This differential resistance to decline of the various components of the responses can be demonstrated by the number of trials required for their habituation (table 2). With the moving shadow stimulus, eye movements are the most resistant to decline, followed by changes in pectoral fin movements, dorsal fin position and head movements. The rates of waning of these components differ using a Friedmans 2-way analysis of variance ($\chi^2 = 13.9$, $n = 7$, $p < 0.02$). The tap stimulus produced a similar result, pectoral and dorsal fin and head movement tending to be the most resistant to decline ($\chi^2 = 8.9$, $n = 7$, $p < 0.2$).

Arousal responses shown by the slippery dick, *Halichoeres bivittatus* are typified by a change in pectoral fin movements and erection of the dorsal fin. Orientation of the head, eyes and body may also occur if the stimulus is visual. These components of arousal are similar to those described for goldfish[8–10] and roach[4].

Early stimuli in a serial presentation can evoke fright responses, especially if the stimulus is a tap. Here, pectoral locomotion ceased, both pectoral and dorsal fins were adducted whilst a wave of contraction passed down the body from head to tail, driving the animal forward. Burrowing behavior often ensued. These vigorous sinusoid movements may be a natural extension of the cyprinid-type tail flip response[8] when performed by this slimmer, more elongate species. Early fright responses disappear to be replaced by arousal, as observed previously[5], though the muscular components of fright are maintained during its expression. The same is not true of the arousal response in which the components of the behavior are reduced during repeated stimulus presentation. Those components which are the most consistent responses to a novel stimulus, whether this be a moving shadow or a tap, are also those most resistant to decline. These are erection of the dorsal fin and change in beat of the pectorals, which take some 3–4 trials to decline. If the stimulus is a moving shadow then detection of the stimulus, indicated by eye movements, may occur for some 8 presentations even though a generalized arousal response is not apparent.

The present work has established a similarity between the behavioral responses of the marine perciform, *Halichoeres* and the previously studied cyprinids. Whereas fright responses are of an 'all' or 'none' nature, arousal responses can differ in the number of behavioral components they entail. Both, however, decline on repeated stimulus presentation so that the response is appropriate both to the environmental circumstances and the previous experience of the individual studied.

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Host cell reactivation capacity of different strains of *E. coli* B resistant or sensitive to ozone

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Summary. Host cell reactivation capacity for ozonated or irradiated phage was determined for different strains of *E. coli* either more sensitive or resistant to ozone than the wild type strain. The results suggest that the *ozr* gene product could be involved in the same repair pathway for ozone-induced lesions on DNA as the *pol*A gene. The possible involment of a specific endonuclease for these lesions is also considered.

Ozone is a natural constituent of clean air at high altitudes and is generated in the stratosphere by reaction of oxygen atoms with molecular oxygen. On the other hand, ozone is the major oxidant found in photochemical smog in the lower atmosphere. This gas is formed in polluted atmospheres by photolysis of NO_2 in the presence of hydrocarbons produced by traffic and chemical plants.

Various toxic effects of ozone on biological systems have been described in recent literature. This strong oxidizing agent has been shown to cause aberrations in the growth of plants[2], membrane damage in erythrocytes[3] and histological changes in animal organs[4]. It has been shown to be capable of inducing chromosomal aberrations in *Vicia faba*[5,6], in human cell culture[7] and in the peripheral lymphocytes of human subjects[8] while similar levels induced chromatid-aberrations in peripheral lymphocytes from Chinese hamsters[9]. In addition, a considerable number of reports concerning the radiomimetic[10,11] and mutagenic[12–14] effects of this gas have been accumulated. Futhermore, ozone has been reported to damage viral[15–17] and bacterial[18–20] nucleic acids. On the molecular level, ozone has been shown to cause lipid peroxidation[21], covalent protein cross-linking[22] and degradation of nucleobases by attacking primarily guanine[23–25].

Some previous work has emphasized the fact that *E. coli* could repair ozone DNA-damage[26,27] and that DNA polymerase I could be involved in the repair process[28,29]. Recently, several mutants of *E. coli* either more sensitive or resistant to ozone than the wild type have been isolated[30–32]. Published data on cell survival and genetic mapping with these different mutants[30–32] indicate the location in the *mal*B region of *E. coli* of 3 genes (*ozr*A, *ozr*B and *ozr*C) that could be involved in the repair of ozone-induced lesions on DNA.

Results presented in a previous paper[29] showed that host cell reactivation (HCR) of ozone-treated phage could be used as a probe to test rapidly the repair proficiency of a given strain against this agent. In a series of experiments reported here, the properties of *E. coli* wild type and of 5 mutants with regard to their sensitivity to ozone were examined by measuring the reactivation factor for ozonated or irradiated phage.

Material and methods. Stocks of T_3 phage were prepared by the confluent lysis method with *E. coli* B251[33]. Ozone treatment was performed by the exposure of 10 ml of T_3 phage water suspensions (10^7 phages/ml) to 10 ppm of ozone for 10 min as described elsewhere[13,29]. UV-irradiation was carried out with an American Ultraviolet CE-30 germicidal lamp emitting mainly at 254 nm wavelength and at an incident dose rate of 1.52 Jm^{-2} sec^{-1}. Water suspensions (2 ml) containing 10^6 phages/ml were irradiated at less than 1 mm thickness for 45 sec[34]. Host cell reactivation (HCR) procedures have been described in a previous paper[29]. The fraction of survivors (S/S°) and the reactivation factor (fraction of survivors with mutant strain/fraction of survivors with wild type strain) were determined for each different bacterial strain used. These bacterial strains, their relevant genotype and their source are listed in table 1.

Results and discussion. In order to simplify the study of the complex relationship between ozone and whole cells, we evaluated the host contribution to the recovery from ozonation of T_3 phage. The host cell reactivation capacity for ozonated phage obtained with different mutants are shown in table 2. According to their host cell reactivation efficiency, these bacterial strains can be classified into 2 groups. Clearly, MQ1812 *ozr*A2 and MQ1817 *ozr*A3 can more efficiently reactivate ozonated phage than the wild type strain B251, while MQ849 *ozr*A1, MQ1844 *ozr*B2 and MQ3060 *ozr*C have a significantly reduced efficiency. This is in agreement with ozonation data which have indicated that MQ1812 and MQ1817 mutants are more resistant to ozone while MQ849, MQ1844 and MQ3060 are more sensitive than the wild type B251[30-32].

Results presented in a previous paper[29] on the host cell reactivation of ozonated phage by *pol*A strains led to the conclusion that DNA polymerase I could play a key role in the repair of ozone-DNA damage. Similar conclusions can be drawn from this study. First, ozone-sensitive mutants (MQ849, MQ1844 and MQ3060) show about the same host cell reactivation capacity for ozonated phage (table 2) as the *pol*A strains[29]. Secondly, as with the *pol*A strains[28], ozone-sensitive mutants degrade their DNA more rapidly after ozonation than the wild type[31]. Finally, the most ozone-resistant mutants (MQ1812 and MQ1817), with a higher reactivation efficiency than the wild type (table 2), degrade their DNA to a lesser extent than the wild type after ozonation[31].

Table 3 summarizes the results of host cell reactivation of UV-irradiated phage with different strains of *E. coli*. It will be seen that in contrast to the results of previous researchers[32,36], which showed that mutants sensitive or resistant to ozone responded in a similar fashion to UV-irradiation[32,37], the ozone-sensitive mutants (MQ849, MQ1844 and MQ3060) can quite efficiently reactivate UV-irradiated phage while ozone-resistant mutants (MQ1812 and MQ1817) have a significantly reduced efficiency.

These results (table 3) reveal a striking paradox when the UV-sensitivity of a certain bacterial strain and its host cell reactivation capacity for irradiated or ozonated phage are compared. On one hand, MQ849 and MQ1844 strains are sensitive to ozone and UV light[31,32,37] while they can quite efficiently reactivate UV-irradiated phage. On the other hand, MQ1812 and MQ1817 mutants are very resistant to ozone and UV light[31,32,37] but have a very much reduced reactivation efficiency for irradiated phage (table 3).

On the basis of these results (table 2 and 3), it seems that strains that are able to efficiently reactivate ozonated phage cannot as efficiently reactivate irradiated phage and vice-versa. Again, this is in agreement with previous published data[29] which have indicated that the *uvr*A mutant, deficient in the host cell reactivation of UV-irradiated phage[38], is able to reactivate ozonated phage more efficiently than the wild type strain. Since it has been proved that the ability of strains to support plaque formation by irradiated phage (HCR) depends upon their possession of a normal excision repair mechanism[38,39], we can argue that maybe MQ1812 and MQ1817 strains have a less efficient excision repair mechanism than *E. coli* B251.

According to the results presented in this paper, *ozr* and *pol*A gene products could be involved in the same repair mechanism of ozone-DNA damage. Futhermore, on the basis of results reported in this and a previous paper[29] on the evident paradox between host cell reactivation of ozonated or irradiated phage, an alternative explanation could be built around the idea that, like the *uvr*A gene product[40], the *ozr* gene product could be a specific endonuclease for ozone-induced lesions on DNA.

Table 1. Bacterial strains

Strain	Relevant genotype	Source
B251	*Escherichia coli* B wild type	Arber and Morse[35]
MQ849	*ozr*A1 derivative of B251	Hamelin and Chung[31]
MQ1812	*ozr*A2 derivative of B251	Hamelin and Chung[31]
MQ1817	*ozr*A3 derivative of B251	Hamelin and Chung[31]
MQ1844	*mal*B, *ozr*B2 derivative of B-Hill	Poliquin et al.[32]
MQ3060	*ozr*C derivative of B251	Côtes and Chung[30]

Table 2. Host cell reactivation capacity for ozonated T_3 phage

Host cell strain		Reactivation factor
B251	(wild type)	1
MQ849	(*ozr*A1)	0.54 ± 0.10^a $(5)^b$
MQ1812	(*ozr*A2)	1.36 ± 0.23 (4)
MQ1817	(*ozr*A3)	2.63 ± 0.55 (4)
MQ1844	(*ozr*B2)	0.56 ± 0.09 (5)
MQ3060	(*ozr*C)	0.45 ± 0.06 (5)

[a] Mean ± SE; [b] number of experiments. Ozone treatment: 10 ppm for 10 min. Reactivation factor: fraction of survivors with mutant strain/fraction of survivors with wild type strain.

Table 3. Host cell reactivation capacity for UV-irradiated T_3 phage

Host cell strain		Reactivation factor
B251	(wild type)	1
MQ849	(*ozr*A1)	0.81 ± 0.03^a $(4)^b$
MQ1812	(*ozr*A2)	0.19 ± 0.04 (4)
MQ1817	(*ozr*A3)	0.21 ± 0.03 (4)
MQ1844	(*ozr*B2)	0.77 ± 0.12 (4)
MQ3060	(*ozr*C)	0.99 ± 0.16 (4)

[a] Mean ± SE; [b] number of experiments. UV-irradiation: 1.54 Jm^{-2} × 45 sec. Reactivation factor: fraction of survivors with mutant strain/fraction of survivors with wild type strain.

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Effects of pressure on oxygen consumption in cottid fish from Lake Baikal

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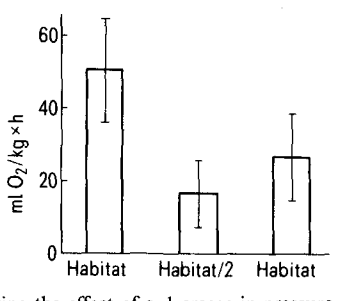
Summary. Rates of oxygen consumption in shallow dwelling cottid fish from Lake Baikal were unaffected by a change in pressure from 11 to 51 ata. The metabolic rate of deep cottids decreased by 72% when the pressure was decreased by 50% from that corresponding to habitat depth. Recovery from decreased pressures was incomplete in deep fish, suggesting that measurements of $\dot{V}O_2$ in deep dwelling fish in the literature may underestimate actual rates.

It has been proposed that adaptation to the deep-sea environment entails a lowering of the metabolic rate in fish compared to shallow water species[2–7]. The low metabolic rates in deep-sea fish are associated with lower caloric densities[8] and reduced activities of glycolytic[9] and tricarboxylic acid cycle[10,11] enzymes. Most studies on the rate of oxygen consumption ($\dot{V}O_2$) of deep-sea fish, however, have been performed at atmospheric pressure (1 ata) rather than at habitat pressures[2–5]. Notable exceptions are 2 studies done in situ at 1230–3650 m[6,7]. In only 2 studies, on *Anoplogaster*[3] and *Melanostigma*[12], were the effects of changes in pressure on $\dot{V}O_2$ investigated; these revealed no significant pressure effects.

The purpose of the present study was to determine the effects of pressure changes on the rates of oxygen consumption in shallow and deep dwelling cottid fish from Lake Baikal, Central Siberia, USSR; and to ascertain whether differences in metabolic rate obtain in closely related species as a function of depth of occurrence as suggested by Childress et al.[2,5].

Materials and methods. Specimens of the 3 deep species of cottid fish, *Batrachocottus nikolskii, Cottinella boulengeri* and *Abyssocottus korotneffi* were collected in the southern and central basins of Lake Baikal from the Soviet research vessel Titov in a 3-m beam trawl from depths of 660–1120 m. The fish were removed from the cod end to a pan of fresh lake water and only undamaged individuals which responded to touch with vigorous swimming were employed. Fish were used immediately after capture. Shallow water cottids *(Cottus kessleri)* were caught by hand from among rocks by the shore. Fish were placed individually in a stainless steel cylindrical pressure vessel equipped with a conical plexiglass window at each end and filled with fresh, aerated lake water. The chamber had a volume of 1250 ml and was connected on one end to the outflow from a pressure amplifier capable of delivering 50 ml of



Histogram showing the effect of a decrease in pressure and subsequent return to habitat pressure on $\dot{V}O_2$ of deep water cottid fish. All groups are significantly different from one another by the Mann-Whitney U-test.