Engineering

Well-matched vibrations cool electronic hotspots

Liwen Sang

Diamond layers can help to dissipate the heat generated by high-power semiconductor devices. This effect has now been enhanced by adding layers of materials and engineering their crystal-lattice vibrations to be compatible at the interfaces.

As computer components become ever smaller and more powerful, managing heat dissipation is becoming crucial to the design of semiconductor transistors. The peak temperature of today's transistors is determined mainly by the localized hotspots that form because of confined electric fields. Adding a layer of synthetic diamond is considered the most efficient strategy for cooling these hotspots, but a resistance to heat flow can build up at the interface between diamond and common semiconductor materials, such as silicon and gallium nitride[1,2]. As reported at the most recent International Electron Devices Meeting, Woo *et al.*[3] have proposed a way of using ultrathin silicon carbide to bring this 'thermal boundary resistance' down to record low values.

Diamond's superior electrical and thermal properties make it an ideal material for dissipating heat in electronic devices. But if the interface between diamond and a semiconductor is imperfect, or the crystal structures of the two materials are mismatched, a large thermal boundary resistance can arise. Strategies for overcoming this problem have been proposed, such as introducing extra layers[4] or growing the diamond from tiny seeds[5], but these approaches have not yielded resistances that are as low as those predicted[6].

In non-metallic solids, crystal-lattice vibrations called phonons act as the main heat carriers. If two materials in contact with each other have well-matched phonon behaviours, the heat transmission between them is optimized. Woo *et al.* have adopted this principle, using silicon carbide as a 'phonon bridge' from a semiconductor device made of gallium nitride or silicon to a layer of diamond (Fig. 1a). The phonons in silicon carbide have a density of states (the proportion of states available at a given energy) that is identical to that of the phonons in diamond. The bridge thus minimizes the thermal boundary resistance to the semiconductors.

Members of the same research group as Woo and colleagues previously found that this technique worked well for the semiconductor material gallium nitride[7], which is a promising alternative to silicon. Gallium nitride has good thermal stability, can switch its logic state faster than silicon can and is able to sustain higher voltages before it breaks down. Transistors made from this material therefore generate a huge amount of heat. Simulations have shown[8,9] that direct contact with diamond could reduce the temperature in gallium nitride devices by 25–50%. However, several of the properties of diamond and gallium nitride differ markedly, which exacerbates the thermal boundary resistance at the interface.

The team's previous work[7] showed that the silicon carbide layer could be generated by inducing carbon atoms to diffuse into a silicon nitride film (Fig. 1b). Silicon nitride can be used to protect gallium nitride from the damaging effects of the hydrogen plasma that forms during epitaxial growth of diamond (in which the carbon atoms adopt the crystal lattice of an underlying substrate). But silicon nitride has poor thermal conductivity, which induces a large thermal boundary resistance. By reducing the thickness of the silicon nitride layer and converting part of it into silicon carbide, the authors achieved a resistance of 3.1 square metres kelvin per gigawatt – less than half than that reported previously for an interface comprising diamond, silicon nitride and gallium nitride[7,10].

In the present work, Woo *et al.* found that they could achieve a thermal boundary resistance as low as 1.89 $m^2 K GW^{-1}$ by growing diamond on silicon, buffered by a silicon carbide layer. Using a form of electron microscopy, they showed that the interface between diamond and silicon carbide was not abrupt, which made it ideal for heat transport because it smoothed the phonon transition between materials. The authors then experimented with a layer of silicon dioxide, which is used widely in the semiconductor industry. Their imaging revealed that carbon atoms diffused into the silicon dioxide in much the same way as they had into silicon nitride, resulting in a thin silicon carbide layer. Although this structure did not achieve as low a resistance as did silicon carbide alone, it demonstrated the immediate applicability of interface engineering.

Although Woo and colleagues' results are impressive, they could be improved further by matching the phonon properties in silicon carbide with those of the semiconductors.

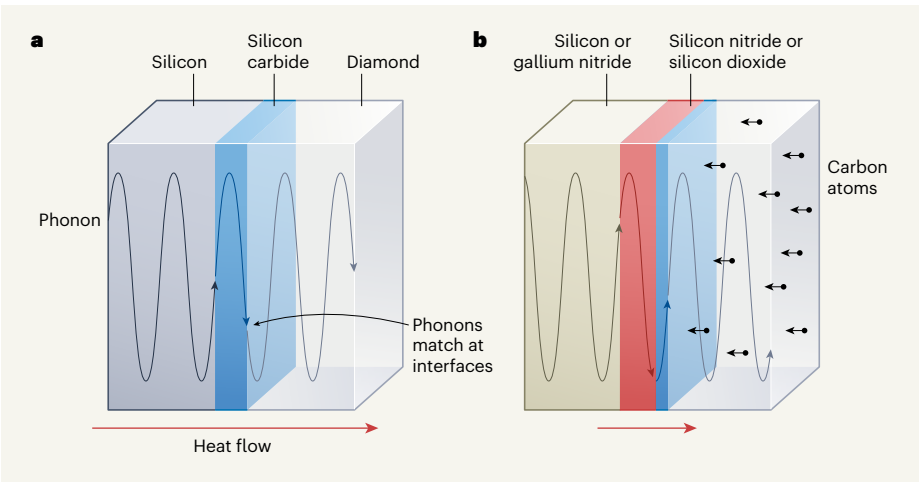

Figure 1 | Matching crystal-lattice vibrations in a semiconductor device. Diamond is widely used to dissipate the heat generated by semiconductor devices, but a thermal boundary resistance can build up at the diamond–semiconductor interface, impeding heat flow. **a**, Woo *et al.*[3] showed that this strategy can be improved by introducing silicon carbide between silicon (a semiconductor) and diamond, and engineering it so that the properties of crystal-lattice vibrations known as phonons were matched at the interfaces. **b**, The same group had previously found[7] that this resistance could be lowered by placing a silicon nitride interlayer between gallium nitride (another semiconductor) and diamond, and in the present work the authors have demonstrated a similar effect by adding silicon dioxide between silicon and diamond. In both cases, diffusing carbon atoms from the diamond formed a thin layer of silicon carbide, leading to enhanced heat dissipation, although not as much as with silicon carbide alone.

Silicon carbide is expected be phonon matched to silicon, but there is no evidence that it is also matched to gallium nitride. The interface between diamond and silicon carbide displayed the lowest thermal boundary resistance, owing to the well-matched phonon modes of these materials, and the thermal boundary resistance at the interface between the silicon and silicon carbide was relatively low, too. But phonon matching at the interface between gallium nitride and silicon carbide is yet to be fully investigated. Engineering this interface could well reduce the 3.1 m^2 K GW^{-1} resistance observed for gallium nitride devices.

It is not yet clear whether the performance of these semiconductor devices will be affected by silicon carbide interface engineering, so the device properties must be carefully evaluated before the phonon bridges developed by Woo and colleagues can be deployed widely. Nevertheless, the team's strategy for maximizing heat dissipation in semiconductor transistors will help to realize the full potential of both silicon and gallium nitride technologies.

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1. Jia, X. *et al. Surf. Interf. Anal.* **51**, 783–790 (2019).
2. Cho, J., Won, Y., Francis, D., Mehdi, A. & Goodson, K. E. In *2014 IEEE Compound Semiconductor Integrated Circuit Symposium (CSICS)* https://doi.org/10.1109/CSICS.2014.6978583 (IEEE, 2014).
3. Woo, K. *et al.* In *2023 IEEE International Electron Devices Meeting* (IEDM) https://doi.org/10.1109/IEDM45741.2023.10413734 (IEDM, 2023).
4. Field, D. E. *et al. ACS Appl. Mater. Interf.* **12**, 54138–54145 (2020).
5. Smith, E. J. W. *et al. Carbon* **167**, 620–626 (2020).
6. Cho, J., Francis, D., Altman, D. H., Asheghi, M. & Goodson, K. E. *J. App. Phys.* **121**, 055105 (2017).
7. Malakoutian, M. *et al. ACS Appl. Mater. Interf.* **13**, 60553–60560 (2021).
8. Nochetto, H. C. *et al.* In *34th IEEE Compound Semiconductor Integrated Circuit Symposium (CSICS)* https://doi.org/10.1109/CSICS.2012.6340106 (2012).
9. Ejeckam, F. *et al. 2014 Lester Eastman Conference on High Performance Devices* https://doi.org/10.1109/LEC.2014.6951556 (2014).
10. Zhou, Y. *et al. ACS Appl. Mater. Interf.* **9**, 34416–34422 (2017).

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Immunology

Astrocyte cells in the brain have immune memory

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The central nervous system's astrocyte cells respond to injury and disease. The finding that they form molecular memories of certain responses, and that these modify inflammatory signalling, sheds light on autommunity. **See p.865**

A history of infection, traumatic injury or disease can influence the onset, progression or severity of central nervous system (CNS) disorders, such as autoimmune disease, stroke and neurodegenerative disease. How this occurs is poorly understood. An emerging potential mechanism is a form of intracellular molecular memory that is mediated by what are called epigenetic changes — modifications of proteins that control the accessibility of DNA for gene expression. Epigenetic immune memory is well characterized in cells of the 'innate' branch of the immune system after defence responses, and the process heightens subsequent defence responses[1]. On page 865, Lee *et al.*[2] reveal that a CNS cell called an astrocyte can acquire epigenetic immune memory that amplifies the cell's pro-inflammatory signalling in response to specific molecular stimuli and during autoimmune disease. The findings open doors to understanding and potentially ameliorating various CNS disorders.

Astrocytes are ubiquitous cells that are present throughout the entire CNS and engage in multiple activities that are essential for healthy CNS function[3]. These cells also respond to all forms of CNS injury and disease. The past few years have seen an explosion of interest in the role of astrocytes in neurological and behavioural disorders, and mounting evidence shows that astrocytes can exert beneficial or detrimental effects that powerfully influence the outcome of these disorders[3]. In this context, astrocytes are emerging as important tissue-resident responders that are directly involved in innate-immune defences and that regulate CNS inflammation in response to infection, injury and disease[4,5].

Vertebrate immunity has long been divided into innate and adaptive arms that comprise different sets of bone-marrow-derived immune cell. Innate immune cells respond rapidly to disease-causing agents and tissue damage through receptors that can recognize general hallmarks of trouble, such as proteins that are common to a range of viruses and bacteria. Adaptive immune cells respond more slowly by comparison and show classic immunological memory by boosting populations of immune cell that can recognize, in a highly specific way, a protein that might be unique to a particular bacterial species, for example.

It has emerged that innate immune cells can also demonstrate a form of cellular memory by undergoing long-term functional reprogramming through epigenetic modification of proteins that control DNA accessibility. This alters subsequent gene expression and functional responses[1,6]. There is a growing appreciation that in addition to bone-marrow-derived cells, there are tissue-resident innate immune cells that can also show such epigenetic immune memory[1,6]. Lee and colleagues' work demonstrates this capacity in CNS astrocytes and identifies underlying molecular mechanisms that could be targeted.

Lee and colleagues first asked whether exposure to an initial pro-inflammatory stimulus might alter the way that astrocytes respond to a second stimulus (a rechallenge) that is identical to the first stimulus. The authors exposed mouse brains *in vivo*, or mouse astrocytes in cell cultures *in vitro*, to the proteins IL-1β and TNF, which are molecules known as cytokines that stimulate pro-inflammatory responses. These particular cytokines were used because previous studies have implicated them in the autoimmune disease multiple sclerosis and a mouse system used to model multiple sclerosis, termed experimental autoimmune encephalomyelitis (EAE). Using RNA sequencing to assess gene expression, the authors found that rechallenge with a second stimulation of IL-1β and TNF caused significantly more potent astrocyte pro-inflammatory responses, which were reflected in both the number and expression levels of upregulated pro-inflammatory genes compared with a single stimulation alone (Fig. 1a). Notably, inflammatory gene expression returned to baseline before the second stimulation, indicating that the increased expression was not simply a cumulative additive effect, but was a true augmentation from baseline.

To examine any potential relevance to autoimmunity, the authors studied the mouse model system of EAE. Astrocytes grown *in vitro* from mice with EAE showed enhanced responses after a single stimulation with IL-1β and TNF compared with that of astrocytes derived from healthy mice. This resembled the response of cultures from healthy mice to a second cytokine challenge, suggesting that EAE exerts a priming effect that amplifies astrocyte pro-inflammatory signalling.

How might exposure to cytokines or autoimmune disease alter astrocyte responses on rechallenge? The authors explored whether