Natural marine phytoplankton assemblages from Bahı́a Bustamante (Chubut, Argentina, 45°S, 66.5°W), mainly consisting of cells in the picoplankton size range (0.2–2 µm), were exposed to various UVBR (280–315 nm) and UVAR (315–400 nm) regimes in order to follow wavelength-dependent patterns of cyclobutane pyrimidine dimer (CPD) induction and repair. Simultaneously, UVR induced photosynthetic inhibition was studied in radiocarbon incorporation experiments. Biological weighting functions (BWFs) for photoinhibition and for CPD induction, the latter measured in bare calf thymus DNA, differed in the UVAR region: carbon incorporation was reduced markedly due to UVAR, whereas no measurable UVAR effect was found on CPD formation. In contrast, BWFs for inhibition of photosynthesis and CPD accumulation were fairly similar in the UVBR region, especially above 300 nm. Incubation of phytoplankton under full solar radiation caused rapid CPD accumulation over the day, giving maximum damage levels exceeding 500 $CPD \cdot MB^{-1}$ at the end of the afternoon. A clear daily pattern of CPD accumulation was found, in keeping with the DNA effective dose measured by a DNA dosimeter. In contrast, UVBR induced photosynthetic inhibition was not dose related and remained nearly constant during the day. Screening of UVBR or UVR did not cause significant CPD removal, indicating that photoreactivation either by PAR or UVAR was of minor importance in these organisms. High CPD levels were found in situ early in the morning, which remained unaffected notwithstanding treatments favoring photorepair. These results imply that a proportion of cells had been killed by UVBR exposure prior to the treatments. Our data suggest that the limited potential for photoreactivation in picophytoplankton assemblages from the southern Atlantic Ocean causes high CPD accumulation as a result of UVBR exposure.