CHAPTER 10

POPULATION DECLINE

The phenomenon of population decline in *Achatina fulica* was first reported upon by Green (1910*c*, 1911*b*) in Ceylon. At the present stage of our investigations, the following words of Green seem to be almost prognosticative: "A newly introduced pest usually increases out of all due proportion during the first few years of its existence in a new country, after which it gradually reverts to more normal conditions. There are already indications that this process is taking place locally. The particular village [Halawegoda] in which the pest is said to have originated, is at the present time not nearly so thickly infested as are some of the neighbouring villages to which the pest has since spread. A similar levelling process has apparently occurred in other countries into which this snail has been introduced." In spite of the early date in which these ideas were advanced, and the clarity with which they were presented, the literature has been essentially devoid of any reference to the phenomenon of population decline until the past few years. In discussing the introduction of the giant toad *Bufo marinus,* Townes (1946) significantly pointed out that the populations of this predator dwindled after an initial build-up and that the average size of the animal diminished. Williams (1951) learned in Zanzibar that the several species of *Achatina* on that island years ago caused damage to crops but that now the farmers pay little heed to them.

It was the words of Green that persuaded the author in 1949 to write to a number of investigators to inquire about the status quo of the problem in the areas where *A. fulica* had been established the longest. Although there were differences of opinion expressed in the

response, even from different people in the same area, by far the majority indicated that there has been noticed in recent years a definite reduction in the number of the snails and, therefore, in the problem itself. Several people, apparently independently, observed that the decline was noticeable in the older populations, but that the newer populations were just as vigorous and damaging as the older populations had been in their early stages. A few reported conditions suggesting an essential decimation of the snail population apparently through the effect of some grossly adverse factor. Most of these reports came from correspondents in Ceylon. In New Britain, however, G. S. Dun (*in litt.* Nov. 5, 1951) stated that in Kavieng the giant snails used to be very abundant, that recently they have decreased so in numbers that vegetables can once again be grown, and that "high piles of moribund snails" have been encountered.

Two main facts, then, came to the fore in these reports, viz., that population decline in *A. fulica* is actually in process at least in some areas, and that an unfavorable factor or combination of factors is responsible for a catastrophic effect upon certain populations. It was eminently apparent that a thorough investigation of the phenomenon of population decline was needed. Because of the preponderance of positive reports from Ceylon, because the infestation in that country was over fifty years old, and because the majority of known predators of this snail was in that country, a proposal was made to set up in that area an extensive research project. This project was conducted by the author under a grant (NSF-G519) from the National Science Foundation, Washington, D.C., for a period of nine months in 1954.

A preliminary survey in Ceylon quickly verified the earlier reports of population decline. In fact, populations were found in surprisingly great variety. Some, such as those in Dambana and Balangoda, were in the early fulminating stage. Others had been in progress for thirty to fifty years and were either stabilized, declining, or apparently extinct. When all information was pieced together, a generalized picture of the population "life cycle" took shape. After firm, unobtrusive, insidious establishment, the giant snail appears almost explosively in great numbers. This is revealed by the fact that first announcements of newly discovered populations are usually made in an urgent, almost frantic manner with descriptions depicting snails literally all over the place. The numbers continue to build up despite control measures in various combinations. The population then seems to stabilize at a high level for an indefinite period of time. At this time, poisoning campaigns in some cases paradoxically seem to favor further build-up of the snail. Ultimately a gradual population decline takes

place. Although there are subsequent periods of recrudescence, after a certain stage the population rarely or perhaps never attains the level of the first peak. With this progressive decline in numbers, there is a concomitant decline in the average size of the individual snails so that instead of a population of true "giant" forms from 4–6 inches, they may be quite modest-appearing forms, ranging from 3–4½ inches. In some cases, however, the population may go into a sudden, unpredictable, and pronounced slump with the result that live specimens can be found only after prolonged hunting, or not at all. More than a simple decline is obviously involved in these latter cases. Somehow the decline has been augmented and intensified until extinction or near-extinction has occurred. Herein lies the very heart of the problem of population control.

The problem of attempting to determine the causal factors in the phenomenon of decline is not going to be an easy one; for it is an ecological problem, and ecological problems are notoriously complex. Several different areas of investigation have been suggested as an approach to the problem. These are treated under separate headings below.

Population Senility Some investigators have attributed decline in the populations of the giant snail to a mythical "population senility." In so doing, they merely beg the question; for in effect they have done nothing more than give an ambiguous term to the phenomenon (Mead 1955*b*). To assume that all populations will eventually go into a state of senility and reduced vigor does not get to the matter of causation.

Sterility On some occasions in the past, investigators have encountered situations wherein, even with diligent hunting, not a single mature snail specimen could be found in the gravid state. Both Kondo and the author have examined snail populations in the Pacific reflecting this characteristic (e.g., the Auluptagel population in the Palau Islands). In attempting to find an explanation for this, some have assumed that a physiological process of sterility had entered the picture and was effecting a population decline through reduced fecundity. The apparently favorable nature of the environmental conditions, however, may have been misleading; for unsuspected factors may have been responsible for precipitating a perfectly normal, temporary period of generalized reproductive inactivity.

There probably is a decline in the reproductive capacity of older individuals in any population; but this, by itself, cannot explain population decline. In the gigantic individuals, which may not necessarily be gerontic, there is fair evidence of sterility. A correlation be-

tween gigantism and sterility in mollusks has been emphasized by Pfeffer (1928). This same correlation has been offered to explain the fact that gigantic forms of *A. fulica* not infrequently appear in the early sigmoid growth stage of the population, but quickly disappear, never to be seen again. It perhaps is therefore relevant that Kondo (1952) found gigantic individuals most prominently at the "forefront" of the Laguna population in Guam.

There has been found in neither the field nor the laboratory any evidence of sterility through metazoan parasitism (cf. Szidat 1941).

Starvation Chamberlin (1952*a*) concluded in his ecological study of *A. fulica* on Tinian that starvation was the main cause for the great number of dead shells found on that island. Although this same idea previously had been less positively expressed (Mead 1950*b*, *c*), it would seem now, in view of the catholic appetite of this snail and its faculty for withstanding for a long time complete lack of food, that starvation could not possibly explain population decline except in limited cases under most unusual circumstances. The same reasoning would apply to the matter of nutritional deficiences, including the very important calcium. As a contributory factor, starvation could have its effect; but alone, it would not be generally decisive.

Exposure Practically without exception, the investigators who have made and reported upon a survey of the problem of the giant African snail have emphasized the apparently high incidence of death by exposure to direct sunlight. This point has been elaborated upon above. Under the prevailing conditions in most infested areas, it is not understandable how this factor could have anything more than an incidental effect except in cases of severe overcrowding in marginal areas. And even in the latter cases, the factor would be self-limiting. The fact remains, however, that there appears to be an inordinate amount of death by this means in some areas. As a possible explanation, an unknown second factor may be contributing to their general unthriftiness causing them to make a less vigorous, and therefore less effective, escape from adverse conditions.

Traumatic Breaks Snails inhabiting rough, rocky terrain are found in a high percentage of the cases to have shells which have been broken and mended and broken and remended. The unsightly appearance of these snails has encouraged some observers to conclude that they were unthrifty and hence were giving way to a population decline. Later, it was suggested that some "other factor," such as a genetic, a pathological, or a nutritional factor, was present to produce the thin, brittle shells which in turn were more prone to fatal or traumatic breaks. But whatever the explanation, the manifest ability

to withstand even severe breaks and the high reproductive potential in this species surely prevent traumatic breaks from producing a decline effect upon the population. In fact, some of the most vigorous populations examined in the Pacific islands were of this fragile, broken-shell type.

Predators Because the predators are the most tangible of the unfavorable factors in the environment of the giant snail, they have been seized upon by both the casual observer and the professional investigator as the most likely factors in effecting the observed decline. The Indian glowworm, for example, is believed by many to be largely if not entirely responsible for the fact that *A. fulica* is now not nearly the pest that it used to be in the areas where this predator is endemic. It is true that the glowworm is obviously more common in some infested endemic areas than it ever could have been without the giant snail. A distinction between cause and effect, however, has not been made. Nonetheless, this predator and other predators believed to be capable of holding *A. fulica* into at least a simulated state of population decline have been used, and will continue to be used, as potential agents of biological control in non-endemic areas. That they will accomplish their mission remains today a moot question indeed. But irrespective of this point, there is still the fact that population decline is manifesting itself in areas where predators are either unknown or nonexistent. Undoubtedly the majority of investigators feel that predators could have an appreciable augmenting effect in the phenomenon of decline, but that predators, by themselves, are not responsible for the current widespread decline in the populations of the giant snail.

Genetics The subject of genetics and its possible relation to the unthrifty nature of some giant snail populations, and therefore to population decline, was first introduced by Mead and Kondo (1949). Subsequent reports by these authors presented further evidence from the field in support of a genetic rather than ecologic explanation for some of the observed phenomena and emphasized the need for a genetic study of this snail species. Variations in individuals, and in populations in particular, were explained on the basis of possible genetic origin. Some of these variations, such as the malformed specimens (roughly in a 1:3 ratio!) in the Army Hill population in Saipan, have been interpreted by some observers as indubitable signs of population degeneration and decline. If the explanation is a genetic one, as suggested above in the discussion of variations, then the variations would contribute to population decline only if they interfered with the normal development of the population. As pointed out

above, gigantic forms show signs of being reproductively sterile. A genetic linkage between gigantism and sterility would have a qualitatively modifying effect upon the development of the population. On the other hand, if the "climax type" in a normal-appearing population possessed a reproductive inadequacy of genetic origin, there would follow a quantitatively modifying effect upon the subsequent development of the population. A population decline would be inevitable and proportional to the severity of the inadequacy. The inferences are tempting in the extreme; for all observed phenomena in the field can be explained plausibly through the medium of established genetic principles. Unfortunately the entire subject, as it pertains to *A. fulica,* has proceeded scarcely beyond the purely speculative stage.

Disease Annandale (1919) in India was the first investigator to report the belief that *A. fulica* was "subject to some kind of fatal epidemic"; but he did not suggest that there was any connection between it and a general population decline. South (1926*b*) accepted this report and listed the "fatal epidemic" as one of the controlling factors in the biology of the giant snail.

Early in 1947, Daniel B. Langford, assistant entomologist of the Trust Territory of the Pacific Islands, reported that many achatina populations in the Pacific area seemed to be changing character; that many of the individuals had thinner, more fragile, often badly distorted, lighter-colored shells; and that reproduction and numbers of individuals were noticeably reduced. This syndrome suggested to him that a disease was present. "Diseased" specimens from Koror, Palau Islands, were sent to investigators at the University of Hawaii in the hopes that disease-producing agents would be found. The findings were inconclusive. According to Langford, "diseased" specimens were then introduced in the achatina populations in Oahu. When these populations were examined by the author in January, 1955, there was no tangible evidence of the described syndrome in any of the specimens.

In January, 1949, Langford introduced from Koror "diseased" specimens in the Anigua population, near Agaña, Guam. Kondo (1950*c*) examined this population and determined that the typical syndrome was present. The shells of specimens that had ben dead for at least several months were compared with the specimens showing the syndrome. There were striking differences. Kondo pointed out that one might be tempted to conclude that the "disease" had been effectively introduced; but he emphasized that there was just as convincing evidence that two genetically different types had interbred.

It should be pointed out, however, that elsewhere in the Pacific islands there were noted just as pronounced differences between the ancestral shells and the living specimens. This suggests that perhaps the so-called "diseased" specimens are nothing more than representatives of a stage in the genetic evolution of the population. If this reasoning is correct, and if the populations are genetically similar, then probably the Anigua population would have appeared just the same had Langford not introduced the Koror specimens. This also would make more understandable the failure of the syndrome to make its appearance in the Oahu populations.

The reports of Langford created the first wave of optimism in the problem of the giant African snail. But this optimism was short-lived. In addition to the failure of investigators to find any pathogens in Hawaii, Lange (1947, 1950) early reported in his survey of the problem that he saw no evidence of an epizootic. He sent preserved specimens to the author for examination; but the gross anatomy showed no signs of pathology (Mead 1950*a*:237). An examination of fifteen of Langford's original "diseased" specimens from Koror revealed the fact that, in spite of their nine months in Guam in open bell jars without food or water, all but one was very much alive and apparently none the worse for the ordeal (Mead 1950*b, c*). During the summer of 1949, many specimens from a number of different populations were examined in the Pacific Islands. Instead of pathology, genetics appeared to form a more logical basis for explaining the common syndrome (Mead and Kondo 1949). With the announcement of these findings, the hope of using a parasitic disease to control *A. fulica* was virtually abandoned and renewed efforts were made to explore further the possibilities of using predators as biological control agents.

That there actually exists a disease of *A. fulica* continued to remain very much a possibility. The reports of Annandale and South, and the frequent reports from Ceylon reasserted that there was still more to the story. An exhaustive check of the literature brought to light the fact that practically nothing is known about gastropod pathology. The works of Szabó and Szabó (1930 *et seq.*), despite their ambitious titles, concern almost entirely anatomical anomalies and gerontic changes. Frömming (1954*a*) makes the same type of contribution. A presumed "contagious disease" of *H. aspersa* is reported upon at length by Boycott and Oldham (1938); but although their discussion is convincing, it is not conclusive. Aside from the fact that no microbiological investigation was made, the factors of genetics and nutrition were not adequately controlled. Muma (1954) states that the tree snail *Drymaeus dormani* in Florida is subject to a dis-

eased condition of possible bacterial origin which causes the snails to turn a greenish color and die. Further work on this problem is currently in progress. Dr. C. Vago of the Station de Recherches Séricicoles in Alès, France, writes (*in litt.* Mar. 23, 1955) that in the past few years he has studied over ten different diseases of several species of snails. His research so far has convinced him that a paracoli occasionally goes into a highly virulent form and produces an epizootic among the commercially raised helicines. Recently, one such epizootic killed over 50,000 snails in "snail parks" where the snails are raised in close confinement.

It was obvious at this point that investigating the possibility of disease being a causal agent in the decline of the giant snail population in Ceylon would have to be essentially a pioneering work.

First of all, the extent and effects of predation had to be determined. This factor was announced early in the investigation as not being decisive (Mead 1955*b*). By far the most effective predator was the Indian glowworm. But this attacked only the smaller specimens of *A. fulica.* It would be expected that any snails that escaped the ravages of the glowworm during the first two months of their lives would be too large to be attacked and, in the absence of other killing factors, would eventually become gerontic individuals in a population manifesting heterogeneity with respect to the size and age range of its individuals. Yet in some areas where this predator was commonly encountered, the giant snail population was in a truly remarkable state of homogeneity in that all individuals were clearly under one and one-half years of age. In others, the snails were not over two to three years of age. Older and larger individuals, which would be immune to the attacks of the glowworm, were completely absent, although their old, worn, dead shells were all too common. It was clear, then, that something more than predation was operative. An epizootic of decimating proportions, for example, or a chronic disease that is aggravated with the increased age of the host, could definitely produce the observed homogeneity in the population. Fitting into this suggestion is the fact that in these same areas in Ceylon there was a serious build-up of the snails in 1951 and such a sharp decline in 1952 that control measures were not undertaken. These populations then had had one and one-half to two years to recover from the decline—a period of time approximately equivalent to the age of the average individual. As a further pertinent point, it should be borne in mind that micro-organisms of disease, unlike predators, have the reproductive capacity to produce quickly a catastrophic effect upon a population (Mead 1955*b*).

With epizootiological evidence building up in favor of the disease hypothesis, a great many specimens were brought into the laboratory to be dissected in the living state for a critical anatomical examination. The results were discouraging, for the viscera and the tissues in general appeared within the range of normal in practically every case. A break finally came with the advent of the summer rainy season. At that time, snails were seen in great numbers during the day crawling about in the rain. As they were being observed in the field, it was noticed that one out of every two to three specimens had leukodermic lesions on the tentacles and, occasionally, on the face and neck. Surely these were symptoms of disease. The gross anatomy of these specimens was examined in the laboratory, but the findings were negative as before. This suggested on the face of it that there was no visceral phase of the disease. At this stage of the investigation, the syndrome was described at the meetings of the Second Pan-Indian Ocean Science Congress in Perth, Australia, in August, 1954. Subsequent histopathological examinations of the hepatopancreas and the kidney, however, revealed pronounced differences between specimens manifesting symptoms and specimens without discernible symptoms. Corresponding changes in other elements of the viscera were suspected.

As more and more specimens were examined, a definite sequence of progressive stages in the disease began to take shape. Leukodermia is caused by the systematic destruction of melanophores in a small locus in the dermis. The melanophores first appear fractured, then reduced to a mass of granules, and finally disappear entirely. Because of concomitant tissue destruction and weakening in the dermis, the lesion may become elevated into ridges, tuberculations, or horns; and as the lesion enlarges, it may coalesce with adjacent lesions. Eventually the tentacle becomes shortened and distorted and, finally, it may remain partially or completely invaginated. Occasionally the initial lesions may diminish in size, but other lesions appear elsewhere on the forepart of the body. It should be remembered that the severity of the "liebespiel" is responsible for leaving pigmentless scars on the surface of the body. Although they are clearly distinguishable from leukodermic lesions, the lesions might easily be confused by the casual observer.

As announced in the preliminary report of this disease (Mead 1956*a*), the whole epizootiologic picture is that of a chronic enzootic disease of uncertain etiology, high incidence (35–68 per cent), and low gastropod host specificity. Observations in the field and laboratory suggest that the disease is spread through contact, that it is highly contagious, and that the immunity is low. A great many natural factors in

the environment favor the development of an epizootic. There characteristically is a high moisture factor during the periods when the snails are active; the traumatic breaks in the shells would allow additional portals of entry for disease agents; the feeding habits of the snails include eating the substrate, consuming dying or dead individuals, and mutual rasping of slime from the surface of the body; and their habit of aggregating in moist, protected niches increases considerably the element of physical contact.

In the areas in Hawaii where *A. fulica* abounds, certain terrestrial arthropods, for example, the amphipod *Orchestia platensis,* the isopods *Porcellio laevis* and *Metoponorthus pruinosus* and unidentified collembolans, frequently have been seen at night crawling over the extended achatina and apparently feeding on mucus on the surface of the body. Their presence implicates them as possible agents of contamination if not actual vectors. A transovarian mode of transmission, however, still remains a strong possibility.

The various stress factors in the environment surely must provide an aggravating influence, perhaps even to the extent of precipitating an acute phase of the disease. Specimens kept for six months under conditions demanding estivation showed a much slower rate of progress of the disease; and, although there were new lesions, in some instances the old lesions appeared to be undergoing tissue repair with irregular areas of excessive melanosis. Apropos of this, Ghose (1959) has reported that tentacular regeneration in this species proceeds much slower during estivation. The course of the symptoms moved noticeably faster in specimens kept under conditions of high humidity and abundant food, which encouraged prolonged periods of activity. The progress of the disease is clearly linked with metabolic rate. From this point, the complications, implications, alternatives, and speculative possibilities literally run out in all directions. Obviously, a great deal of pioneering research is required to produce any sort of conceptual perspective in this complex problem. As a good start in this direction, a United States Public Health grant (E-1245 [C3]) has been made in support of a projected investigation of the etiology and pathology of this disease. The etiological research completed in Ceylon has essentially eliminated from further consideration protozoans, malacogenous fungi, yeasts, and spirochetes. Hence, attention is being focused on the bacteria and viruses.

Similar leukodermic lesions were found in specimens of *A. fulica* in Singapore, Hong Kong, and Hawaii (cf. van Zwaluwenburg 1955). Because of the high rate of infectivity in the Ceylon populations, and because the snail infestations to the east of Ceylon undoubtedly were

started by specimens from Ceylon, it has been provisionally assumed that the disease has been carried with the snails and that similar symptoms mean the same etiology. There is evidence, however, suggesting that different "strains" of the pathogen exist. For example, the infected specimens in Hawaii show a conspicuous paucity of tuberculations and an inordinately high incidence of large lesions, 3–6 mm. in diameter, located at the base of the ocular tentacles (67 per cent of positive individuals). Even in Ceylon, groups of infected specimens from different populations displayed noticeably different tendencies in their symptoms; in some, facial lesions were common, in others, they were almost completely absent; in still others, the lesions were conspicuously absent from the ventral tentacles. An explanation for these differences conceivably cannot rest entirely with the pathogen; for there must be differences in host response stemming from the different genetic make-up of the various populations. The difficulty in distinguishing between possible strain difference and difference in host response was most dramatically brought out in an experiment wherein the pathogen was transferred from *A. fulica* to *Limax flavus* to *Helix aspersa*. Out of 106 inoculated specimens of *H. aspersa*, 14.15 per cent were unmistakably positive; but the heretofore unobserved symptoms in *H. aspersa* were vastly different from those of *A. fulica* inasmuch as the leukodermia was generalized rather than localized and the tentacles were atrophied rather than distorted.

From what was known of the distribution of the disease, it was assumed that it would probably be found in all of the infestations east of Ceylon and India (Mead 1956*a*). More recent support for this assumption was obtained when specimens of *A. fulica* were collected in Bangkok, Thailand, in December, 1957, and found to be diseased (Mead 1958*a*, *b*). In contrast to the syndrome in the Hawaiian specimens, however, leukodermia was much less pronounced and tuberculation was severe. At that time, the only inkling we had regarding the possible existence of the disease in the Pacific island specimens was a casual remark of Chamberlin (1952*b*:12) tucked away in a footnote, viz., "Occasional individuals had pigmentless blotches on the skin but examination of these showed no coincident anomalies." As good fortune would have it, the return trip from Thailand was routed through Guam and it was possible to examine a number of giant snail specimens in the vicinity of the military airport. These were found to have the disease syndrome more nearly like that of the Thailand specimens than the Hawaiian specimens. This information suggests strongly that

all other Pacific island populations will be found to have the disease in one form or another. It is of interest to note that A. J. Kohn, during the 1957 Yale Seychelles expedition, found typical symptoms on specimens collected on Hitadu Island of the Addu Atoll in the Maldive Islands. On the other hand, Ghose (1960) failed to find any specimens in India with discernible symptoms; but his field work seems to have been quite limited. It is not known whether the disease occurs in the East African achatinas; but it is felt that more than likely it does. Finding a strikingly similar syndrome in the introduced achatinid *Rumina decollata* in central Arizona (Mead 1959*b*) has precipitated the feeling that the disease of the giant African snail is not unique, but one of a whole class of diseases of varying pathogenicity found in many species and in many parts of the world. It is a strong temptation to extrapolate further to assume that it is these diseases which successfully hold most snail populations in check and produce a major effect in population fluctuations. Actually, it would be a paradox indeed if snails did not have their own diseases. It is easy to predict that a number of snail diseases will be found and reported upon in the next relatively few years. When the effects of these are known, infinitely more will be understood about establishing balance in invading populations.

After the etiological agent has been discovered, it will be important to determine its affinity for forming different strains of varying pathogenicity. An understanding of the pathology must be sought at the histopathological level; but as Pan (1958) has emphasized, this has as its prerequisite an understanding of the normal histology. Thanks to the meticulous and exhaustive work of Ghose (1960), we know now much about the gross anatomy, developmental anatomy, and histology of *A. fulica*. Similarly, an understanding of the epizootiology depends upon a rather considerable knowledge of the ecology, and particularly of the population dynamics, of this important species. We are less fortunate here; for although there have been recorded many observations made at the time the invading populations were at their fulminating stage, practically nothing is known about the normal course of development of a population. The field investigations of Mead and G. D. Butler, Jr., in Hawaii are contributing to our knowledge in this respect; but considerably more basic work is urgently needed.

So far, the epizootiological data recorded from examinations of the unusually heavy population of snails in the Mahinui area of Oahu since 1955 have given us our best, but by no means clear, conception

of the course and effects of the disease on a relatively recently established giant snail population. In January, 1955, the incidence of the disease was 14.5 per cent and not a single specimen could be classed as having "severe" symptoms. One hundred adult specimens collected at random in August, 1957, indicated that the incidence had increased to 57.0 per cent and specimens with advanced symptoms were common. Exactly one year later, the incidence had raised to 68.6 per cent and the majority of the positive specimens had prominent lesions. Throughout this period of time, the snail population level remained essentially stable. In December, 1957, 400 specimens collected at random were measured, weighed, examined to determine and record the presence and extent of disease symptoms, and released in the area where they were collected. In the following August, 132 (33 per cent) of these specimens were recovered and reexamined. The new data demonstrated that in the intervening months negative specimens had become positive and positive specimens had either become more severe or had disappeared. In essence, a far bigger population turnover had been taking place than was ever suspected; and the high reproductive potential had apparently insured the maintenance of the population load (Mead 1959*b*). This information is compatible with the earlier conclusion that the disease reduces life expectancy or, conversely, that age acts as a decisive stress factor in the presence of the disease.

The disease, however, has not just been spreading from one achatina to another; experimental evidence supports the assumption that natural transmission has taken place between *A. fulica* and *Bradybaena similaris* and *Subulina octona* in this area. These latter two species were completely negative for symptoms in other areas in Oahu which had not been invaded by *Achatina.*

Identical sampling techniques in the Mahinui population in September, 1959, revealed the following facts: the incidence had increased to an amazing 83.0 per cent; the vast majority of diseased specimens were of the "severe" type with one or more large (5 mm.+) leukodermic lesions; for the first time several specimens were found to have multiple lesions (12+) on the exposed parts of the body; and, also for the first time, there was a marked population decline. There is good evidence that the disease becomes an increasingly more effective limiting factor only after the snail population has begun to level off following the sigmoid growth stage. Research is being continued in the Mahinui area, but unfortunately the predatory snail *Euglandina* has recently invaded the experimental site as an incalculable variable.

Multiple Factors It is obvious that under natural conditions, unfavorable environmental factors are mutually augmentative but operate wholly independently. At any time in any one place, some may be producing the maximum unfavorable effect while others are temporarily ebbing or remaining neutral in their effect. But, as in any other system of independently operating variables, there are inevitable periods of synchronization. If a period of synchronization is at a time when the unfavorable factors have reached their maximum intensity, then the effect upon the population will be most severe. In some animal groups, this could mean local extermination, particularly when man-made unfavorable factors are added to the natural compliment.

Possible instances of this nature have been observed in Ceylon and in the Pacific islands. A case in point is the population of *A. fulica* at the Dodangoda post of the Godahene estate near Neboda, Ceylon. In 1951, the giant snail was so abundant that the leguminous cover crop *Pueraria* was almost completely denuded. A program of poisoning the snails with metaldehyde bait was immediately initiated. By 1952, the population of the snails had diminished to the point where further poisoning was not considered necessary. By 1953, the snails were scarcely in evidence and the local people literally "forgot about them" in that area. In 1954, the author made a lengthy and exhaustive search of the area during the rainy season and in the rain. Not a single live specimen could be found. Nor could any other snail be found alive, including the ubiquitous and common *Subulina octona*. Empty shells of *A. fulica* were there in abundance, but their weathered condition indicated in every case that death had occurred many months previous to that time. No predators were found; but a large number of weathered but characteristically broken shells near bandicoot burrows indicated that this mammalian predator had been active in the past. More recently, the bandicoots had turned to the large, rich seeds of the rubber plant for their food. With no live snail specimens present, positive evidence of the disease was not discoverable. However, it seemed more than significant that live snails in the nearby Pettigola section of this estate had the highest infectivity rate (64 per cent) that was found in Ceylon. Surely, then, the chief unfavorable factors were poisoning, predation, and disease. Possible unfavorable meteorological conditions could not be determined beyond the fact that rain had been unusually abundant during the preceding months.

There appears to have occurred in that area virtual or actual extinction not only of *A. fulica* and other introduced snails but of

the endemic snails as well. Cause for this cannot be laid wholly at the feet of the poisoning program as their program was of such a scale that it could not possibly have been eradicative. In the absence of any further information, one can scarcely escape the plausibility of the assumption that the disappearance of the snails was caused by the compounding of unfavorable factors. But, more than a simple summation of the unfavorable effects, there actually may be a synergistic action in certain combinations of factors. If there is a formula for producing an eradication of this snail pest, it probably will be found in the realm of multiple unfavorable environmental factors.