before hatching[2]. Thus, Lanza states that DNA treatment extends the life span of such melanocytes. Because of related studies in my laboratory, I would like to offer two possible interpretations of Lanza's observations.

We have reported that injection of chicken DNA into White Leghorn eggs induces two types of effects on the embryos, depending on incubation conditions: a lethal effect which may be mediated by a genetic-like mechanism and a survival-enhancing effect involving a non-genetic mechanism[3]. Both effects are inducible by isologous DNA. Highly polymerised DNA can also enhance mammalian cell viability *in vitro* under conditions excluding a genetic-like mechanism[4]. Such a phenomenon might account for the extended survival of melanocytes in Lanza's system. While we did not observe induction of pigment in more than 200 hatched chickens whose eggs had been injected with non-degraded DNA (either isologous or Rhode Island Red), our experimental procedures differed extensively from Lanza's.

Second, several groups, including ours, have reported induction of melanin in mammalian cells by exogenous DNA[5-7]. Detailed analysis of our data indicated that our effect was the result of alteration of a regulatory gene instead of a structural gene[8,9]. This would seem to be the case for Lanza's system if it involved genetic transformation for pigmentation (instead of survival). As noted above, his target cells presumably contained genetic information for melanin production before DNA treatment.

J. Leslie Glick
Associated Biomedic Systems, Inc.,
872 Main Street,
Buffalo, New York 14202

Dr Lanza replies—The points made by Dr Glick are well taken. A survival-enhancing effect similar to that cited[3], could indeed have extended the life span of the melanocytes reported.

I would like to point out that all five of the embryos which had patches of pigmented feathers were examined between days 14 and 16 of incubation. Although one of the newborn chicks developed some pigmented feathers, no pigment was observed in the juvenile down of any chickens hatched in earlier experiments. This suggests that all the melanocytes were not altered permanently, and those that were, were too few in most cases to synthesise enough melanin to be detected in the juvenile down of a newborn chicken.

Glick's experimental procedures differ extensively from the experiment reported. Alterations in the embryonic stage would have gone undetected because he observed only hatched chickens. It should be emphasised that the pigmentary genes in the White Leghorns referred to in Glick's response are considered dominant, whereas those in White Plymouth Rocks are considered recessive[10]. I am led to conclude that any genetic transformation would be non-pigmentary and would directly involve the genes responsible for the degeneration of the melanocytes.

Although Glick's analysis of induced melanin synthesis in mammalian cells indicated that the effect was due to a regulatory gene instead of a structural gene[8], he should not preclude the possibility of a structural gene defect in White Plymouth Rock melanocytes. The lack of pigmentation in most mammalian cells results from a gene–enzyme defect, in which tyrosinase fails to catalyse the reaction in which tyrosine is hydroxylated to dihydroxyphenylalanine. This is not the case, however, with White Plymouth Rocks as Glick stated, the melanocytes in that system were initially capable of melanin synthesis. Therefore geretic transformations involving pigmentation in this breed, would not necessarily be the result of a regulatory gene.

College of Liberal Arts,
Boston University

1 Lanza, R. P., *Nature*, **252**, 597–598 (1974).
2 Hamilton, H. L., *Anat. Rec.*, **78**, 525–548 (1940).
3 Glick, J. L., Salim, A. P., and Goldhamer, R., in *Informative Molecules in Biological Systems* (edit. by Ledoux, L.), 353–365 (North-Holland, Amsterdam, 1971).
4 Glick, J. L., *Cancer Res.*, **30**, 138–146 (1970).
5 Glick, J. L., and Salim, A. P., *J. Cell Biol.*, **33**, 209–212 (1967).
6 Ottolenghi-Nightingale, E., *Proc. natn. Acad. Sci. U.S.A.*, **64**, 184–189 (1969).
7 Lipkin, G., *J. invest. Derm.*, **57**, 49–65 (1971); *ibid.*, **60**, 381–398 (1973).
8 Glick, J. L., in *Uptake of Informative Molecules by Living Cells* (edit. by Ledoux, L.), 327–346 (North-Holland, Amsterdam, 1972).
9 Glick, J. L., and Majumdar, A., *J. theor. Biol.*, **36**, 503–512 (1972).
10 Rawles, M. E., *J. Invest. Derm.*, **28**, 390 (1948).

Schwarzschild orbital topography and high Doppler blueshifts

Chitre, Narlikar and Kapoor[1] discuss the high Doppler blueshift of forward light emission from material particles in circular orbit in a Schwarzschild field at and near $r = 3GM/c^2$, which they claim is the radius of the unstable circular orbit (meaning, apparently, "the marginally stable circular orbit").

This supposed effect rests on a basic confusion concerning the orbital topography and energetics in the Schwarzschild field. Outside the event horizon, at $r = 2GM/c^2$, there are three important circular orbits[2]: the marginally stable circular orbit at $r_{ms} = 6GM/c^2$, inside which there can be no stable circular orbits (Chitre *et al.*[1] mistakenly put this at $r = 3GM/c^2$); the marginally bound circular orbit at $r_{mb} = 4GM/c^2$, inside which there can be no bound circular orbits; and the photon orbit at $r_{ph} = 3GM/c^2$, which has an infinite energy per unit mass and requires a tangential velocity $v_{tang} = c$. Chitre *et al.*[1] seem to have confused the marginally stable circular orbit with the photon orbit. So it is not surprising that they obtain an infinite blueshift there, since any particle in such an orbit must have $v_{tang} = c$. But, of course, massive particles are barred from such orbits; and those in circular orbits of radii approaching $r = 3GM/c^2$ are not only in an unstable configuration but also highly unbound ($E \gg 1\, mc^2$). In any realistic physical situation they will all move along outward spiral orbits.

Near $r_{ms} = 6GM/c^2$ particles will make many revolutions, but there will be no net Doppler blueshift, since for these $v_{tang} \simeq c/2$. As particles reach $r = r_{ms}$ through, for example, viscous drift, they plunge towards the black hole along spiral orbits. For a test particle moving along such a spiral geodesic, conserving total energy ($E_{ms} = 0.943\ mc^2$) and angular momentum ($L_{ms} = 3.46GMm/c$), where m is the mass of the test particle, 4.85 revolutions will be executed between $r = r_{ms}$ and $r = 2GM/c^2$ (W. R. S., unpublished); and the first four revolutions will be between $r = r_{ms}$ and $r = 5GM/c^2$. If dissipative and radiation processes are important, the spiral orbits will have even fewer revolutions. So there seems to be little reason to expect that there could be a thin disk of massive particles executing even approximately circular orbits in the "high blueshift region" near $r = 3GM/c^2$.

W. R. Stoeger
Institute of Astronomy,
University of Cambridge,
Madingley Road,
Cambridge CB3 0HA, UK

1 Chitre, S. M., Narlikar, J. V., and Kapoor, R. C., *Nature*, **252**, 460 (1974).
2 Bardeen, J. M., Press, W. H., and Teukolsky, S. A., *Astrophys. J.*, **178**, 347–369 (1972).

Drs Chitre, Narlikar and Kapoor reply—We meant what we said in our paper. The orbit in question is close to $r = 3GM/c^2$ as stated, and is not near to $r = 6GM/c^2$ as Stoeger would have us suppose. We are aware that these orbits are unstable and it is not intended that the particle should circulate in such orbits for ever. The circular orbit is meant to be an approximation to the geodesic trajectory of the infalling particle with high energy and small impact parameter, as discussed by Misner *et al.*[1]. Those authors have discussed gravitational synchrotron radiation from particles transiting close to $r = 3GM/c^2$. These and related references have already been cited in our paper so no further clarification is necessary.

Tata Institute of Fundamental Research,
Bombay 400 005

Indian Institute of Astrophysics,
Bangalor 560 006, India

1 Misner, C. W., *et al.*, *Phys. Rev. Lett.*, **28**, 998 (1972).