Selection was practiced for 69 generations prior to this work. Reciprocal crosses were carried out between the selection lines and the base population. In order to reduce the environmental variance, samples of 100 eggs were seeded in bottles with 20 ml of food avoiding larval competition during development. For each cross four replications were set up, and 20 females were scored per bottle. All experiments were conducted at 19± 1 °C.

The average dominance was calculated from the ratio of deviations of the F_1 (selection line × basis population, and reciprocal cross) with respect to the mean value between the selection lines and the base populations.

Results and discussion. The means of interocellar bristle number for females from the selection lines, the base populations (control lines) and the reciprocal crosses between them are given in table 1. From the differences between the reciprocal crosses, we can determine the contribution of maternal components and cytoplasmic effects to response of selection. The values of average dominance are presented in table 2. The differences between the means of reciprocal crosses, except in B1H and B1L, are not significant. Therefore we can conclude that generally there was no significant directional contribution of maternally inherited factors.

The values of average dominance indicate that the alleles determining interocellar bristle number are recessive. The recessiveness, which is partial in alleles increasing the number of interocellar bristles, is practically absolute in alleles decreasing them. Consequently, in our populations the response to selection would be expected to be greater in high lines that in low lines. This is in agreement with the data obtained (see table 1). However it must be pointed out that the response in low lines is limited by the morphological threshold of 'zero bristles'.

So far, no other data are available about the average dominance of interocellar bristle polygenes. However, we can compare our results with those obtained by other authors for genes determining abdominal bristle number. For this trait, Frankham[4] also indicates recessive behaviour in the Canberra population, while other results suggest that alleles were additive or slightly dominant[7-10]. Consequently, it seems evident that the average dominance may differ considerably depending on the bristle system as much as on the population analyzed. Thus the results obtained with 1 specific experiment can never be generalized.

Table 1. Mean interocellar bristle number for females from the selection lines, the base populations (control lines) and the reciprocal crosses

Line	Mean	(A) F_1 Selection line* × control line	(B) F_1 Control line* × selection line
A1H	15.00±0.22	8.96±0.14	8.77±0.10
A2H	24.69±0.37	12.76±0.33	12.78±0.37
B1H	17.40±0.25	8.85±0.11	8.13±0.12
B2H	23.94±0.30	8.96±0.12	8.81±0.10
AC	7.10±0.07		
BC	6.16±0.09		
A1L	0.21±0.05	5.98±0.12	6.00±0.08
A2L	0.16±0.05	6.02±0.09	6.04±0.08
B1L	0.41±0.08	5.72±0.11	5.36±0.11
B2L	0.22±0.07	5.66±0.13	5.71±0.14

* Female parent.

Table 2. Average dominance from different lines

Line	d/a (1)	d/a (2)
A1H	0.52	0.57
A2H	0.35	0.35
B1H	0.52	0.64
B2H	0.68	0.70
A1L	0.79	0.80
A2L	0.78	0.78
B1L	1.13	1.00
B2L	0.97	0.99

(1) refers to cross (A) and (2) refers to cross (B), see table 1. d, genotypic values of the F_1; a, genotypic values of the selection lines.

1 A. Robertson, Proc. R. Soc. Lond.B *153*, 234 (1960).
2 L.P. Jones, R. Frankham and J.S.F. Barker, Genet. Res. *12*, 249 (1968).
3 S. Wright, Am. Nat. *103*, 561 (1969).
4 R. Frankham, Aust. J. biol. Sci. *27*, 683 (1974).
5 R. Marcos, Drosophila Inf. Serv. *52*, 61 (1977).
6 R. Marcos, Thesis, Univ. Autónoma de Barcelona, 1978.
7 G.A. Clayton, J.A. Morris and A. Robertson, J. Genet. *55*, 131 (1957).
8 J.F. Kidwell and O. Kempthorne, Züchter *36*, 163 (1966).
9 J.F. Kidwell and M.M. Kidwell, Can. J. Genet. Cytol. *8*, 207 (1966).
10 M. Rasmuson, Acta zool. *33*, 277 (1952).

Effect of L-methionine-DL-sulphoximine on the photoproduction of hydrogen by *Rhodospirillum rubrum*[1]

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Summary. The repression of photoproduction of hydrogen by ammonia could be relieved by L-methionine-DL-sulfoximine. In the absence of ammonia, hydrogen evolution was inhibited by concentrations of L-methionine-DL-sulfoximine higher than 0.1 mM.

Apart from the reduction of several substrates, the photoproduction of hydrogen by cyanobacteria and photosynthetic bacteria also is catalyzed by nitrogenase. In whole cells of photosynthetic bacteria this enzyme is quickly inhibited by low concentrations of ammonium salts[2-5]. It has also been shown that glutamine synthetase activity decreases rapidly after addition of NH_4^+[6,7]. The glutamate analog L-methionine-DL-sulfoximine (MSO), a potent inhibitor of glutamine synthetase, is able to relieve the repression exerted by exogenous ammonia on nitrogenase activity[8-10]. Furthermore, studies on mutants of *Rhodospirillum rubrum*[11] and recently of *Rhodopseudomonas capsulata*[12] have also demonstrated that glutamine synthetase plays an important role in the regulation of nitrogenase activity.

The photoproduction of hydrogen by photosynthetic bacteria is under investigation as a potential source of fuel[5,13]. The efficient repression of nitrogenase by NH_4^+ poses severe restrictions when waste material containing com-

bined nitrogen as a nutrient source and hydrogen donor is used.

The results presented here show that the rate of hydrogen evolution in a system with NH_4^+ in the presence of MSO was almost the same as in a non-repressed system.

Material and methods. In order to obtain comparable cell material for all experiments, *Rhodospirillum rubrum* strain S-1 was grown in continuous culture in a fermenter of a volume of 1000 ml as previously described[13]. Growth conditions were the following: Anaerobic, photosynthetic, illuminated by tungsten lamps (light intensity on the average 30 mW cm^{-2}) at 30 °C. The synthetic medium of Ormerod et al.[14] was used. Unless otherwise stated L-lactate (50 mM) and L-glutamate (15 mM) were the carbon and nitrogen sources respectively. Chemostatic growth was limited by glutamate. The culture attained a steady state absorbance of 5.0–5.5 at 660 nm (measured on a Hitachi 101 spectrophotometer).

18 ml cell suspension of a hydrogen producing fermenter culture were incubated in rubber stoppered bottles provided with hypodermic needles for gas outlet. The bottles were flushed with He to eliminate repression of hydrogen evolution by air. The cultures were made 50 mM with lactate and incubated in a shaking water bath at 30 °C illuminated by tungsten lamps (light intensity 30 mW cm^{-2}). The gas produced was trapped in calibrated cylinders. CO_2 was absorbed by an alkaline solution. The trapped gas was analysed by gas chromatography.

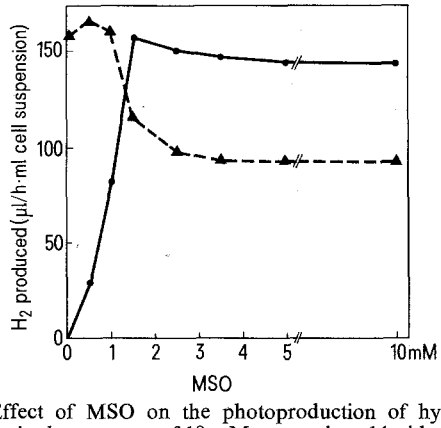

Fig. 1. Effect of MSO on the photoproduction of hydrogen by *R. rubrum* in the presence of 10 mM ammoniumchloride (solid line) and in the absence of ammoniumchloride (dashed line). Hydrogen evolution was measured during 24 h and average rate was determined.

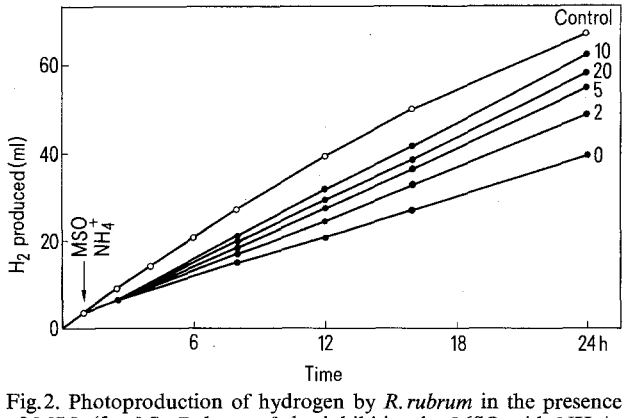

Fig. 2. Photoproduction of hydrogen by *R. rubrum* in the presence of MSO (5 mM). Release of the inhibition by MSO with NH_4^+. NH_4^+ was added at the concentrations indicated (mM). Control: without MSO and NH_4^+.

Results and discussion. An initial concentration of 10 mM NH_4^+ completely inhibited hydrogen production for 24 h. The strong repression could be relieved by simultaneous addition of MSO as indicated in figure 1. The degree of derepression was dependent on the concentration of MSO. Weare and Shanmugam[9] described a concentration-dependent derepression of nitrogenase activity measured as acetylene reduction. The highest nitrogenase activity in the presence of 10 mM NH_4^+ was reported to be at a MSO concentration of 10–20 mg/ml. In our work, however, we achieved a maximum hydrogen production in the presence of 10 mM NH_4^+ with a MSO concentration of only 0.27 mg/ml (1.5 mM). This difference may be due to different experimental conditions, mainly the cell material used. On the other hand different catalytic sites for acetylene or H^+ reduction cannot be excluded[15]. In a recent work Jones and Monty[10] found in short time experiments with *Rhodopseudomonas sphaeroides* no inhibition of the photosynthetic hydrogen evolution after the additon of 0.13 mM ammonia in the presence of 0.1 mM MSO.

Figure 1 indicates that hydrogen evolution, especially in the absence of NH_4^+, was inhibited by the higher MSO concentrations, while low concentrations were slightly stimulating. This inhibition of hydrogen production by MSO could be relieved to a great extent by simultaneous addition of NH_4^+. It was dependent on the concentration of the added ammonia (figure 2). This indicates that the nitrogenase inhibitor is not NH_4^+ per se, as suggested by the findings of Stewart and Rowell in *Klebsiella*[8] and Jones and Monty in *R. sphaeroides*[10].

Cells of *R. rubrum* grown in continuous culture with NH_4^+ as nitrogen source have an active nitrogenase and evolve hydrogen, if ammonia is the growth limiting factor. Using cells grown in this way, hydrogen evolution was repressed by NH_4^+ and derepressed by MSO in a way similar to that described in the experiments with cells grown with glutamine as nitrogen source. Weare[11] proposes the use of a mutant of *R. rubrum* which is not repressed by NH_4^+ for the photoproduction of ammonia and hydrogen. Glutamine auxotroph mutants of *Rhodopseudomonas capsulata* have been described, which synthesize nitrogenase and produce hydrogen in the presence of exogenous ammonia[12]. Apart from genetically altered photosynthetic bacteria, chemical regulations could be of great interest for the photoproduction of hydrogen with waste substrates containing combined nitrogen.

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2 H.-J. Schick, Arch. Mikrobiol. *75*, 102 (1971).
3 P. Hillmer and H. Gest, J. Bact. *129*, 732 (1977).
4 W.G. Zumft and F. Castillo, Arch. Microbiol. *117*, 53 (1978).
5 J. Meyer, B.C. Kelley and P.M. Vignais, Biochemie *60*, 245 (1978).
6 P. Rowell, S. Enticott and W.D.P. Stewart, New Phytol. *79*, 41 (1977).
7 P. Hillmer and K. Fahlbusch, Arch. Microbiol. *122*, 213 (1979).
8 W.D.P. Stewart and P. Rowell, Biochem. biophys. Res. Commun. *65*, 846 (1975).
9 N.M. Weare and K.T. Shanmugam, Arch. Microbiol. *110*, 207 (1976).
10 B.L. Jones and K.J. Monty, J. Bact. *139*, 1007 (1979).
11 N.M. Weare, Biochim. biophys. Acta *502*, 486 (1978).
12 J.D. Wall and H. Gest, J. Bact. *137*, 1459 (1979).
13 H. Zürrer and R. Bachofen, Appl. envir. Microbiol. *37*, 789 (1979).
14 J.G. Ormerod, K.S. Ormerod H. Gest, Archs Biochem. Biophys. *94*, 449 (1961).
15 J.M. Rivera-Ortiz and R.H. Burris, J. Bact. *123*, 537 (1975).