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Racial Variations in Sweat Gland Distribution. (26501)

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Ogata(1) reported the existence of both active and inactive sweat glands in man. Although no difference between these 2 types of glands is seen histologically, only the active glands appear to be stimulated to secretion by either pharmacological agents or thermal stress. Because the number of sites of secretions was used as an index of the number of sweat glands, the data reported here are assumed to be applicable to only the active sweat glands, by Ogata's distinction.

In Japanese subjects Kawahata(2) showed that the number of sweat glands is smaller at birth than at 2 years of age, and suggested that initially inactive glands may be recruited into use during the first years of life. In support of this suggested environmental influence on eventual number of sweat glands, he subsequently demonstrated that Japanese born and living in tropical areas have a greater number, and consequently a greater density of sweat glands than members of the same racial group inhabiting temperate zones. Seemingly further weight is added to these suggestions by Kawahata's finding that Caucasian (Russian) subjects living in northern Manchuria have fewer active sweat glands, despite larger average body size, than do Japanese from temperate climates. In making the latter comparison, however, Kawahata noted that possible racial differences might have influenced these data. Kawahata and Sakamoto(3) have shown that Ainos in Hokkaido, Japan, have fewer sweat glands than do Japanese inhabiting the same island. Both factors modifying sweat gland distribution, racial and environmental, merit additional study.

Methods. The subjects sampled consisted of male Negroes and male and female Caucasians and Eskimos. The Negro subjects were born and lived in the midwestern United States. The origins of the Caucasian group were varied as shown in Table II. The Eskimo group was sampled in their native village (Anaktuvak Pass) in the Brooks mountain range in interior Alaska. The ages of all subjects are reported with the observed sweat gland data in Tables I to III.

Sweat gland measurements were made in heated chambers maintained at 41°-42°C after the subject had been in the room for 30-50 minutes and was sweating maximally. Sweat glands were counted by Jurgensen's method as modified by Kawahata(2). This method consists of counting under magnification (10×) the sites of sweat secretion on the skin surface covered with tinted cedar oil. The secretion sites corresponding to the openings of the underlying glands are counted within a 0.09 cm^2 area stamped onto the skin

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TABLE I. Male Caucasian and Male Negro.

Subj. No.	Age (yr)	Total sweat glands ($\times 10^3$)	No. of sweat glands (per cm^2)
A. Male Negro (mean S.A. = 1.86 M^2)			
1	25	2024	115
2	20	2046	109
3	21	2058	114
4	22	2138	109
5	23	2199	124
6	31	2203	125
7	20	2256	132
8	19	2313	136
9	36	2371	100
Mean ± S.E.		2179 ± 115	117 ± 11
B. Male Caucasian (mean S.A. = 1.88 M^2)			
1	25	1800	92
2	26	2304	125
3	21	2364	119
4	21	2369	130
5	23	2405	126
6	16	2523	139
7	21	2579	147
8	18	2640	126
9	22	2815	179
10	24	2894	128
Mean ± S.E.		2469 ± 290	131 ± 21

before application of the oil. Estimation of active glands depends on averaging sample points(3-5) at 20 different skin regions over the body. Eight regions were used for the Eskimo group (forehead, cheek, upper and lower arm and leg, and dorsum of hand and foot). From these data both total number and distribution of active sweat glands may be estimated. The surface area for each meas-

TABLE II. Female Caucasian.
(Mean S.A. = 1.63 M^2)

Subj. No.	Age (yr)	Total sweat glands ($\times 10^3$)	No. of sweat glands (per cm^2)	Geographical origin
1	19	2478	170	Denmark
2	22	2501	171	Germany
3	21	2572	147	U.S.A.
4	22	2663	182	Germany
5	20	2849	154	U.S.A.
6	18	2984	178	"
7	19	3010	182	"
8	19	3069	185	"
9	20	3111	200	Germany
10	18	3155	184	U.S.A.
11	20	3254	183	Turkey
12	25	3279	221	"
13	20	3533	204	U.S.A.
14	17	3551	219	Turkey
15	24	3611	240	"
16	21	3679	211	Holland
17	29	3827	236	Hungary
Mean ± S.E.		3123 ± 410	192 ± 25	

TABLE III. Eskimo.

Subj. No.	Age (yr)	Total sweat glands ($\times 10^3$)	No. of sweat glands (per cm^2)
A. Male Eskimo (mean S.A. = 1.77 M^2)			
1	27	1727	102
2	44	2077	118
B. Female Eskimo (mean S.A. = 1.58 M^2)			
1	18	2452	157
2	23	1771	105
3	20	2592	165
4	14	2725	192
5	16	3068	206
6	35	2476	155
7	29	1934	124
8	36	2071	141
	Mean	2386	156

ured body region was derived from a linear formula suggested by DuBois and modified by Kawahata(4). Whole body surface areas for the Eskimo group were estimated from height and weight, according to the method of DuBois(5). The accuracy and reproducibility of measurements of active sweat glands during maximal sweating have been confirmed by Randall(6).

Results. Tables I to III contain the data for all subjects. The density and total number of sweat glands for Caucasian men exceeds those for the Negro subjects ($P < 0.02$),† even though these 2 groups have similar average whole body surface areas (1.88 and 1.86 m^2) (Table I). Further, comparison of these data with those in Table II shows that Caucasian women exceed both Caucasian and Negro men in these measures ($P < 0.01$). Even though average whole body surface areas (1.63 m^2) for the Caucasian female subjects are smaller ($P < 0.01$) than those of the males of the same race (1.88 m^2), the total number of glands appears to be greater ($P < 0.01$).

Both of these measurements are smaller for the Eskimo than for the Caucasian women ($P < 0.05$) whereas no apparent differences ($P > 0.05$) exist either within the Eskimo group or between these two races for male subjects, although the sampling of Eskimo men was too small to support conclusive statements (Table III).

Discussion. According to Randall(6)

† Fisher T test.

higher environmental temperatures are required to produce observable sweat secretions from normal glands than from those affected by mecholyl iontophoresis. He suggests on this basis that the "inactive glands" described by Ogata(1) were simply not responding to the thermal stimulus. Ogata reported earlier, however, that the same total number and distribution of sweat glands were measured with either extreme heat or locally injected pilocarpine. Using injected acetylcholine instead of pilocarpine, Kawahata(7) obtained similar results.

Other evidence also argues against the assumption that less active sweat glands may be mistaken for inactive glands in these measurements(8). These data show that sweat production follows predictably with increasing degrees of locally applied thermal stress, although total number or position of active glands does not change.

Since a tropical environment influences early activation of sweat glands, it might appear surprising that the Caucasian males showed a greater number of activated sweat glands than did the male Negroes, members of a racial group with essentially a tropical origin. However, the Negro subjects in this study were born and reared in temperate climates and these physiological measurements might more predictably reflect environmental than genetic factors. This might also be pertinent to an interpretation of Thomson's report(9) of no differences in sweat gland density between native Negroes and immigrated Caucasians in tropical Africa. These data, however, may not have been gathered under conditions of maximal sweating, as indicated by Kuno(10). Ages of the Caucasians at time of immigration must be considered. In view of the similarities of environmental influences on Negro and Caucasian groups in this study, the reported differences in sweat gland density most reasonably appear to be of a racial origin.

The sex difference in sweat gland distribution in the Caucasian group may appear paradoxical considering the observations of Tanaka(11) and others(12-15) that the male Caucasian response to thermal sweating is greater than that of the female Caucasian. However, this difference may be one of response threshold to stimulation and activity level of the activated sweat gland and not one of distribution. Additional data on other racial groups must be obtained before these observations can be attributed to a sex difference, rather than to environmental or other factors.

It is interesting that the measurements indicate that Eskimo women had fewer active sweat glands than female Caucasians have; however, both environmental and racial effects may be operating. The efficient microenvironment of the Eskimo even in the extreme thermal stress of their natural climate must be considered before these differences are attributed to environmental factors.

Summary. Total number and distribution of active sweat glands were determined for American Negro, Caucasian and Eskimo subjects under conditions of maximal thermal sweating. Both sexes were included in Caucasian and Eskimo groups, whereas only adult, male Negro subjects were investigated. A greater density and total number of active sweat glands were found in Caucasian males than in Negro males; Caucasian women appear to exceed any other group in these measurements, regardless of race or sex. The measurements in Eskimo women did not follow this pattern and they appear to be like the others measured.

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Embolic Trophoblast in Peripheral Circulation During Pregnancy.* (26502)

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Pulmonary embolism by trophoblastic fragments during pregnancy was first demonstrated by Schmorl(1), and is now considered a commonplace finding in normal gestation. Park(2), as well as Bardawil and Toy (3) have implied that one may anticipate uncomplicated trophoblastic embolism to the lungs in roughly half of all seemingly physiological pregnancies. The fate of ectopic trophoblasts has aroused particular interest as a possible factor in the genesis of choriocarcinoma. Experiments such as those by Park(2) have suggested a destiny no more threatening than ultimate degeneration and disappearance from the host.

Until recently, free trophoblastic embolism has been observed only in the pulmonary capillaries. Douglas *et al.*(4) recently reported isolation of syncytial masses at time of Caesarean section from the veins of the broad ligament in 8 of 13 cases, the ovarian veins of one patient, and the inferior vena cava in 3 samples from an unstated number of cases out of a total of 33 explored. Attempts to isolate trophoblasts from the antecubital vein failed to reveal positive results. The desirability of a statistical perspective on the problem of trophoblastic embolism and its analysis has induced the authors to submit their results of a similar survey.

Materials and methods. Blood samples were drawn from a total of 155 patients in various stages of pregnancy, 10 of whom were in active labor at time of sampling. Specimens were obtained in all cases from the antecubital vein and in 22 of these patients blood was also secured from the placental site during Caesarean section. Ten smears and a serially sectioned cell block were examined in each of the first 130 cases; 5 smears were evaluated in each of the remaining 25 cases. The latter included 5 samples from the placental site.

Early attempts to apply the method of Sandberg and Moore(5) yielded unsatisfactory results. The majority of samples have been processed according to the technic of Malmgren and his group(6) as later modified by Long and his colleagues(7). Heparinized blood is centrifuged and the cellular sediment treated with streptolysin O to destroy erythrocytes and polymorphonuclear leukocytes. The remaining cellular debris is concentrated by centrifugation and smeared out on slides for staining by the Papanicolaou technic.

All slides were scanned systematically with a mechanical stage; all positive or equivocal cell forms were marked and restudied until classified.

Results. In no case was a cell remotely resembling ectopic trophoblasts retrieved from the antecubital vein of a pregnant patient. Occasional bizarre forms were observed but rejected as blood cell debris upon close examination. In 3 cases, all sampled at

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