than cv. Maui Lehua, BC99-6, and P1 at 3 d after inoculation (Fig. 3E–F). There was no significant difference in average lesion diameter between transgenic line g5 and cv. Pa'akala (Figs. 3F).

Discussion

Transformation of taro with the OxO gene *gf2.8* from wheat showed great promise in controlling the major taro pathogen *P. colocasiae*, particularly in taro cultivars



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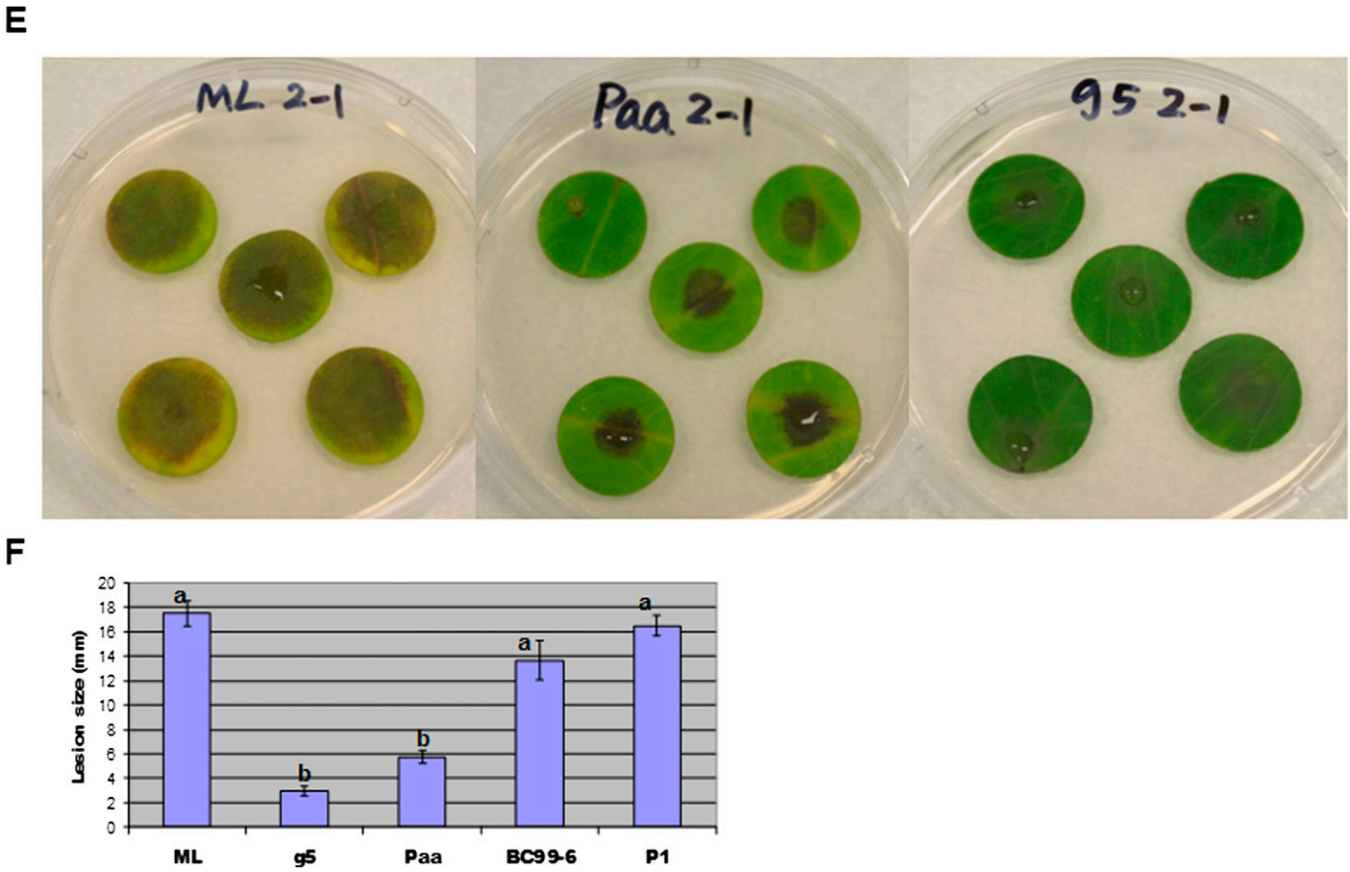

Fig. 3. (**A**) One plantlet of transgenic line g5 (**g5**, right) and one non-transformed plantlet control (NT, left) at 12 d after inoculation with *P. colocasiae*. The NT plantlet showed necrotic, water-soaked lesions extending from the cut shoot base to the leaf blades, and plants appeared dead. Plantlet of line g5 appeared green and healthy at 12 d and remained alive through 30 d when observations were ended. The arrow indicates where the agar disks containing spores were placed next to the cut shoot base. (**B**) Lesion length (mm) of *P. colocasiae*-inoculated plantlets of non-transformed control (NT) or transgenic line g5 measured at 12 d after inoculation. Graph bars represent the mean ± SD values of three replicates (each replicate consisted of three NT plantlets and three plantlets of g5). Different letters (a, b) indicate significant differences ($P < 0.05$) using analysis of variance (ANOVA). (**C**) Leaf-disk bioassay of one-year-old potted plants of transgenic line g5 (g5, right) or non-transformed plantlet control (NT, left) challenged by *P. colocasiae* at 3 d after inoculation. Zoospore suspensions were pipetted onto the center of each leaf-disk and the lesion diameter measured at 3 d after inoculation. Note that the chlorotic and necrotic, water-soaked lesions expanded throughout the whole leaf disk of NT. In contrast, lesions expanded very little beyond the pipetted spore suspensions in leaf disks of line g5. (**D**) Lesion length (mm) of *P. colocasiae*-inoculated leaf disks of non-transformed control (NT) or transgenic line g5 measured at 3 d after inoculation. Graph bars represent the mean ± SD values of three replicates (each replicate consisted of the averages of six leaf disks in three plates). Different letters (a, b) indicate significant differences ($P < 0.05$) using ANOVA. (**E**) Leaf-disk bioassay of transgenic line g5 (g5, right) and cvs. Maui Lehua (ML, left) and Pa'akala (Paa, middle) challenged by *P. colocasiae* at 3 d after inoculation. Zoospore suspensions were pipetted onto the center of each leaf-disk and the lesion diameter measured at 3 d after inoculation. Note that the chlorotic and necrotic, water-soaked lesions expanded throughout the whole leaf disk of ML. In contrast, lesions expanded to a much lesser extent in leaf disks of line g5 and cv. Paa. (**F**) Lesion length (mm) of *P. colocasiae*-inoculated leaf disks of commercial cv. Maui Lehua (ML), transgenic line g5, and putative TLB-resistant cvs. Pa'akala (Paa), BC99-6, and Ngesuas (P1) measured at 3 d after inoculation. Graph bars represent the mean ± SD values of three replicates (each replicate consisted of averages of three plates × five leaf disks). Different letters (a, b) indicate significant differences ($P < 0.05$) using ANOVA. TLB = Taro leaf blight.

that do not lend themselves to conventional cross-breeding. The spread of this oomycete pathogen was completely stopped in transgenic plantlets and greatly inhibited in leaf blades of one-year-old cv. Bun Long plants. To our knowledge, this is the first report of a transgenic plant with an inserted OxO gene that exhibited enhanced resistance to a pathogen (*P. colocasiae*) that does not secrete OA. These results are of particular interest, because Taro leaf blight caused by *P. colocasiae* is one of the worst, most invasive diseases affecting the sustainability of taro production in the world.

This study provides evidence in support of either a direct effect of H_2O_2 (e.g., hypersensitive-like cell death or antimicrobial activity) or an indirect effect of H_2O_2 (e.g., cell wall lignification or induction of PR genes) on plant responses to pathogens. We have observed hypersensitive-like responses on transgenic line g5 plantlets (Fig. 3A, right); in addition, mycelium of *P. colocasiae* appeared to grow on the surface of the leaf disk but did not penetrate into the leaf tissue (Fig. 3C, right). Interestingly, Urs et al. (2006) reported that localized H_2O_2 levels and peroxidase activity increased when gametophytes of the fern *Ceratopteris richardii* were exposed to pathogen *Pythium infestans*, indicating that in this plant species, peroxidases may be involved in production of H_2O_2 and resistance to an oomycete pathogen.

One advantage of genetic transformation is that disease resistance gene(s) can be inserted into the genome of an elite cultivar, while maintaining all of its other desirable characteristics. Cultivar Bun Long is commercially important in Hawaii and the Dominican Republic; however, it flowers rarely under the environmental conditions found in these two islands. As a result of the difficulty in the induction of flowering of cv. Bun Long (even with the use of gibberellic acid that is effective on other taro cultivars), conventional breeding of this particular cultivar for increased disease resistance is extremely slow.

In contrast, commercially important cv. Maui Lehua is more amenable to conventional breeding, because it flowers naturally in Hawaii and in response to gibberellic acid. Although cv. Maui Lehua showed significantly less resistance to TLB than the transgenic line g5, its progeny, Pa'akala, exhibited similar resistance to the transgenic line g5. These results indicate that conventional breeding for increased TLB resistance is possible

for elite cultivars that flower more readily such as cv. Maui Lehua.

Sharma et al. (2009) did not report up-regulation of OxO genes in resistant taro cv. Muktakeshi compared with susceptible taro cv. UL-56 when each was infected by *P. colocasiae*. This result indicates that other PR proteins (e.g., chitinases) may be involved in TLB resistance of that particular taro cultivar. Interestingly, in previous research, taro transformed with the chitinase gene *RICCHI11* showed no change in resistance to the oomycete pathogen *P. colocasiae* (He et al., 2008, 2010).

Another advantage of genetic transformation to increase disease resistance is the ability to stack genes of interest (Sridevi et al., 2008). In previous research, transgenic taro with an inserted chitinase gene from rice showed moderately increased tolerance of the fungal pathogen *Sclerotium rolfsii* (He et al., 2008, 2010). Insertion of the OxO gene into taro also resulted in a modest increase in resistance to *S. rolfsii* (He, 2006). Perhaps, disease resistance could be enhanced further in future transgenic taro plants by the combined expression of a chitinase gene with the OxO gene as found in other plant species.

Despite the potential advantages of genetic transformation of taro with the OxO gene to confer enhanced disease resistance, such research in Hawaii has been suspended as a result of controversies discussed briefly by He et al. (2010). Research in our laboratory now focuses on identification of naturally occurring disease resistance genes in taro and marker-assisted selection to accelerate introgression of such genes into elite germplasm.

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