

Figure 1 | Persistent spin helix. In a semiconductor quantum well, a thin layer of semiconductor material sandwiched between two other semiconductors, electrons are confined in the dimension perpendicular to the plane of the layer — that is, they move only along the layer (yellow). By a process known as optical orientation², electron spins (arrows) can be made to orient out of the plane (a) or along one of the plane's dimensions (b). In both cases, the electrons are subject to random forces that, in conjunction with an interaction called spin–orbit coupling, cause their spins to flip. Koralek and colleagues³ show that by combining these two spin orientations to form a helical wave of rotating spin orientation (c), and by fine-tuning the structural properties of the quantum well, the spins become largely protected against decay.

becomes inhibited. The authors show that the collective spin-orientation wave persists for much longer than its individual spin components. That is, spins in the helical wave become immune against relaxation: spin–orbit coupling is effectively absent, making the spin unaware of the random velocity kicks. This so-called persistent spin helix, which was theoretically introduced by Bernevig *et al.*⁹, is based on a dynamical symmetry of the entire spin ensemble that is formally akin to the rotational symmetry a single electron spin enjoys in the absence of spin–orbit coupling (Fig. 1).

Despite the appeal of the theoretical ideas behind the persistent spin helix, a theorist's notion of fine-tuning spin–orbit coupling and creating rather special spin helices is a far cry from the experimental effort required to realize them. And yet Koralek and colleagues have succeeded in doing just that. Their experimental demonstration of the persistent spin helix is a remarkable feat.

To create spin-orientation waves of the required wavelength, the authors used a technique known as transient spin grating^{10,11}. In their experiment, two non-collinear laser beams of light linearly polarized in orthogonal directions interfere at the plane of the quantum well and produce a sinusoidal pattern of light helicity: stripes of alternating circular polarization (helicity) of light. Such a pattern of helicity can orient electrons' spins through a process called optical orientation² and generate a spin-orientation wave. Say that right- or left-circularly polarized light creates spin-down and spin-up electrons, respectively. The pattern of light helicity then translates into an identical pattern of electron spin orientation. The wavelength of such a spin-orientation wave can be

tuned by changing the angle between the two interfering laser beams.

The resulting (linearly polarized) spin-orientation wave can be viewed as composed of two spin helices — waves of rotating spin orientation. One helix rotates clockwise, the other anticlockwise. Under the right conditions, only one of them is the persistent spin helix. The other helix decays as usual. By watching the temporal evolution of the spin-orientation wave pattern with probe laser beams, we should in principle spot an initial fast decay of the normal (non-persistent) helix, followed by a much slower decay of the persistent one.

This is exactly what Koralek and colleagues

find in their experiments. By suitably tuning the structural composition of the quantum wells, achieved by varying both the width and the degree of doping asymmetry of the quantum well, the authors show that the emergent persistent spin helix lasts a hundred times longer than the normal one. The spins curl themselves up to ward off spin relaxation. The slow decay of the persistent spin helix is caused by residual spin–orbit interactions.

Koralek and colleagues' experimental realization of the persistent spin helix is a breakthrough towards minimizing and controlling spin relaxation in electronic systems. The next chapter in the field of spintronics is one that deals with ways of controlling the spin's lifetime electrically. That could be achieved by turning spin helices on and off with an electrical gate, or by demonstrating their role in the predicted drastic increase of electrical spin injection efficiency, an essential part in the operation of spintronic devices¹². For the spin, this is as good as it gets — at least for now. ■

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DNA REPAIR

New tales of an old tail

Jiri Lukas and Jiri Bartek

Modifications of DNA-associated histone proteins maintain genome integrity. On damage to DNA, phosphorylation of histone H2A.X determines whether repair is justified or if the damaged cell must die.

Chromosomal DNA wraps around histone proteins to form a complex scaffold called chromatin¹. The reorganization of these proteins following DNA damage is crucial for repairing the damage, and so maintaining genomic integrity and reducing the likelihood of cell death or cancer. One such histone modification — known as γ -H2A.X — follows DNA double-strand breaks (DSBs) and involves phosphorylation by the enzyme ATM of serine

residue 139, which is located in the carboxy-terminal tail of the histone variant H2A.X (ref. 2). γ -H2A.X generates a chromosomal microenvironment that promotes recruitment of repair proteins³ and facilitates DNA repair to reduce the risk of mutations⁴. But how this modification is regulated and how it affects cell fate have remained elusive. Two papers^{5,6}, including one on page 591 of this issue, provide insights into these questions.

The discovery of DSB-induced γ -H2A.X sparked enormous efforts to decipher how repair and signalling proteins assemble into foci on chromatin marked by this modification. The search concentrated mainly on identifying repair factors and other histone modifications operating downstream of γ -H2A.X — hence ‘moving away’ from this priming DSB-associated histone mark. But it emerges that another key chromatin modification in response to DSBs also occurs in the H2A.X tail, just three amino acids away from serine 139 (S139).

Indeed, Xiao *et al.*⁵ and Cook *et al.*⁶ have now independently discovered that tyrosine residue 142 (Y142) of H2A.X is also phosphorylated (Fig. 1a). Both groups show, however, that unlike S139, Y142 is already phosphorylated in unstressed cells and becomes gradually dephosphorylated after DNA damage. Even more unexpectedly, dephosphorylation of Y142 seems to be a prerequisite for the γ -H2A.X modification, indicating that the phosphorylation status of the Y142 residue of H2A.X regulates what has been considered the main trigger of the entire DSB-induced chromatin pathway. Such a twist in our thinking about genome-maintenance mechanisms clearly deserves a closer look.

The starting point for Xiao *et al.*⁵ was the observation that the evolutionarily conserved Y142 in human H2A.X is phosphorylated *in vivo*. They then found that components of the WICH chromatin-remodelling complex^{7,8} interact with the carboxy terminus of H2A.X, where Y142 is located. Strikingly, they showed that the WSTF component of WICH has tyrosine-kinase activity, enabling it to phosphorylate Y142. The authors also found that, after DNA damage, WSTF dissociates from chromatin — consistent with a decrease in Y142 phosphorylation — making way for the γ -H2A.X modification (Fig. 1b).

Cook *et al.*⁶ observed that, during embryonic development of mouse kidney, deletion of either *Eya1* or *Eya3* — genes encoding protein-phosphatase enzymes that dephosphorylate tyrosine residues — coincides with increased γ -H2A.X. The authors also found that the *Eya1* and *Eya3* enzymes bind to and co-localize with γ -H2A.X at foci of DSBs in the nucleus, leading them to consider that H2A.X might be phosphorylated on a tyrosine residue; indeed, they identified Y142 as the target. What's more, in agreement with the observations of Xiao and colleagues, Cook *et al.* report that after DNA damage there is an *Eya1*- or *Eya3*-dependent decrease in tyrosine phosphorylation of H2A.X (Fig. 1b).

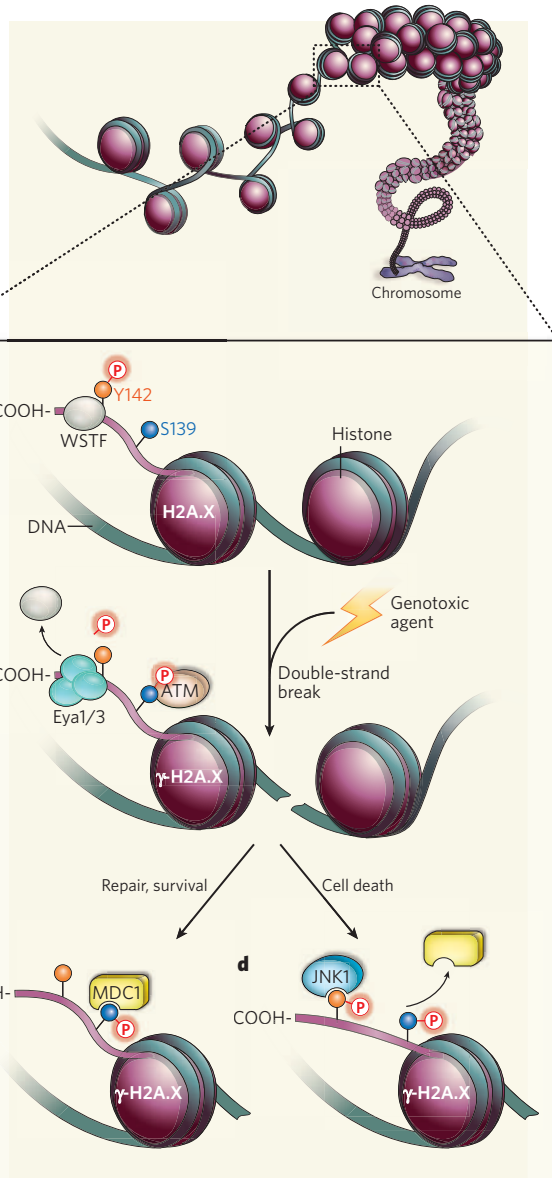


Figure 1 | A matter of life or death^{5,6}. **a**, Normally, the WSTF kinase associates with the carboxy terminus of the histone variant H2A.X and phosphorylates its Y142 residue. Thus, chromatin remains in a ‘standby’ mode with no unnecessary DNA repair events. **b**, When DNA double-strand breaks occur after exposure to genotoxic agents, WSTF dissociates and is replaced with the *Eya1/3* phosphatases, which dephosphorylate Y142, facilitating S139 phosphorylation (the γ -H2A.X modification) by the ATM enzyme. What happens next depends on whether the damage is repairable. **c**, If repair is possible, phosphorylated S139 recruits MDC1 and other repair factors. **d**, If it is not, the γ -H2A.X tail might undergo conformational changes that allow maintenance or re-phosphorylation of Y142. This would prevent retention of repair factors, and instead attract the JNK1 complex, which promotes apoptosis.

Reducing *Eya* levels prevented DNA-damage-induced dephosphorylation of Y142 and the proper interaction of γ -H2A.X with MDC1 — an adaptor protein that senses γ -H2A.X and orchestrates the assembly of repair proteins on the chromatin at DSBs^{9,10}.

Together, these findings^{5,6} make a compelling case for Y142 phosphorylation as a new modification of H2A.X and suggest that a balance between the kinase activity

of WSTF and the phosphatase activity of *Eya* proteins regulates both the formation of γ -H2A.X-marked chromatin and the recruitment of repair factors to DSBs. And, besides uncovering another dimension of the chromatin response to genotoxic stress, each paper provides other surprising results.

First, the WAC catalytic domain that Xiao and colleagues⁵ identified in the amino terminus of WSTF shares no sequence similarity with other known kinase enzymes⁴ — an intriguing finding, the significance of which extends beyond DNA repair. WSTF probably also phosphorylates substrates other than H2A.X, and the identification of these might help explain the clinical symptoms associated with Williams–Beuren syndrome, a neurodevelopmental disorder linked to deletions of the *WSTF* gene. Furthermore, other proteins might contain a WAC domain, and a search for such hitherto unrecognized tyrosine kinases could be rewarding.

Second, Cook *et al.*⁶ report that peptides derived from the carboxy-terminal tail of H2A.X that were phosphorylated on both S139 and Y142 did not bind MDC1, consistent with the fact that Y142 dephosphorylation is required for γ -H2A.X–MDC1 interaction. What was unexpected, however, was that the doubly phosphorylated H2A.X peptide binds the protein kinase JNK1 — an established inducer of programmed cell death (apoptosis). It seems, therefore, that phosphorylated Y142 might function as a decision-maker, determining cell fate after DNA damage. When repair is possible, Y142 is dephosphorylated, allowing the γ -H2A.X modification and the recruitment of repair factors (Fig. 1c). Otherwise, Y142-phosphorylated H2A.X persists, recruiting the JNK1 complex to ‘switch’ to the pro-apoptotic mode, and eliminate cells with irreversibly damaged genomes from the organism (Fig. 1d).

As with all inspiring discoveries, the work of Xiao *et al.*⁵ and Cook *et al.*⁶ raises yet more questions. As WSTF is the kinase responsible for Y142 phosphorylation — and could thus be viewed as a negative regulator of γ -H2A.X — one would predict that reducing WSTF levels could facilitate γ -H2A.X formation. In fact, the opposite happens: in the absence of WSTF, γ -H2A.X and focus formation cannot be sustained, and MDC1 recruitment to DSBs is inhibited⁵. To explain this conundrum, Xiao *et al.*⁵ propose that WSTF might also help adjust local chromatin structure for maintenance

of γ -H2A.X. This is plausible, as the WICH complex also has chromatin-remodelling activity during DNA replication^{7,8}.

The main conceptual issue arising from Cook and colleagues' results⁶ is the proposed role of phosphorylated Y142 in promoting cell death. On one hand, the authors provide evidence for increased H2A.X-JNK1 interaction in cells exposed to high doses of radiation. This indeed supports the switch model, as such Y142-mediated recruitment of JNK to sites of DSBs helps direct cells towards apoptosis as a last resort. On the other hand, they show that Y142 is dephosphorylated after DNA damage, resulting in the loss of the 'docking site' for JNK1. At first glance at least, this finding does not fit the switch model, calling for more work to reconcile it with the observed pro-apoptotic effects of Y142 phosphorylation. It may be, however, that Y142 is re-phosphorylated after futile attempts to repair excessive DNA damage.

Clearly, the issue of the efficiency of DSB repair and the role of posttranslational chromatin modifications in this process is here to

stay. Nevertheless, the two papers^{5,6} provide a fresh conceptual framework and tools to tackle this challenge, which should enable us to better understand the genesis of major genome-instability diseases, including cancer, premature ageing and neurodegeneration. ■
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ENVIRONMENTAL SCIENCE

Clean coal and sparkling water

Werner Aeschbach-Hertig

Subsurface storage of carbon dioxide is a major option for mitigating climate change. On one account, much of the gas sequestered in this way would end up as carbonic acid in the pore waters of the host rock.

Atmospheric concentrations of greenhouse gases, especially carbon dioxide, continue to rise at an alarming rate. We seem unable to tame our appetite for fossil fuels on a meaningful timescale, and the concept of carbon capture and storage has emerged as a serious option for reducing CO₂ emissions to the atmosphere. A 'clean coal' technology, in which CO₂ is collected from coal-fired power plants and stored safely below ground, might enable us to continue using this comparatively cheap and abundant energy source without climatic worries.

However, little is known about the long-term fate of large quantities of CO₂ put into geological storage. Gilfillan *et al.*¹ (page 614 of this issue) illuminate this crucial matter by showing that dissolution in groundwater is by far the most important trapping mechanism for CO₂ in the subsurface environment. In other words, sequestering CO₂ in geological formations would probably produce vast quantities of highly CO₂-enriched sparkling water.

The safety of geological storage of CO₂ is obviously a central concern in planning carbon sequestration on a large scale. When CO₂ is injected into the subsurface, it will be retained by physical and geochemical mechanisms². Physical trapping is provided by the presence

of sealing, low-permeability rock formations above the targeted layer. Such cap rocks are essential features of natural gas and oil reservoirs, and are a primary requirement for CO₂ storage sites. A further level of safety is added by geochemical interactions that remove the pure CO₂ phase, either through dissolution in water (solubility trapping) or by precipitation of carbonate minerals (mineral trapping). Clearly, mineral trapping is the preferable pathway, as it promises to store the carbon over geological timescales.

To assess the risk of leakage from storage reservoirs, an expansive programme for monitoring underground CO₂ injection in a variety of geological settings has been called for³. There are only a few currently active pilot sites, and more are needed. But that apart, such monitoring programmes can reveal the effects of carbon sequestration only on the engineering timescale — they do not yield a direct answer to questions regarding the long-term behaviour of CO₂ in geological storage.

In this respect, the approach taken by Gilfillan *et al.*¹ is logical and informative. The authors used CO₂-rich gas fields as natural analogues for future carbon-storage sites. Other researchers have exploited this idea⁴. But in offering a self-consistent evaluation of noble



50 YEARS AGO

It often happens that investigators, particularly in the social sciences, must try to collect the information which they need by using questionnaires. One of the many problems that are apt to arise concerns the reliability of answers to questions which require an exercise of detailed and specific memory. Recently, the Tobacco Manufacturers Standing Committee issued a Research Paper (No. 2) entitled "The Reliability of Statements about Smoking Habits" by G. F. Todd and J. T. Laws ... The authors show how statements about current smoking habits are generally reconstructed from a sort of 'mental picture' that the informant has of himself 'in his role as a smoker'. Changes in smoking habits are far more frequent than is generally thought to be the case, and so any information about them which refers to the past, based, as it must be, upon a general and personal assessment of current practices, is very likely to be in error ... recall is frequently mistaken both as regards the amount and the kind of smoking carried on. Apart from the special topical interest of this study, it has wide methodological implications which ought to be considered by all users of questionnaires.

From *Nature* 4 April 1959.

100 YEARS AGO

The influence of breed on egg-production in poultry is well seen in a report recently issued by Messrs. E. and W. Brown from University College, Reading. Danish, American, and English Leghorns were kept under comparable conditions for twelve months, and careful record was kept of the number of eggs laid. The Danish birds had been bred to yield a large number of eggs of moderate size; the English birds, on the other hand, had been largely bred for exhibition purposes, for which egg-producing capacity is not needed ... The profit on the English birds is shown to be much less than that on the Danish or American birds.

From *Nature* 1 April 1909.

50 & 100 YEARS AGO