

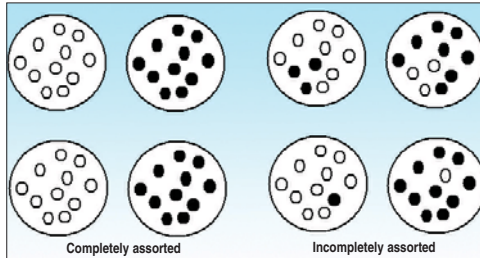
Being alike helps being good neighbours

Social structure has been seen to affect the advent of cooperative behaviour, says s ananthanarayanan

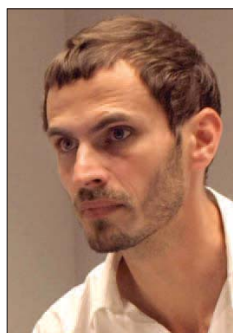
THE dynamics through which organisms, whose objective is survival, still opt for cooperative strategies that may involve short-term losses or uncertainties have been of interest in the fields of biology, economics, psychology and sociology. While some of the cooperative behaviour is genetically wired in through evolution — for instance, in ants or bees and wasps — in most cases it seems to arise from intelligent choices based on mathematical game theory principles. There is, in fact, a paradox here as the logical choice would appear to be selfish behaviour, but still conditions work to tilt the preference towards cooperation. One of the reasons is clearly that selfish behaviour would induce retaliation or even the avoidance of interactions. Matthijs van Veele, Julián García, David G Randa and Martin A Nowaka, at Harvard, Amsterdam and the Max Planck Institute of Evolutionary Biology in Plön, Germany, report in the *Proceedings of the National Academy of Sciences* (USA) that while repeated encounters may induce cooperation as the choice, it is also important that the population form groups of members with like behaviour for the effect of repetition to be sustained.

The classic example where participants in a two-player game have to make a choice is the *Prisoner's Dilemma*. In this example, the police do not have full evidence to convict two prisoners whom they have caught in the act of committing a serious offence. So they make the prisoners an offer to induce them to talk — if one of you confesses, we'll let him/her off while the other will get the full three years. If both of you confess, you both get a lighter sentence of one year. If you both stay silent, of course, you still get three months with the evidence we already have. The best choice is for the prisoners to confess — this closes the door to getting only three months, but it eliminates the risk of three years if one should hold out while his/her partner in crime should confess. Variations of the example, where the prisoners have been faced with this choice many times before or belong to a gang that is sworn not to confess, or even where this may be the last time they are faced with the choice, can favour different strategies and probabilities of outcomes. But the best course, generally, seems to be the one where one plays for one's own safety, cooperation with the other player being a typically "high energy" choice. The incentive for cooperation, of course, is that playing for oneself would induce the same behaviour in the other player, in the form of "retaliation" and would finally turn out to be a losing course.

"Direct reciprocity" can thus lead to the evolution of cooperation as the probability of another encounter between the



Matthijs van Veele.



Julián García.

same two individuals exceeds the cost-benefit ratio of being altruistic. Repetitive encounters, then, is a strong influence that favours cooperation and so also different probabilities of meeting the same players in future encounters. Even in an isolated instance of the *Prisoner's Dilemma*, there can be cooperation if the players know the other player. And in such a case, repetition could even induce selfish behaviour as an "assured better strategy".

Simulations

In the paper in *PNAS*, the authors examine the dynamics of the interaction of these two influences for cooperation, in a series of computer simulations that first considered the effect of many repetitions on sustaining different strategies. There could, for instance, be the *Tit For Tat* strategy that would stop the *Always Defect* strategy in its tracks if there were sufficient assurance of repetition. But with an *Always Cooperate, Tit For Tat* would be a natural match and *Always Cooperate* would become abundant in a *Tit For Tat* environment. But widespread *Always Cooperate* would also be the invitation to be exploited by the revival of *Always Defect* — unconditional cooperation becomes cooperation's worst enemy!



Martin A Nowaka.

The simulations show that no strategy is robust against such indirect invasions, which are seen to dominate, destroying and establishing cooperation, and there was a constant weaving in and out of different strategies. But what the simulations show additionally is that in a well mixed population, the paths out of cooperation are more likely than paths that return to cooperation. Even for relatively high continuation probabilities, evolution consistently leads only to moderate levels of cooperation when averaged over time (unless the benefit-to-cost ratio of cooperation is very high).

The simulations then allowed changing of other factors to see what conditions could actually increase cooperation. It was found that making even modest changes in the level of structure in the population increased cooperation substantially over repeated transactions. To study the effect of structure, the simulation model was modified to include a given probability of a player meeting another player who followed the same strategy. The extreme case would be when all cooperators are together and meeting a player of same strategy and also of never meeting one of the other strategy is certain.

This is depicted in the first case in the picture, where cooperators are grouped together and defectors are grouped together. The less extreme grouping is in the second case — where there are the same numbers of cooperators and defectors, grouped differently. It can be worked out that the probability of a player meeting another of the same kind, in this case, is 38.5%; and of meeting another of the different kind is 17.5%.

Simulations were carried out using a different mix of assortments and also with a different probability of repetition. The case of no repetition, of course, was the "single trial" *Prisoner's Dilemma*, with the strategy assured as "defection". But with an increasing structure of the population — that is, when the probability of meeting a cooperator in place of a defector increased — there was a cut-off after which cooperative behaviour commenced. When the probability of repetition was also increased, the minimum structure for commencement of cooperation came down, till it was almost zero at nearly full assurance of repetition. On the other hand, even at nearly full assurance of repetition, cooperation did not commence unless there was at least some structure.

Cooperation increased, of course, when both repetition and structure increased. "Without population structure, cooperation based on repetition is unstable," Julián García, one of the authors of the paper, explains. This is especially true for humans who comprise fluid but not totally unstructured populations, where repetition occurs regularly. Experimental studies show that humans are highly cooperative in repeated games and use conditional strategies. "Therefore, the recipe for human cooperation might be: a bit of structure and a lot of repetition," says García.

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Influencing activity

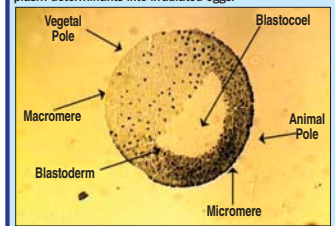
tapan kumar maitra explains the importance of cytoplasmic localisation in development

HOW does one discern the first differences between the cells of an early embryo? The cytoplasm of oocytes and other cells contains molecules that influence the activity of the nucleus. Eggs have such substances in their cytoplasm, and in some cases these are localised in specific regions. As development proceeds, these substances, called determinants of development, are unequally distributed in the cytoplasm of certain groups of cells, which then become committed to a particular type of cell differentiation.

Eggs are generally very large cells that stockpile many of the molecules required for early development. For example, a *Xenopus* egg contains about 100,000 times more RNA polymerases, histones, mitochondria and ribosomes than does a normal adult *Xenopus* somatic cell. One reason for accumulating these ready-made materials rather than making them *de novo* during early embryogenesis is that cell division is extraordinarily rapid during cleavage. At the mid-blastula stage, one of the first stages of development, *Xenopus* replicates its DNA every 10 minutes, a process that takes 1.9 hours in the somatic cells of adults. Similarly, *Drosophila* blastulae double their DNA content in 3.4 minutes.

This rapid pace allows little time for new RNA and protein synthesis, but it is during this period that the first differences between nuclei are established. At least in some cases, these differences result from the presence of the determinants mentioned above. The best example of determinants in development is provided by the germ plasm. Amphibian cells contain in their so-called vegetal (yolky) pole a specialised region of cytoplasm that can be recognised morphologically by the presence of special granules. This cytoplasm has the property of inducing germ cell formation; that is, those cells that contain the germ plasm will eventually become the germ cells of the new organism.

When the posterior poles of the eggs are irradiated with ultraviolet light, sterile (but otherwise normal) animals are obtained. The effect of ultraviolet treatment can be reversed or rescued by injecting cytoplasm containing pole plasm determinants into irradiated eggs.



Blastula stage of frog embryo.

Although determinants are undoubtedly very important in establishing early differences between cells, they cannot entirely explain development. For example, there is no evidence of cytoplasmic localisation in mammalian eggs. Further, as development advances, cell interactions become increasingly important. At the stage of gastrulation, extensive cell movements and migrations occur and different types of cells interact with each other in the phenomenon known as *embryonic induction*. Notochord tissue, for example, induces the overlying ectoderm to become neural tissue, and the optic vesicles (an outgrowth of the brain) induce nearby ectoderm to become the eye lens. These inductions are mediated by diffusible substances but in spite of numerous attempts to isolate them their chemical nature remains unknown.

It is possible that the same principles involved in the action of egg determinants could also apply to adult cells. All cells contain in their cytoplasm molecules that can reprogramme gene expression. Cells continually exchange information between the nucleus and cytoplasm so that gene products accumulated in the cytoplasm can subsequently modify nuclear activity. If these substances are localised in certain regions of the cytoplasm, upon cell division they can become unequally distributed between daughter cells, giving rise to two different cell types. In the differentiation of adult tissues it is frequently observed that only one of the daughter cells becomes specialised; the other remains as a stem cell, which is able to divide again. This occurs in red blood cell differentiation and could occur in skin and intestinal epithelium, in which the dividing cells are located in certain regions of the tissue (attached to the basal membrane or at the bottom of the intestinal crypts).

The hypothetical mechanism is supported by experimental evidence. During nerve cell differentiation in grasshoppers, some cell divisions result in the formation of a neuron (ganglion cell) and a stem cell (neuroblast) which are always in the same position and morphologically recognisable. By introducing a needle at mitosis in one experiment, it was possible to rotate the spindle and chromosomes by 180 degrees, but in spite of this manipulation the resulting daughter cells had the neuron and stem cell in the normal positions. This shows that the ability to become a neuron does not depend on a particular chromosome set but rather on the type of cytoplasm inherited by the daughter cell.

The idea that the cytoplasm contains the determinants that can become unequally distributed in the daughter cells and affect nuclear activity is by no means new. In the 1896 edition of his classic book, *The Cell in Development and Heredity*, EB Wilson viewed development as follows: If chromatin be the idiolplasm (ie, an old term referring to the genes) in which there is the sum total of hereditary forces, and if it be equally distributed at every cell-division, how can its mode of action so vary in different cells as to cause diversity of structure (ie, differentiation)? Through the influence of this idiolplasm (ie, the genes) the cytoplasm of the egg, of the blastomeres derived from it, undergoes specific and progressive changes, each change reacting upon the nucleus and thus inciting a new change. These changes differ in different regions of the egg because of pre-existing differences, chemical and physical, in the cytoplasmic structure; and these form the conditions under which the nucleus operates.

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Nature, nurture... or neither?

Epigenetics is the new twist in an age-old argument. A combination of genes and our environment makes us what we are. Or so we always thought... jeremy laurance reports

IT is a shibboleth of family life — that every individual is the product of their genes and environment, the one an immutable inheritance, the other a mutable array of influences and pressures with unpredictable outcomes. But new research has demonstrated that genes can change. Identical twins with the same genetic inheritance can turn out completely different and the impact of environmental influences can be passed down the generations. The new science of epigenetics has shown that in addition to nature and nurture, what makes us who we are is also determined by biological mechanisms that can switch genes on or off. These epigenetic (above the gene) "light switches" can affect characteristics as fundamental as autism and sexual orientation. But they are also subject to environmental influences and thus, in theory, are within our control.

Professor Tim Spector, head of the department of twin research at Kings College, London, who has undertaken the most detailed studies in the world, cited the case of Iranian twins Ladan and Laleh, who were joined at the head and shared identical genes

and environment and yet had different personalities. The differences led him to question the influence of genes.

"Up to a few years ago I believed genes were the key to the universe. But over the last three years, I have changed my mind," he said at the launch of his book *Identically Different: Why You Can Change Your Genes*, which challenges the view that an individual's genetic inheritance is immutable.

Studies of the effects of famines in Holland in the 1940s, in China in the 1950s and in the USA over a century ago show they changed the lifespan and obesity rates in subsequent generations. They switched on genes that increased the accumulation of body fat in times of plenty, in order to improve survival chances in times of famine.

In the modern world, with calorie-dense fast foods more freely available than at any time in history, the seeds of the current obesity epidemic



Iranian twins Ladan and Laleh.

may thus have been sown in the 19th century. "The risk of obesity can come not just from your own environment or your mother's but higher up (the ancestral chain)," he said. "Four drugs with epigenetic effects that can switch genes on or off are already on the

market and 40 more are in development, he added.

But there are other, natural, ways of controlling them, too. Exercise has been shown to switch off the FTO gene, a key driver of obesity. Diet can also affect gene expression. "We and our genes are more flexible than we thought," Spector said.

The Independent, London



Tim Spector.