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9. Briefly, the ethanol diet was prepared by mixing the proper proportion of a stock ethanol solution (63.3 percent by volume) with Nutrament (Mead Johnson) to result in a diet with 35 to 39 percent ethanol-derived calories. The sucrose control diet was prepared similarly, except that an isocaloric stock solution (87 percent weight to volume) was substituted for the stock ethanol solution. Both diets were additionally fortified with Vitamin Diet Fortification Mixture (0.3 g/dl) and Salt Mixture XIV (0.5 g/dl) (both from ICN Nutritional Biochemicals). Both the ethanol- and sucrose-containing liquid diets contained 1.3 kcal/ml.
10. The daily consumption of ethanol averaged over the 5-month period of exposure [mean ± standard error (S.E.)] for group E was 13.2 ± .33 g per kilogram of body weight. This is comparable to the level of ethanol consumption that results in residual behavioral deficits in rats (*7*). There were no statistically significant differences in body weight among the three groups at any point during the experiment. The body weights (mean ± S.E.) at the beginning of liquid diet administration were 245.0 ± 4.9 g for group E, 242.0 ± 4.1 g for group S, and 242.0 ± 5.6 g for group LC. The body weights at the end of the 5-month experimental diet period were 523.0 ± 22.5 g for group E, 517 ± 16.8 g for group S, and 497.0 ± 10.1 g for group LC.
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14. The number of granule cells per square millimeter was determined by calculating the average number of granule cells contained within a grid area of 0.0025 mm² at a magnification of ×1500. Six samples of granule cell density were taken in each section, three samples from each of the dorsal and ventral blades of the dentate gyrus granule cell layer. The total area (square millimeters) of the granule cell layer was determined by projecting each section onto paper by use of a projecting microscope, tracing the boundary of the granule cell layer and measuring the area with a compensating polar planimeter (Keuffel and Esser).
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Do Corals Lie About Their Age? Some Demographic Consequences of Partial Mortality, Fission, and Fusion

Abstract. *Population dynamics of corals and other colonial animals are complicated by their modular construction and growth. Partial colony mortality, colony fission, and colony fusion distort any simple relationship between size and age among reef corals.*

Life expectancies of scleractinian corals and other colonial animals are poorly known, largely because their modular mode of growth presents difficulties not evident in the demography of more conventional, solitary organisms (*1–5*). The few longevity estimates available for corals are primarily based on extrapolation from data on short-term growth and the untested assumption that colony size is proportional to colony age (*5*, *6*). In this report, we show how relationships of coral size to age can be severely distorted by three modular processes (partial colony mortality, colony fission, and colony fusion) and point out some of the more important consequences of these phenomena to population studies of reef corals.

The numbers and sizes of five species of foliaceous scleractinian corals were recorded for 1 year at Rio Bueno, Jamaica. More than half of the 662 colonies censused were *Agaricia agaricites*. The

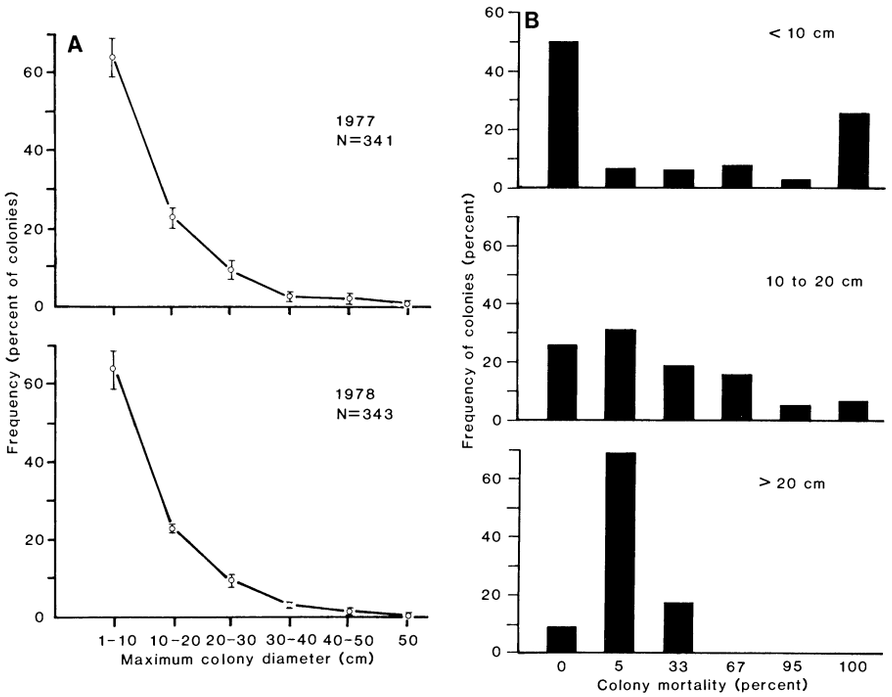

Fig. 1. (A) Size-frequency distributions of colonies of *Agaricia agaricites* on a vertical reef face just west of Rio Bueno Harbour, Jamaica, West Indies. Six quadrats (1 m²) were tied to the reef at −10 m, −15 m, and −20 m. Nails were fixed adjacent to corals for spatial reference. All corals larger than 1 cm in diameter were photographed and measured in situ in July 1977 and again in July 1978. Changes in living coral dimensions over the year were determined to an accuracy of about 1 cm. Error bars indicate 1 standard deviation from the mean frequency for corals from the three depths combined. (B) Frequency distribution of the percentage of colony mortality over 1 year for three size classes of *Agaricia agaricites*. Coral sizes are maximum colony diameters. The sample size for corals < 10 cm is 216, for corals 10 to 20 cm it is 78, and for corals > 20 cm it is 47.

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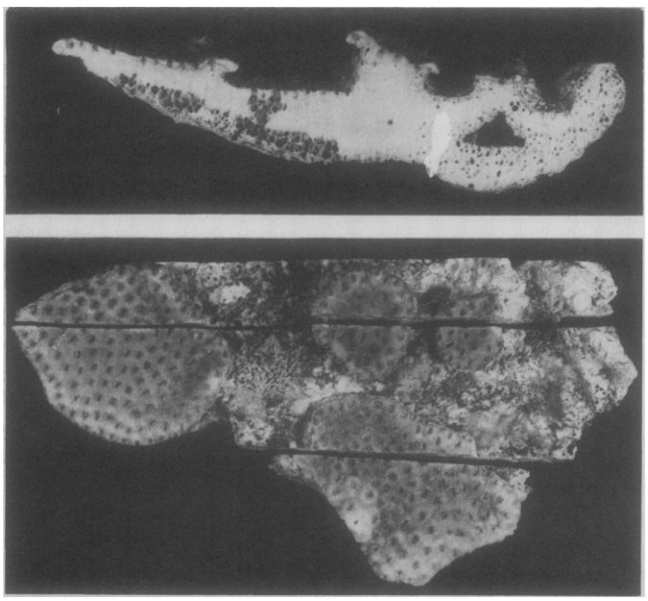

Fig. 2. Surface and cross-sectional views of three colonies of *Montastrea annalaris* showing their origin from a single parent colony. The specimen length along the saw cut is 18 cm.

platelike growth form of this and other common species at Rio Bueno rendered them amenable to photographic analysis of processes affecting colony area and size. The pooled size-frequency distributions for *A. agaricites* in 1977 and in 1978 are shown in Fig. 1A. Although the smallest corals are numerically important, accounting for more than half the total colony count, they constitute less than 5 percent of the total area of living coral. If colony diameter and colony age were directly related, then Fig. 1A would approximate a type 3 survivorship curve in which high juvenile mortality gives way to low mortality rates for older individuals (*1*). Following known corals in photographs for successive years, however, demonstrates that size and age are seldom directly related. This means that the stability apparent in Fig. 1A does not result from a simple balance between births and deaths in the manner characteristic of solitary animals.

Many of the colonies recorded in 1977 were reduced in size during the year by various physical and biological processes, especially sedimentation and ephemeral overgrowth by algae. Such partial mortality is apparent in time series photographs but difficult or impossible to detect in situ because coral skeleton bared by death of overlying tissue is readily overgrown by other sessile organisms or abraded by grazers. The frequencies of such injury for three size classes of *A. agaricites* are shown in Fig. 1B. Colony mortality ranges from zero (escape from injury) to 100 percent (whole colony mortality). Nearly 60 percent (209 of 341) of all *A. agaricites* colonies suffered partial or complete mortality in 1 year.

Patterns of mortality are strikingly related to colony size (Fig. 1B). Most small colonies died completely or escaped injury entirely, whereas most large corals suffered partial mortality.

Partial mortality can also result in formation of two or more similar colonies by fission of a previously existing large colony (*7*). Thirty-nine physically distinct colonies of *A. agaricites*, *Agaricia lamarcki*, and *Helioseris cucullata* were formed from 18 parent colonies during the year by such fission (2.2 colonies per square meter). Twenty-one of these new colonies were less than 5 cm in diameter. During the same year 62 newly settled coral colonies survived to 1 cm in diameter or larger (3.6 colonies per square meter). Thus fission produced small colonies at about one-third the rate of observed larval recruitment at this site.

Fission forms adjacent colonies which presumably are of identical genotype. Subsequent lateral growth may reunite these colonies and eventually fusion may occur. Eleven cases of intraspecific colony fusion involving 22 colonies of *A. agaricites* or *H. cucullata* were detected in the quadrats during the year. Thus, in a single year at Rio Bueno, 40 of 662 corals (6 percent) showed a dramatic change in size due to fission or fusion. Fusion was always perfect, involving both tissue and skeleton. Such fusion has been demonstrated experimentally only between coral fragments of the same original colony (*8*).

Fission and fusion can be observed in sectioned colonies by tracing patterns of growth, orientation, and spacing of calyces. The coral skeleton, then, may provide an accurate account of colony ontogeny which often cannot otherwise be documented in a practical (human) time scale (*6*). For example, the three neighboring colonies of *Montastrea annularis* shown in Fig. 2 obviously had a common origin. Despite their different size they are of identical genetic age. From their rates of vertical and lateral growth we estimate these colonies had been isolated for 3 to 5 years, and would not have grown together again for at least 10 years. At some earlier time the original parent colony was larger than all three of the present colonies combined.

Partial mortality, fission, and fusion occur in many organisms other than corals. Shrinkage or fission due to partial mortality also occurs in Porifera (*9*), Hydrozoa (*10*), Gorgonacea (*11*), Ectoprocta (*12*), and a wide variety of terrestrial plants (*2, 3*). Similarly, fusion between separated parts of the same colony has been observed in Porifera (*13*), Hydrozoa (*14*), other Anthozoa (*15*), Ectoprocta (*16*), Ascidiacea (*17*), and terrestrial plants (*2, 3*). Such modular processes have an ancient pedigree. Growth patterns resulting from partial mortality, fission, and fusion are clearly evident in skeletal sections of Ordovician and Silurian Ectoprocta (*18*), Stromatoporoidea (*19*), and Tabulata (*20*).

Our results show that partial mortality, fission, and fusion occur often enough to profoundly distort any simple relationship between size and age among reef corals. The remarkable stability of the coral population during the year at Rio Bueno masks the dynamics of underlying processes affecting colony growth and shrinkage. Similar size-frequency distributions of colonial animal populations can be derived in many different ways. A colony of *Agaricia agaricites* 10 cm in diameter which by chance had never suffered partial mortality might be only 5 years old. Alternatively, the same colony might be a remnant of a once much larger colony that settled hundreds of years before. Partial mortality and fission which reduce colony size occur much more frequently than fusion which tends to offset colony shrinkage. We conclude that estimates of coral age based on colony size and growth data are probably far too low, especially for the largest corals.

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Environmental Influences on Body Weight and Behavior in Developing Rats After Neonatal 6-Hydroxydopamine

Abstract. *There is less hyperactive motor activity and better avoidance performance in rat pups treated with 6-hydroxydopamine as neonates and reared with vehicle-treated littermates than in pups reared in litters composed solely of other 6-hydroxydopamine-treated animals. Thus, in this experimental model of hyperactivity, an environmental manipulation provides an alternative to pharmacologic agents in reducing activity and improving learning performance.*

In recent years, evidence from several lines of investigation has indicated that both genetic endowment and environmental determinants contribute to the behavioral repertoire of the developing mammalian central nervous system. Although such environmental alterations as modification in litter size have been shown to produce enduring effects on behavior (*1*), the consequences of other aspects of litter composition on subsequent development have received less attention. One such aspect has been the behavioral effects of rearing developing mice with foster mothers of a different species or strain. Denenberg *et al.* (*2*) noted reductions in open-field activity and aggression in mice reared by rat foster mothers. McCarthy and Southwick (*3*) showed that reciprocal cross-fostering of mice of an aggressive strain with a more passive strain decreased the aggressive behavior of the more aggressive pups but had no effect on the more passive strain.

Administration of 6-hydroxydopamine (6-OHDA) to the neonatal rat pup results in hyperactive motor activity that abates with maturity and cognitive deficits that persist into adult life (*4*, *5*). In recent studies we were able to attenuate such hyperactivity by placing the pups with their anesthetized mother (*6*). The subsequent reduction in activity was even more pronounced than that observed after administration of amphetamine or methylphenidate. Furthermore, Randall and Campbell (*7*) noted that hyperactivity in the developing rat pup may be attenuated by the presence of an anesthetized lactating female, and Smith and Spear (*8*) showed that home environmental cues, such as litter shavings, are critical determinants of early learning and retention. These findings suggest a complex interaction between biological factors and environmental influences during early development.

In the present experiment, we examined the effects of varying the litter composition on activity and cognitive performance in normal and 6-OHDA-treated rat pups. Sprague-Dawley rat pups (Charles River) were cross-fostered at 2 days of age and maintained with the same mother in litters of nine to ten animals throughout the 33-day experiment. They were divided into four distinct groups: (i) vehicle homogeneous (V-Hom), pups administered saline and reared with similarly treated pups; (ii) treated homogeneous (T-Hom), pups treated with 6-OHDA at 5 days of age and reared with similarly treated pups; (iii) vehicle heterogeneous (V-Het), pups given vehicle and reared in litters in which 50 percent of the pups received vehicle and 50 percent 6-OHDA; and (iv) treated heterogeneous (T-Het), pups treated with 6-OHDA and reared in litters in which, again, 50 percent of the pups received vehicle and 50 percent 6-OHDA. There were 12 litters in all (three litters for each of the four groups), with 22 to 26 animals in each experimental group. Brain dopamine was selectively depleted at 5 days of age by administering desmethylimipramine (20 mg/kg, intraperitoneally) followed 1 hour later by intracisternal 6-OHDA (100 μg in 20 μl of saline, calculated as the free base; Regis Chemical).

Activity was recorded in a soundproof room when the pups were 12, 15, 19, 23, 27, and 30 days of age. Each pup was randomly assigned to one of ten plastic cages placed on the floor of the room, and recording was begun immediately with a television camera coupled to a time-lapse tape recorder. Activity was scored by playing the tape back at a speed equivalent to six times that of real time, and was determined for alternate 5-minute periods throughout a 60-minute observation period. Activity was defined as any detectable movement. Duration was determined by activating an electric timer at the onset of any movement and stopping it when the movement ceased. The cumulative duration of movements for each 5-minute interval was thus obtained, and the percentage of time in which the pup was active during each 60-minute observation period was then calculated and recorded. Avoidance performance was determined in a shuttle box at 27 days (*9*). All animals were killed at 33 days, and their brains were analyzed for dopamine and norepinephrine by high-pressure liquid chromatography (*10*). Activity and avoidance learning data were then subjected to analyses of variance, with age and trial blocks representing within-group factors and 6-OHDA (or vehicle) and litter composition representing between-group factors.

Figure 1 shows the effect of 6-OHDA

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