to achieve the same equilibrium body temperature. Therefore, at low wall emissivities a relatively high surface temperature means that an animal may not be under nearly as great a cold stress as might be assumed from measurements of air temperature alone. Interpretations of an animal's tolerances to cold environments may lead to overestimates of an animal's capabilities. Interpretations of tolerances to warm environments can also be misleading, since at low wall emissivities the sum of emitted and reflected thermal radiation from the walls may be only slightly higher than the energy radiating from the surface of the animal.

Since physiological studies of metabolism are usually correlated to air temperature, Fig. 2 shows the changes in metabolism that might be expected at a fixed air temperature for different thicknesses of fur or feathers of model spherical animals. I determined the points on these graphs by using a computer program that simultaneously solves Eqs. 8 and 9.

$$T_r = T_b - \frac{(M - E_{ex})\,A_s}{4\pi r_b r_s k_b/(r_s - r_b)} - \frac{(M - E_{ex} - E_{sw})\,A_f}{4\pi r_f r_s k_f/(r_f - r_s)} \quad (8)$$

Equation 8 was derived from Eq. 7, Porter and Gate's figure 4 (*2*), and Birkebak's equation (*4*) for heat conducted to a sphere's surface. Equation 8 describes the steady-state heat transfer between the core of a sphere and its surface. Equation 9 describes the steady-state heat transfer between the surface of the sphere and its environment.

$$M = Q_{12} + C + E_{ex} + E_{sw} \quad (9)$$

In these equations M is the metabolic heat production, E_{ex} is the energy lost by evaporation during breathing (in calories per square centimeter per minute), E_{sw} is the energy lost by water evaporating from the skin, T_r is the surface temperature, T_b is the body (core) temperature, A_s is the skin area, r_s is the radius of the sphere to the skin, r_b is the radius out to the point in the body wall where temperature begins to drop from the core temperature (*2*), k_b is the conductivity of flesh (*9*), A_f is the surface area of fur or feathers, r_f is the radius to the effective radiating temperature of the fur (*2*), k_f is the fur conductivity, and C is the convection from a sphere at the appropriate Reynolds number (*3*). Conductive heat loss is assumed to be negligible and has not been included.

The assumptions used in Fig. 2, a and b, are that the animal has curled into a ball whose outside diameter is 5 cm (about the body diameter of a cardinal). The only difference in the values used to compute the points in Fig. 2, a and b, is the air temperature. The vertical line, $\varepsilon_2 = 1.0$, indicates the energy absorbed from black surfaces at −20°C and 0°C, respectively. A comparison of Fig. 2a with Fig. 2b indicates that over the full range of emissivities the change in metabolic requirements at a given thickness of insulation is dependent on the magnitude of the difference between the core and the air temperature.

Figure 2c is an extreme, hypothetical example which shows how a large animal curled into a spherical shape could be expected to respond to changes in wall emissivity. For identical environments (Fig. 2, b and c) metabolic requirements are less for a 60-cm spherical shape than for a 5-cm spherical shape because a larger object has a thicker boundary layer (*2*, *10*) that insulates its surface more effectively from air temperature. Since a larger object with little fur is not cooled as much by convection, less metabolic heat is required, for example, at $\varepsilon_2 = 1.0$, to maintain a body temperature of 38.5°C at an air temperature of 0°C.

Thus, in order to keep Q/Q_{max} (Fig. 1) as close to 1 as possible and thereby to minimize complications involving view factors and reflected thermal radiation in metabolic chambers, wall emissivity should approach 1. Alternatively, if material of lower emissivity is essential for chamber construction, the container should be as large as possible relative to the organism's size so as to keep the ratio of surface areas, A_1/A_2, small. If neither course is possible, area ratios and wall emissivities must be determined to accurately establish an animal or plant's absorbed thermal radiation.

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Virus of the 1918 Influenza Pandemic Era: New Evidence about Its Antigenic Character

Abstract. *In serums of unusually isolated Pacific islanders whose only exposure to influenza occurred during the era of the 1918 pandemic the residual neutralizing antibody was greatest to the PR/8 and BH strains of human type A influenza virus, significantly lower to swine influenza virus, and absent to equine or later human type A virus strains. The pandemic virus was thus antigenically closer to human type A strains isolated during the middle 1930's than to other known influenza virus types.*

During the course of our studies of the immune response to influenza vaccines in isolated Pacific island populations (*1*), we discovered in 1964 that the population of one particularly inaccessible western Caroline island, Fais, had not experienced type A influenza for several decades. In a total population of over 200, not a single individual born after 1924 and who had never been off the island had neutralizing antibody to any of several strains of virus spanning the subgroups of influenza virus type A. Antibody to the early subtype A strains, however, was evenly spread throughout the older age groups in approximately half of the 80 people born before 1924.

Table 1. Geometric mean titers of antibody at various test doses of several strains of influenza virus in ten individuals whose only exposure to influenza occurred during the era of the 1918 pandemic (*12*).

Virus strain	Units of virus in test			
	4*	8	16	32
SW	37	25	20	9
WS	48	15	13	9
PR/8	111	70	37	13
BH	86	65	ND	ND
HICK	2	0	0	0
CAM	1	0	0	0
AA/1	2	0	0	0
TW/1	0	0	0	0
EQ-1	0	0	0	0
EQ-2	0	0	0	0

* Some individual titers unreliable at this low dose of virus.

Although most of the world was afflicted during the three successive waves of influenza in 1918–19, a few insular regions, mainly in the southwestern Pacific, are known to have escaped: the Solomon, Gilbert, and Ellice islands, the New Hebrides, New Caledonia, and New Guinea (*2*). Localized epidemic "trailers" continued to occur throughout the early 1920's, and, around the western Pacific basin, severe epidemics were recorded in parts of Australia in 1923, of China in 1924, and of the Philippine Islands in 1925 (*3*). A Japanese medical survey dated a severe influenza-like outbreak in 1924 on the island of Yap (*4*), from which most ships to Fais depart; according to native accounts the population of Fais at about this time suffered a single, devastating influenza-like epidemic.

These historical facts, together with our serological findings, indicate that the population of Fais experienced in 1924 its first contact with a strain of virus that was probably closely related or identical to that which initiated the worldwide pandemic of 1918 and that the population has not since been exposed to any further influenza virus type A infection.

From the approximately 40 people over the age of 40 whose serums contained type A antibody, ten individuals were born in the decade preceding the epidemic and so would have been least likely to have experienced any earlier infection. The following analysis is based on results obtained in virus neutralization tests with serums from these ten people, in which residual antibody may therefore be said to reflect infection only by the influenza virus of the 1918 era, unmodified by either earlier or later influenza infection.

Serums were examined for neutralizing antibody to ten strains of influenza virus (certified research reagents) (*5*), namely, seven strains of human influenza virus encompassing the major antigenic varieties identified between 1933—the date of the original human isolate—and 1968, the virus of swine influenza, and viruses of the two major subtypes of equine influenza (*6*). Box titrations of the serums in virus neutralization tests revealed that the optimum combination of reproducibility and sensitivity occurred when eight tissue culture infective doses (TCID) of virus were used (Table 1), and it is at this unitage that antibody titers of the ten individuals to the ten virus strains tested are shown (Table 2). Titers of neutralizing antibody were highest to the A/PR/8/34 and A/BH/-/35 strains of virus. Lower titers occurred to the A/Swine/1976/31 and A/WS/-/33 strains, and no antibody was detected to the more recent subtype A, A1, or A2 human strains, or to either of the equine strains of influenza virus.

Our finding that the antigenic relationship of the 1918 influenza strain was closer to human type A strains than to the swine strain of influenza virus is not in agreement with conclusions based on serological surveys in cosmopolitan populations with frequent exposures to influenza. Such surveys have shown a peak titer of antibody to the PR/8 or WS strains of influenza virus in the age cohort which was in its childhood during the middle 1930's when these strains were prevalent, and a peak titer of antibody to swine influenza virus in the age cohort which was in its childhood during the era of the 1918 pandemic (*7, 8*). Thus, a close relationship of the 1918 strain to the virus of swine influenza has been inferred (*8*).

Complicating this interpretation, however, is the fact that, although antibody to swine influenza virus occurs very infrequently after a single infection by the PR/8 strain of influenza virus, antibody develops against both swine and human strains of influenza after repeated immunization with the PR/8 strain (*9*). These observations have been supported by evidence from antibody absorption experiments on childhood (single exposure) and adult (multiple exposure) human serums (*10*). They have suggested that antibody to swine influenza virus in cosmopolitan populations might alternatively result from the broadening effect of cumulative infections with human type A strains of virus (*10, 11*).

Our study is unique in that our analysis rests upon antibody patterns resulting solely from infection by the virus under scrutiny, and it thereby circumvents the entire issue of the extent to which additional infections may have modified the original antibody response. Our results indicate that the virus circulating in the 1918 pandemic era was

Table 2. Residual neutralizing antibody titers in serums of ten individuals whose only exposure to influenza occurred during the era of the 1918 pandemic. The numbers identify each individual. Statistically, $P > .05$ for SW versus WS and PR/8 versus BH; $P < .05$ for SW or WS versus PR/8 or BH, and for SW, WS, PR/8, and BH versus the other six strains (*13*).

Strain	Year	Persons with antibody titer:							Geometric mean titer
		< 10	10	20	40	80	160	320	
Subtype A									
Swine	1931		1, 2	3, 4, 5, 6	7, 8, 9	10			25
WS	1933	6	2	1, 4, 7, 8, 10	3, 5	9			15
PR/8	1934		4	1, 7		2, 3, 5, 6	10	8, 9	70
BH	1935			1	2, 3, 6, 8	4, 7	5, 9, 10		65
HICK	1940	all							< 10
CAM	1946	all							< 10
Subtype A_1									
AA/1	1957	all							< 10
Subtype A_2									
TW/1	1964	all							< 10
Equine A									
1	1956	all							< 10
2	1963	all							< 10

more closely related to human type A strains circulating in the middle 1930's than to other known influenza virus strains, including the virus of swine influenza.

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13. Statistical analysis was performed by Dr. P. Shaughnessy, who used several parametric and nonparametric tests which showed uniform agreement on the following results: $P > .05$ for SW versus WS and PR/8 versus BH; $P < .05$ for SW or WS versus PR/8 or BH, and for SW, WS, PR/8, and BH versus the other six virus strains.
14. We thank C. Shaw and Miss D. Stein for technical assistance.

2 June 1969

Reef Coral from Aldabra: New Mode of Reproduction

Abstract. *A unique mode of asexual reproduction in recently collected specimens of* Goniopora *(Scleractinia) is reported. Skeleton is absent from new polyps; the skelton develops independently of the parent colony as the new polyps themselves increase. The young colonies eventually become detached. The cycle seems to be a response to a sandy habitat, a conclusion reached by analogy with* Fungia *and* Manicina.

The shallow waters of the atoll of Aldabra are generally poor in coral growth (*1*). Areas of densest growth are most often found just inside the lagoon, in the region of the passages through the atoll rim. Current action is generally stronger here, and the better circulation is presumably an important factor favoring coral growth. In one area just inside the lagoon at Passe Gionnet, consisting of scattered coral patches and open sand, there is a patch of coral measuring approximately 20 by 5 m; the patch is composed almost entirely of the coral *Goniopora stokesi* Edwards and Haime (a new species record for Aldabra and the region). This coral occurs as small hemispherical colonies, between 2 and 8 cm in size, which lie unattached on a soft substrate consisting of a clean, white, detrital calcareous sand.

Where it grows *Goniopora* covers about 80 percent of the substrate surface. Small growths of the marine phanerogam *Halophila* and the algae *Caulerpa* and *Halimeda* also occur. The coral patch extends up to the prop roots of a mangrove fringe surrounding a lagoon islet, and for much of the day the corals are shaded by mangrove trees. At low spring tides the area is covered by about 1 m of water (tidal range approximately 2.5 m).

The *Goniopora* polyps are usually expanded during the day (at low water), a habit which is unusual in corals (*2*), but which has been recorded (*2*, *3*) in other species of this genus in other regions. The columns of the polyps may extend by as much as 5 cm from the corallum and are brown-mauve in color. Another feature is that in the retracted state, the polyps are still not withdrawn entirely into the skeletal calice; the columns project about 2 to 3 mm. Even the action of a preservative fails to increase retraction (Fig. 1), and this condition must, therefore, be taken to be normal for these specimens.

By far the most unusual feature, however, is that of small, closely attached, spherical masses of polyps (referred to here as polyp-balls) borne by the principal colonies. In one of our preserved specimens (Fig. 1) measuring 6 cm in diameter, about 25 percent of the surface area is obscured by some 40 polyp-balls. These polyp-balls are of all sizes between 3 mm and 2 cm, and bear anything from 1 to about 30 polyps. Each possesses its own small spherical calcareous corallum (skeleton) (Fig. 2), but surprisingly, there is no rigid connecting tissue between the principal corallum and the polyp-ball coralla; they are held in place by soft tissue alone. Each polyp-ball is derived from an individual polyp. No feature of this kind in corals has ever been described before, or otherwise made known to us (*4*).

Examination of two sets of dried specimens (originally two colonies bearing polyp-balls) and seven preserved specimens (several with polyp-balls), together with the facts of their occurrence, leads to the conclusion that the polyp-balls are part of the life cycle of the species. The attached nature of what are evidently young colonies suggests that they represent a form or phase of asexual reproduction. By an unusual mode of budding, young colonies could develop and grow in the attached polyp-ball state until they are about 2 cm in diameter, after which they detach. They then gradually assume a hemispherical shape with an epithecal base. Development of epitheca results from the partial withdrawal of fleshy tissues which previously surrounded the entire polyp-ball skeleton. Epitheca also serves as a useful means of dis-

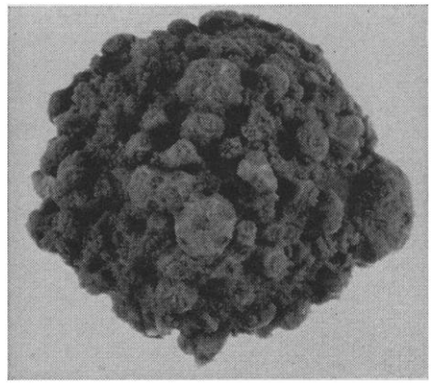

Fig. 1. Preserved colony of *Goniopora stokesi* showing the protuberances of the polyp-balls (× 0.75).