

# **Numerical Simulation of Mortality of Zooplankton Caused by Direct Injection of Carbon Dioxide in the Ocean**

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**Abstract**

The direct injection of CO<sub>2</sub> in the deep ocean is a promising way to mitigate global warming. One of the uncertainties in this method, however, is its impact on marine organisms in the near field. Since the concentration of CO<sub>2</sub>, which organisms experience in the ocean, changes with time, it is required to develop a biological impact model for the organisms against the unsteady change of CO<sub>2</sub> concentration. In general, the LC<sub>50</sub> concept is widely applied for testing a toxic agent for the acute mortality. Here, we regard the probit-transformed mortality as a linear function not only of the concentration of CO<sub>2</sub> but also of exposure time. A simple mathematical transform of the function gives a damage-accumulation mortality model for zooplankton. In this article, this model was validated by the mortality test of *Metamphiascopsis hirsutus* against the transient change of CO<sub>2</sub> concentration. This model was installed in a computational simulation code for the reconstruction of small-scale ocean turbulence. The results suggest that the biological effect is not significant when the ship speed is 4 knots and CO<sub>2</sub> is injected at 0.1 ton/sec in the form of a spray through 100 nozzles provided vertically on a pipe at 10m intervals. It is therefore considered that the moving-ship method is effective for direct CO<sub>2</sub> injection.

**1. Introduction**

The Intergovernmental Panel on Climate Change (IPCC) reported that the partial pressure of carbon dioxide (*PCO<sub>2</sub>*) in the atmosphere will range from 550 ppm to 1000 ppm at the end of the 21st century. However, Thornton and Shirayama (2001) pointed out that even a *PCO<sub>2</sub>* of 550ppm causes nontrivial damages to benthos, such as sea urchins, in surface water. This implies that even the IPCC's most conservative scenario may not be adequate for the sustainability of marine ecosystems in the surface water. What we need is certain methods to supply large amounts of cheap energy in place of fossil fuels and, otherwise, to sequesterate CO<sub>2</sub>. One of the promising techniques for the latter option is the direct injection of CO<sub>2</sub> in the deep ocean (e.g. Ohsumi,1995; Broecker, 1997; Sato and Sato, 2002; Herzog *et al.*, 2003). An uncertainty in this method is its impact on marine organisms near injection points before CO<sub>2</sub> is diluted widely in the ocean. Since field experiments cost enormously, computational models are alternatively expected to simulate the biological impact near the injection points.

Recent researches, such as Kikkawa *et al.* (2004), elucidated that the biological impacts of CO<sub>2</sub> in the ocean should not be related to *pH* but to *PCO<sub>2</sub>* itself, because the same low *pH* caused by CO<sub>2</sub> as that by some strong acid agents, such as hydrochloric and sulphuric acids, result in more damages on marine organisms than the cases by the above acids. Therefore, currently, collecting data on biological damage by CO<sub>2</sub> is a pressing need in the marine biological society (e.g. Kita and Ohsumi, 2004; Portner *et al.* 2004; Riebesell, 2004; Kurihara *et al.*, 2004a; Kurihara *et al.*, 2004b).

This article presents a model to predict the acute mortality of zooplankton caused by *PCO<sub>2</sub>* in seawater and computational simulations to predict the acute biological impact of CO<sub>2</sub> on marine organisms using a combination of the mortality model of zooplankton and a computational fluid dynamics (CFD) method for the reconstruction of small-scale ocean turbulence.

**2. Modelling of Acute Mortality**

When marine organisms are in the rising plume of CO<sub>2</sub> droplets, which dissolve into seawater during the rise, as was shown by Sato and Sato (2002), and when they are in eddies in ocean turbulence, as was simulated by Sato (2004), they experience various CO<sub>2</sub> concentration, which changes with time. It is, therefore, important to consider the impact when the concentration changes with time. As Sato (2004) proposed, if the concepts of both LC<sub>50</sub> and LT<sub>50</sub> hold, a linear function for the mortality is obtained as follows.

$$Y = a \log(t) + b \log(x) + c, \tag{1}$$

where *Y* is the mortality in probit unit, *t* is the exposure time, and *x* is the concentration of toxicity agent. Fig. 1 shows the LC<sub>10</sub>, LC<sub>50</sub>, and LC<sub>90</sub> data for *Metridia pacifica*, *Calanus pacificus*, and epipelagic copepods, plotted in

$\log(t)$ - $\log(PCO_2)$  diagrams. Lines were depicted by multiple linear regression. It can be seen that  $Y$  can become linear with respect to both  $\log(t)$  and  $\log(x)$  within a couple of weeks. Auerbach *et al.* (1997) also proposed a linear correlation for  $LC_{50}$  in a  $\log(t)$ - $\log(pH)$  diagram for this time range.

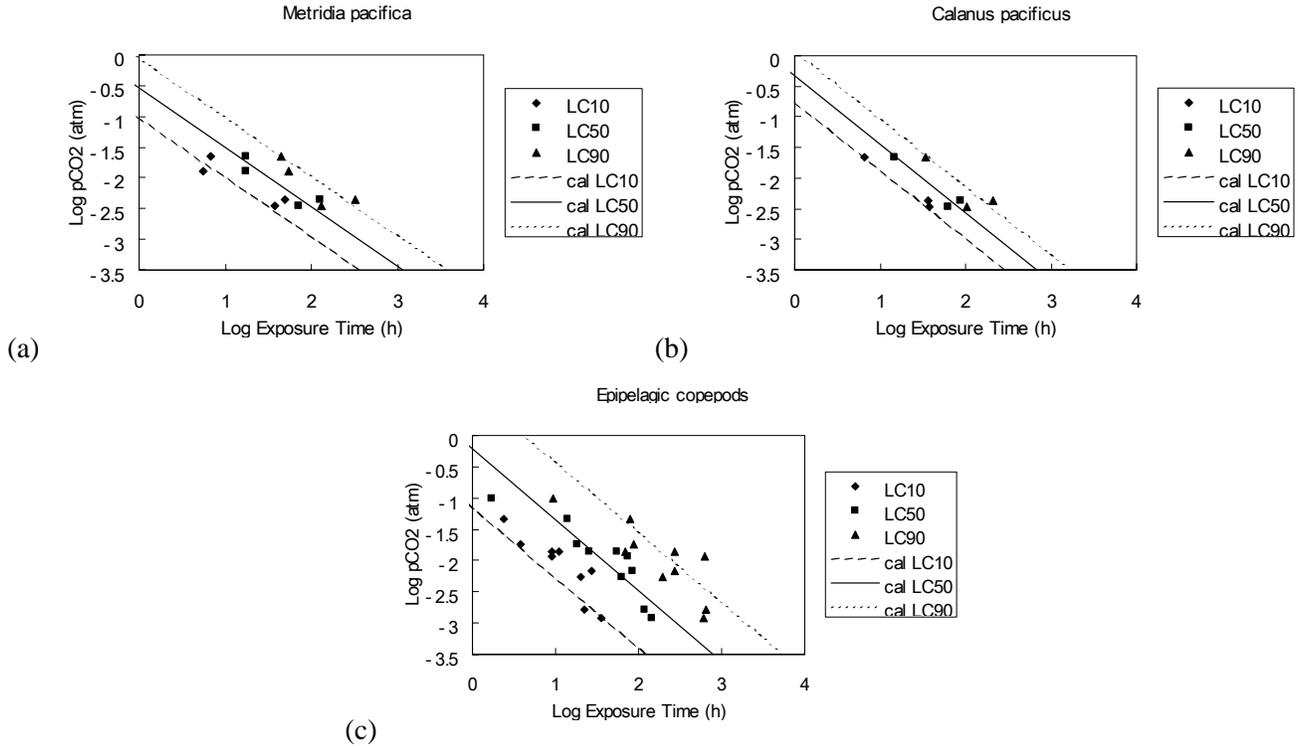


Fig. 1 Measured and calculated  $\log(t)$ - $\log(PCO_2)$  curves for *Metridia pacifica* (A), *Calanus pacificus* (B), and Epipelagic copepods (C).

Eq. (1) can be reformed by simple mathematics as follows.

$$Y = a \log\left(t \cdot [x]^{\frac{b}{a}}\right) + c. \quad (2)$$

This form has an advantage when extending the model to unsteady experience of  $x$ :

$$Y = a \log\left(\int_0^t [x]^{\frac{b}{a}} d\tau\right) + c. \quad (3)$$

The first term on the RHS of Eq. (3) indicates cumulative damage in acute mortality. The coefficients  $a$ ,  $b$ , and  $c$  can be determined by mortality experiments with various  $PCO_2$ , which is constant with time.

### 3. Experimental

#### 3.1 Set-up

The zooplankton used in the experiment was *Metamphiascopsis hirsutus* propagated in a culture tank (capacity of 5 m<sup>3</sup>) of the Miyazu branch of The General Environmental Technos, Japan. *M. hirsutus* is benthic species and their habitats differ from the planktonic copepods, which may be suitable for investigating the impact caused by the CO<sub>2</sub> direct injection. However, they are easy to kept steadily and the slopes of the  $LC_{50}$  lines in the  $\log(t)$ - $\log(PCO_2)$  diagram, like Fig. 1, for harpacticoid copepods are almost the same as those for the pelagic copepods in subtropical ocean (Watanabe, unpublished). Therefore we thought *M. hirsutus* is an analogue of the pelagic copepods in terms of the mortality against  $PCO_2$ .

The seawater in the tank was pumped up from the coast of the Japan Sea at the Miyazu branch. The seawater drawn from the tank was filtered using a GF/F glass fiber filter (Whatman) and transferred to 2 flasks (3 L), where the N<sub>2</sub>-O<sub>2</sub> (O<sub>2</sub>: 12.5 %) mixture gases with the concentration of CO<sub>2</sub> of 0 % and 20 %, respectively, were bubbled at the flow rate of 50 mL/min more than 6 h until  $PCO_2$  was equilibrate to the mixture gases. After that,  $PCO_2$  was adjusted to planned values by mixing the seawaters. The resultant waters were introduced to glass vials (30 mL). Just before the experiment, the zooplankton were acclimated in a thermostatic incubator at the experimental

temperature more than 1 day. Then, each individual zooplankton was put in each vial, which was, thereon, sealed up to prevent the water from exposing to the air and the zooplankton from being trapped at the gas-water interface. In advance, it had been confirmed that the vial capacity is large enough for the zooplankton to survive normally for the time period of the present experiment.

The vials were kept in the thermostatic incubator at  $25.5 \pm 1$  °C in the dark condition during the experiment. Table 1 denotes water quality measured before and after the experiment. The  $PCO_2$  of 0.05 % (the natural concentration of  $CO_2$ ) was used as the control (Case I) against the other. The transient changes of  $PCO_2$  were examined for the mortality of the zooplankton by sudden changes of  $PCO_2$  from 4 % to 2 % (Case III-2) and from 4 % to 8 % (Case III-3) at 48 h after the start of measurements. Although  $PCO_2$  in Case IV was originally planned to be 8 % when the vials were filled with the mixture water, the calculated  $PCO_2$  from the measured  $pH$  and alkalinity was 9 %. However, this causes no problem when obtaining the coefficients of Eq. (1).

Table 1 Experimental conditions

Case		I	II	III-1	III-2	III-3	IV	
$PCO_2$ (%)		0.05	2	4	4 to 2	4 to 8	9	
Salinity (‰)		32.3	32.3	32.3	32.3	32.3	32.3	
Time (h)	0	Temperature (°C)	25.6	25.8	25.5	25.5	25.5	25.3
		$pH$	8.03	6.53	6.32	6.32	6.32	5.75
96		Temperature (°C)	25.6	25.8	25.1	26.0	25.6	24.6
		$pH$	8.03	6.63	6.13	6.85	6.22	5.84

### 3.2 Results and Discussion

In Fig. 2, where the time-accumulated mortalities in decimal fraction,  $M$ , are denoted, these scattered data were averaged and indicated by error bars. Case I indicated no particular influence on the mortality. Data were scattered in Case III-1, III-2, and III-3, although these cases have the same  $PCO_2$  until 48 h after the start of the measurement. Fig. 3 shows  $Y$  calculated by Eq. (3), with respect to exposure time (A) and to  $PCO_2$  (B). The coefficients,  $a$  and  $b$ , in Eq. (1) were extracted from the data of accumulated mortalities for the  $PCO_2$  of 4 % after 48 h in Case III-1 and 9 % in Case IV. The section,  $c$ , in Eq. (1) was also calculated to fit the mortality for the  $PCO_2$  of 4 % after 48 h. As a result, we obtained the model coefficients:  $a=1.5308$ ,  $b=4.3894$ , and  $c=7.8362$ .

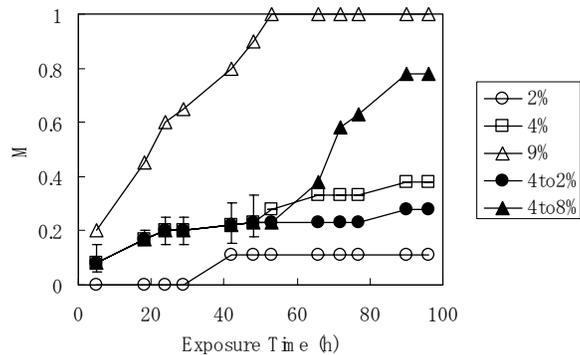


Fig. 2 Measured mortalities for *Metamphiascopsis hirsutus* against various  $PCO_2$  with respect to exposure time, including the cases of transient change of  $PCO_2$ .

By adopting these coefficients, we applied our model to the mortality of *M. hirsutus* against the transient change of  $PCO_2$ . It is seen that the model simulated the measurements moderately well (Fig. 4). The coefficient of correlations between the measured and the calculated  $M$  in Cases III-2 and III-3 are 0.9183 and 0.9519, respectively. The validity of the present model may suggest that the concept of the mathematical accumulation of damage works for zooplankton.

Very recently, Kikkawa (2004) obtained the acute mortality data of fish against the transient change of  $PCO_2$ , part of which was presented in Yoshimoto *et al.* (2005). The data indicated that, after experiencing the step-wise increases of  $PCO_2$  with time, the fish can be tolerant against some level of  $PCO_2$ , which is lethal without such gradual increases. Ishimatsu *et al.* (2004) pointed out that fish can compensate the effects of  $CO_2$  by accumulation

of bicarbonate ions in body fluid as long as  $PCO_2$  remains sublethal. It is believed that this possibly results in the acclimation of fish against  $PCO_2$  in mortality. It is inferred that the physiological buffer mechanism of fish against  $PCO_2$  is more developed than that of zooplankton. It is, therefore, safe to say that the applicability of the present model is limited to zooplankton.

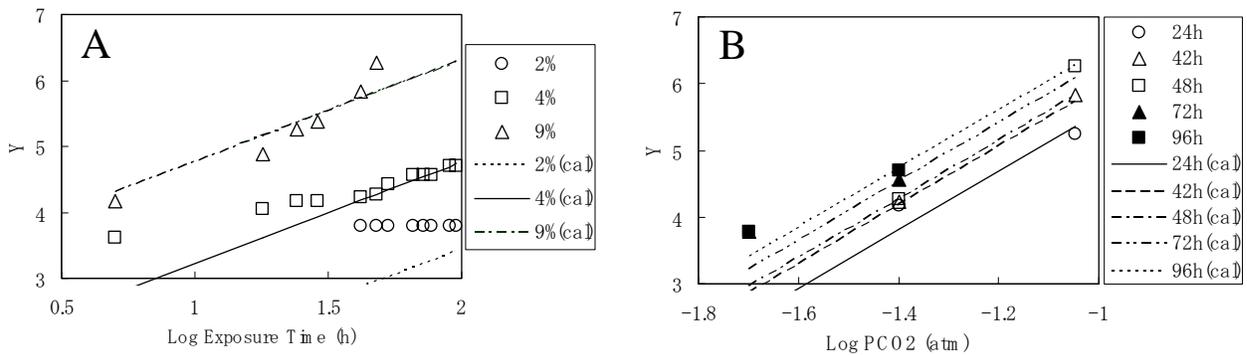


Fig. 3 Measured mortalities in probit unit for *Metamphiascopsis hirsutus* shown by dots and their linear curve-fittings with respect to exposure time (A) and to  $PCO_2$  (B).

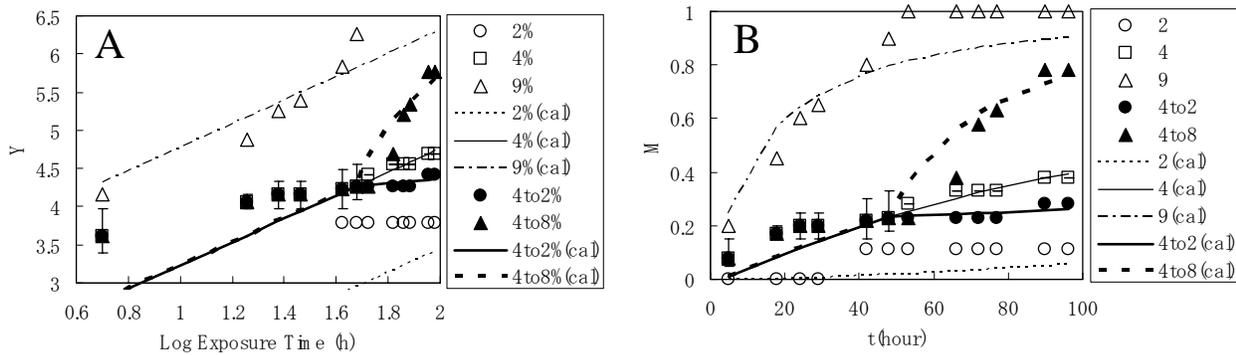


Fig. 4 Calculated and measured mortalities in probit unit (A) and in decimal fraction (B) for *Metamphiascopsis hirsutus* with respect to exposure time.

## 4. Computational Simulation of Biological Impact of $CO_2$ in Ocean Turbulence

### 4.1 Numerical Turbulent Simulator

One of the difficulties in computational simulation of ocean turbulence is that the large scale of eddies, which supply energy to smaller eddies, are commonly larger than the computational domain in most cases. Therefore, it is necessary to introduce appropriate fluctuating flow into the computational domain. Sato (2004) developed a numerical turbulent simulator by low-wavenumber forcing, where large-wavenumber components are expected to generate in the computation by the energy cascade of 3D turbulence. In this study, as the low-wavenumber components, we tentatively used an existing data of the real sea, which is the time sequence of 3 Cartesian components of velocity, temperature, and salinity measured offshore, west of Keyhole Point, Hawaii Island, for a week in October 2001.

### 4.2 Computational Conditions

The computational conditions are listed in Table 2, where the wavenumber is normalized by the size of the computational domain  $L=80$  m without multiplying by  $2\pi$ . The length scales and velocities are nondimensionalised by  $L$  and the reference velocity,  $U=1.0$  m/sec, respectively. The grid size is 2.5 m, which is almost the same order of magnitude as the smallest eddy detected in the measurements. Since the order of the magnitude of the Kolmogorov microscale  $\eta$  is about 0.003 m, it is believed that the grid size is within the inertia subrange. The nondimensional threshold wavenumber  $k_c$  between the high- and low-wavenumber components is

2. Since  $|U^0|$  is about 0.065 m/sec within the selected time range, the minimum nonzero dimensionless wavenumber introduced in the computation is about 0.04, which is less than the unity and small enough for the present study.

Table 2 Computational conditions.

calculation domain (x,y,z)	1.0,1.0,1.0
number of grid (x,y,z)	32,32,32
Reynolds number	$8.0 \times 10^7$
Froude number	0.0357
Prandtl number	10.26
Schmidt number	745.0
threshold wavenumber $k_c$	2
maximum wavenumber of $\mathbf{u}^H, \mathbf{T}^H$	16

We consider spray-type injection of liquid CO<sub>2</sub> from 100 nozzles arranged along a vertical pipe with an interval of 10m. The total amount of CO<sub>2</sub> released is 0.1 ton/sec and, consequently, the flow rate from a single nozzle is 1.0kg/sec. Because the vertical size of the computational domain is 80 m, only 8 nozzles exist in the domain and it is assumed that an infinite number of nozzles are arranged in the vertical direction because of the periodic boundary condition. The diameter of the pipe is supposed to be about 0.3 m, which is smaller than the computational grid size, and, therefore, the volume of the pipe is ignored and there is no vortex shedding from the pipe. The nozzles move at the ship speeds,  $U_{ship}$ , which are 0 and 4 knots in the  $x$  direction in the present simulation.

The coordinate migration velocity is calculated so that a target marine organism always stays at the centre of the computational domain, which means that the computational domain moves in accordance with the passive transfer of the organism due to the computed turbulent flow. Therefore, the injection points (volumeless nozzles) move in the opposite direction to the flow at the centre of the domain together with the ship speed and disappear after they come to the edge of the domain. Although the computational domain is small (80m×80m×80m), the domain moves and covers a couple of km when the current speed is 0.065 m/sec and the time range is 10 h.

The initial depth of the centre of the computational domain is set to be 1500 m, which varies due to the Lagrangian movement of the computational domain. Salinity is constant to be 34.6 permil and the ambient CO<sub>2</sub> concentration is  $2.3142 \times 10^{-3}$  mol/l, which is equivalent to the  $PCO_2$  of about 1000  $\mu\text{atm}$  at the given pressure, salinity, and temperature.

The target marine organism is *Metridia pacifica*, the parameters of which for the present mortality model, Eq. (3), are  $a= 3.26355$ ,  $b= 0.32943$ , and  $c= -0.00619$ . *M. pacifica* demonstrates the poorest survival rate among the zooplankton examined by us and, hence, is regarded as the most conservative case.

### 4.3 Results and Discussions

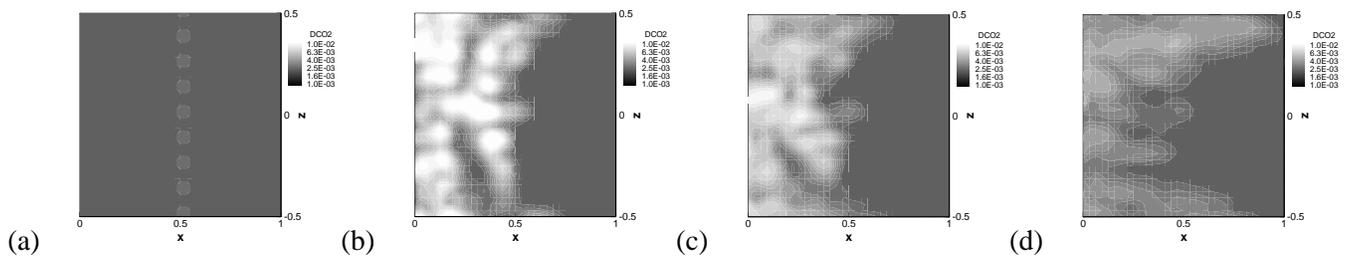


Fig. 5 Time sequence of contour maps of computed total carbon concentration on the central vertical plane in the case that ship speed is 0 knot at t=0h (a), t=2h (b), t=4h (c), t=6h (d).

Fig. 5 shows the time sequence of the contour maps of total carbon  $C$  every 2 hours in the case that  $U_{ship}=0$  knot. The arranged injection points are well observed at  $t=0$  h in Fig. 5(a). These  $CO_2$  sources migrate to the left due to the Lagrangian movement of the computational domain and hence already disappeared in Fig. 5(b). After the disappearance of the injection nozzles, we observe that the numerically reproduced turbulence diffuses the dissolved  $CO_2$  in Fig. 5(b)-(d).

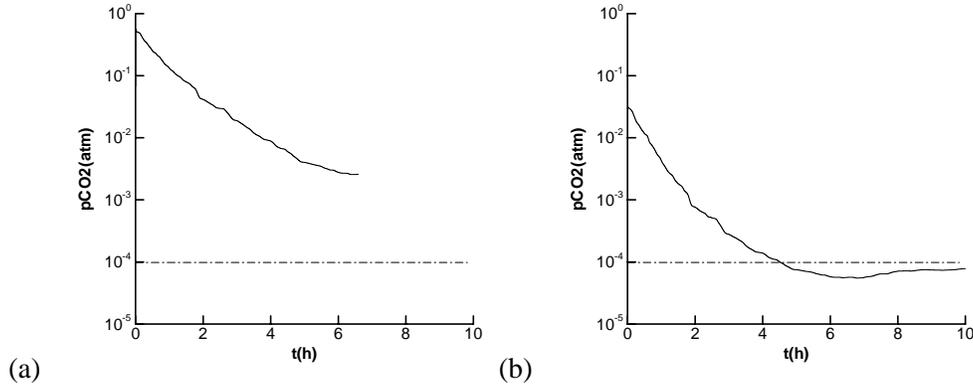


Fig. 6 Time variations of computed  $PCO_2$  difference from the background in the cases that the ship speeds are 0 (a) and 4 (b) knots.

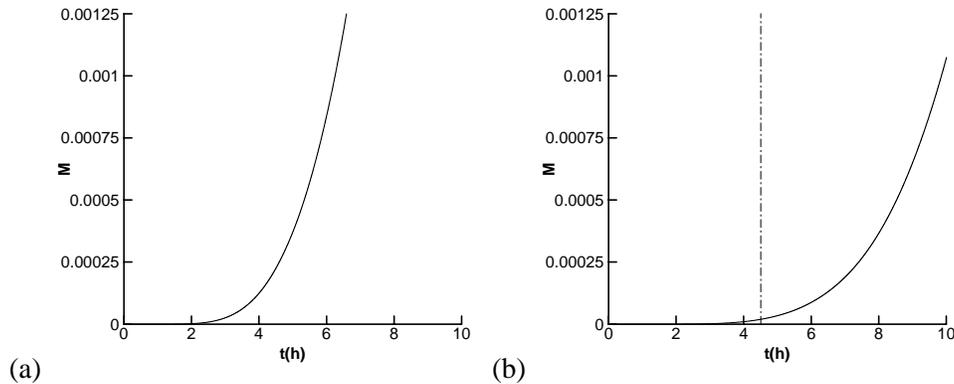


Fig. 7 Time variations of computed mortality of *Metridia pacifica* in the cases that the ship speeds are 0 (a) and 4 (b) knots.

Fig. 6 denotes the time variations of the difference between  $PCO_2$  and the background  $PCO_2$ . In the case that  $U_{ship}=0$  knot,  $PCO_2$  decreases with time because of turbulent diffusion, but still remains much higher than the background, while it almost reaches the background and does not change much thereafter in the case that  $U_{ship}=4$  knots. The measured  $PCO_2$  fluctuation is about  $\pm 200 \mu\text{atm}$  around the set background value,  $1000 \mu\text{atm}$ , at the target depth in the target ocean. The dash-dotted lines indicate  $100 \mu\text{atm}$ , which we regard as the threshold for the necessary dilution for nonsignificant biological impacts. In Fig. 6(a), the  $PCO_2$  difference does not reach the fluctuation range, while it becomes lower than the range at about 4.5 h after the start of injection in the case that  $U_{ship}=4$  knots in Fig. 6(b).

The time variations of the mortality of *M. Pacifica* are shown in Fig. 7 in the cases of the ship speed of 0 and 4 knots, corresponding to Fig. 6. It is obvious that the mortality reaches the lower three-sigma limit, 0.125%, at about  $t=7$  h in Fig. 7(a). This suggests that the biological impact predicted by the present method is nontrivial in the case that  $U_{ship}=0$  knot. In Fig. 7(b), the mortality also increases almost to the threshold of mortality 10 h after the start of injection. However, according to Fig. 6(b), the  $PCO_2$  difference reaches its threshold at  $t=4.5$  h, which is denoted by a dash-dotted line in the figure, and at that time, the mortality is still lower than the mortality

threshold. Therefore, it is thought that the target zooplankton is still safe, based on the results from the present computational simulation.

The mortality shown in Fig. 6(b) continues to increase quite rapidly after  $t=4.5$  h, even though the  $PCO_2$  is lower than  $100 \mu\text{atm}$ . This is because the mortality is very sensitive to the tiny level of  $PCO_2$  in this range due to the probit transform. It should also be noted that the mortality data shown in Fig. 1 are obtained for a  $PCO_2$  of more than  $1000 \mu\text{atm}$ , so that a  $PCO_2$  difference of  $100 \mu\text{atm}$  is beyond the meaningful range. If the mortality curve is not linear below the  $PCO_2$  of  $1000 \mu\text{atm}$  and becomes parabolic, just like the mortality curve against  $pH$  of Auerbach et al. (1997), the linear approximation is more severe to marine organisms and our standpoint is more conservative.

## 5. Conclusions

A model has been proposed for predicating the acute effect of  $CO_2$  on the mortality of zooplankton. This acute mortality model was validated by the mortality test of *Metamphiascopsis hirsutus* against the transient change of  $CO_2$  concentration and installed on a CFD method for the reproduction of small-scale ocean turbulence, in which low-wavenumber eddies are forced and high-wavenumber eddies are generated by the turbulent energy cascade-down. The simulation results suggest that the acute biological impact is nontrivial when the ship speed is 0 knot and  $CO_2$  of 0.1 ton/sec is injected in the form of a spray through 100 nozzles provided vertically on a pipe at 10m intervals. However, in the case that the ship speed is 4 knots, the difference of  $PCO_2$  from its background value becomes lower than its fluctuation range 4.5 h after the start of injection, before the mortality reaches a threshold, the lower 3-sigma limit. It is therefore believed that the moving-ship method is effective, according to the present acute mortality model in the present ocean turbulence simulator. It is also deduced that some engineering measures to achieve greater initial dilution are strongly recommended for greater confidence in the technique.

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## References

- Auerbach, D. I., Caufield, J. A., Adams, E. E. (1997), Impacts of ocean  $CO_2$  disposal on marine life, *Environ. Modelling Assess.*, **2**, 333.
- Broecker, W.S. 1997. Thermohaline circulation, the Achilles heel of our climate system: Will man-made  $CO_2$  upset the current balance? *Science* 278, 1582-1588.
- Herzog, H., Caldeira, K., Reilly, J. 2003. An issue of permanence: assessing the effectiveness of temporary carbon storage. *Climatic Change* 59, 293-310.
- Ishimatsu, A., Kikkawa, T., Hayashi, M., Lee, K.-S., Kita, J. 2004. Effects of  $CO_2$  on marine fish: larvae and adults. *J. Oceanogr.* 60, 731-742.
- Kikkawa, T. (2004), Effects of  $CO_2$  on the early development stages of marine fish, *Rep. Mar. Ecol. Res. Inst.*, **7**, 1-33 (in Japanese).
- Kikkawa, T., Ishimatsu, A., Kita, J. (2003), Acute  $CO_2$  tolerance during the early development stages of four marine teleosts, *Environ. Toxicol.*, **18**, 375-382.
- Kikkawa, T., Kita, J., Ishimatsu, A. 2004. Comparison of the lethal effect of  $CO_2$  and acidification on red sea bream during the early developmental stages. *Mar. Pollut. Bull.* 48, 108-110.
- Kita, J., Ohsumi, T., 2004. Perspectives on biological research for  $CO_2$  ocean sequestration. *J. Oceanogr.* 60, 695-704.
- Kurihara, H., Shimode, S., Shirayama Y. 2004a. Effects of raised  $CO_2$  concentration on the egg production rate and early development of two marine copepods. *Mar. Pollut. Bull.* 49, 721-727.
- Kurihara, H., Shimode, S., Shirayama Y. 2004b. Sub-lethal effects of elevated concentration of  $CO_2$  on planktonic copepods and sea urchins. *J. Oceanogr.* 60, 743-750.
- Ohsumi, T. 1995.  $CO_2$  disposal options in the deep sea. *Mar. Technol. Soc. J.* 23, 58-66.
- Portner, H.O., Langenbuch, M., Reipschlag, A. 2004. Biological impact on elevated ocean  $CO_2$  concentrations: lessons from animal physiology and earth history. *J. Oceanogr.* 60, 705-718.
- Riebesell, U. 2004. Effect of  $CO_2$  enrichment on marine phytoplankton. *J. Oceanogr.* 60, 719-730.

- Sato, T. 2002. Modelling of biological impact in direct injection of carbon dioxide in the ocean. Proc. 6th Int. Conf. On Greenhouse Gas Control Technol. B3-3, 1-6.
- Sato, T., Sato, K. 2002. Numerical prediction of the dilution process and its biological Impacts in CO<sub>2</sub> ocean sequestration. J. Mar. Sci. Technol. 6, 169-180.
- Sato, T. 2004. Numerical simulation of biological impact caused by direct injection of carbon dioxide in the ocean. J. Oceanogr. 60, 807-816.
- Thornton, H., Shirayama Y. 2001. CO<sub>2</sub> ocean sequestration and its biological impacts, III-1: effects of CO<sub>2</sub> on benthic organisms. Bull. Japanese Soc. Scient. Fish. 67, 756-757 (in Japanese).
- Yoshimoto, N., Sato, T, Kikkawa, T. 2005. Mechanical mortality model for fish caused by CO<sub>2</sub> considering acid-base regulation. Proc. 18th Ocean Engrg. Symp. CD-ROM (in Japanese).