TABLE II. Passive Transfer after Injection of Separated Portions of Sonic Vibrated Leucocytes into Recipient Animals.

No. of donors	Time of vibration (min.)	WBC/aliquot*	Reactions 24 hr Sup.	24 hr Sed.	24 hr Cells	48 hr Sup.	48 hr Sed.	48 hr Cells
		×1000						
15	6.5	1653000	++	+	++++	++	+	++++
15	7.5	1440827	+	0	+	+	0	++
14	7.5	1285920	+	0	++	++	0	+++
13	7.5	1151702	++	0	++	+++	0	++++
10	7.5	875448	+	0	+++	+	0	+++
10	7.5	625000	+	0	+	+	0	+
10	10	1341316	+	0	+	+	0	+

* Equal numbers of exudative leucocytes were treated with sonic vibration or injected intact for control.

0 = no reaction. + = slight erythema. ++ = patchy erythema. +++ = homogeneous erythema. ++++ = marked homogeneous erythema.

Sup. = Animals receiving aqueous supernatant fluid. Sed. = Animals receiving sediment + lipid fractions. Cells = Animals receiving intact cells.

fore, any particles remaining would be under a size of 0.5 μ.

Attempts to extend this study to other delayed systems have been unsuccessful. In several experiments using tuberculin sensitive donors results were questionable. This may be due to a fundamental difference in the mechanisms of simple chemical and tuberculin sensitivity. However, in none of the tuberculin experiments was an exquisite donor sensitivity achieved, and the degree of sensitivity transferred in control animals was minimal. In experiments in progress we hope to achieve a higher quality of donor sensitivity to enable us to determine whether transfer of tuberculin sensitivity can be accomplished with cellular extracts.

Summary. Passive transfer of delayed cutaneous hypersensitivity to 2,4-dinitrochlorobenzene in guinea pigs has been accomplished with extracts of peritoneal exudative leucocytes disrupted by sonic vibration. Crude extracts of cells treated and subsequently centrifuged were effective as transfer media. Sedimented material was essentially ineffective.

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Hematological Changes Influenced by Short and Long Exposure to Cold. (21065)

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Because of the present interest in studies involving stress and resultant adaptive changes we wish to report our findings with short and long exposures of mice to cold. Selye's adaptation syndrome explanation(1) has met with both acceptance and criticism. Various forms of stress are commonly used, including heat and cold, since these stimuli are easily varied quantitatively. The duration as well as the severity of the stress is undoubtedly important for adaptation may not be a simple linear function. Emphasis at

TABLE I. Comparison of Erythrocyte Number, Hemoglobin and Blood Sugar in Control Mice, after Short and Long Exposure to Cold.

	Controls			Short exposure			Long exposure		
	RBCs (M)	Hb (g %)	BS (mg %)	RBCs (M)	Hb (g %)	BS (mg %)	RBCs (M)	Hb (g %)	BS (mg %)
	7.88	13.3	185	7.98	14.0	194	8.32	13.8	190
	8.02	13.6	184	8.53	13.5	232	8.69	14.2	200
	7.53	13.4	172	8.21	12.9	208	9.23	13.9	170
	8.56	13.8	180	7.55	13.6	178	8.02	13.8	184
	8.10	13.5	177	7.69	14.0	215	8.56	13.6	182
	7.99	13.4	186	8.03	13.3	226	7.98	13.6	202
	8.32	13.4	194	7.92	14.2	198	8.88	14.0	198
	8.25	13.6	182	8.26	13.2	204	8.36	14.4	188
	8.23	13.5	175	7.89	13.8	202	8.40	14.0	172
	7.95	13.3	168	7.68	13.8	225	8.29	13.6	178
Avg	8.08	13.5	180.3	7.97	13.6	208.2*	8.47	13.9	186.4

* Only the short exposure blood sugar (208.2 mg %) is statistically significant, having a value of $P < .001$.

present is placed on the adrenal cortex(2) and thyroid(3). Controversial reports have resulted from thyroid investigation(4,5) indicating no change in level of activity of animals (rats and men) exposed to cold. The effect of cold exposure on blood sugar level has received less attention. Exposure to cold depresses the vago-insulin system while stimulating the sympathetico-adrenal system bringing about a rise in blood sugar(6). Immersion of dogs in cold water raised blood sugar(7). Prolonged exposure to low temperature results in fluid migration from blood to tissues, anhydremia(8). Others(9) believed that there is little evidence of hemoconcentration in man with acclimatization to cold. The effect of cold on glycemia level may vary with species. Thus, in the rabbit(10) no change in blood sugar with exposure to cold was found. Also the same effect was claimed for the dog (11). The same investigators(11) noted a fall in blood sugar in the fasted rabbit exposed to cold and a rise in blood sugar in the fed rabbit with cold exposure. Others(12) believe that no change in blood sugar occurs with exposure to cold unless body temperature is altered, whereupon hyperglycemia results. A seasonal variation in fasting blood sugar occurs in man(13), being higher in winter than in summer. No references were found to the effect of cold on erythrocyte number or hemoglobin but it has been shown that exposure to low temperature increases anoxic tolerance as measured by barometric decompression(14,15). Mobilization of blood sugar in short periods of time results from severe anoxia as a response to sympathetic stimulation(16,17). A similar response might result from cold stress. Anoxia causes an early elevation in blood hemoglobin which is accounted for by splenic contraction. This would also cause an apparent rise in erythrocyte number.

Material and methods. Mice of the Purdue Swiss strain were used and these were divided into 3 groups of 10 each. Group I comprised untreated controls. Group II was exposed to 5°C temperature for 15 minutes. Group III was kept in a constant temperature room of 5° for a period of 5 days. The animals were killed by decapitation with a razor blade and blood drained out by holding the animal over a watch glass. Erythrocytes were counted on a "Brite-Line" hemacytometer, hemoglobin was measured by the Sheard-Sanford method (18) using a phototelometer, and glycemia determinations were made by the Folin-Wu micro method(19).

Results. The results of the 3 groups are shown in Table I where it can be seen that the short (15 minute) exposure to cold caused a 15% rise in blood sugar while the long exposure (5 days) did not alter the blood sugar from the control level. The increase in blood sugar following short time exposure was in all likelihood a sympathetic effect resulting from cold stress and increased thermogenesis. It is interesting that blood sugar levels had returned to normal in 5 days. No long time

effect of cold on either hemoglobin or erythrocyte number was seen. Possibly 5 days are not a long enough time in which to expect acclimatization to appear, or possibly no adaptive change to low temperature occurs in hemoglobin or erythrocyte number. With acclimatization to cold some hemoconcentration results(8), which could account for the increase seen with the long cold exposure. The number of erythrocytes found in this study falls within the range listed for different strains of mice(20). Hemoglobin and blood sugar levels also agree with those reported for the same strain of mice(21).

Summary. Mice were exposed to cold (5°C) for periods of 15 minutes and 5 days and the effect on erythrocyte number, hemoglobin concentration and blood sugar level determined. The only significant change occurred in the blood sugar level during the short period of cold exposure. Statistically this change had a P value of $<.001$. Possible physiological changes are discussed.

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A Plasma Extract with Erythropoietic Activity. (21066)

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Speculation still exists regarding the precise mechanism by which anoxia, the so-called fundamental erythropoietic stimulus, evokes the formation of red cells in the higher organism. Mediation through a humoral factor, termed "hemopoietine", has been proposed as one type of explanation(1-3). Erythropoietic activity in sera obtained from rabbits rendered anemic by bleeding or exposed to lowered barometric pressures was not detected in another experiment(4). However, the largest quantities administered by these workers to intact or anemic rabbits did not exceed 15 ml daily. More recent experiments (5) have tended to renew interest in this problem because of the finding that larger quantities of sera (50 ml/day), obtained from bled rabbits, induced significant rises in the