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Kammerer's Midwife

In his concern with re-delivering Kammerer's work, Koestler has assumed that if Kammerer's experimental integrity were accepted, his Lamarckian conclusions would be reasonable[1], a flaw unnoticed by a recent review[2]. However, known phenomena are sufficient to explain Kammerer's results. Most controversy followed work on the terrestrial midwife toad, eggs from which were immersed in water for several generations. Most eggs died but eventually some male descendants were produced with nuptial pads, characteristic of aquatic anurans. Kammerer suggested acquired inheritance, but intensive selection had occurred and a third explanation is that an environmentally-triggered gene switch had operated.

In Kammerer's other work he confuses adaptation at the individual level with evolutionary adaptation. For example, the European salamander changes its colour in response to background. The change is hormone-induced[3] and the salamanders are viviparous, so it is not surprising that at birth young salamanders have the same colouring as their mothers. The factor that is inherited is the ability to change colour; the birth colour is a maternal effect. Superficially the Lamarckian explanation is merely a rather unlikely alternative. Considered further it is even less likely, for there is no model to explain how an acquired character could be incorporated into the genome. If environmentally triggered cellular changes are to direct evolution then either the cell must be able to distinguish *a priori* which changes are beneficial and therefore to be incorporated, or numerous random changes occur with only a few beneficial changes eventually surviving. The former is magic; the latter is a theory uncommonly close in spirit and execution to that of random mutation and natural selection.

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Faecal Pollution of Our Beaches — How Serious is the Situation?

Smith[1] determined the incidence of antibiotic resistant coliform bacteria in various rivers, paying particular attention to *Escherichia coli* with transmissible resistance to chloramphenicol; this resistance is potentially dangerous because it may be transferable to *Salmonella typhi* and so render the treatment of typhoid fever more difficult. An important conclusion that emerged from his work was that most of the antibiotic resistant coliforms and R^+ *E. coli* in rivers examined came from urban sewage. These types were present even in sewage from areas with no abattoirs; human beings were thus shown to be the main source of antibiotic resistant types in the rivers. Smith[2] subsequently determined the incidence of antibiotic resistant coliforms and R^+ *E. coli* in coastal bathing waters and reported that the ratio of the concentrations of the different kinds of antibiotic resistant coliform organisms to each other and to the antibiotic sensitive organisms in the seawater resembled that in human sewage previously examined[1]. He concluded[2] that R^+ *E. coli* in seawater provided "stronger evidence of contamination with human sewage", and that "the present undesirable situation could be corrected immediately if proper disposal of sewage from seaside towns were practised".

We would like to look further at three aspects of the implications of this work. First, does the presence of R^+ *E. coli* and other antibiotic resistant coliforms in fact provide "stronger evidence of contamination with human sewage"—presumably as opposed to contamination by animal faeces *via* farm wastes and rural drainage? Second, is there evidence that the situation is undesirable from the public health point of view? Third, what constitutes proper disposal of sewage from seaside towns?

Antibiotic resistant coliforms including *E. coli* are present in animal faeces. Although contamination by animal faeces may be as aesthetically undesirable as contamination by human faeces, it is less dangerous because large numbers of faecal bacteria from an animal would have to be ingested to cause serious illness, while the ingestion of only a few human intestinal pathogenic bacteria can cause disease.

Recent work in America[3], where admittedly more animals receive antibiotics than in UK, showed that from 16 to 96% of *E. coli* strains isolated from animals on various farms showed multiple resistance. Smith[4] found R^+ *E. coli* in calves, pigs and fowls as well as in human beings, and Walton[5] isolated strains of *E. coli* showing multiple resistance from the faeces of healthy pigs and calves. Studies such as these suggest there is no *prima facie* evidence for assuming that there are more antibiotic resistant coliforms in human sewage than in rural (i.e. predominantly animal) sewage and surface run-off. Bearing this in mind we recalculated Smith's data[1] to obtain for each antibiotic the resistant coliforms expressed as a percentage of the total coliform count (Tables 1, 2) and concluded that the number of resistant coliforms isolated by Smith had depended more on the total number of coliforms (resistant + sensitive) present in each sample than on the source. Thus the results do not support the idea that counts of antibiotic resistant coliforms and of R^+ *E. coli* distinguish between contamination from urban sewage and contamination from rural sources; so more work is required.

Our recalculation, although showing that Smith's survey[2] provides no bacteriological evidence that contamination was from human beings, does not reduce his demonstration of faecal contamination, sometimes heavy and probably from human beings, of several beaches and so we have to consider if there is any scientific evidence that such contamination is dangerous in sea bathing water.

There is no generally accepted bacteriological standard for bathing waters in the UK, not because of apathy, but because a great deal of research by government scientists, local authority health officials and others, has indicated that a reliable standard cannot be formulated. There are two main reasons for this. First, many factors, especially roughness of sea and amount of sunlight, affect the numbers of coliform bacteria recovered on successive days; there may be ten-fold differences[6]. Second, there is no epidemiological evidence that disease is transmitted by sea bathing, except perhaps when the sea contains recognizable faeces. In other words[7], "The possibility of contracting an enteric disease as a result of bathing in polluted seawater cannot be entirely ruled out. On the other hand, when there are no aesthetic objections the probability of contracting a serious disease by bathing in sewage polluted water is so small as to be epidemiologically undemonstrable".

There remains the possibility that R^+ *E. coli* may become established in the gut as a result of bathing in sewage polluted water and so be available to transfer their resistance to pathogenic organisms. This possibility seems slight, because large doses are required for transfer to occur[8], and not particularly important, because many people already carry R^+ *E. coli*[9].