properties differentiate the physical chemical characteristics of DST from SDS. The CMC of SDS was 4 m*M* under the conditions of these experiments, whereas the CMC of DST was 21 m*M*, and cholesterol was more efficiently solubilized by the longer DST molecule. The bulky hydrated head group should stabilize the DST micelle, and reduce the hydrophobic chain interactions, thus opening the palisade layer of the micelle for interactions with the bulky, nonpolar parts of sterols. For the same reason, the strong, bulky, polar head group may render the molecules so soluble in water that they are poor oil-water emulsifiers. The rigidity of the steroidal hydrophobic parts of simple bile salt micelles reduces their efficiency for cholesterol solubilization. However, once lecithin is incorporated into the micelles, the acquired liquid hydrocarbon core increases cholesterol solubility significantly (Fig. 3).

Whatever the precise explanation for those differences, the results establish that the crustacean detergent is not an efficient emulsifier but exhibits a marked capacity to solubilize cholesterol and lecithin as mixed micelles. While further studies will have to be performed to see the effect of free fatty acid and other constituents of the postcibal intestinal milieu on solubilization, our results support our hypothesis that these detergents promote the intestinal absorption of ingested sterol. The high capacity of crustacean detergent for cholesterol solubilization ensures the maintenance of cholesterol in solution in the exocrine secretion of hepatopancreas even at low concentrations of lecithin, and promotes the efficient solubilization of dietary sterols prior to absorption. The results also suggest that DST may serve as a model for detergent replacement in bile salt deficiency syndromes in humans.

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References and Notes

1. C. R. Treadwell and C. V. Vahouny, in *Handbook of Physiology*, C. F. Code, Ed. (American Physiological Society, Washington D.C., 1968), vol. 3, p. 72; A. F. Hofmann, *ibid.*, vol. 5, p. 2507; M. C. Carey and D. M. Small, *Am. J. Med.* **49**, 590 (1970).
2. H. Danielsson, in *Bile Salt Metabolism*, L. Schiff, J. B. Carey, J. M. Dietschy, Eds. (Thomas, Springfield, Ill., 1969), p. 91; in *The Bile Acids*, P. P. Nair and D. Kritchevsky, Eds. (Plenum, New York, 1973), vol. 2, p. 1; E. H. Mosbach, *Arch. Intern. Med.* **130**, 478 (1972); G. A. D. Haslewood, in *Handbook of Physiology*, C. G. Code, Ed. (American Physiological Society, Washington, D.C., 1968), vol. 5, p. 2375; M. C. Carey and D. M. Small, *Arch. Intern. Med.* **130**, 506 (1972).
3. H. O. Wheeler, *Arch. Intern. Med.* **130**, 533 (1972); S. Nilsson and T. Scherstén, *Gastroenterology* **57**, 525 (1969); A. F. Hofmann and M. S. Mekhjian, in *The Bile Acids*, P. P. Nair and D. Kritchevsky, Eds. (Plenum, New York, 1973), vol. 2, p. 103; V. L. Sallee and J. M. Dietschy, *Science* **174**, 1031 (1971); W. G. M. Hardison and J. T. Apter, *Am. J. Physiol.* **222**, 61 (1972); H. O. Wheeler and K. K. King, *J. Clin. Invest.* **51**, 1337 (1972); R. H. Dowling, E. Mack, D. M. Small, *ibid.* **50**, 1917 (1971).
4. H. J. Vonk, in *The Physiology of Crustacea*, T. M. Waterman, Ed. (Academic Press, New York, 1960), vol. 1, p. 291; *Arch. Int. Physiol. Biochim.* **70**, 67 (1962); H. Brockerhoff, J. E. Stewart, W. Tacreiter, *Can. J. Biochem.* **45**, 421 (1967); H. Brockerhoff and R. J. Hoyle, *ibid.*, p. 1365; ———, P. C. Hwang, *J. Fish Res. Board Can.* **27**, 1357 (1970); A. H. A. van den Oord, thesis, University of Utrecht (1965).
5. *Scientific Tables* (Ciba-Giegy, Basle, Switzerland, 1970), p. 498; A. Kanazawa, N. Tanaka, S. Teshima, K. Kashiwada, *Comp. Biochem. Physiol.* **37**, 211 (1971).
6. A. H. A. van den Oord, *Comp. Biochem. Physiol.* **13**, 461 (1964); D. I. Zandee, *Nature (Lond.)* **202**, 1335 (1964); *Comp. Biochem. Physiol.* **20**, 811 (1967); *Arch. Int. Physiol. Biochim.* **74**, 435 (1966); L. Gosselin, *ibid.* **73**, 543 (1965).
7. D. R. Idler and P. Wiseman, *Comp. Biochem. Physiol.* **26**, 1113 (1968).
8. A. H. A. van den Oord, H. Danielsson, R. Ryhage, *Nature (Lond.)* **203**, 301 (1964); D. A. Holwerda and H. J. Vonk, *Comp. Biochem. Physiol.* **45B**, 51 (1973).
9. S. Teshima, *Comp. Biochem. Physiol.* **39B**, 815 (1971); ——— and A. Kanazawa, *ibid.* **38**, 603 (1971); A. Kanazawa and S. Teshima, *Bull. Jap. Soc. Sci. Fish.* **37**, 891 (1971); M. Florkin and B. T. Scheer, Eds., *Chemical Zoology* (Academic Press, New York, 1970), vol. 5, pp. 241–242.
10. A. H. A. van den Oord, H. Danielsson, R. Ryhage, *J. Biol. Chem.* **240**, 2242 (1965).
11. ———, *Comp. Biochem. Physiol.* **17**, 715 (1966).
12. H. J. Vonk, *ibid.* **29**, 361 (1969).
13. S. R. Dowd, J. M. Little, R. Lester, in preparation.
14. E. Krafft and H. Wiglow, *Ber. Deutsch. Chem. Ges.* **28**, 2566 (1895); F. Lachampt and R. Perron, in *Traité Chim. Organ.*, V. Grignard, G. Dupont, R. Locquin, Eds. (Masson & Cie, Paris, 1953), vol. 22, p. 837.
15. The apparent *pK* of DST was calculated by taking the *p*H corresponding to the point on the curve where half an equivalent of the detergent was titrated. The equivalent weight was calculated from the gravimetric weight and the formula molecular weight of the detergent. Titration of the sulfonate group of the detergent was assumed to commence at the inflection point of the curve.
16. F. Rosevear, *J. Am. Oil Chem. Soc.* **31**, 628 (1954); *J. Soc. Cosmet. Chem.* **19**, 581 (1968); F. Lachampt and R. M. Vila, *Rev. Fr. Corps Gras* (February 1969), No. 2, p. 87; J. M. Corkill and J. F. Goodman, *Adv. Colloid Interface Sci.* **2**, 297 (1969).
17. D. M. Small, M. C. Bourges, D. G. Dervichian, *Biochim. Biophys. Acta* **125**, 563 (1966).
18. B. Isaksson, *Acta Soc. Med. Upsal.* **56**, 177 (1951); *ibid.* **59**, 277 (1953–54); thesis, University of Gothenberg, Sweden (1954); W. M. Admirand and S. M. Small, *J. Clin. Invest.* **47**, 1043 (1968); M. C. Carey and S. M. Small, in *Bile Acids in Human Diseases III*, S. Matern and J. Hackenschmidt, Eds. (Schattauer Verlag, Stuttgart, in press).
19. *Handbook of Chemistry and Physics*, R. C. West, Ed. (Chemical Rubber Company, ed. 54, Cleveland, Ohio, 1973), F–186.
20. A considerable physiological advantage may be provided by these straight-chain detergents over bile salts in the presence of high concentrations of divalent cations. K. Hofmann [thesis, University of Lund, Sweden (1964)] demonstrated that the common vertebrate bile salts are readily precipitated from solution by calcium salts, whereas we found that synthetic DST in concentrations found in crab juice was soluble in artificially prepared seawater. It is of interest that better detergency in washing is reputed to be associated with detergents with branched head groups [A. M. Schwartz and J. W. Perry, *Surface Active Agents* (Interscience, New York, 1949), pp. 102, 385], and a group of industrial detergents (Igepons) which are mixtures of *N*-acyl-*N*-methyltaurines have been developed for use in conditions of acidity and hard water in the textile industry [M. L. Kastens and J. J. Ayo, *Ind. Eng. Chem.* **42**, 1626 (1950)].
21. Marine crustacea, like other marine animals, have high concentrations of long-chain polyunsaturated fatty acids in both tissue triglycerides and phospholipids [*Fish as Food*, G. Börgström, Ed. (Academic Press, New York, 1961), vol. 1, pp. 164, 213]. In the lobster hepatopancreas, these unsaturated fatty acids are located predominantly at the 2-position of triglycerides, phosphatidyl cholines, and phosphatidyl ethanolamines [M. Brockerhoff, R. C. Ackman, R. J. Hoyle, *Arch. Biochem. Biophys.* **100**, 9 (1963)].
22. C. R. Bartlett, *J. Biol. Chem.* **234**, 406 (1959); J. J. Carr and I. J. Drekter, *Clin. Chem.* **2**, 353 (1956); L. L. Abell, B. B. Levy, B. B. Brodie, F. E. Kendall, *J. Biol. Chem.* **195**, 357 (1952) (the concentration of detergents was calculated by the difference between the deproteinized dry weights and the analytical sums of cholesterol, lecithin, and electrolytes in each sample).
23. Supported by PHS grants AMHD17847, HD-08954, AM11453, AM18559, and AM01128 and a grant from the Medical Research Foundation, Inc., Boston, Massachusetts. Facilities of the Biophysics Division, Department of Medicine, Boston University School of Medicine, and the Marine Biological Laboratory, Woods Hole, Massachusetts, were used. We thank Drs. Klaus Hofmann and D. M. Small for their advice, assistance, and encouragement.

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Arteriovenous Anastomoses in the Skin of the Weddell Seal, *Leptonychotes weddelli*

Abstract. *Arteriovenous anastomoses of epithelioid type were demonstrated in Weddell seal skin. The majority occurred just beneath the epidermis and among the hair follicles. There was no significant variation in density of these anastomoses between body and flipper skin. These observations suggest that arteriovenous anastomoses are important in thermoregulation in the Weddell seal, particularly as heat dissipating structures when the animal is out of the water, and that the entire body surface is involved rather than specific regions such as the flippers.*

In this report we describe the structure, distribution, and density of arteriovenous anastomoses (AVA's) of epithelioid type (*1*) in the skin of the Weddell seal, *Leptonychotes weddelli*. To our knowledge, AVA's have not been described previously in the skin of marine mammals, although their presence was suspected in two species of seals (*Callorhinus ursinus* and *Phoca vitulina*) by Tarasoff and Fisher (*2*).

Skin samples of a 2-day-old female pup and an adult female Weddell seal were taken from the dorsal midline between the scapulae, and from the dorsal aspect of the

carpal region in the foreflipper, and fixed in 10 percent neutral buffered formalin. Serial sections (6 μm) were stained with hematoxylin and eosin and examined to determine the size, structure, and distribution of AVA's. In the determination of the density of AVA's, each anastomosis was identified from its arterial origin through its epithelioid segment to its venous termination, and its location was entered on a diagram of the skin sample to insure that it was counted only once.

Arteriovenous anastomoses in the body and flipper skin of the Weddell seals resembled the simple type of AVA's described in the skin of other mammals (*1*). In the seal the anastomoses were C-shaped or slightly coiled vessels in which the characteristic segments, artery, epithelioid segment, and vein could be recognized (Fig. 1).

Anastomoses occurred throughout the dermis and hypodermis, the majority (65 percent) occurring superficially beneath the epidermis and among the hair follicles.

In both the pup and the adult there was no significant variation in density of AVA's between body and foreflipper skin (Table 1). In the pup the AVA's were smaller, but of a higher density, than in the adult. Assuming that the total number of AVA's in the skin is established at birth, these differences may indicate merely a relationship between density of AVA's and total skin area.

Table 1. Density of arteriovenous anastomoses (AVA's) in Weddell seal skin.

Animal	Region	Skin area (cm^2)	AVA's	
			(N)	(N/cm^2)
Pup	Body	0.049	69	1408
Pup	Flipper	0.119	153	1286
Adult	Body	0.098	93	949
Adult	Flipper	0.080	78	975

The structure, distribution, and density of AVA's in the skin of the seal differ from those in a terrestrial mammal, the sheep. In the sheep, the majority of AVA's occur at the dermal-hypodermal junction, and the greatest complexity and density of AVA's is found in forelimb skin (*3*), which has been shown to have a thermoregulatory function (*4*). In contrast, our study has shown that in the Weddell seal there is no difference in structure, distribution, and density of AVA's in body and flipper skin. All the AVA's are relatively simple in structure, the majority are in a superficial position just beneath the epidermis and among the hair follicles, and their density is many times greater than that in the sheep.

Weddell seals inhabit Antarctic coastal waters associated with sea ice, where the water temperature varies little from its freezing temperature of -1.7°C (*5*). Heat stress in the aquatic environment is expected to be virtually nonexistent, while heat conservation is of major concern. Seals are well adapted to conserve body heat, having a heavy blubber layer which is a most effective insulator (*6*) and a vascular pattern in the flippers that suggests a heat-conserving mechanism (*2*). It is unlikely that AVA's are involved in heat conservation, as suggested by Tarasoff and Fisher (*2*), because they are too superficial to be effective in this way. It has been demonstrated that general peripheral vasoconstriction in the extremities conserves body heat (*7*). However, on the rocks or ice where these seals haul out there is a wide variation in ambient temperature, and heat stress can occur on occasions (*8*).

The high density of the AVA's in Weddell seal skin and their position superficial to the blubber suggest that they are important in dissipation of heat, particularly when the animal is out of the water. Dilation of AVA's accompanied by heat loss has been described in the ear of the rabbit (*9*). A similar relationship of AVA's to heat loss in the leg of the sheep has been suggested (*3*). In the seal, if the large numbers of AVA's present were to open there would be a considerable increase in blood

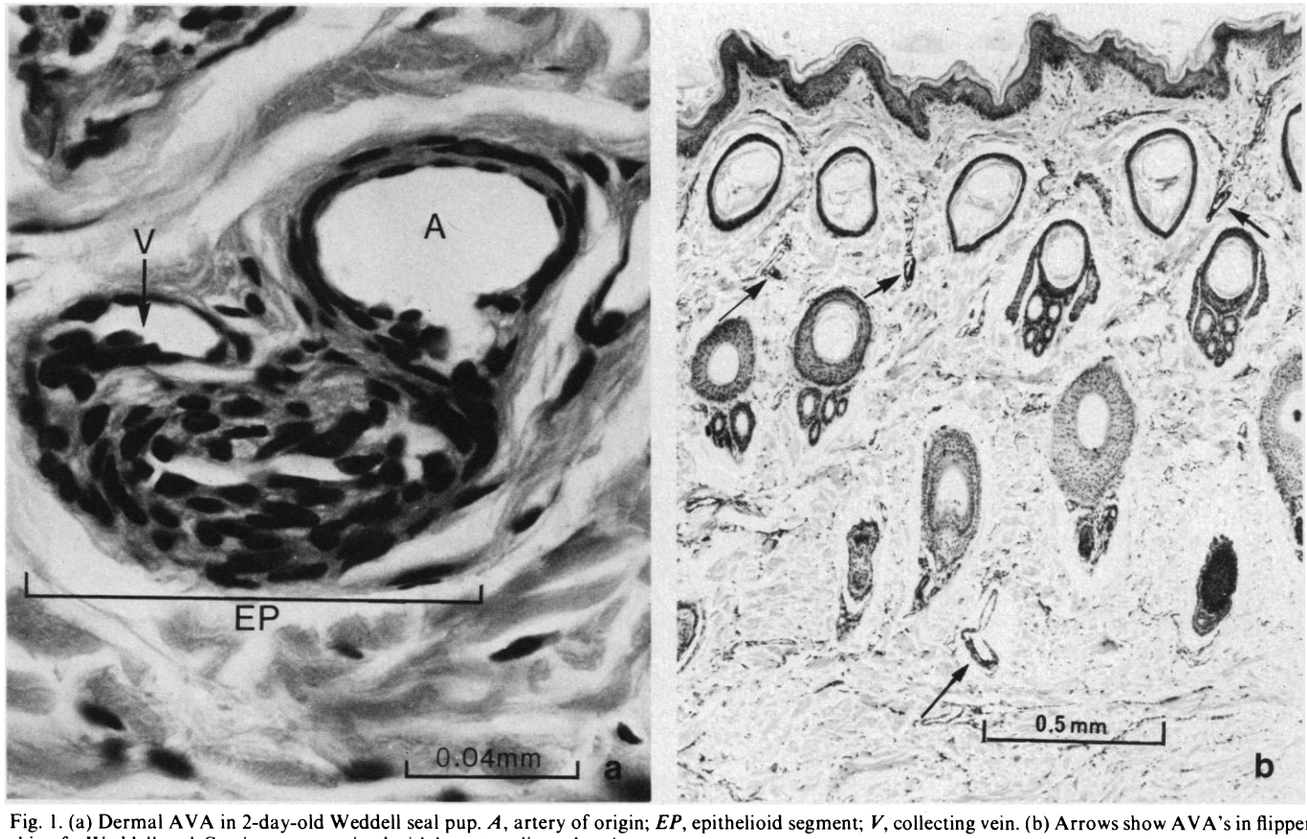

Fig. 1. (a) Dermal AVA in 2-day-old Weddell seal pup. *A*, artery of origin; *EP*, epithelioid segment; *V*, collecting vein. (b) Arrows show AVA's in flipper skin of a Weddell seal. Sections were stained with hematoxylin and eosin.

circulation through the skin, allowing heat loss. In this respect, our findings suggest that dissipation of body heat may occur from the entire skin surface or from local regions of it, rather than from specific peripheral areas such as the flippers.

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References and Notes

1. S. von Schumacher, *Arch. Mikrosk. Anat. Entwicklungsmech.* **71**, 58 (1908).
2. F. J. Tarasoff and H. D. Fisher, *Can. J. Zool.* **48**, 821 (1970).
3. G. S. Molyneux, in *Biology of the Skin and Hair Growth*, A. G. Lyne and B. F. Short, Eds. (Angus & Robertson, Syndey, 1965), p. 591.
4. M. E. D. Webster and K. G. Johnson, *Nature (Lond.)* **201**, 208 (1964).
5. J. S. Hart and H. D. Fisher, *Fed. Proc.* **23**, 1207 (1964).
6. M. M. Bryden, *Nature (Lond.)* **203**, 1299 (1964).
7. P. F. Scholander, *Hvalradets Skr.* **22** (whole issue) (1940).
8. C. Ray and M. S. R. Smith, *Zoologica* **53**, 33 (1968).
9. E. R. Clark and E. L. Clark, *Am. J. Anat.* **54**, 229 (1934).
10. This project was financed by a grant from the Australian Research Grants Committee, with field support generously supplied by the National Science Foundation, Washington, D.C. We thank R. A. Tedman for assistance with field collection of tissue samples and L. Bell for technical assistance.

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Color Vision and Brightness Discrimination in Two-Month-Old Human Infants

Abstract. *A red or white bar, embedded in a white screen, was systematically varied in intensity. Infants consistently located and stared at the white bar unless it closely matched the screen in intensity. They also stared at* all *intensities of the* red *bar, presumptively including the red-white brightness match, and hence must have some form of color vision.*

If an organism can discriminate a colored light from a "white" light, solely on the basis of their difference in wavelength composition, then the organism is said to have color vision (*1*). In this report we present evidence that 2-month-old human infants can make such a discrimination.

It has been demonstrated several times that infants can discriminate between objects or lights having different wavelength compositions (*2*). The difficulty lies in proving that the discriminations are being made on the basis of wavelength (or chromatic) differences rather than just on the basis of infant luminance (or brightness) differences.

Infants' spectral sensitivity curves—the relative sensitivity to different wavelengths of light—are known to be quite similar to those of human adults, especially in the middle- and long-wave regions of the spectrum (*3*). Hence a heterochromatic brightness match made by a color-normal adult provides a good first approximation to the brightness match for an infant, but does not guarantee the complete elimination of brightness differences.

Our approach toward eliminating the brightness cue was to use a long wavelength (red) light and test the infant's capacity to discriminate it from a white light. We started from the adult red-white brightness match, and explored a *range* of relative intensities centered around this match. We explored this range in small enough intensity *steps* to ensure that in at least one case the red and white lights would have to be indiscriminable in brightness for the infant. If the infant could discriminate between red and white for *all* of the relative intensities used (including, then, whichever one is a brightness match), the infant must have color vision.

It is extremely likely that, for red light, the range ±0.4 log unit around the adult's red-white brightness match will somewhere contain each individual infant's red-white brightness match (*3*). Thus, we chose intensities about 0.4 log unit above and below the adult brightness match as the end points of the range, for a total range of a little more than 0.8 log unit.

In order to choose the size of the intensity steps needed for detailed examination of the 0.8 log unit range, we decided to leave color aside temporarily, and find out how sensitive the infant is to small brightness differences, using only white lights.

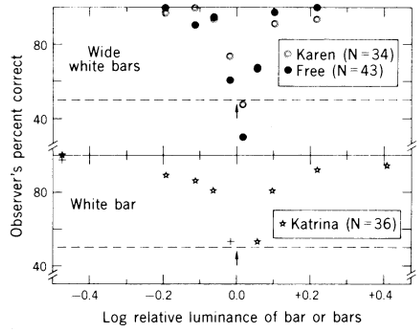

Fig. 1. (Top) Brightness discrimination functions in two 2-month-old human infants, Karen and Free. Zero on the abscissa represents the intensity at which a set of wide white bars matched a surrounding white screen. Both infants are sensitive to very small intensity differences. (Bottom) Same as top, but the four wide bars were replaced by a single narrower white bar, and a third infant, Katrina, was used. The brightness discrimination function is broadened somewhat. The plus marks (+) indicate data collected during the last day's session.

In this experiment, each of two 2-month-old female infants (*4*) was held 34.5 cm from a 0.1 log mlam white screen of a color temperature of about 2650°K. An observer watched the infant's face through a peephole in the center of the screen. On either side of the peephole (centered 16.5 cm, or 24.2°, laterally) four vertical rectangular openings (8.4 by 1.2 cm, or 13.9° by 2.0°) were cut in the screen. The openings formed four cycles of a square-wave grating of about 0.25 cycle/deg.

Diffusing screens were located about 10 cm behind the openings, and could be independently back-illuminated. On every trial, the back illumination was arranged to make the light coming through one set of openings match the screen in brightness and hue, so that the screen looked virtually homogeneous (to us) on that side of the peephole. The light from behind the other set of openings could be set to a variety of intensities, above or below that of the screen, and formed (for us) a set of readily visible bars. The intensity of these bars, and the side on which they were presented, varied randomly across trials.

When the intensity of the bars differs enough from that of the screen, an infant will stare fixedly in the direction of the bars (*5*), and this behavior forms the basis of our response measure (*6*). The observer, looking through the peephole, was not told the position or intensity of the bars. On each trial, the observer was required to judge the side on which the bars were located by observing the pattern of the infant's eye and head movements. If the observer performs better than chance at judging the location of the bars, it follows that the infant can see the bars. Thus, percent correct on the part of the observer was our dependent measure and above-chance values indicate that the infant sees the stimulus. When the intensity of the bars approaches that of the screen, the infant's staring behavior becomes random and the observer's performance drops to chance.

Figure 1 (top) shows the observer's percent correct in naming the position of the bars, as a function of the log relative luminance of the bars. For intensity differences of about 25 percent (0.1 log unit) and above, the observer's performance was always 90 percent or better. Of the intensities we used, only the increment of 5 percent (0.02 log unit) above the background intensity was small enough that the infants failed to stare at the bars. Under the stimulus conditions of the experiment, then, the U-shaped dip in the discrimina-