

UV-B irradiation disrupts obligate endosymbioses and reduces fitness in agricultural pest insects

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Abstract

Sap-sucking insect pests, such as aphids and whiteflies, depend on obligate bacterial endosymbionts for survival. Driven by evolutionary genome reduction, these symbionts often lose essential DNA repair pathways, potentially leaving them defenseless against UV-induced damage—a vulnerability that remains untested as a pest control strategy. Here, we evaluated ultraviolet-B (UV-B) efficacy against three strawberry pests (*Chaetosiphon fragaefolii*, *Aphis gossypii*, and *Bemisia tabaci*) and linked host susceptibility to symbiont DNA repair capacities. UV-B irradiation significantly reduced pest survival and fecundity, with *B. tabaci* exhibiting extreme susceptibility even at low intensity. In *B. tabaci* and *C. fragaefolii*, UV-B depleted primary endosymbiont titers (*Portiera* and *Buchnera*), disrupted bacteriome morphogenesis, and impaired oocyte development. Comparative genomics, including a newly assembled *Buchnera* genome, confirmed that all examined obligate symbionts had lost most dark-repair pathways, with *Portiera* lacking almost all repair mechanisms. This repair-gene erosion extends to obligate symbionts of other major agricultural and medical pests. Our findings demonstrate that UV-B impairs pest fitness by exploiting DNA repair-deficient obligate endosymbionts, establishing a novel paradigm for pest management that targets the evolutionary 'Achilles' heel' of insect endosymbiosis.

Introduction

Insect pests severely impact agricultural quality and yield, causing an estimated 23% loss in annual global crop production [1]. While the application of chemical insecticides remains a primary line of defense in modern agriculture, its heavy reliance on these chemicals has manifested as severe adverse effects on surrounding ecosystems [2, 3]. Furthermore, the global emergence of pest populations that have developed resistance to multiple classes of insecticides poses a critical threat to food security [4–6]. Therefore, developing eco-friendly, sustainable pest control alternatives to chemical insecticides is an urgent imperative for modern agriculture.

Ultraviolet (UV) radiation represents a highly promising physical control method. UV radiation is conventionally divided into three wavebands: UV-A (315–400 nm), UV-B (280–315 nm), and UV-C (100–280 nm). While UV-C is highly germicidal and generally requires strict safety protocols to mitigate operational risks [7], artificial UV-B irradiation has also emerged as a powerful tool, demonstrating significant efficacy against destructive pests such as spider mites [8, 9], the Oriental fruit fly [10], and the Western flower thrips [11]; for broader perspectives on its wide-ranging effects across diverse insects, see Beard [12] and Holford et al. [13]. Beyond arthropod management, UV-B irradiation effectively suppresses plant pathogens like powdery mildew [14–16], making it an excellent component for multi-target Integrated Pest Management (IPM) systems [17]. Furthermore, because UV-B irradiation highly integrates with Internet of Things (IoT) technologies and automated systems, it represents an exceptionally efficient approach for modern greenhouse management [15, 18]. Consequently, this physical method is widely anticipated to play a pivotal role as a next-generation pest control strategy, particularly in developed nations facing critical agricultural labor shortages driven by depopulation [19].

Among the diverse pest complexes in protected agriculture, sap-sucking insects and mites—most notably aphids, whiteflies, thrips, along with spider mites—pose some of the most severe economic threats to greenhouse environments [20, 21]. These devastating pests exploit the stable, predator-free microclimates of high-tech greenhouses, causing catastrophic yield losses worldwide through both direct feeding damage and the transmission of plant pathogens. Crucially, a specific group of these primary insect pests, most notably aphids and whiteflies, strictly relies on hidden microbial partners to sustain their life cycles. Almost all aphids harbor *Buchnera aphidicola* (the prefix "Candidatus" is omitted throughout this paper), whereas whiteflies possess *Portiera aleyrodidarum* alongside strain-dependent co-obligate symbiotic bacteria within their specialized cells for endosymbiosis [22–24]. Although these host insects are highly diverse, encompassing more than 5,700 aphid and 1,500 whitefly species worldwide [25, 26], this obligate endosymbiotic system is preserved with constancy in whiteflies, and maintained with only few exceptional replacements in aphids [27, 28]. This symbiotic alliance has persisted for over 100 million years, driven by a strict mechanism of vertical transmission from the maternal ovaries to the offspring [29]. These endosymbionts provide their hosts with essential nutrients, such as essential amino acids and vitamins, which are critically deficient in the plant phloem-sap diet [30]. Hence, the elimination or disruption of these obligate symbionts inevitably leads to developmental delays and sterility [30]. Notably, like other obligate endosymbionts, these bacteria have undergone extensive genome reduction over their long co-evolutionary history, a process expected to have eroded not only redundant metabolic pathways [23, 31] but also DNA repair systems [31]—leaving them potentially defenseless against UV-induced DNA damage, a vulnerability that has not yet been experimentally tested as a pest control strategy.

To test this hypothesis and determine whether UV-B irradiation can disrupt these critical symbiotic relationships to serve as an effective control measure. Specifically, we evaluated the control efficacy of UV-B irradiation against three major greenhouse pests with differing host ranges—*Chaetosiphon fragaefolii* (a specialized pest of strawberries), and *Aphis gossypii* and *Bemisia tabaci* MED Q1 (both highly polyphagous pests)—using strawberry plants as a shared experimental host system. To elucidate the potential physiological mechanisms behind their UV-B susceptibility, we focused on the obligate endosymbionts of *C. fragaefolii* and *B. tabaci*, quantifying their titers. For the more susceptible *B. tabaci*, its spatial localization within the host bodies was also analyzed following UV-B exposure. Furthermore, to substantiate these physiological responses from a genomic perspective, we conducted a comprehensive comparative analysis of DNA repair pathways among these symbionts—including the newly determined complete genome of *Buchnera* from *C. fragaefolii*—using the fully capable genome of *Escherichia coli* as a baseline. Through these integrated approaches, we demonstrate that the genomic erosion of DNA repair mechanisms renders these ancient endosymbionts highly vulnerable to UV-B-induced damage, highlighting a critical physiological bottleneck that can be exploited for targeted pest management.

Results

Impact of UV-B irradiation on the fitness of three phloem-feeding insects

UV-B irradiation gradually reduced the survival of *Chaetosiphon fragaefolii* compared to the control, with significant differences detected from day 4 onward (Fig. 1A). Offspring were first observed on day 7 (i.e., at 9 days of age), in 6 of the 9 control dishes (Fig. 1B). In contrast, UV-B exposure significantly delayed the onset of reproduction. No offspring were produced under UV-B conditions by the end of the experiment (Fig. 1C).

For *Aphis gossypii* Generation 1, UV-B irradiation had no significant effect on survival or the time to first reproduction (Additional file 6A and B), but it slightly reduced fecundity ($p < 0.05$; Additional file 6C). In contrast, the suppressive effects of UV-B became more evident in Generation 2; survival was modestly but significantly reduced (Additional file 6D). While the time to first reproduction remained unaffected in Generation 2 as well (Additional file 6E), the impact on fecundity was further intensified, with the median number of offspring per dish decreasing by approximately half (Additional file 6F).

UV-B irradiation significantly reduced adult emergence in *B. tabaci*, with the emergence rate decreasing as the UV-B intensity increased from $180 \mu\text{W cm}^{-2}$ to $470 \mu\text{W cm}^{-2}$ (Fig. 2A). Moreover, exposure to a lower UV-B intensity of $15 \mu\text{W cm}^{-2}$ caused a modest yet statistically significant reduction in the adult emergence rate of *B. tabaci* (Fig. 2B). Extending the duration of UV-B exposure from 3 h day^{-1} to 5.5 h day^{-1} resulted in a lower adult emergence rate at 1 day of treatment compared with the control, although no significant difference was detected between the two UV-B exposure durations (Fig. 2B). After 2 and 3 days of treatment, both UV-B treatments continued to show significantly lower emergence rates than the control. By day 3, while both UV-B treatment groups still exhibited significantly lower emergence compared to the control, the median emergence rates for the 3-h and 5.5-h exposure treatments had converged to nearly identical levels; i.e., at this specific intensity ($15 \mu\text{W cm}^{-2}$), extending the daily exposure duration beyond 3 h provided no additional suppressive effect on the final emergence rate.

Effects of UV-B treatments and exposure time on abnormal pigmentation of the whitefly

To evaluate the overall impact of UV-B radiation, the proportions of all individuals exhibiting any of these abnormal pigmentation patterns (types B–D combined; Additional file 2) were tracked over time. A generalized linear mixed model revealed significant main effects of both treatment ($p = 0.049$) and time ($p < 0.001$) on the proportion of individuals exhibiting abnormal pigmentation (Fig. 3). The proportion of individuals with abnormal pigmentation tended to increase over time under higher UV irradiation conditions; however no significant interaction between treatment and time ($p = 0.116$). Post hoc comparisons among treatments within each time point showed no significant differences on day 1 or day 2. On day 3, however, the proportion of individuals with abnormal pigmentation was significantly

higher in the $470 \mu\text{W cm}^{-2}$ UV-B treatment than in the control. In contrast, the intermediate treatment group exhibited a dose-dependent trend but did not differ significantly from the other groups.

Carry-over effects of nymphal UV-B exposure on adult whitefly fecundity

A generalized linear mixed model revealed significant main effects of both treatment ($p = 0.031$) and time ($p < 0.001$) on the cumulative number of eggs laid by individuals that had been exposed to UV-B ($15 \mu\text{W cm}^{-2}$) for 3 h per day over a 2-day period during the 4th instar stage until emergence (Fig. 4): UV-B treatment significantly decreased the number of eggs, and this inhibitory effect tended to become more pronounced as time progressed. Although the interaction between treatment and time was not significant, post-hoc comparisons indicated a significant difference between the control and UV-B groups specifically on day 5 ($p < 0.05$).

Quantification of symbiont titer following UV-B treatment

A two-way ANOVA revealed that UV-B irradiation tended to decrease symbiotic bacterial titers in *B. tabaci*. In the control group, the overall abundance of both *Portiera* and *Hamiltonella* tended to be higher in females than in males over the experimental period. *Portiera* titers in both sexes were significantly affected by UV-B treatment, whereas neither time nor their interaction had a significant effect (Fig. 5A and B). In contrast, *Hamiltonella* titers were significantly affected by both UV-B treatment and time exclusively in males (Fig. 5D), while no significant main effects or interactions were detected in females (Fig. 5C).

In *C. fragaefolii*, a two-way ANOVA demonstrated that *Buchnera* titers were significantly affected by both UV-B treatment and time (Additional file 7). In the control group, *Buchnera* titers tended to increase from day 1 (3-day-old) to day 4 (6-day-old). Conversely, this increasing tendency was no longer observed in the UV-B treatment group, although no significant interaction between treatment and time was detected.

Influence of UV-B treatment on the symbiont localization in Bemisia tabaci

To capture the macro-scale organization of the symbiotic system, Additional file 8 illustrates the wide-field spatial distribution of symbiotic organs on day 2 in a control female and three UV-B-irradiated females. In the control group, a well-developed, single central bacteriome was observed, reflecting normal developmental aggregation of the two lateral pupal bacteriomes toward the midline upon adult emergence [24, 41]. Within this matured bacteriome, the primary symbiont *Portiera* was distinctly localized throughout its cytoplasm. In these control samples, well-developed oocytes were also observed adjacent to the bacteriome. Conversely, the UV-B-treated individuals exhibited a markedly altered morphology; in all three replicates, the bacteriome consistently failed to fuse into a single central organ and remained divided into two separate lateral entities within the abdomen; simultaneously, no

developed oocytes were detected in their abdomen. This structural non-coalescence indicates a clear developmental arrest in the maturation of the symbiotic organ and the host reproductive system, even though the external body had transitioned into the adult stage.

To further examine the intra-cellular dynamics of the endosymbionts at a higher magnification, the non-treated control group showed female adults with well-developed bacteriocytes densely packed with *Portiera* signals, which were consistently observed in all individuals examined from days 2 to 4 (Fig. 6). Conversely, UV-B irradiation induced stage-dependent degenerative changes in female bacteriocytes: while no apparent morphological differences were detected on day 2, a distinct loosening of individual *Portiera* signals—characterized by increased intracellular gaps—was observed on day 3. By day 4, the abundance of *Portiera* signals within the bacteriocytes of UV-B-treated females was visibly and drastically reduced. In these female samples, *Hamiltonella* signals were overall modest and exhibited no treatment-specific alterations in their localization.

In contrast, male adults displayed distinct symbiont dynamics (Fig. 6). Even in the control group, the baseline intensity of *Portiera* signals was generally weaker in males than in females, and a rapid, age-dependent decline in *Portiera* abundance was observed over the sampling period. UV-B irradiation further exacerbated this reduction, leading to a profound depletion of *Portiera* signals in treated males. Although *Hamiltonella* signals remained at a modest baseline intensity, UV-B irradiated males occasionally exhibited an aberrant localization of *Hamiltonella* that was never observed in the control group. This phenotypic shift is discernable in the overview (Fig. 6) and further visualized with enhanced signal contrast in Additional file 9 (showing the same sample with optimized contrast for *Hamiltonella*).

General Feature of the reconstructed endosymbiont genomes

Through the de novo assembly of PacBio HiFi reads using Flye, we successfully reconstructed the high-quality genome sequence of the primary endosymbiont, *Buchnera aphidicola* in *C. fragaefolii*. The assembly achieved an exceptionally high genomic coverage of 33×, ensuring a robust base accuracy for subsequent analyses. The chromosome was successfully resolved into a single circular contig of 620,959 bp with no sequencing gaps. Additionally, one plasmid sequence was successfully reconstructed. The general genomic features determined by the DFAST pipeline are summarized in Additional file 4.

DNA repair profiles in the essential endosymbionts of *C. fragaefolii*, *A. gossypii* and *B. tabaci*

In contrast to the free-living model bacterium *Escherichia coli*, which retains a complete and fully functional set of all core DNA repair enzymes, the genomes of the analyzed endosymbionts exhibited distinct patterns in the retention and absence of these enzymes (Additional file 5). The presence of the enzymes varied substantially among the examined symbionts. *Portiera* of *B. tabaci* MED Q1 lacked almost all repair pathways as well as the photoreactivation system. In contrast, *Hamiltonella* of *B. tabaci* MED Q1 retained all functional pathways and the photoreactivation system, except for a single step in

the NER and the double-strand break repair in HR. *Buchnera* of *A. gossypii* and *C. fragaefolii* exhibited an intermediate level of enzyme loss. In both *Buchnera* species, photoreactivation was the only repair mechanism predicted to be fully active, whereas all other multi-step dark repair pathways (such as BER, NER, and MMR) as well as the HR pathway were disrupted by the loss of essential constituent enzymes at one or more steps.

Cross-species comparison of DNA repair profiles across diverse pest symbionts

The pattern of enzyme retention was also evaluated in other agricultural and medical/veterinary pests (Additional file 5). *Portiera* and *Hamiltonella* from the MEAM1 genotype of *B. tabaci* exhibited profiles identical to those observed in the MED Q1 genotype. In other dual-symbiotic systems—the *Tremblaya*–*Moranella* pair in the mealybug and the *Karelsulcia*–*Baumannia* pair in the glassy-winged sharpshooter—the primary symbionts lacked almost all repair pathways as well as the photoreactivation system. In contrast, the co-obligate symbionts retained a larger number of enzymes in the dark repair pathways. *Carsonella*, the primary symbiont of the Asian citrus psyllid, lacked all core DNA repair mechanisms.

Among the medical and veterinary pest symbionts, *Wigglesworthia*, the essential symbiont of the tsetse fly (the vector of African trypanosomiasis, or sleeping sickness), retained a relatively large number of repair enzymes, though photoreactivation was the only mechanism predicted to be intact. The obligate *Wolbachia* symbionts of the bed bug and the human filarial nematode responsible for onchocerciasis also retained numerous core enzymes; however, neither their dark repair pathways nor the photoreactivation mechanism was complete.

Discussion

In this study, we investigated the physiological impacts of UV-B irradiation on three important phloem-feeding insect pests that pose severe threats to global agriculture and commonly infest strawberry fields: *Chaetosiphon fragaefolii*, *Aphis gossypii*, and *Bemisia tabaci*. In *C. fragaefolii*, exposure to UV-B at $470 \mu\text{W cm}^{-2}$ for 9.5 h per day resulted in significant negative effects on both survival and reproduction (Fig. 1). In contrast, *A. gossypii* G1 generation showed relatively low sensitivity to this UV-B regime, exhibiting only a slight reduction in fecundity (Additional file 6A– C). Interestingly, despite the robust tolerance observed in the G1 generation of *A. gossypii*, the subsequent second generation (G2) reared under UV-B conditions suffered pronounced reductions in both survival and fecundity (Additional file 6D– F). Because G2 individuals were exposed to UV-B transgenerationally from the embryonic stage within the maternal body, this prolonged, multi-generational exposure likely exerted a cumulative stress that led to the amplified suppressive effects.

A remarkably high sensitivity to UV-B radiation was also observed in *B. tabaci*. Even when the UV-B intensity and exposure time were reduced to a low-dose regime ($15 \mu\text{W cm}^{-2}$ for 3 h/day), the cumulative adult emergence rate was significantly suppressed to approximately 75% (Fig. 2B). Although high-dose

UV-B irradiation typically suppresses crop growth and productivity [42] and can cause visible injury such as leaf scorch or fruit abnormalities [43], such a low-dose condition (e.g., $\sim 15 \mu\text{W cm}^{-2}$ for 3 h/day) falls within the recommended safety margin for crop production, effectively controlling pests and diseases without adverse effects on plant health [44, 45]. Under this non-phytotoxic regime, *B. tabaci* nymphs exposed during the fourth instar still exhibited an increasing trend in abnormal pigmentation, albeit not statistically significant (Fig. 3). Furthermore, when nymphs were exposed to this low-dose regime just twice during the fourth instar, the total number of offspring produced by 5 days post-transfer was significantly reduced (Fig. 4).

Collectively, these findings clearly indicate that susceptibility to UV-B radiation varies substantially among insect pests, revealing a profound discrepancy in the effective dosages required for suppression. While *B. tabaci* demonstrated high susceptibility to the low-dose regime, a much higher UV-B intensity ($470 \mu\text{W cm}^{-2}$ for 9.5 h per day) was necessary to achieve sufficient suppressive effects on the aphid species, particularly *A. gossypii*. A primary hypothesis for these interspecific differences points to the physical and phenotypic traits of the insects, where variations in body size, structural thickness, and cuticle pigmentation may modulate the permeability of UV-B radiation into their internal tissues, as suggested in early evaluations [12]. Consequently, for practical field applications, optimizing and shifting the UV-B irradiation regime based on the dominant pest composition in the greenhouse will provide a highly viable, versatile, and customizable IPM framework.

Various insect species, including pest insects, harbor intracellular symbiotic bacteria that provide essential nutrients lacking in their host plants and are crucial for the insects' survival and reproduction [22, 23]. To support these vital nutritional roles, the population dynamics of such symbionts are strictly regulated within the host to meet heightened nutritional demands [24, 46–50]. This regulatory principle is also maintained in *B. tabaci*. *Bemisia tabaci* possesses two distinct symbiotic bacteria, *Portiera* and *Hamiltonella*. These symbionts co-localize within the same host bacteriocytes—specialized cells dedicated to endosymbiosis—forming an obligate metabolic partnership that co-synthesizes essential nutrients through the reciprocal exchange of metabolic intermediates [24]. Typically, both *Portiera* and *Hamiltonella* multiply throughout nymphal development and reach their peak abundance immediately after adult emergence [24]. According to this report, both sexes exhibit a subsequent decline in titers as they age, although females sustain higher symbiont populations for a longer duration than males. Notably, our control group closely mirrored these established baseline dynamics (Fig. 5), thereby validating the reliability of our experimental setup. However, this highly coordinated symbiotic infrastructure was severely disrupted by UV-B treatment: UV-B irradiation significantly reduced *Portiera* titers in both sexes (Fig. 5A and B). Furthermore, *Hamiltonella* titers were also significantly decreased by UV-B exposure exclusively in males, which already exhibited lower baseline symbiont abundance compared to females (Fig. 5C and D). Because *Portiera* serves as the indispensable metabolic backbone of this tripartite symbiosis, such a profound reduction in its population inevitably disrupts the shared metabolic pathways required for synthesizing essential nutrients. Therefore, the prominent suppressive

effects of UV-B on whitefly fitness are likely triggered, or at least accelerated, by this symbiont-mediated nutritional collapse.

Remarkably, a highly consistent phenomenon was observed in *C. fragaefolii*. *Buchnera* titers in the control group of *C. fragaefolii* exhibited a clear increasing trend from day 1 to day 4 (corresponding to 3-day to 6-day-old) (Additional file 7). This observation aligns perfectly with the well-established developmental dynamics of aphid endosymbiosis; *Buchnera* cells typically proliferate rapidly alongside host nymphal growth, reaching their maximum density at the teneral adult stage [46, 47]. Crucially, however, this natural developmental proliferation was substantially suppressed in the UV-B treatment group.

These congruent outcomes across both whiteflies and aphids strongly suggest that UV-B irradiation profoundly depresses the expected developmental proliferation of these endosymbionts. Importantly, these findings present two non-mutually exclusive possibilities regarding the underlying mechanism. On one hand, UV-B radiation may directly damage the endosymbionts or their containing host cells (bacteriocytes). On the other hand, it is equally plausible that the initial UV-B damage to the host's own physiological systems secondarily impairs its ability to regulate and sustain its microbiota.

Our FISH observations provided spatial and visual confirmation that UV-B radiation disrupts the symbiotic system across both macroscopic and microscopic scales, highlighting a dual impact on both the host symbiotic organs and the endosymbionts. At the organ level, repeated UV-B exposure severely arrested structural maturation, as evidenced by the failure of the two lateral pupal bacteriomes to fuse into a single central organ upon adult emergence (Additional file 8). Recent studies in various insects have revealed that the proper morphogenesis and differentiation of symbiotic organs are often actively induced by their endosymbionts [51, 52]. Given this biological precedent, it is highly plausible that in *B. tabaci*, the rapid, UV-B-induced depletion of the symbionts during the late nymphal stages may lead to insufficient critical bacterial cues to trigger bacteriome development.

This macro-scale developmental arrest directly correlated with cellular-level degeneration. The abundance of *Portiera* signals decreased progressively as the duration of UV-B exposure extended, with the most drastic reduction observed on day 4 of treatment (Fig. 6), indicating a cumulative detrimental effect over time. Furthermore, this extreme physiological stress destabilized the spatial regulation of the symbiotic system, as evidenced by the aberrant localization of *Hamiltonella* expanding beyond the bacteriocyte boundaries in treated males (Additional file 9). Taken together, the host's nutritional homeostasis likely suffered a catastrophic failure, driven by both the structural collapse of the bacteriome and the profound depletion of its essential endosymbionts. Because whitefly reproduction depends entirely on the essential nutrients provided by these symbionts, this mutual collapse of both organogenesis and symbiont maintenance provides a logical explanation for the arrested internal oocyte development (Additional file 8) and the subsequent reduction in fecundity (Fig. 4).

To discern whether the observed symbiont reduction derives from direct UV-B damage or host-mediated physiological stress, we comprehensively analyzed and compared the retention of DNA repair-related

genes among the genomes of the symbionts in *C. fragaefolii*, *A. gossypii*, and *B. tabaci* MED Q1. It is well-documented that UV-B radiation critically damages cellular components primarily by inducing DNA lesions such as cyclobutane pyrimidine dimers (CPDs) and (6 – 4) photoproducts [53]. In free-living bacteria, these cytotoxic lesions and subsequent replication blocks or DNA double-strand breaks are routinely repaired and counteracted by specialized DNA systems. These include not only light-dependent photoreactivation and nucleotide excision repair for direct lesion removal but also mismatch repair and homologous recombination pathways to restore genomic integrity during and after replication [53–55]. Therefore, the intrinsic DNA repair capability of these bacterial endosymbionts—and how far they have deviated from the fully capable baseline seen in *E. coli*—was expected to provide important clues regarding their survival under UV-B stress.

Our genomic comparison revealed a striking contrast in the repair repertoires between the free-living baseline represented by *E. coli* and the endosymbionts, as well as among the symbionts themselves (Additional file 5). All analyzed endosymbionts generally exhibited a marked deficiency in their DNA repair toolkits compared to *E. coli*. However, the degree of gene retention varied substantially among the species. Crucially, the primary symbiont of *B. tabaci* MED Q1, *Portiera*, has lost almost all major DNA repair pathways. In sharp contrast, *Hamiltonella* retained almost all functional pathways and the photoreactivation system. This genomic disparity may partly explain the differential susceptibility and varying reduction patterns observed between the two symbionts under UV-B irradiation (Figs. 5A–D and 6). The complete absence of DNA repair pathways in *Portiera* aligns with its significant and consistent decline across both sexes. In contrast, *Hamiltonella*—equipped with a functional repair toolkit—exhibited a more resilient trend in females, where its reduction was not statistically significant. However, this genomic advantage was likely overwhelmed in males, leading to a significant depletion of both symbionts. However, because *Hamiltonella* operates in close metabolic concert with *Portiera* to form an obligate co-synthesis network that supplements the host's nutritional deficiencies [24], any severe depletion of *Portiera* would inevitably undermine *Hamiltonella* itself over time. Therefore, UV-B-induced collapse of *Portiera* acts as a lethal metabolic bottleneck, triggering a systemic breakdown of the entire symbiotic infrastructure that cannot be sustained by *Hamiltonella* alone.

Buchnera from the aphid species retains a key repair mechanism, including a gene involved in photoreactivation. This clear genomic disparity may, at least in part, explain why aphid species exhibit much higher baseline tolerance to UV-B radiation than the highly susceptible *B. tabaci*. It should be noted, however, that *Buchnera* strains from *C. fragaefolii* and *A. gossypii* possess nearly identical configurations of these DNA repair systems (Additional file 5), despite the significant difference in UV-B tolerance observed between the two host aphids in our experiments (Fig. 1 and Additional file 6). This finding strongly reinforces our initial hypothesis that host-mediated physical factors—such as the dark green to nearly black cuticle of *A. gossypii* versus the pale, translucent cuticles (pale green in *C. fragaefolii* and pale yellow in *B. tabaci*)—critically modulate actual UV-B penetration and subsequent tolerance in the field.

It is well-established that obligate endosymbionts universally undergo massive and irreversible genome reduction, characterized by the catastrophic loss of non-essential metabolic pathways and cellular machinery [23, 31]. This genome erosion is further accelerated by the non-adaptive fixation of deleterious mutations through relaxed selection and strong genetic drift [56], which frequently involves the loss of fundamental DNA replication and repair systems [31]. In line with this universal trend, the genome of *Buchnera* from *C. fragaefolii* analyzed in this study also exemplified this severe genomic erosion, possessing a highly streamlined chromosome of approximately 621 kb (Additional file 4). As a consequence of this extreme reductive evolution, genes redundant to basic cellular survival or those compensated for by the host environment have been entirely purged from these symbiont genomes.

Building on this evolutionary premise, other obligate symbionts identified in agricultural pests exhibited a similarly pronounced deficiency in their DNA repair toolkits (Additional file 5). Intriguingly, within multi-symbiont systems, secondary co-obligate symbionts consistently retained a larger number of enzymes in the dark repair pathways, likely reflecting their relatively shorter evolutionary history of endosymbiosis [23, 56]. In sharp contrast, the primary symbionts—which have undergone much longer periods of intimate association—consistently lacked almost all major repair pathways.

Essential endosymbionts are also widely distributed across filarial nematodes as well as medical and veterinary pest insects, where they are renowned for synthesizing critical micronutrients such as B vitamins that are fundamentally lacking in the hosts' restricted diets [57–59]. Remarkably, our comprehensive genome-wide analysis revealed that the DNA repair systems in these medical and veterinary pest symbionts are also systematically incomplete (Additional file 5). *Wigglesworthia*, the essential symbiont of the tsetse flies which vectors devastating African sleeping sickness, retained a relatively large number of repair enzymes, though photoreactivation was the only mechanism predicted to be intact. The obligate *Wolbachia* symbionts of the bed bug and the human filarial nematode responsible for onchocerciasis also retained numerous core enzymes; however, neither their dark repair pathways nor the photoreactivation mechanism was complete.

Ultimately, while these endosymbionts provide indispensable metabolic support that allows their hosts to exploit nutrient-deficient dietary niches, their extreme genomic erosion renders them entirely devoid of environmental stress responses. Although the concept of endosymbionts acting as an "Achilles' heel" for insects has been previously proposed [60–62], our study demonstrates that UV-B radiation serves as a highly potent and decisive physical trigger that directly exploits this profound physiological vulnerability. Given that massive genome reduction and the subsequent loss of DNA repair systems are universal hallmarks of obligate microflora, a vast range of agricultural pest insects—and potentially various medical and veterinary pests—that rely on such fragile symbiotic infrastructures could be susceptible to this UV-B-mediated disruption. Consequently, these insights highlight the exceptional utility of UV-B irradiation as a next-generation pest management strategy, offering a universally applicable, non-chemical approach to selectively undermine host fitness by targeting the evolutionary weaknesses of their hidden microbial partners.

Conclusion

In conclusion, this study demonstrated that UV-B irradiation severely reduces the fitness and survival of important agricultural pest insects. Crucially, our findings reveal that this physical stress operates not merely through direct damage to the host insects, but via the targeted disruption of their obligate bacterial endosymbionts, such as *Portiera* and *Buchnera*. Driven by evolutionary genome reduction and the consequent loss of essential DNA repair mechanisms, these endosymbionts represent a critical, unbuffered vulnerability within the host-symbiont complex. By linking micro-level genomic erosion to macro-level host phenotypes, this work establishes a novel paradigm for pest management that exploits the evolutionary Achilles's heel of insect endosymbiosis. Ultimately, these insights open new avenues for developing sustainable, non-chemical pest control technologies. Moreover, they advance our fundamental understanding of how obligate symbioses balance stability and fragility under evolutionary and environmental constraints.

Methods

Insect and plants

Aphis gossypii was derived from a population collected on April 28, 2021, from a strawberry field at the Tochigi Agricultural Experiment Station and was confirmed to harbor *Buchnera*. The aphids were maintained under laboratory conditions through continuous rearing. For rearing, hydroponically grown eggplant seedlings with 4–5 true leaves were used as host plants. Infested eggplants were placed in plastic containers (15 cm wide × 15 cm deep × 25 cm high; Fukushima Container: Okado PET) with two opposing sides cut out (10 cm wide × 8 cm high) and covered with nylon mesh to allow ventilation. These containers were kept in an incubator (KCLP-1000IICT; Nihon Medical & Chemical Instruments Co., Ltd., Osaka, Japan) set at 25°C under a 16 h light, 8 h dark photoperiod (16L:8D). *Chaetosiphon fragaefolii* was obtained from a naturally occurring population on strawberry seedlings (cv. 'Yotsuboshi') in the garden of the University of Toyama, harboring *Buchnera*, and was also maintained under laboratory conditions. Hydroponically grown strawberry seedlings with 5–9 true leaves were used as host plants. The aphid-infested strawberry plants were kept in the same type of plastic containers as described above and maintained in the same model of incubator (KCLP-1000IICT) set at 20°C under a 16L:8D photoperiod. Host plants for those aphid species were replaced every two weeks by placing aphids at the base of fresh seedlings.

Bemisia tabaci MED Q1 used in this study was the laboratory strain with *Portiera*, *Hamiltonella* and *Cardinium* [24]. This strain was maintained on hydroponically grown cabbage (*Brassica oleracea*) (cv. 'YR Yuryoku' or 'Kinshun') leaves at 25 ± 1°C and 40–60% relative humidity under a 16L: 8D in an incubator (BiOTRON LPH-411PFD-S; Nippon Medical & Chemical Instruments Co., Ltd., Osaka, Japan).

UV-B irradiation experiment for the two aphid species

For *C. fragaefolii* and *A. gossypii*, experiments were conducted using 3-cm glass Petri dishes (FS-30; Flat Co., Ltd., Japan) at their respective rearing temperature (as described above) under 12L:12D photoperiod. To synchronize the developmental stage of the experimental insects, only apterous adults were allowed to larviposit for 24 h, and the resulting 1-day-old apterous nymphs were reared until they reached 3 days of age before being used in the trials. Each dish contained a 3-cm-diameter leaf disc (strawberry for *C. fragaefolii* or eggplant for *A. gossypii*) placed on a 5-mm layer of 1% agar (FUJIFILM Wako Pure Chemical Corporation, Japan) supplemented with a 0.1% Hyponex fertilizer solution (N–P–K = 6.5–6–19; HYPONeX Japan Corp Ltd., Osaka, Japan). Leaf discs were replaced every three days to ensure freshness.

For *C. fragaefolii*, five synchronized 3-day-old nymphs were grouped in each dish, and UV-B treatment was initiated and continued until they reached 10 days of age. For *A. gossypii*, the effects were tracked across two successive generations. In the first generation (G_1), five individuals (starting at 3 days of age) were grouped per dish and treated with UV-B until 11 days of age. The second generation (G_2) was established using nymphs born on the second day after the onset of reproduction in G_1 . These G_2 nymphs were grouped (five per dish) and subjected to UV-B treatment from 1 to 6 days of age.

UV-B with $470 \mu\text{W}/\text{cm}^{-2}$ was applied using DE-H042-UV310 (Nichia Corp., Japan; peak wavelength, 308 nm; full width at half maximum, approximately 9 nm) for 9.5 h per day. Across all UV-B irradiation experiments in this study, the irradiance at the sample position was measured and verified prior to each trial using an X1 optometer with a UV370 detector (Gigahertz Optik GmbH, Germany) to ensure precise target intensity. To prevent photoreactivation of UV-induced DNA damage, which can occur under white LED illumination, a 2-hour dark period was imposed immediately after each UV-B exposure as described [8]. Survival and fecundity were recorded daily to evaluate the cumulative impact of chronic UV-B treatment relative to an untreated control group (maintained under standard 12L:12D condition without UV-B exposure).

Influence of UV-B irradiation on the adult emergence rate of B. tabaci

For all UV-B experiments using *B. tabaci*, fourth-instar nymphs (Additional file 1A) were maintained at 25°C under constant darkness within a custom-built irradiation chamber (Additional file 1B and C) equipped with the same UV-B light source described for the aphid experiments. To evaluate the impact on adult emergence, 15 to 30 nymphs were placed in each chamber and subjected to one of four UV-B irradiation regimens: 470, 180, or $15 \mu\text{W cm}^{-2}$ for 5.5 h/day, and $15 \mu\text{W cm}^{-2}$ for 3 h/day. The UV-B treatment was applied once daily, and the number of emerged adults was recorded after 1, 2, and 3 days of treatment (corresponding to 1, 2, and 3 separate exposure sessions, respectively). The results were evaluated relative to an untreated control group (no UV-B exposure).

Assessment of UV-B-induced abnormal pigmentation in B. tabaci

Thirty fourth-instar nymphs per chamber were subjected to the experimental setup described above under four different UV-B irradiation regimes: 470, 180, or 15 $\mu\text{W cm}^{-2}$ for 5.5 h /day, and 15 $\mu\text{W cm}^{-2}$ for 3 h /day. UV-B irradiation induced distinct visual symptoms of abnormal pigmentation, which were classified into four phenotypic categories based on stereomicroscopic observations: normal appearance (Additional file 2A), presence of small black spots on the body (Additional file 2B), partial black discoloration of the body (Additional file 2C), and whole-body discoloration (Additional file 2D). Individuals exhibiting any of these symptoms (types B to D) were recorded at 1, 2, and 3 days of treatment. The results were evaluated relative to an untreated control group (no UV-B exposure).

Influence of UV-B irradiation on the fecundity of B. tabaci

To evaluate reproductive potential, 10 fourth-instar nymphs per chamber were subjected to UV-B treatment with 15 $\mu\text{W cm}^{-2}$ for 3 h /day. Adults that emerged between 24 and 48 h after the onset of treatment were collected and paired with an individual of the opposite sex. Each pair was then transferred to a new chamber and allowed to oviposit under UV-B-free conditions. The number of eggs deposited was recorded every 24 h for five days post-transfer. The results were evaluated relative to an untreated control group (no UV-B exposure).

Quantification of symbiont titer following UV-B treatment

Fourth-instar nymphs of *B. tabaci* were exposed to UV-B radiation (15 $\mu\text{W cm}^{-2}$ for 3 h /day) over two, three or four separate days (2-, 3-, or 4-day treatments). Adults that emerged during this period were collected on the final day of each respective treatment. DNA was extracted from each insect as previously described [32]. Quantitative PCR (qPCR) was performed to estimate *Portiera* and *Hamiltonella* titers based on bacterial gene copy numbers using the CFX Connect Real-Time PCR Detection System (Bio-Rad Laboratories, Inc., USA) with the KOD SYBR qPCR Mix (TOYOBO Co., Ltd., Japan) and specific primer sets (Additional file 3).

For 3-day-old nymphs of *Chaetosiphon fragaefolii*, the daily UV-B exposure condition (470 $\mu\text{W cm}^{-2}$ for 9.5 h /day) was applied and administered either once (1-day treatment) or on four separate days (4-day treatment). Individual DNA extraction was conducted using the NucleoSpin Tissue XS kit (Takara Bio Inc., Japan). The qPCR assays were performed to estimate *Buchnera* titers using a Mx3005P qPCR system (Agilent Technologies, Inc., USA) with specific primer sets (Additional file 3).

For both insect species, the standard curve method with a dissociation curve analysis was applied for quantification as previously described [24]. The qPCR cycling conditions for all assays were identical: 98°C for 2 min; 40 cycles of 98°C for 10 s, primer-specific annealing temperature for 10 s, and 68°C for 30 s.

Fluorescence in situ hybridization (FISH)

Fourth-instar nymphs of *B. tabaci* were exposed to UV-B radiation ($15 \mu\text{W cm}^{-2}$, 3h/day) over two, three, or four separate days (2-, 3-, or 4-day treatments). Adults that emerged during this period were collected on the final day of each respective treatment. All samples were initially fixed using Carnoy's solution (ethanol: chloroform: glacial acetic acid, 6:3:1, v/v/v) overnight with gentle mixing. Then, the head-thorax and abdominal regions of each sample were separated using a needle and forceps to facilitate optimal fixative and probe penetration. All tissue parts were then re-fixed in Carnoy's solution overnight and bleached in 6% hydrogen peroxide in ethanol for seven days at room temperature in the dark. The samples were subjected to whole-mount FISH, as described [33]. Fluorochrome-labelled oligonucleotide probes used in this study are listed in Additional file 3. The host cell nuclei were counterstained with 4',6-diamino-2-phenylindole (DAPI). For microscopic assessment, observations and image acquisitions were focused on the abdominal regions, where bacteriocytes are localized, using a laser scanning confocal microscope LSM880 (Carl Zeiss, Germany). To ensure valid qualitative and intensity comparisons across all treatments, all images were captured under identical acquisition settings, including laser power, detector gain, and pinhole diameter, and analyzed using ZEN2 (black edition) or ZEN2.5 (blue edition) software (Carl Zeiss, Germany). The specificity of *in situ* hybridization was confirmed by the following control experiments: a no-probe control and an RNase digestion control, as previously described [34].

Genome sequencing data retrieval and de novo assembly

Public high-throughput sequencing data for the *Buchnera* genome of *C. fragaefolii* were retrieved from the NCBI Sequence Read Archive (SRA) [35] under accession number SRX27876456. The data consisted of high-quality PacBio HiFi reads and were downloaded using the prefetch tool (v3.4.1) within the SRA Toolkit. The retrieved files were converted to raw FASTQ format using fasterq-dump. To optimize computational efficiency and achieve high-quality assembly of the target endosymbiont genome, the dataset was randomly subsampled using seqkit v 2.0.0 [36]: the run SRR32566845 (associated with SRX27876456) was downsampled to 2% of its total volume. *De novo* genome assembly of the subsampled datasets was subsequently performed on a high-performance computing system using Flye v2.9.6 [37] via Singularity containers [38]. The assembly was executed using the specialized PacBio HiFi mode (--pacbio-hifi) to optimize the reconstruction and potential circularization of the bacterial endosymbiont genomes. The general genomic features of the newly reconstructed *Buchnera* genome are summarized in Additional file 4.

Functional annotation and pathway reconstruction

The newly assembled genome of *Buchnera* was structurally and functionally annotated using the DFAST pipeline (DDBJ Fast Annotation and Submission Tool; <https://dfast.ddbj.nig.ac.jp/>) to predict protein-coding genes. To systematically assign functional categories and reconstruct metabolic capabilities, officially assigned KEGG Orthology (KO) identifiers were retrieved directly from the KEGG database for established reference genomes. For unannotated endosymbiont sequences, including the newly sequenced *Buchnera* and specific reference strains detailed in the footnotes of Additional file 5, the predicted amino acid sequences were subjected to the BlastKOALA server [39] for *de novo* KO assignment.

Identification and Comparison of DNA Repair Enzymes

To evaluate the retention of DNA repair pathways across genomes of essential symbionts in representative agricultural and medical pests (fully listed in Additional file 5), we systematically searched for enzymes involved in photoreactivation, base excision repair (BER), nucleotide excision repair (NER), mismatch repair (MMR), and homologous recombination (HR). For photoreactivation, the search was strictly limited to enzymes with officially assigned KEGG Orthology (KO) identifiers to ensure annotation accuracy. For the other four major repair pathways (BER, NER, MMR, and HR), specific genes were identified by referencing the *Escherichia coli* K-12 (MG1655) reference pathway maps in the KEGG database: eco03410 (Base excision repair), eco03420 (Nucleotide excision repair), eco03430 (Mismatch repair), and eco03440 (Homologous recombination).

Statistical analysis

To test for differences in cumulative adult emergence rates among treatments at each time point, we employed generalized linear models (GLMs) with a binomial error distribution and logit link function, using the cumulative number of emerged adults versus the number remaining as the response variable. Because the data exhibited near-complete separation at later time points, we applied bias-reduced logistic regression (Firth's method) as implemented in the *brglm2* package in R to obtain stable estimates. Post hoc multiple comparisons among treatments were conducted using estimated marginal means (*emmeans*) with Tukey's adjustment for multiple testing. Analyses were performed separately for 1, 2, and 3 days of treatment.

To compare the proportion of individuals exhibiting abnormal pigmentation, statistical analyses were conducted using generalized linear mixed models (GLMMs) with a binomial error distribution. Treatment, time, and their interaction were included as fixed effects, and sample identity was included as a random effect to account for repeated measurements. Because all treatments showed zero abnormal pigmentation at time 0, this time point was excluded from inferential analyses. Post hoc comparisons among treatments within each time point were performed using estimated marginal means with Sidak adjustment.

To evaluate the effect of UVB irradiation on the number of eggs laid over time, we employed a GLMM with a negative binomial distribution to account for overdispersion in the count data. In the model, treatment (control and UV-B irradiation), time (days), and their interaction were included as fixed effects, while individual ID was incorporated as a random effect to account for repeated measurements from the same individuals. The significance of the fixed effects was assessed using Type II Wald chi-square tests. For post-hoc analysis, pairwise comparisons between treatments at each time point were performed.

Quantitative data for symbiont titers (*Portiera*, *Hamiltonella*, and *Buchnera*) were analyzed separately for each combination of symbiont and host group. For *Portiera* and *Hamiltonella*, data were analyzed for each host sex (female or male), while for *Buchnera*, data from *Chaetosiphon fragaefolii* were used. For each dataset, a series of generalized linear models (GLMs) were fitted with different error distributions

and link functions (Gaussian with identity and log links; Gamma with log and inverse links). The optimal model for each dataset was selected based on the lowest Akaike Information Criterion (AIC) value. Subsequent two-way ANOVA was performed using GLM with a Gamma error distribution and log function for consistency across symbiont species, to examine the effects of the treatment (non-treated control and UV-B) and time (1 and 4 h for *Buchnera*; 2, 3, and 4 days for others). All analyses were conducted in R software version 4.2.1 or later [40].

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

H.O., T.O., and T.Y. are employees of NTT EAST, Inc. This study was funded through a joint research agreement with NTT EAST, Inc. The remaining authors declare that they have no competing interests.

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Author Contribution

Conceptualization: T.T. and A.F.; Investigation: S. F., R. K., K. K., M. N. and A. F.; Formal analysis: T. T.; Methodology: T. T., H. O., T. Y., T. O. and A. F.; Project administration: T. T., H. O., T. Y., T. O. and A. F.; Supervision: T. T. and A. F.; Writing—original draft: T. T. and A. K.; Writing—review and editing: T. T. and A. K.

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Data Availability

The complete genome assembly and functional annotations generated in this study have been deposited in the DDBJ/EMBL/GenBank databases under the accession numbers BR002639 (Chromosome) and BR002640 (pLeu plasmid). This genome assembly was reconstructed using the previously published sequencing data retrieved from the NCBI Sequence Read Archive (SRA) [35] under the accession number SRX27876456.

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