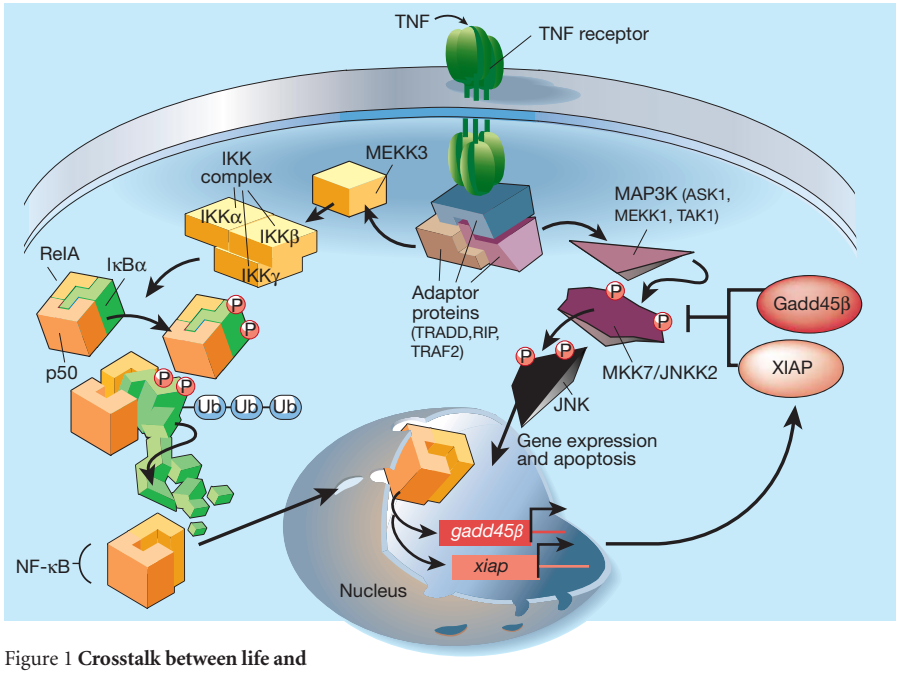

Figure 1 **Crosstalk between life and death signalling pathways. The inflammatory molecule tumour-necrosis factor (TNF) recruits two pathways, which culminate in the activation of JNK or NF-κB. Both begin (top) with the binding of TNF to its receptors, which are coupled to downstream events by adaptor proteins[3]. Left, in the NF-κB pathway, the next step[14] is the activation of the enzyme MEKK3. This in turn activates the IKK complex, which adds phosphate groups (P) to IκBα, ensuring that this protein is modified with ubiquitin groups (Ub) — a prerequisite for proteolytic destruction. NF-κB is then free to move to the nucleus, where it activates target genes. Right, in the JNK pathway, a cascade of protein kinases leads to the activation of JNKs, some of which move to the nucleus and influence the expression of a different set of genes. Tang *et al.*[1] and De Smaele *et al.*[2] have found that NF-κB induces the expression of *gadd45β* and *xiap*, which blunt the JNK pathway and so promote cell survival.**

the Gadd45 proteins couple signalling pathways that are recruited by DNA damage to JNK activators. Moreover, in T lymphocytes, disruption of *gadd45γ* dampens the activation of JNK by the T-cell antigen receptor[12]. However, these results pertain primarily to Gadd45γ, not Gadd45β.

Meanwhile, XIAP was identified as a 'bridging' protein that indirectly couples receptors for bone morphogenetic proteins (a group of cytokines important in development) to the activation of JNK and other MAPKs[3]. How, then, might it blunt the activation of JNK by TNF? Perhaps, when bone morphogenetic protein is absent, XIAP instead sequesters elements that are needed for TNF to activate JNKs. One such element could be TAK1, a protein kinase that associates indirectly with XIAP and has been implicated[13] in the activation of both IKKs and JNK by interleukin-1. Yet NF-κB does not affect[1] the recruitment of JNK in response to interleukin-1. Further studies are clearly needed.

How might these new results[1,2] fit into a dynamic, context-dependent model for TNF-induced signalling? Under different physiological circumstances, different adaptor proteins could be recruited to TNF receptors, favouring the activation of NF-κB or JNK and so influencing the contribution of the 'crosstalk' effect. For example, apoptosis-signal-regulating kinase-1 is a protein kinase required for the sustained activation of JNK by TNF in fibroblast cells[6]. Moreover, prolonged activation of NF-κB triggers the expression of its inhibitor, IκB, reducing further activation[4]. The contribution of these two effects might ensure that long-term exposure to TNF preferentially activates JNK-dependent apoptosis. It will be interesting to see how cell- and context-specific signalling elements are integrated with the newly discovered crosstalk between these general pathways of life and death signalling. ■

John M. Kyriakis is in the Diabetes Research Laboratory, Massachusetts General Hospital, 149 13th Street, Charlestown, Massachusetts 02129, USA.
e-mail: kyriakis@helix.mgh.harvard.edu

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Daedalus

Slippery light aircraft

The drag on an aeroplane, says Daedalus, is largely due to the impact of its 'wetted surface' with still air. Each colliding air molecule is momentarily adsorbed and later re-emitted at some unpredictable angle, running away with energy. The unwetted ideal, in which each molecule bounces off directly like light from a mirror, almost never happens. A rusty wing, resisting the attacking oxygen, seems worth trying; but this still leaves the nitrogen in the air. In any case, most aircraft wings are aluminium. This is superficially oxidized to transparent aluminium oxide, but is still well wetted by oxygen in the air. Surface tension is well understood in liquids, but has been largely ignored in gases. Daedalus recalls the chemical notion of anti-bonding, in which one molecule is repelled by another if the bond between them is raised by light or near-ultraviolet irradiation to an anti-bonding state.

Illuminated aircraft were actually tried during the Second World War to reduce the contrast between plane and sky as seen from the ground. Daedalus reckons that a non-wetted, anti-bonding plane should fly. DREADCO physicists are therefore fitting an aluminium plane with two sets of lights: one to raise the bond between aluminium oxide and oxygen to anti-bonding, the other to do the same for the bond between aluminium oxide and nitrogen. Such an aircraft would then remain untouched by the chief constituents of the air. Illuminated wings and fuselage should simply not be wetted.

Daedalus predicts a dramatic loss of drag. Only the windows would remain to contribute to the normal drag. The new slippery aircraft would require much less power. Even better, the propellers or the inlet fans of the jet engines could also be illuminated. The drag of these fast-moving elements would be cancelled as well, greatly increasing their efficiency. The whole plane should consume a small fraction of the usual power.

Aerodynamicists will at last be able to predict the behaviour of wings and fuselage simply. All the problems of the boundary layer will vanish: there won't be one. Molecules will bounce off an unwetted aircraft perfectly. Daedalus reckons that there should be a great saving of fuel, helping to reduce carbon dioxide emissions. Sadly, the usual painted logos must be abandoned, otherwise more light wavelengths will have to be added to reverse the bonds between the paint and the air's oxygen and nitrogen. David Jones