ONLINE SUPPLEMENTAL INFORMATION

More phototron methods

The phototron in experiments #1-#3 used UV-B but also UV-A and PAR. A UV-B lamp (Spectronics Spectroline BLE-1T158 15 W lamp, Westbury, NY) was covered with a fresh piece of cellulose acetate to remove wavelengths less than 295 nm and suspended 24 cm above the rotating wheel. Photorepair radiation (PRR, predominantly longer wavelength UV-A and PAR) was supplied from below the rotating wheel using two 40-W cool-white fluorescent bulbs (primarily PAR) and two 40-W Q-Panel 340 bulbs (primarily UV-A). The spectra of these lamps have been published (Williamson *et al.* 2001). Only exposure to the UV-B lamp was manipulated in radiation treatments. PRR, when present, was kept constant across treatments. Since we did not know if *M. bicuspidata* employed photoenzymatic repair, we conducted the initial experiment (experiment #1) with photorepair radiation (PRR) as well.

Statistical methods

We analyzed infection and survival data using more standard GLM-based statistics. In Table S1, we present full results from models fit to the multi-factorial experiments, #1 and #5. In the text, unless we refer to non-significant interactions, we present results from best-fitting models (determined using AICc). Here, we characterize performance of the full models built with all possible interactions. We also fitted competing functions for transmission and survival to the data using maximum-likelihood-based and information-theoretic techniques. Since the latter approaches are less standard, we expand on them here (see also Table S2 for results of the model competition and Table S3 for parameter estimates, with likelihood profiled confidence intervals, estimated for the winning models).

<u>Lab-based susceptibility assays (Experiments 1, 3, 4):</u>

We estimated susceptibility of hosts and infection success of parasites by fitting

differential equations to the data produced from the various infection assays. Those equations estimated the per host, per spore transmission rate, β, from a standard density-dependent rate epidemiological model for infection. In this model, susceptible hosts (S) become infected (I) by contacting fungal spores (Z) at rate β, or:

$$dS/dt = -\beta_j(U)SZ\ ;\ dI/dt = \beta_j(U)SZ\ . \qquad \text{(S1)}$$

This transmission rate parameter, in turn, is a function of exposure of parasites to ultraviolet radiation, U. We modeled the mapping of U to $\beta_j(U)$ using one of three assumptions:

'Null': $\beta_j(U) = \beta_{0,j}$ (S2.a)

'Linear UV': $\beta_j(U) = \beta_{0,j}\exp(b_j U)$ (S2.b)

'Power UV': $\beta_j(U) = \beta_{0,j}\exp(b_j U^{c_j})$. (S2.c)

In the 'null' model (equ. S2.a), transmission rate does not depend on exposure of fungal spores to ultraviolet radiation. In the 'linear UV' model (equ. S2.b), transmission rate increases or decreases (exponentially) according to slope parameter b_j. In the 'power UV' model (equ. S2.c), the influence of UVR on transmission rate depends on slope parameter b_j and exponent c_j; the added parameter permits $\beta_j(U)$ to take on more flexible shapes. The exponentiation function, exp(…), prevents transmission rate from becoming negative. The 'j' subscripts allow for separate parameters to be estimated for a *Daphnia* exposure effect (0 for no direct exposure, 1 for direct exposure to UVR). We use separate parameter estimates as opposed to a function here because *Daphnia* were exposed to only two levels of UVR. If we assume no difference among *Daphnia* treatments, then we just estimate the same parameter for both treatments (i.e., only β_0 is estimated, assuming $\beta_{0,0} = \beta_{0,1}$; similarly, only b is estimated, assuming $b_1 = b_2$, etc.).

Thus, with this structure, we readily tested six competing models for Experiment #1 (Tables S2, S3). The simplest null model assumed that UVR did not diminish the spores' per spore infectivity, even though it could increase or decrease the *Daphnia*'s susceptibility (1 vs. 2 parameter versions). We then created the two corresponding versions of both the 'linear' and the 'power' models, too (see Table S2). In the other two assays of transmission rate, we did not expose *Daphnia* to UVR. Hence, we only fit three models to each dataset (i.e., no j subscripts

were needed when fitting the models in equ. S2).

Survivorship:

We competed several models to quantify effects of UVR on survival of *Daphnia*. Our approach used a standard method (McCallum 2002) for estimating instantaneous per capita mortality rate of hosts, ρ. If we assume that this death rate is a function of ultraviolet radiation, U, then two survival models merit consideration:

Exponential model: $S(t,U) = \exp\left(-\rho(U)t\right)$ (S3.a)

Weibull model $S(t,U) = \exp\left(-\left[\rho(U)t\right]^k\right)$. (S3.b)

where survival, S, is a function of time of exposure, t, and level of exposure, U. In the exponential model (equ. S3.a), death rate $\rho(U)$ is assumed constant per unit time. In the Weibull model (equ. S3.b), death rate increases or decreases through time, depending on the value of dimensionless parameter, k. Since we use survival data up to day five, the exponential and Weibull model both provide identical fits with different parameter estimates (although the Weibull model gets penalized in the AIC calculation for having an extra parameter to estimate). Thus, we only present the results from the exponential model (Tables S2, S3). However, the death rate parameter can become a function of the intensity of UVR in one of three ways:

'Null model': $\rho(U) = \exp(a)$ (S4.a)

'Linear UV': $\rho(U) = \exp(a)\exp(bU)$ (S4.b)

'Power UV': $\rho(U) = \exp(a)\exp(bU^c)$. (S4.c)

In the null model (equ. S4.a), death rate does not depend on UV (U) radiation but instead remains at a fixed background rate (i.e., $\rho = \exp(a)$). In the linear model (equ. S4.b), death rate depends on this fixed background rate times an exponentiated function of UV, governed by linear slope parameter b. In the power model (equ. S4.c), this function of UV has two parameters, slope b and exponent c. The exponentiations, exp(…), ensure that death rate, $\rho(U)$, remains positive for all values of a, b, c, and U.

Field surveys:

We use a partial regression analysis with the field data. This simple method (Legendre and Legendre 1998) involves computing a multiple linear regression of maximum prevalence (P_{max}) against a_{d320} and start date (D_S) together, a linear regression of P_{max} against a_{d320}, and a linear regression of P_{max} against D_S. These yield, respectively, the total fraction variation explained [a+b+c], the sum of [a+b], and the sum of [b+c]. Variation explained solely by a_{d320} is found by the difference of [a+b+c] - [b+c]. Similarly, that explained by start date alone is found by the difference of [a+b+c] - [a+b], while the variation explained by the interaction is [a+b] + [b+c] - [a+b+c].

Estimates of UVR exposure in the field experiment (#5)

It is challenging to equate lab-based levels of UVR exposure to that received by organisms in the field. Simply put, solar radiation has a different spectral composition than artificial bulb-produced radiation. The very strong differences in biological effectiveness of photons of different wavelengths in the UV waveband thus prohibit comparison of solar UV and UV from artificial bulbs on an energy basis (KJ) without a weighting function. To deal with this issue, we measured incident UVR during the field experiment (#5) and used prior knowledge of *Daphnia* biological weighting functions to assure that UV exposure levels were reasonable in our phototron experiments. The BIC radiometer (Biospherical Instruments, Inc., San Diego, CA) measured radiation at one-minute intervals at wavelengths that span the UV-B, UV-A, and PAR regions of the light spectrum (specifically, 305 nm, 320 nm, 380 nm, and 400-800nm). On a cloudless day near summer solstice, under normal ozone, we estimate that the daily amount of 320 nm UVR exposure incident on the lake surface (320nm exposure day) would be 11.4 KJ m^{-2}, the highest level chosen in our lab-based experiments (see Cooke & Williamson 2006 for details of the calculations using biological weighting functions). However, at the shallowest incubation depth in the field experiment, mixed meteorological conditions led spores to receive a total of only 7 to 7.25 KJ m^{-2} during the entire four day experimental period (from 30 July to 3 August

2010). This exposure is less than two-thirds of the maximum level they could receive on a single summer day. Thus, the strong effects seen in the field experiment were produced under even very low supply of incident UVR. We also used depth profiles from the BIC radiometer to estimate diffuse attenuation coefficients (extinction coefficients) of 320 nm radiation, where k_{d320} is the slope of the relationship between the natural log of 320 nm radiation vs. depth.

More data from the field survey: DOC, *Chaoborus*, and light extinction (PAR)

During our lake surveys, we also estimated three additional, pertinent factors related to the UVR-epidemic size patterns presented in the text.

Dissolved organic carbon (DOC):

We measured dissolved organic carbon (DOC) using samples from which we estimated dissolved absorbance (a_{d320}). Filtered water samples (Whatman GF/F, 0.7 µm) were analyzed for DOC (using a Shimadzu TOC-V_{CPH} Total Organic Carbon Analyzer). The DOC and a_{d320} metrics were strongly and positively correlated ($r = 0.74$, $P = 0.0005$): the UV penetration index was smaller, i.e., lakes were more transparent to UVR, when DOC was lower.

Light environment:

During lake visits, we estimated penetration of photosynthetically active radiation (PAR). The laboratory and field-based assays showed sensitivity of fungal spores to PAR. In the field, PAR penetration is indexed using a light meter (Li-Cor, Lincoln, Nebraska, USA) and fitting a linear regression between ln-transformed irradiance, $I(z)$, measured at 1 m intervals (0-4 m, duplicate measurements), and depth, z: $\ln(I[z]) = a - kz + \varepsilon$ (with slope a and residual errors ε). The light extinction coefficient, k, is the slope of that relationship; high values of k indicate low penetration of PAR. Lakes with more light extinction (higher k, lower PAR penetration) had higher values of a_{d320} (lower UV penetration; Fig. S1.A); however, lakes with more DOC did not have significantly more light extinction (although the trend was positive: $R = 0.38$, $P = 0.12$).

These lakes with more light extinction (less PAR penetration) also had larger epidemics (Fig. S1.B) that started earlier in the season (Fig. S1.C).

Chaoborus densities:

We estimated densities of *Chaoborus punctipennis* in the zooplankton samples collected in August. We present counts of third and fourth instars of this predator. Analysis of the relationship between UVR transparency and *Chaoborus* density was slightly complicated due to the outlier denoted with the arrow (Fig. S1.D). To reduce the impact of that outlier, we fitted a regression model using iteratively weighted least squares and the Huber weighting function (see Neter *et al.* 1996 for details). Using this procedure, lakes with more *Chaoborus* had higher values of $a_{d320,}$ i.e., less UVR penetration. Additionally, lakes with higher *Chaoborus* density had larger epidemics (Fig. S1.E). However, start date of the epidemics were not correlated with *Chaoborus* density (Fig S1.F).

Reference cited only in the *Online Supplementary Information*

Burnham K.P. & Anderson D. (2002). *Model selection and multi-model inference*. Springer, New York.

Cooke S.L. & Williamson C.E. (2006). Positive effects of UV radiation on a calanoid copepod in a transparent lake: do competition, predation or food availability play a role? *J. Plankton Res.*, 28, 171-179.

Legendre P. & Legendre L. (1998). *Numerical ecology*. 2nd English edn. Elsevier, Amsterdam ; New York.

McCallum, H. 2000. Population parameters: estimation for ecological models. Wiley-Blackwell.

Neter, J., M. H. Kutner, C. J. Nachtsheim, and W. Wasserman. 1996. Applied linear statistical models, 4th ed. WCB McGraw-Hill.

Table S1. Analysis of deviance results from GLM analyses of Experiments #1 and #5. (* indicates results significant at 0.05 level).

165

Experiment	Df†	Deviance	F†	Pr(>F) †
Experiment 1: Both Exposed				
UVR-fungus	2	105.66	60.99	<0.01*
Spore density	1	28.42	32.81	<0.01*
UVR-host	1	0.39	0.63	0.43
UVR-fungus x spore density	2	3.17	2.55	0.08
UVR-fungus x UVR-host	2	0.22	0.18	0.84
Spore density x UVR-host	1	1.30	2.09	0.15
UVR-fungus x spore density x UVR-host	2	1.74	1.40	0.25
Experiment 5: Field Experiment				
Radiation	2	224.02	75.60	<0.001*
Depth	1	2.23	1.51	0.22
Lake	1	15.14	10.22	0.002*
Radiation x depth	2	10.10	3.41	0.04*
Radiation x lake	2	10.81	3.65	0.03*
Depth x lake	1	0.94	0.64	0.43
Radiation x depth x lake	2	0.63	0.21	0.81

† Df: degrees of freedom, F: F statistic, Pr(>F): p-value.

Table S2. Results from competition among hypotheses that model how transmission rate (host susceptibility and per spore infectivity) depends on exposure of fungal spores and/or host *Daphnia* to different levels to ultraviolet radiation (UV) and photorepair radiation (PRR).

	Daphnia	**Information theoretic statistics**				
Model	**UV effect***	**NLL†**	***K*†**	**AIC_c†**	**AIC Δ†**	**AIC *w*†**
Experiment 1: Both exposed						
Power UV	N	**70.18**	**3**	**146.59**	**0.00**	**0.88**
Linear UV	N	73.90	2	151.91	5.32	0.06
Power UV	Y	69.72	6	152.27	5.68	0.05
Linear UV	Y	73.40	4	155.20	8.61	0.02
Null	N	125.81	1	253.66	107.07	0.00
Null	Y	125.55	2	255.21	108.62	0.00
Experiment 2: Host survival						
Power UV	--	**102.2**	**3**	**210.4**	**0**	**0.995**
Linear UV	--	108.6	2	221.1	10.7	0.005
Null	--	178.4	1	358.8	148.3	0
Experiment 3: Parasite exposure, +PRR						
Power UV	--	**41.6**	**3**	**89.2**	**0**	**0.998**
Linear UV	--	48.9	2	101.9	12.7	0.002
Null	--	68.6	1	139.2	50.0	0
Experiment 4: Parasite exposure, -PRR						
Power UV	--	**93.7**	**3**	**193.7**	**0**	**0.84**
Linear UV	--	96.4	2	197.0	3.3	0.16
Null	--	114.9	1	231.8	38.1	0

* An effect of UV exposure on *Daphnia*, estimated with different parameters for each exposure

treatment (j = 1 for exposure, j = 0 for no exposure).

175 † NLL: negative log-likelihood; K = number of parameters estimated; *AICc* = small sample size-corrected Aikaike Information Criterion; *AIC Δ*: AIC deltas, the difference between smallest AIC_c and all other models; *AIC w*: Aikaike weights, the relative likelihood of the model given the data and the other models (Burnham and Anderson 2002).

Table S3. Maximum likelihood-based parameter estimates for the best fitting models described in Table S2. For each experiment, the three-parameter 'power' model provided the best fit. Each parameter estimate is accompanied by profiled, 95% confidence intervals.

Expt. number	Description of model	Winning model*	Coefficient β , a †	Slope b ‡	Exponent c §
1	Both exposed	'Power'	1.63×10^{-6} (1.25×10^{-6}, 2.08×10^{-6})	-1.36 (-0.41, -3.62)	0.33 (0.00, 0.80)
2	*Daphnia* survival	'Power'	-5.02 (-6.58, -3.99)	0.65 (0.20, 1.65)	0.49 (0.32, 0.73)
3	Fungus exposure, +PRR	'Power'	1.45×10^{-6} (8.29×10^{-7}, 2.35×10^{-6})	-1.90 (-1.06, -2.86)	0.29 (0.079 , 0.56)
4	Fungal exposure, -PRR	'Power'	1.10×10^{-6} (8.32×10^{-7}, 1.48×10^{-6})	-7.84×10^{-4} (-1.04×10^{-7}, 3.34×10^{-2})	2.91 (1.21, 6.20)

* For experiments 1, 3, and 4, the power model is equ. S2.c; for experiment 2, it is equ. S3.c.

† Units: for experiments 1, 3, and 4 (β): $(\text{spores/L})^{-1} \cdot (\text{hosts/L})^{-1} \cdot \text{day}^{-1}$; for experiment 2 ($a$): day^{-1}. ‡ Units: for experiments 1-4: $\text{m}^2 \cdot \text{kJ}^{-1}$. § Units: none.

Figure S1. Relationships between the UV transparency index, a_{d320}, and metrics of fungal epidemics (maximal prevalence and start time [ordinal date]) with two factors, (A)-(C) light extinction coefficient, and (D)-(F) density of the invertebrate predator *Chaoborus*. *Light extinction coefficient* (high levels mean low penetration of PAR): (A) Lakes with higher a_{d320} (lower UVR penetration) have higher light extinction (shallower PAR penetration). Epidemics (B) grew to larger size and (C) started earlier in lakes with higher light extinction. *Chaoborus*: (D) Lakes with higher a_{d320} (lower UV penetration) have more *Chaoborus*. (The *P*-value here was calculated using weighted least squares but included the outlier to which the arrow points). (E) Lakes with more *Chaoborus*, in turn, have larger epidemics. (F). No relationship emerged between density of *Chaoborus* and the start date of epidemics.