College and Bishop Heber College rank much above the rest. If a productivity measure such as exergy per faculty (*X*/*F*) score is chosen, Miranda House ranks third among this list of ten. If an efficiency measure such as exergy per crore (Rs) of spending (*X*/*S*) score is considered, we find that Miranda House drops to fifth place. It also seems that higher spending or more faculty does not necessarily increase productivity or efficiency in translating money to scientific wealth.

1. Savithri, S. and Prathap, G., *Curr. Sci.*, 2015, **108**(10), 1922–1926.
2. Abramo, G. and D'Angelo, C. A., *Scientometrics*, 2014, **101**(2), 1129–1144.
3. Abramo, G. and D'Angelo, C. A., *J. Informet.*, 2016, http://dx.doi.org/10.1016/j.joi.2016.04.006
4. Prathap, G., *Scientometrics*, 2011, **87**(3), 515–524.
5. Prathap, G., *Curr. Sci.*, 2017, **112**(6), 1240–1242.

Gangan Prathap is in the Vidya Academy of Science and Technology, Thrissur 680 501, India; and also with A. P. J. Abdul Kalam Technological University, Thiruvananthapuram 695 016, India.
e-mail: gangan_prathap@hotmail.com

OPINION

Polarity, asymmetry and aging: are there Yayatis among bacteria?

Ulfat Baig, Milind Watve and Uttara Lele

Yayati was a Hindu mythological king who exchanged his age with his son so that the father became young and the son old. Is this possible anywhere in the world of biology?

Bacteria have been shown to age. In an exponentially growing population some cells progressively slow down and stop dividing[1]. This is thought to be due to asymmetric damage segregation in which old pole cells retain damaged components and the new pole cells receive newly synthesized components[2]. Polarity implies functional asymmetry with a predefined direction with or without morphological difference. Cellular polarity and division asymmetry are common to yeast, bacteria and stem cells of multicell organisms[3]. A number of processes in bacteria, including formation of endospores, flagella, stalks or buds show clear polar biases[4].

Experiments in morphologically symmetric rod-shaped *Escherichia coli* showed that the cells inheriting old pole exhibited decreased growth rate, less offspring production, and increased probability of death[1,2]. Although damages could potentially be of many types, a major component that shows demonstrable asymmetric segregation is protein aggregates[5]. Protein aggregates frequently occupy polar positions, although they are also observed at other locations[6].

Often in the context of bacterial aging, the terms 'polarity' and 'asymmetric damage segregation' have been used interchangeably. In principle, asymmetric damage segregation should be possible without predefined polarity. Even if the damaged components go randomly to one of the daughter cells, all the presumed advantages of asymmetric division would be obtained[7–10]. The old pole–new pole axis (OPNPA) is not necessary for this advantage. However, it is possible that the mechanism of asymmetric segregation is such that the old pole receives the damaged components either invariably or with a greater probability. Therefore, there may or may not be a one-to-one association between old pole and old age.

Stewart *et al.*[1] observed 7953 pairs of sister cells among which 54% of the time the new pole divided faster than the old pole, 15% of the time there was no difference and 31% of the time the old pole divided faster than the new pole. Lele *et al.*[11] showed that old pole cells divided slower than the new pole cells in 12 out of 18 experiments, while in the remaining six a reversed pattern was seen. Lindner *et al.*[5] observed that under non-stressed conditions, 28% of the time protein aggregates were localized at mid-cell position, 30% of the time at the new pole and 31% of the time at the old pole when first formed with a noticeable size. It is possible that with subsequent divisions the aggregates end up being at the old pole. Baig *et al.*[6] and Lele *et al.*[11] showed that protein aggregation and symmetry of cell division in *E. coli* is not hard-wired but responsive to environmental conditions and even reversible under certain conditions. There is substantial plasticity as well as evolvability in protein aggregation and functional asymmetry.

All these results suggest that while asymmetric damage segregation is at the centre of aging in bacteria, its association with OPNPA may not be indispensable. If OPNPA is central and critical to asymmetric division and aging in bacteria, then spherical organisms that change their plane of division and thereby do not have a fixed OPNPA could be immune to aging[3,12]. Baig *et al.*[13] showed that cumulative cell division asymmetry exists in *Staphylococcus aureus*. Also, there is no evidence for any equivalence of polarity in these organisms[12]. OPNPA does not seem to be a necessary prerequisite for asymmetric damage segregation and thereby cell senescence.

This might be the solution for an unresolved riddle. Wang *et al.*[14] followed the old pole cell for 200 generations using a microfluidic device and showed that the

growth rate of the old pole cell remained unchanged; nevertheless, the probability of death increased with age. These results are not surprising if we realize that there is a small probability of pole flipping in the functional sense. The membrane is a fluid and therefore even if the aggregated damage foci are anchored to it theoretically, they can drift. The position of protein aggregates could change in a small proportion of cells as noted by Lindner *et al.*[5] which may be sufficient to cause pole flipping with a small probability. If this happens, long-term conservation of growth rate of the mother cells is possible by periodic rejuvenation. Furthermore, the probability of pole flipping may be different in fluid media and on agar surfaces, since the fluid dynamics of the two systems can be substantially different. Pole flipping by protein aggregates has been demonstrated in yeast[15], further strengthening our speculation. If there is functional pole flipping, the old pole cells can become fresh and the new pole cells old. This has a close parallel to the Yayati story of Indian mythology, where an old individual becomes young by exchanging age with a younger individual. By flipping poles the relationship of protein aggregation with other polar functions such as antibiotic persistence[16,17] can change increasing population variability and thereby bet hedging advantage. Thus there is likely to be greater biological relevance to pole flipping, which needs to be explored further.

1. Stewart, E. J., Madden, R., Paul, G. and Taddei, F., *PLoS Biol.*, 2005, **3**, 295–300.
2. Ackermann, M., Stearns, S. C. and Jenal, U., *Science*, 2003, **300**, 1920.
3. Macara, I. G. and Mili, S., *Cell*, 2008, **135**, 801–812.
4. Cameron, T. A., Zupan, J. R. and Zambryski, P. C., *Trends Microbiol.*, 2015, **23**, 347–353.
5. Lindner, A. B., Madden, R., Demarez, A., Stewart, E. J. and Taddei, F., *Proc. Natl. Acad. Sci. USA*, 2008, **105**, 3076–3081.
6. Baig, U. I., Bhadbhade, B. J., Mariyam, D. and Watve, M. G., *PLoS One*, 2014, **9**, e107445.
7. Chao, L., Rang, C. U., Proenca, A. M. and Chao, J. U., *PLoS Comput Biol.*, 2016, **12**, e1004700.
8. Watve, M., Parab, S., Jogdand, P. and Keni, S., *Proc. Natl. Acad. Sci. USA*, 2006, **103**, 14831–14835.
9. Kysela, D. T., Brown, P. J. B., Huang, K. C. and Brun, Y. V., *Annu. Rev. Microbiol.*, 2013, **67**, 417–435.
10. Vedel, S., Nunns, H., Košmrlj, A., Semsey, S. and Trusina, A., *Cell Syst.*, 2016, **3**, 187–198.
11. Lele, U. N., Baig, U. I. and Watve, M. G., *PLoS One*, 2011, **6**, e14516.
12. Turke, P. W., *Q. Rev. Biol.*, 2008, **83**, 243–256.
13. Baig, U., Sunny, R., Watve, M. and Lele, U., *Matters*, 2016, **2**, e201603000022.
14. Wang, P., Robert, L., Pelletier, J., Dang, W. L., Taddei, F., Wright, A. and Jun, S., *Curr. Biol.*, 2010, **20**, 1099–1103.
15. Nakaoka, H. and Yuichi, W., *BioRxiv.*, 2017, **1**, 128298.
16. Bergmiller, T. *et al.*, *Science*, 2017, **356**, 311–315.
17. Leszczynska, D., Matuszewska, E., Kuczynska-Wisnik, D., Furmanek-Blaszk, B. and Laskowska, E., *PLoS One*, 2013, **8**, e54737.

ACKNOWLEDGEMENT. U.B. and U.L. were supported by the Rajeev Gandhi Science and Technology Commission, Maharashtra during the study.

Ulfat Baig, Milind Watve and Uttara Lele are in the Indian Institute of Science Education and Research, Dr Homi Bhabha Road, Pune 411 008, India.*
**e-mail: ulfat@iiserpune.ac.in*