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Ultraviolet radiation affects invasibility of lake ecosystems by warm-water fish

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Abstract. Predicting where species invasions will occur remains a substantial challenge in ecology, but identifying factors that ultimately constrain the distribution of potential invaders could facilitate successful prediction. Whereas ultraviolet radiation (UVR) is recognized as an important factor controlling species distribution and community composition, the role of UVR in a habitat invasibility context has not been explored. Here we examine how underwater UVR can regulate warm-water fish invasion. In Lake Tahoe, California and Nevada, USA, established populations of exotic bluegill sunfish (*Lepomis macrochirus*) are currently limited to turbid, low-UVR embayments. An in situ incubation experiment that manipulated incident UVR exposure of larval bluegill, combined with an assessment of UVR exposure levels in nearshore habitats around Lake Tahoe, demonstrates that UVR can mediate habitat invasibility. Our findings suggest that the susceptibility to invasion by UVR sensitive species may increase in transparent aquatic systems threatened by declining water quality, and they highlight the importance of abiotic factors as regulators of invasion risk in ecosystems.

Key words: abiotic factors; aquatic invasion; bluegill sunfish; DNA dosimeters; habitat invasibility; Lake Tahoe, USA; Lepomis macrochirus; *ultraviolet radiation.*

Introduction

The proliferation of invasive species is one of the most important anthropogenic impacts in freshwater systems (Naiman et al. 1995). The problem is largely a byproduct of human development, with its tendency to deconstruct biogeographic barriers (Rahel 2007) and fundamentally alter the biotic and abiotic components of environments that foster distinct populations of plants and animals and regulate the susceptibility of habitats to invasion. Consequently, habitat invasibility is generally thought to be high in areas characterized by extensive human impact. For example, among California, USA watersheds, the number of nonnative fish species is positively correlated with anthropogenic landscape-level changes related to watershed disturbance and altered hydrology (Marchetti et al. 2004). Reservoirs are also a notable example of how human activity may promote invasion (Havel et al. 2005). These examples highlight important factors that are likely to control invasibility in some habitats but they are driven by more traditional notions of human impact, such as the stabilization of flow regimes related to habitat alteration or the influence of a high degree of environmental variability through time. Whereas changes in water transparency with anthropogenic disturbance are widely recognized in aquatic habitats, little attention is given to how such disturbances can mediate exposure to damaging wavelengths of ultraviolet radiation (UVR). Here we demonstrate the potential importance of UVR exposure as a factor controlling habitat invasibility of a warm-water fish in Lake Tahoe.

Lake Tahoe is a sub-alpine lake in the northern Sierra Nevada range spanning the California–Nevada border. The lake is renowned for its deep blue water and high transparency, afforded by the combination of great depth, small watershed-to-lake-area ratio, and granitic geology of the basin (Jassby et al. 1994). However, the transparency has decreased over time with the average annual Secchi transparency declining from 31 m in 1968 to 21 m by 1998 (Jassby et al. 1999). During this same 30-year interval, a number of nonnative warm-water fish species established populations in some portions of Lake Tahoe (Reuter and Miller 2000).

The establishment of these warm-water species may be directly related to the significant changes in water transparency observed in recent decades (see Plate 1). For example, larval bluegill sunfish (*Lepomis macrochirus*) perish within a single day when exposed to incident UVR at the surface of transparent lakes (Williamson et al. 1999). Yet the requirement for warmer spawning temperatures constrains bluegill nests

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to the shallow surface waters in the littoral zone of lakes and rivers (Kitchell et al. 1974). Thus the transparency of the water as well as the depth and location of nests are critical determinants of reproductive success in bluegill (Olson et al. 2006). Currently, the only well-established bluegill populations in Lake Tahoe are limited to sites in the southern end of the basin characterized by extensive development and in close proximity to some of the lake's largest tributaries (Kamerath et al. 2008). Water transparency at these sites is low and may explain their suitability for the UVR-sensitive bluegill. Our primary aim was to explicitly test the hypothesis that UVR controls the suitability of nearshore habitats for the earliest life history stages of exotic bluegill.

We were also interested in understanding what controls the UVR transparency of nearshore habitats in Lake Tahoe. The decline in visible light transparency has been attributed to increases in both biological (i.e., phytoplankton and detritus) and inorganic (i.e., terrestrial sediment) particulate matter (Swift et al. 2006) resulting largely from human impacts in and around the basin related to eutrophication (Goldman 1988) and stream bank erosion (Byron and Goldman 1989). However, the attenuation of UVR in freshwater lakes is strongly regulated by chromophoric dissolved organic matter (CDOM) (Morris et al. 1995, Williamson et al. 1996). CDOM may be especially important in nearshore habitats where fish spawning occurs, since CDOM inputs are likely to be concentrated in those areas. For example, in Lake Tahoe stream water inputs of CDOM are approximately 10 times higher than CDOM levels offshore where most of the long term transparency monitoring has been conducted (Swift 2004). An understanding of the mechanisms underlying UVR transparency in Lake Tahoe could enable us to better understand how regional and global environmental changes related to the factors that mediate UVR transparency could in turn affect habitat invasibility in this large, highly transparent lake.

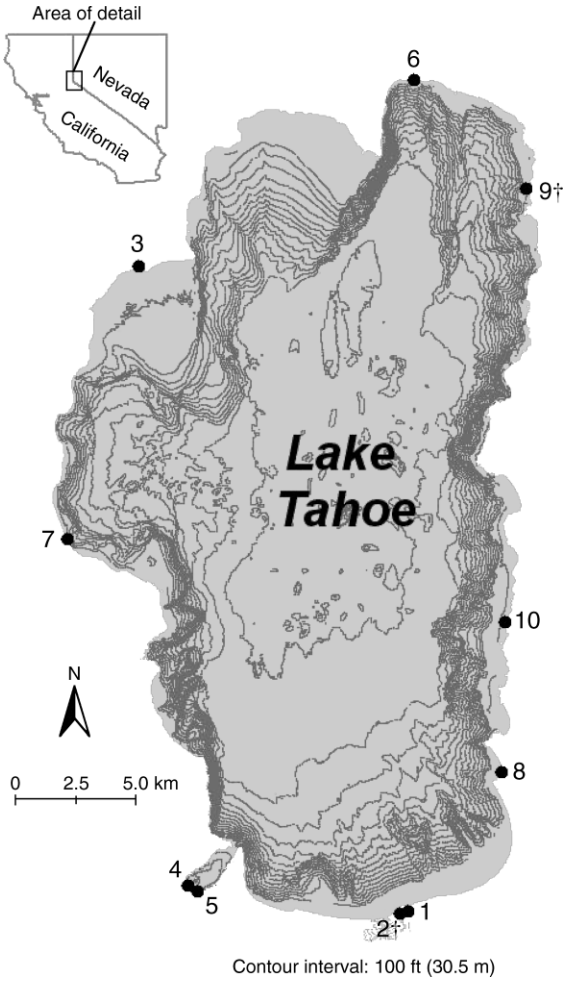

FIG. 1. Map of Lake Tahoe indicating the location of nearshore sites where DNA dosimeters were deployed. Site numbers correspond to those plotted in Fig. 4. Sites where larval incubations were deployed are indicated by a dagger.

METHODS

To test the hypothesis that UVR controls the suitability of nearshore habitats for bluegill invasion we measured UVR exposure at multiple nearshore locations around the perimeter of the lake using DNA dosimeters (Fig. 1). In two of these nearshore locations, we carried out a four day in situ incubation experiment that manipulated the incident UVR levels to which larval bluegill were exposed. DNA dosimeters were also deployed with the larval bluegill in these in situ incubations as a means for comparing levels of DNA damage in dosimeters with larval bluegill mortality. Standardized DNA damage values obtained from dosimeters incubated alone around the lake were compared to DNA damage values from dosimeters included with larval bluegill to evaluate the potential for larvae to survive in multiple nearshore locations. To assess the relative importance of dissolved organic carbon and chlorophyll as regulators of the UVR environment in nearshore areas of Lake Tahoe we measured levels of these light attenuating components at 13 nearshore sites, including each of the sites where we deployed dosimeters.

Larval incubation experiment

Larval yolk sac bluegill were collected from a single nest at approximately 1 m depth in the Tahoe Keys on 17 July 2007. Larvae ($n = 5$) were placed in Whirl-Pak bags (Nasco, Fort Atkinson, Wisconsin, USA) filled with 100 mL of 48-μm filtered lake water to exclude most zooplankton. To isolate the effect of UVR between incubation sites, the Whirl-Pak bags were either shielded from incident UVR in Courtgard (CP Films, Martinsville, Virginia, USA) sleeves or exposed to incident UVR in Aclar (Honeywell International, Morristown, New Jersey, USA) sleeves. Courtgard is a long-wave-pass plastic that transmits photosynthetically active radiation (PAR; 95% 400–800 nm in water) but

PLATE 1. Nearshore sites in Lake Tahoe with contrasting (left) high and (right) low UVR transparency. Photo credits: left, Carrie Kissman; right, A. J. Tucker.

blocks most UVR (transmits no UV-B 295–319 nm, and only 9% of UV-A 320–400 nm with a sharp wavelength cutoff and 50% transmittance at 400 nm). Aclar is a long-wave-pass plastic that in water transmits both PAR (100% 400–800 nm) and most UVR (98% of UV-B 295–319 nm, 99% UV-A 320–399 nm, with a sharp wavelength cutoff and 50% transmittance at 212 nm). The two incubation sites for the larval exposure experiment included waters with low and high UVR transparencies, that is, the Tahoe Keys and Sand Harbor areas, respectively. Four replicates of each of the UVR shielded and unshielded treatments were deployed at dusk on 17 July at one meter depth in both the high (Sand Harbor) and low (Tahoe Keys) UVR sites and retrieved early on the morning of 21 July. After collection, larvae were examined under a dissecting microscope and scored as live if a heartbeat was observed. The 84 hour incubation period used here is similar to the time it takes larvae to reach swim-up stage and leave the nest (Gross and MacMillan 1981). Average daily water temperature at 1 m over the course of the incubation was 20.4°C in Sand Harbor and 22.4°C in Tahoe Keys. These temperatures are well within threshold temperatures for bluegill spawning (Wallus and Simon 2008). All procedures involving animals were in accordance with the policies set forth by Miami University's Institutional Animal Care and Use Committee (IACUC protocol #683).

Both incident and submersible radiometers and DNA dosimeters were deployed to measure incident UVR and water transparency during the incubation. Underwater solar radiation was measured at each site with a BIC profiling UVR-PAR radiometer (BICLogger; Biospherical Instruments, San Diego, California, USA). This instrument quantifies incident solar irradiance at three different UVR wavelengths (305, 320, and 380 nm) as well as visible wavelengths of photosynthetically active radiation (PAR, 400–700 nm). Transparency data from BIC profiles were used to calculate diffuse attenuation coefficients (K_d) for each site and were combined with cumulative surface irradiance data measured with a Biospherical Instruments BICLogger, a multichannel, internally recording radiometer of a similar design and specifications to the BIC, to estimate total exposure for the duration of the incubation experiment. Two DNA dosimeters were included with fish in each of the four UVR unshielded bags and in two of the four UVR shielded bags at each site during larval fish incubations.

Logistic regression analysis of a 2^2 factorial design was performed using SAS 9.1 (SAS Institute 2005) to test for main effects of site, and UVR+ or UVR− microcosm on larval survival.

DNA dosimeters

The DNA dosimeters were 10 mm diameter by 40 mm long quartz cuvettes filled with 0.4 mL of raw salmon testes DNA solution diluted to 100 μg/mL in double

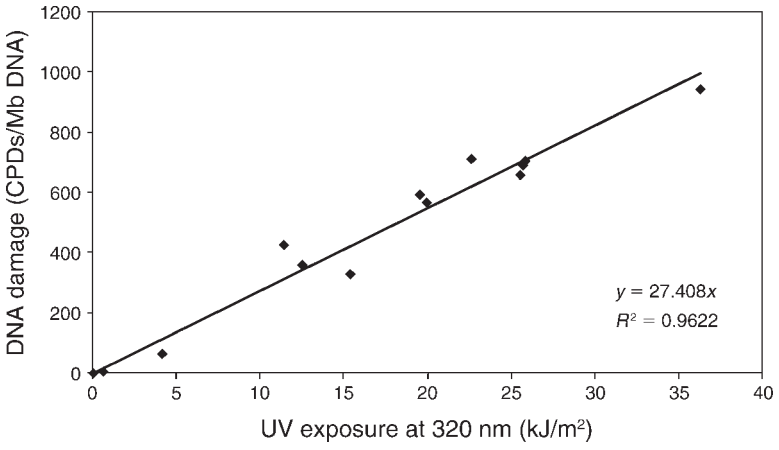

FIG. 2. Relationship between measured DNA damage and 320-nm UV exposure at 10 sites in Lake Tahoe. DNA damage values were plotted for each site as the average of dark-corrected dosimeter values (cyclobutane pyrimidine dimers [CPDs]/Mb DNA from the radioimmunoassay [RIA]) for dosimeters deployed during the one-week sampling period, 15–20 July 2007. The 320-nm UV exposure for the duration of the dosimeter deployment at a given site was derived by combining transparency data from BIC UVR-PAR profiles at a given site with cumulative surface irradiance data measured with the BICLogger, according to the equation $E_z = E_0 \times (e^{-K_d Z})$, where E_z = 320-nm UVR irradiance in kJ/m^2 at depth z (m) and E_0 = 320-nm UVR irradiance in kJ/m^2 at the water surface. Dosimeters were deployed at more than one depth at some sites.

distilled water and sealed on each end with silicone stoppers and parafilm. DNA in dosimeters accumulates damage as a function of UVR exposure, and the frequency of cyclobutane pyrimidine dimers (CPDs) per mega-base (Mb) of DNA, the most common photoproduct, were estimated using radioimmunoassay (RIA) as described by Mitchell (Mitchell 1996, 1999).

Dosimeters were included with fish in larval incubation experiments as described in *Methods: Larval incubation experiment*. Dosimeters ($n = 4$) were also deployed alone for approximately four days during the period between 15–20 July 2007 at 1- and 2-m depths at 10 sites around Lake Tahoe, including the two larval incubation sites. These dosimeters were placed in Whirl-Pak bags filled with 100 mL of 48-μm filtered lake water and then inserted into Aclar sleeves. Because the dosimeters deployed alone were incubated at multiple depths and for various times a relationship between 320-nm UVR exposure and DNA damage values was derived and then used to estimate DNA damage for each site around the lake based on a standard 320-nm UVR exposure. We used a standard exposure equivalent to the cumulative incident (i.e., surface) 320-nm UVR exposure for larval incubation experiments, so that estimated DNA damage values for sites around the lake are comparable to DNA damage values from dosimeters that were included in the larval fish incubations. In more detail, the estimated DNA damage values based on a standard exposure were derived as follows.

First, using UVR exposure data and DNA damage values obtained during dosimeter incubations 15–20 July, the relationship of 320-nm UVR exposure to measured DNA damage was derived (Fig. 2). A linear relationship between measured DNA damage and UV 320-nm exposure was observed and the equation describing this relationship was derived by simple linear regression ($y = 27.408x$, $R^2 = 0.9622$). The cumulative 320-nm UVR exposure for the duration of the dosimeter deployment at each given site was derived as described above for the larval incubation experiment, combining transparency data from BIC profiles at a given site with cumulative surface irradiance data measured with the BICLogger. Measured DNA damage values for each site were plotted as the average of dark corrected dosimeter values in CPDs/Mb DNA from the RIA.

Next, the 320-nm UVR exposure at 1 m depth for a given site was calculated for a standard surface exposure. The 1 m depth is equivalent to the depth at which dosimeters were deployed with the larval fish. The standard surface exposure was 26.3 kJ/m^2, the mean value of 320-nm surface exposure for the duration of the larval fish incubations. For reference, the highest incident UVR exposure that we observed with the BIC logger at Lake Tahoe for a single, full day of 320-nm UVR exposure was 9.73 kJ/m^2 on 17 July 2007. This is essentially equivalent to one exposure day (sensu Williamson et al. 1999). The estimated 320-nm UVR exposure at 1 m depth for each site (E_{1ms}) for the 26.3 kJ/m^2 standard surface exposure was then derived from the equation that describes the attenuation of light in water:

$$E_{1ms} = E_{0std} \times (e^{-K_d z})$$

where E_{0std} = 26.3 kJ/m^2, integrated 320-nm surface irradiance for the duration of the larval fish dosimeter deployment; K_d = diffuse attenuation coefficient, derived as the slope of the semi-log plot from vertical UVR profiles at a given site; and $z = 1$ m, standard exposure depth.

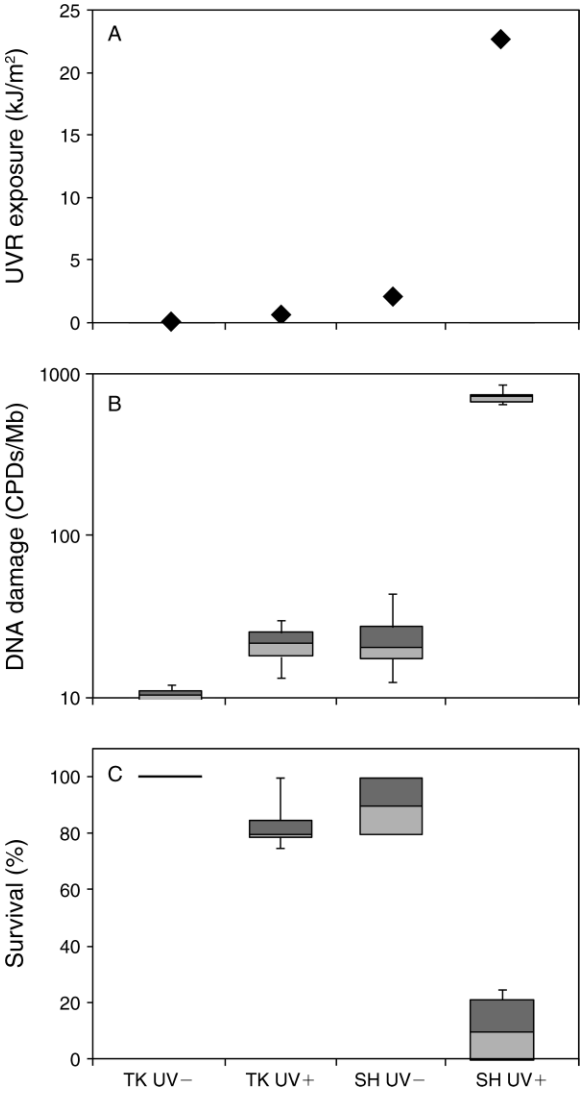

FIG. 3. (A) Ultraviolet radiation (UVR) exposure levels, (B) DNA damage, and (C) survival of bluegill larvae in experimental microcosms. UVR exposure levels in the experimental microcosms were estimated from incident UVR measurements. DNA damage was measured in DNA dosimeters incubated in microcosms with larvae. Survival of bluegill larvae was measured after an 84-hour incubation in low (TK, Tahoe Keys) and high (SH, Sand Harbor) UVR sites when shielded (−) and unshielded (+) from incident UVR. For panels B and C, bars indicate maximum and minimum values within treatments. Boxes indicate the median and 25th and 75th percentiles.

Finally, this E_{1ms} value was inserted into the equation describing the relationship of 320-nm UVR exposure to DNA damage (i.e., $y = 27.408x$). The resulting value (y) was the estimated DNA damage value based on this relationship (from Fig. 2).

DOC and chlorophyll a *analysis*

Water samples were collected in pre-rinsed 1-L polyethylene bottles from within the mixed layer. Water used in dissolved organic carbon (DOC) analysis was filtered through pre-ashed 25-mm 0.7-μm Whatman GFF filters within 8 hours of sample collection using a glass filter support. The filtered sample was stored in the cold and dark in 40-mL glass bottles until analysis. The DOC samples were analyzed with a Shimadzu TOC-V_{CPH} analyzer (Shimadzu, Columbia, Maryland, USA) within one week post sampling. For chlorophyll *a*, 100 mL of the water sample was filtered through pre-ashed 25-mm 0.7-μm Whatman GFF filters within 8 hours of collection and the filter was immediately frozen until chlorophyll analysis. Chlorophyll *a* extraction was completed with an acetone–methanol mixture and chlorophyll *a* concentration was completed via fluorometry within one month of sample collection. UVR attenuation was also measured at each site with the BIC profiling radiometer, and diffuse attenuation coefficients (K_d) were calculated for each site from the slope of the natural log relationship of UVR irradiance vs. depth. Using SAS v. 9.1, we performed a likelihood ratio test to compare models that predicted $K_{d,320nm}$ from DOC and/or chl *a* values.

RESULTS

For the larval incubation experiment, exposure to 320-nm radiation in unshielded treatments was nearly 40× higher in the Sand Harbor site (22.65 kJ/m^2) compared to the Tahoe Keys site (0.60 kJ/m^2, Fig. 3A). The mean DNA damage levels, measured in DNA dosimeters as the frequency of cyclobutane pyrimidine dimers (CPDs), at the Sand Harbor site were more than 30× higher than those measured at the Tahoe Keys site (729 vs. 22 CPDs/Mb DNA, Fig. 3B). Larval survival was inversely related to UVR exposure with 84% of larvae surviving in unshielded microcosms in the low UVR site and only 11% survival in the high UVR site (Fig. 3C). For bluegill in unshielded UVR microcosms, there was a statistically significant effect of site on larval fish survival (PROC LOGISTIC; $P < 0.0001$). In the UVR-shielded microcosms, larval survival was high (90–100%) at both sites. DNA damage measured in the dosimeters also increased with increasing UVR transparency across the ten sample sites (Fig. 4). In seven of the 10 sample sites DNA damage levels were higher than those measured at the Tahoe Keys, where bluegill survival was high. Indeed, the majority of sites showed DNA damage levels above the threshold for larval survival (Figs. 3B and 4), implying high potential UVR-induced mortality in bluegill at most sample sites.

The 1% attenuation depths, that is the depth where 320-nm UVR reaches 1% of surface irradiance, show the wide range of UVR transparency of nearshore sites in Lake Tahoe (Table 1). UVR (320 nm) transparency of the near shore sites was strongly dependent upon DOC ($K_{d,320nm} = [2.57 \times DOC^{2.53}]$; $R^2 = 0.81$). However, a model that included both DOC and chlorophyll *a* ($K_{d,320nm} = [1.95 \times DOC^{3.01}] + (0.02 \times \text{chl } a]$) was the best predictor of UVR attenuation ($R^2 = 0.98$) for the

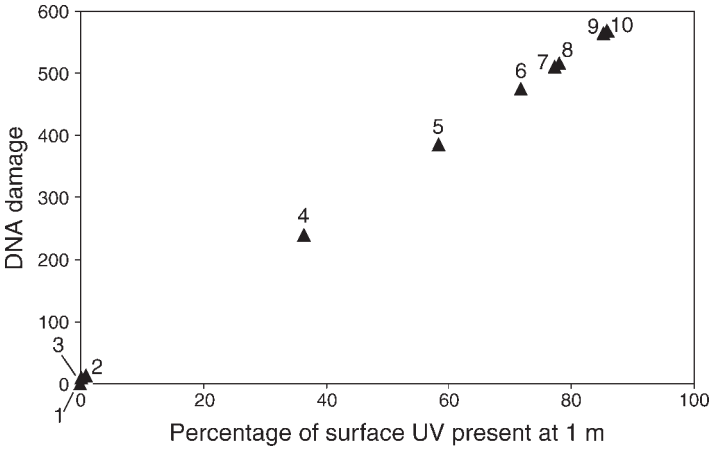

FIG. 4. Relationship showing the increase in DNA damage (measured as CPDs/Mb DNA) with increasing UVR transparency (indicated here by the percent of incident surface 320-nm UVR present at 1 m depth). DNA damage values are estimated from a derived exposure vs. dosimeter value relationship and are standardized for depth and deployment duration for comparison to larval fish incubation experiments. Site numbers appear above data points: site 2 is the low-UVR Tahoe Keys; site 9 is high-UVR Sand Harbor.

sites sampled (likelihood ratio chi-square = 11.2, df = 1, $P = 0.0008$).

DISCUSSION

In this study, dosimeters of raw DNA in solution were used as tools to assess potential UVR effects on larval bluegill by relating DNA damage levels in dosimeters with larval fish mortality. The observed levels of DNA damage in the dosimeters support the hypothesis that UVR is a potent force contributing to the suitability of nearshore habitats for successful bluegill reproduction. Current UVR conditions were substantial enough to reduce reproductive success of bluegill in the majority of nearshore sites sampled. Both DOC and chlorophyll *a* were important regulators of variation in the UVR environment in nearshore areas of Lake Tahoe. This suggests that effective regulation of chlorophyll and DOC inputs could stem future declines in UVR transparency in Lake Tahoe and in turn help mediate habitat invasibility.

Our study was motivated by a framework for predicting species invasion that highlights the importance of identifying the specific abiotic factors that will ultimately constrain distribution in an invaded range. Current approaches for predicting habitat invasion tend to rely on correlating species' distribution with selected habitat parameters that implicitly incorporate biotic constraints on distribution. These biotic constraints may not always be present in an uninvaded range (Kearney and Porter 2004). It has been argued that a more powerful approach is to identify specific abiotic factors with demonstrable fitness consequences for an organism, and then map the fitness consequences (e.g., survival or reproduction) at various levels of the abiotic factor onto the landscape (Kearney 2006). This kind of approach is fundamental if we wish to improve our confidence in extrapolating species' potential distributions to novel circumstances under climate change scenarios, and it could be especially useful for predicting invasions in systems where a specific factor regulating invasion (e.g., UVR) is closely tied to a global change element (e.g., climate driven changes in DOC). In our study, we have accomplished the crucial first step in this approach by demonstrating that UVR is a key abiotic factor with the potential to constrain the reproductive success of bluegill in Lake Tahoe. By identifying some of the key mechanisms underlying UVR transparency we have also increased our understanding of how regional and global environmental changes related to the factors that mediate UVR transparency could in turn affect habitat invasibility in this lake. We suspect that this framework and our results could be directly relevant to other transparent lakes.

Whereas few lakes are as highly transparent as Lake Tahoe, estimates from DOC measurements in North American lakes indicate that UVR transparency is relatively high throughout western, northwestern, and southeastern portions of the USA (Williamson et al. 1996). For example, based on modeling the relationship between DOC concentration and UVR attenuation, the depth to which 1% of 320-nm UVR surface irradiance penetrates is greater than 1 m in 75% of lakes sampled in the western United States. About 25% of these lakes

TABLE 1. Attenuation depths, Z, for 320-nm ultraviolet radiation (UVR) at nine sites in Lake Tahoe.

Site number	$Z_{1\%320nm}$ (m)	DOC (mg/L)	Chl *a* (μg/L)
1	0.4	1.77	2.47
2	1.3	1.24	12.20
3	1.1	1.00	144.70
5	8.6	0.66	0.95
6	14.0	0.53	1.81
7	17.8	0.58	0.42
8	18.6	0.58	0.58
9	28.8	0.53	0.32
10	30.3	0.51	0.52

Notes: $Z_{1\%320nm}$, is the depth where 320 nm UVR reaches 1% of surface irradiance. Site numbers correspond to those plotted in Figs. 1 and 4. The 1% attenuation depths were estimated from the diffuse attenuation coefficient K_d as: $Z_{1\%} = 4.605\,K_d^{-1}$ where $K_d(\lambda, z) = [\ln(I_0/I_Z)]/Z$; Z is depth (m), and I is down-welling irradiance for a given wavelength (λ) at the surface (I_0) and at depth Z (I_Z). Also shown are dissolved organic carbon (DOC) and chlorophyll *a* values for each of the nearshore sample sites (excluding site 4, where only UVR data were collected).

exhibit 1% UVR depths greater than 4.75 m. This is noteworthy because bluegill generally nest at depths less than 4 m (Carlander 1977), and other studies have demonstrated significant UVR effects on reproductive success of temperate fish species (including bluegill) in the eastern USA in lakes with a 1% UVR depth not in excess of 4.9 m (Huff et al. 2004, Olson et al. 2006).

The DOC concentration in most of the transparent lakes referenced above is quite low (i.e., <1 mg/L), suggesting that even small changes in DOC could significantly reduce current UVR levels in these lakes (Williamson et al. 1996). Although there are no specific predictions for future DOC concentrations in western and southeastern U.S. lakes, widespread and strong trends of generally increasing DOC concentrations have been observed in lakes and rivers elsewhere (Evans et al. 2005, Monteith et al. 2007). Therefore it is reasonable to consider the potential for substantial changes in UVR transparency, and consequently habitat invasibility in these transparent lakes. Just as relevant and better documented in high elevation transparent lakes like Tahoe are trends of increased algal growth and reduced water clarity as a consequence of increased nitrogen deposition (Jassby et al. 1994, 1995, Sickman et al. 2003). These trends, documented in the western United States, are predicted to continue across that region (Lamarque et al. 2005). Chlorophyll has a proportionately greater effect on UVR attenuation in low-DOC systems (Laurion et al. 2000, Sommaruga and Augustin 2006). Consequently variations in chlorophyll levels, like changing DOC concentrations, have the potential to modify transparency in very low DOC lakes. This in turn could facilitate the establishment of exotic species in formerly unsuitable habitats.

One critical question pertinent to the role of UVR in mediating habitat invasibility in transparent lakes is whether adult bluegill are able to respond to these selective pressures by reducing UVR exposure through either nesting deeper or shifting their spawning time to coincide with periods of decreased water transparency. For Lake Tahoe, this seems an unlikely possibility. First, in this study the 1 m depth and the seasonal timing of our experiments were consistent with actual nest depths and nesting times in Lake Tahoe. Moreover, later spawning times, coincident with increasing water temperatures that might allow bluegill to nest at greater depths, are unlikely to decrease UVR exposure because UVR transparency (320 nm) actually increases on the order of 20–90% from spring to summer as allochthonous inputs decrease in the nearshore (Rose et al. 2009). On the other hand, accelerated spawning phenologies that could potentially enable bluegill to benefit from decreased water transparency earlier in the growing season are likely constrained by thermal conditions required for spawning. Bluegill are reported to spawn at temperatures from 15.6°C to 32°C, with optimum spawning temperatures in the range of 21°C to 24°C (Wallus and Simon 2008). Surface water temperatures measured at an index site in May 2007 and 2008 never exceeded 11.1°C, well below the minimum spawning temperature. Even in June, the maximum surface water temperature over this two year period was 17.2°C (C. E. Williamson, *unpublished data*), still below the optimal spawning temperature for bluegill. Thus the primary opportunity for invasion is likely to be in the shallow nearshore embayments where both water temperatures are high enough and transparency to UVR is low enough to permit adult spawning and larval survival.

It is also important to note that we have used the most severe response metric (i.e., mortality) in evaluating the role of UVR for regulating habitat suitability for larval bluegill invasion. Consequently, our study likely underestimates the full extent of UVR induced effects on larvae when considered in terms of the interactions of sublethal effects with sources of background mortality in developing larvae and other "life history bottlenecks" that young fish face. For example, UVR exposure impedes larval growth in a variety of fish species (Hunter et al. 1979, Winckler and Fidhiany 1996, Vehniainen et al. 2007) and body size in young fish, including bluegill, is a critical determinant of overwinter survivorship and mortality due to predation (Belk and Hales 1993, Cargnelli and Gross 1996). Other potential sublethal UVR effects that may ultimately reduce bluegill survival include diminished immune system function and increased incidence of infectious disease resulting from "sunburn" (Salo et al. 1998, Nowak 1999), developmental anomalies that might increase susceptibility to predators (Vehniainen et al. 2007), indirect trophic mediated UVR effects on food availability (Williamson et al. 1994, Zagarese and Williamson 2001), or phototoxic effects (Bullock and Roberts 1979, Oris and Geisy 1987).

It is unclear to what extent UVR may play a role in the invasion ecology of other invasive species or other life history stages. We contend that it could have relevance for any UVR sensitive species that is constrained to shallow water environments by, e.g., requirements for warmer spawning temperatures in clear, cold-water lakes. For older more tolerant and mobile life history stages other biotic and/or abiotic factors (e.g., food availability or habitat structure) likely play a more important role in determining habitat suitability. However, we have emphasized the earliest life history stages here for two reasons. First, early life history stages are less pigmented, less mobile, and thus likely to be less well-defended against UV damage. Second, in a biological invasion context the naturalization and eventual invasion of a species in a novel environment depends critically on the ability of that species to establish self-perpetuating populations through successful reproduction (Richardson et al. 2000). Whereas other among-habitat characteristics may be important in regulating species invasions, we have shown that for these critical early life history stages UVR alone is an adequate

determinant of habitat suitability and thus a potential regulator of habitat invasibility.

The extent to which UVR ultimately controls bluegill invasion in Lake Tahoe or any other system will depend upon the potential for these organisms to adapt to local conditions. It is possible for example, that constitutive levels of maternally derived photoprotective compounds (PPCs) could increase in larval fish spawned in high UVR environments, thereby increasing UVR tolerance and the ability to spread into new habitats. High UV environments tend to stimulate PPC synthesis by algae and bacteria. These can be transferred in food chains and accumulated at higher trophic levels by organisms that have such capability, which may in turn be enhanced by UV exposure (e.g., copepods [Tartarotti et al. 2004, Moeller et al. 2005, Tartarotti and Sommaruga 2006]; coral reef fish [Zamzow 2004]).

Environmental stress is often considered a driver of adaptation during invasion and it has been demonstrated that abiotic conditions can select for adaptive genotypes in invasive species (Lee et al. 2007). Our data suggest that UVR can similarly act as a selective force in highly transparent systems, and the potential for the development of more resistant genotypes could be tested. Future research concerning the role of UVR in controlling biological invasion should consider these and other possibilities. Nevertheless, we have shown that for the current bluegill population in Lake Tahoe UVR is a potent stressor that mediates habitat suitability for larval fish in nearshore areas and therefore controls habitat invasibility.

Further efforts to quantify the effect of abiotic controls on the growth, survival, and reproduction of organisms and to map those effects onto the landscape will help us to more accurately predict the full potential of species invasion in imperiled environments. Knowledge of the particular levels of important abiotic factors that reduce the fitness of non-natives could also enable us to manage abiotic conditions in habitats for the prevention of species invasion (Alpert et al. 2004). In lakes, for example, one goal might be to establish and manage UVR transparency thresholds that prevent the establishment of non-native species by inhibiting successful reproduction. We suggest that future studies in highly transparent aquatic ecosystems consider UVR and other abiotic habitat features as important factors controlling habitat invasibility and invasion risk.

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