

RESPONSE OF WINTER SWIMMERS TO BRIEF IMMERSION IN ICE WATER

Kyllikki Kauppinen¹⁾ and Marja Pajari-Backas²⁾

¹⁾President Urho Kekkonen Institute for Health Promotion Research, Tampere; and ²⁾United Laboratories, Ltd., Helsinki, FINLAND;

INTRODUCTION

According to Brück (1), people react to continuous or repeated cold exposure in one of two ways or their combination: (a) So-called hypothermic-insulative response, where an ample layer of subcutaneous white adipose tissue (WAT) is developed and, should the insulation be insufficient to maintain body core temperature, it is allowed to decline; or (b) metabolic response, where brown adipose tissue is developed and a continuously even body core temperature is maintained through shivering thermogenesis and brown fat nonshivering thermogenesis so long as the energy stores permit. The present study was undertaken to investigate the possibility of discernible hypothermic-insulative and metabolic responses of winter swimmers (2) to acute extreme cold exposures.

METHODS

Active winter swimmers served as informed volunteer subjects for the study. The ages of the subjects ranged from 55 to 69 years and they had practised winter swimming from seven to 30 years (22.8 ± 8.1 yrs). Based on their body mass indices (BMI) they were grouped as 'fat' or 'slim'. Their measurements are given in Table I.

TABLE I. Measurements of experimental subjects in 'fat' and 'slim' groups.

	HEIGHT m	WEIGHT kg	BMI kg/m ²
'FAT' (n = 4)	1.78 ± 0.1 (1.66 - 1.90)	102.8 ± 19.9 (83 - 130)	32.1 ± 3.3 (28.7 - 36.0)
'SLIM' (n = 6)	1.69 ± 0.1 (1.57 - 1.77)	72.7 ± 7.2 (64 - 81)	25.5 ± 1.0 (23.8 - 26.3)

The field experiments, conducted in Tampere, Finland, were run in January between 1600 and 1900 hrs. From a cabin at 22°C the subject, in swim suit, walked 10 meters to the ice covered lake, stayed immersed in ice water up to the neck for 20 seconds. He/she then returned to the cabin. Three sets of blood samples were taken: B = sample before exposure; A1 = 1st sample after exposure, immediately upon entry into room temperature; and A2 = 2nd sample after exposure, following a 10-minute rest at room temperature. The serums and plasmas were separated and stored at -70°C until analysis.

Skin surface temperatures were recorded continuously with copper-constantan thermocouples from (a) between the scapulae at level T4 on the spine, and (b) on the chest about 5 cm above the left nipple. The thermocouples were attached onto the skin as described previously (2). The probes were connected to a battery operated portable datalogger (weight 700 g) placed on a platform on top of the subject's head.

The blood samples were analyzed for free fatty acids (FFA), cortisol, epinephrine, and norepinephrine concentrations, which were corrected for individual plasma volume changes to represent concentrations at initial (B) plasma volumes. The results were statistically tested with the Friedman's two-way analysis of variance by ranks and the Student's *t*-test for unequal sizes.

RESULTS.

No abrupt rises were seen in the interscapular skin temperature in comparison to the pectoral skin temperature. At the end of the 10-minute rest period in the 'slim' group the mean interscapular surface temperature was 30 ± 1.1 °C and the mean pectoral surface temperature was 28 ± 1.9 °C. In the "fat" group the respective means were 28 ± 1.4 °C and 28 ± 3.1 °C. The results of the blood sample analyses are given in Table II.

The mean FFA concentrations in the 'fat' and 'slim' groups did not differ, nor was any change due exposure observed. The mean epinephrine concentrations were not significantly different from one another either within groups or between groups.

The mean cortisol concentrations in the two groups did not differ, nor did the mean concentration in the 'fat' group change as a result of the experimental cold exposure. In the 'slim' group the mean cortisol concentration at A2 was significantly ($P < 0.01$) higher than before (B) or immediately after (A1) exposure.

TABLE II. Serum free fatty acid, serum cortisol, plasma epinephrine, and plasma norepinephrine concentrations (mean \pm S.D.) of 'fat' and 'slim' groups before (B) and immediately after (A1) ice water immersion, and following a 10-minute rest (A2) at room temperature.

	B	A1	A2
FREE FATTY ACIDS, mEq/l			
'FAT'	0.41 \pm 0.11	0.41 \pm 0.11	0.45 \pm 0.15
'SLIM'	0.37 \pm 0.24	0.36 \pm 0.24	0.41 \pm 0.21
CORTISOL, nmol/l			
'FAT'	329 \pm 125	393 \pm 95	462 \pm 177
'SLIM'	333 \pm 264	325 \pm 234	** 464 \pm 278
EPINEPHRINE, nmol/l			
'FAT'	0.15 \pm 0.06	0.18 \pm 0.07	0.14 \pm 0.09
'SLIM'	0.13 \pm 0.05	0.17 \pm 0.09	0.27 \pm 0.19
NOREPINEPHRINE, nmol/l			
'FAT'	1.58 \pm 0.38	1.88 \pm 0.43	2.05 \pm 1.13
'SLIM'	1.88 \pm 0.55	** 3.54 \pm 3.30	2.66 \pm 1.45

** = $P < 0.01$

The mean norepinephrine concentrations between the two groups showed no statistically significant differences, but in the 'slim' group the change of the mean norepinephrine concentration from B to A1 was significant ($P < 0.01$).

CONCLUSIONS

The relatively low FFA and epinephrine concentrations in both groups of winter swimmers suggest that all subjects were adapted to cold (3) and relaxed about ice water immersions (4).

The 'fat' winter swimmers displayed the same surface temperatures on the interscapular and pectoral skin at the end of the experiment and showed no hormonal response to cold exposure. The 'slim' group, on the other hand, tended toward a warmer interscapular than pectoral skin surface temperature and showed a significant rise in the cortisol and norepinephrine concentrations following the cold water immersion. These findings lend support to the previously presented view (1) of two types of human responses to cold exposure: (a) A hypothermic-insulative response of our 'fat' group and (b) a metabolic response of our 'slim' group.

ACKNOWLEDGMENT: The work was financially supported by the Yrjö Jahnsson Foundation, Helsinki, Finland.

REFERENCES

1. Brück K. and Wünnenberg W. 1970, "Meshed" control of two effector systems: Nonshivering and shivering thermogenesis, in J.D. Hardy, A.P. Gagge and J.A.J. Stolwijk (eds.) *Physiological and behavioral temperature regulation* (Thomas, Springfield), 562-580.
2. Kauppinen K. 1989, *Sauna and Winter Swimming. Winter swimmers, their health status, and physiological responses to heat, cold, and alternating heat and cold.* Academic Dissertation, University of Helsinki, ISBN 952-90039-8-6.
3. Itoh S. 1974, *Physiology of cold-adapted man.* Hokkaido University Medical Library Series, Sapporo. Vol 7, p. 37.
4. Euler U.S. von 1964, *Commentary: Quantitation of stress by catecholamine analysis.* Clin Pharmacol Ther 5:398-400.