LETTERS TO THE EDITOR

supply system. These data also stress the need to prevent contamination of divers’ skin, ears, and mucous membranes, and to develop the deep diver’s resistance to infections.

A. N. Victorov
V. K. Ilyin
N. A. Policarpov
M. P. Bragina
V. G. Sobolevski
A. D. Syssoev
T. J. Norkina
I. N. KornushenkoVA
Institute of Biomedical Problems
Moscow, USSR

REFERENCES

1. Sakhno PN, Guljar SA. Mikroflora kozhi naruznikh obolochek nosa i glotki avkanautov uslubov-
   jakh prerobivania v podvodnikh laboratorijakh. In: Autoflora zdrovogo i bolnogo organizma.
3. Money KE, Buckingham IP, Calder IM, et al. Damage to the middle ear and the inner ear in
4. Agoli B, Bozanio V, Arsenievic S, Rilovic P. Open sea saturation diving at 100 m with excursion
   diving in 120 m—some characteristics. Vojnosanit Pregl 1980; 37:347.
5. Doig P, Todd T, Sastri PA, et al. Role of pili in adhesion of Pseudomonas aeruginosa to human

To the Editor:

Simulation of a microbial epidemic on a diving ship

Divers who work in harbors and near sewer outlets often are at risk of contamination by infectious agents, followed by development of infectious diseases (1, 2). Studies confirm the danger to divers during their contact with contaminated water, in spite of protective features of the divers’ equipment (3–5).

The literature suggests that Escherichia coli M-17 strain can serve as an indicator of epidemic processes and infectious agent transport (6). Known to be harmless to human health, the Soviet drug Kolibacterin is made from this strain.

The purpose of our work was to study the process of epidemic spread of test strain Escherichia coli M-17 among divers and crew members of ships with diving crews. The crew consists of 10 persons, including 3 divers.
<table>
<thead>
<tr>
<th>Days of Experiment</th>
<th>Divers</th>
<th>Engine Operator</th>
<th>Cook</th>
<th>Water Closet</th>
<th>Deck</th>
<th>Living Rooms</th>
<th>Intestine</th>
<th>Hand</th>
<th>Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>x</td>
<td>0</td>
<td>x</td>
<td>0</td>
<td>x</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>x</td>
<td>0</td>
<td>x</td>
<td>0</td>
<td>x</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>0</td>
<td>0</td>
<td>x</td>
<td>0</td>
<td>x</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>0</td>
<td>0</td>
<td>x</td>
<td>0</td>
<td>x</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>0</td>
<td>0</td>
<td>x</td>
<td>0</td>
<td>x</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- **x** = the sample contains test-strain
- **0** = no test-strain detected
- **no studies provided**
LETTERS TO THE EDITOR

External surfaces of divers' clothes and equipment, suits, boots, weights, and signal belts were contaminated with test strain fresh broth culture, diluted to a concentration of 10 colony forming units/ml. All the contaminated equipment was put on the divers, who then imitated underwater work in a freshwater lake. The supporting team members took the divers aboard from the water and helped them to undress. They then washed the divers' equipment and clothes with soap and disinfectant.

Bacteriologic samples were obtained before the study and then daily. Samples were taken by cotton swabs from divers' clothes, hands, and from deck equipment, kitchen tables, living and dining rooms, and door knobs. In addition, at Day 0 and at Days 6 and 20 of the investigation, intestinal flora studies of the crew were done. The test strain was identified according to its serologic features, biochemical peculiarities (saccharose reducing), and susceptibility to *Fredericq colicine*.

The data are presented in Table 1. On the day of contamination, the test strain was isolated from the divers' clothes, from the deck floor, from knobs of deck diving mechanisms, from boots and hands of supporting divers, from the divers' living-room door knobs, and from bathroom door knobs. However, by Days 2 and 3 of the investigation we had failed to isolate the test strain anywhere.

On Day 6 we isolated the test strain from the intestines of the engine operator and the supporting diver. After Day 12 of the investigation positive samples obtained from living and dining rooms, kitchen, and bathroom were markedly increased. Moreover, at Day 20, three other intestinal carriers were identified: two divers and the cook.

**TABLE 2**

**BACTERIAL SURVIVAL ON DIVING SHIPS (DAYS)**

<table>
<thead>
<tr>
<th>Organism</th>
<th>Diver Suit Material</th>
<th>Metal Parts of Diver Equipment</th>
<th>Metal of Ladder</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Salmonella typhimurium</em></td>
<td>9</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td><em>E. coli</em></td>
<td>3</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

We have managed to imitate an epidemic spread of *E. coli* strain among the crew members of the diving ship. This strain not only appeared and settled in the intestines of half of the crew members, but also developed on furnishings and equipment. Apparently a test-strain epidemic spreads from the primary contaminated objects via crew members' hands to crew members' intestines and to the kitchen. Having contaminated equipment and some of the crew members' intestines, the test strain may cycle on board the ship, involving more and more objects and more crew members as its carriers. This corresponds to data in the literature, where the possibility of exogenous spread of *E. coli* among the isolated individuals was shown (7). The situation may be more dangerous the more pathogenic the specimen circulating on board the ship, not only because of the grade of its pathogenicity but also on account of its ability to stay longer on the surface of metallic and nonmetallic objects (Table 2). Thus, we speculate that colonization and spread of *Salmonella* rods on the diving
ship are more intensive than for \textit{E. coli}, because the former may stay on contaminated surfaces for a long time, so \textit{S. typhymurium}-contaminated surfaces may serve as additional sources of infection longer than surfaces contaminated with \textit{E. coli}.

\begin{flushright}
A. B. SYSSOEV
V. K. ILYIN
V. I. PUTOW
\textit{Institute of Biomedical Problems}
\textit{Moscow, USSR}
\end{flushright}

\section*{REFERENCES}

\textit{To the Editor}

Decompression sickness risk in women

The incidence of decompression sickness (DCS) in women remains controversial. The early work of Bangassar and Bassett has been challenged by Zwingleberg et al. (1) who claim that there is no increased incidence of DCS in women. Robinson (2) disputes this finding, on statistical grounds, and asserts that the incidence rate of DCS in women could still be between 0 and 3 times that of men. However, there is another aspect to this question. Although the question of incidence has been investigated, there has presently been no research into whether women are more susceptible to type 2 than men.

I have examined 111 cases of DCS treated at the recompression unit at the Royal Australian Navy base at HMAS Stirling. Using linear logistic regression, the association between sex and disease type was considered. From this study, women have a 4.3 times greater risk of having type 2 DCS than men (95\% confidence intervals: 1.2–15.8, \(P < 0.05\)). There was no significant association between the variable of age, diving experience, compliance with recognized dive tables, absence of diving qualifications, or repetitive diving practices and type of disease.

Zwingleberg et al. (1) postulate that the increased incidence of DCS in women may be due to their increased adiposity when compared to men. Cole (3) notes that women
LETTERS TO THE EDITOR

may have increased clotting tendency, irrespective of the contraceptive pill, as well as differences in blood flow through adipose tissue, which may impede inert gas transfer. These physiologic mechanisms, while possibly contributing to an increased incidence of DCS as claimed (1, 3), may increase the risk of type II DCS in women. Further research into this area needs to look at the different susceptibilities of males and females to serious DCS as well as looking at the incidence rates and the pathophysiologic mechanisms involved.

A. G. ROBERTSON MB BS MPH
HMAS Stirling
PO Box 228
Rockingham
Western Australia 6168

REFERENCES
