of these compounds (17). It is therefore possible that cancer may result from synergistic interactions between normal cellular UV light, bacteria-produced UV emissions and pollutants such as PAHs.

The suggested hypothesis would help explain why cancer is not infectious in the normal sense, since it implies that the UV produced by bacteria growing within the cell would operate as a 'cancer switch' which would also be influenced by both hereditary and environmental factors. The internal production of UV might be one of many ways by which intracellular bacteria might influence the operation of such a 'switch'.

Of course, 'the devil lies in the detail' and the main problem with this hypothesis relates to dose-effect. That is would sufficient UV be produced by intracellular bacteria to effect DNA mutagenesis, bearing in mind that any UV emissions would be subject to adsorption by cellular contents, including membranes?

Should the present hypothesis be correct, then vaccines might be developed to prevent the growth of intracellular bacteria. Alternatively, other agents might be found which may prevent such bacteria from emitting UV, or counteract its mutagenic effects. Such agents would be invaluable in preventing both the development or spread of cancer.

**Microbiology Comment**

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In summary, this is an interesting hypothesis. However, if it is an established fact that micro-organisms do produce UV light, then it is worth considering.

**Reviewer's Comments**

At first sight, this hypothesis appears unlikely. However, if it is an established fact that micro-organisms do produce UV light, then it is worth considering.

One possible way of testing this hypothesis is to look at the profile of mutations induced in a gene such as p53 in tumour cells. Skin cancers show significant levels of mutations at adjacent pyrimidines indicative of UV-damage. If this was shown to be the case for internal tumours this would suggest UV-damage at di-pyrimidine sites, a finding that would strongly support the hypothesis. However, the prevailing data do not show this, certainly not at significant levels. For example, lung tumours, associated with smoking, most often show transversions at G residues, consistent with UV-damage. If this was shown for di-pyrimidines this would suggest an interaction between the UV light produced by the micro-organisms and the DNA in the cell.

It has been argued that in electron micrographs of mycoplasma cells (PG18) with human polymorphonuclear leukocytes (PMNL) which show mycoplasmas clustered within phagosomes and also single mycoplasma cells apparently free within the cytoplasm. The frequency with which intracytoplasmic mycoplasmas were seen was greater where mycoplasma cells were non-opsonized than when non-specifically opsonized by incubation for 45 min in 10% human serum. In contrast, when mycoplasma cells were opsonized, intracellular mycoplasmas were almost always internalized within phagolysosomes.

It has been argued that in electron microscopic studies the appearance of mycoplasmas within host cells might be artefactual and due to the presence of the mycoplasmas within imaginations of the cell membrane. Thus, Taylor-Robinson et al. (2) claimed that unequivocal evidence of the intracellular location of mycoplasmas required specific staining of both the mycoplasma and host cell surface. This was achieved in their study using gold-labelled anti-mycoplasma antibody and ruthenium red, which bound to the exposed poly-saccharide surface of both mycoplasma and eukaryotic cell surfaces, but not to the membranes of cell vacuoles. Jensen et al. (3) found ruthenium red staining of the Vero cell membrane to be weak and of little value in confirming the apparent intracellular location of M. genitalium cells. They argued that where mycoplasma cells appeared close
to the cell nucleus, this was good evidence for an intracellular location. Interestingly, the apparently intracytoplasmic *M. genitalium* cells seen in their electron micrographs were embedded within electron dense material. This reaction may be similar to that for *M. fermentans* cells within human PMNL, suggesting a phagosome-independent uptake route for mycoplasmas and confirming an intracellular location.

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