Silicon integrated circuits shine

David A. B. Miller

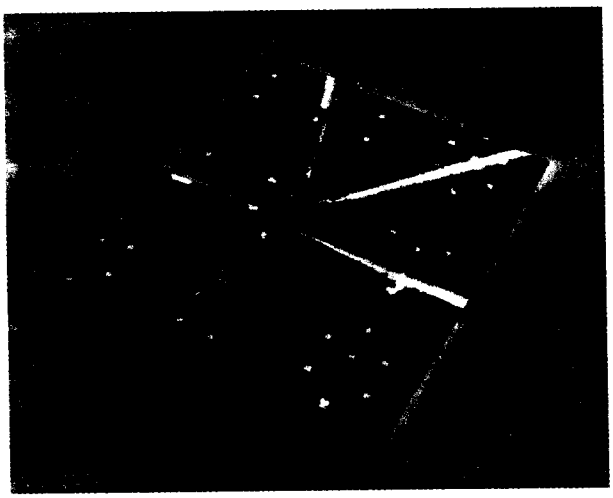
Mixing light-emitting diodes and ordinary electronics on the same silicon chip could create new possibilities for cheap, smart optoelectronics, and inspire new applications.

ACCORDING to Gordon Moore of Intel, we now make more transistors per year than raindrops fall in California, and it costs less to make one than to print a character on this page. The reason is the astonishing power of silicon electronic integration technology to manufacture sophisticated yet cheap integrated systems. But silicon has historically had one embarrassing failing — it cannot emit light. Recent work has raised hopes that there may be ways to get useful light from silicon, but until now there has been no cheap way to integrate such silicon-based light emitters with silicon electronics.

The paper on page 338 of this issue[1] by Hirschman *et al.* from the University of Rochester reports the successful integration of silicon-based light emitters into silicon electronics in a way that appears compatible with mass-manufacturing techniques — an important step in turning on the power of silicon integration technology for a broad range of new optical applications. In doing so, the group have addressed the 'mundane' issues of device lifetime and integration that can be so crucial in turning a scientific curiosity into a viable technology.

The very visible failing of silicon to emit useful light has been all the more galling because optoelectronics — the combination of optical and electronic functions — is becoming crucial in handling the information that ordinary silicon technology processes. Optoelectronic light emitters or modulators are essential for the visual display of changing information. Laser diodes read compact disks and drive laser printers. Optics already dominates in sending information over long distances. It is used in networks over shorter distances as demand for information rises, and may be used increasingly for interconnection within computers. And optical isolators are already used inside electronic apparatus to avoid voltage isolation problems.

There is little reason, however, to develop silicon light emitters merely as substitutes for existing individual devices.



An array of silicon displays. The chemical stability of oxidized porous silicon that allows integration with electronic components also allows many optical emitters to be created on a single silicon wafer. Here there are more than 150 seven-segment displays, each 2.5 by 4 mm. Reflection and emission images are superimposed to show electrical probes in contact with one display, and another one lit up.

The 'III–V' semiconductors, such as gallium arsenide, are naturally very good light emitters, and are the basis of a mature, inexpensive technology that is a high-volume business, with more than 30 billion devices manufactured each year. III–V light emitters are so efficient that they are starting to replace tungsten light bulbs, for example in car stop lights. But the benefits of silicon-based light emission probably lie not in competing with silicon's brilliant III–V cousins, but instead in cheap integration with silicon electronics.

The reason why silicon does not emit efficiently is very basic. Silicon is an 'indirect-gap' semiconductor. To conserve momentum, the silicon crystal has to vibrate to allow a photon to be emitted, a requirement that a 'direct-gap' semiconductor (such as gallium arsenide) does not have. A variety of valiant attempts to solve this problem have failed, though there are some promising approaches using quantum-confined microcrystals[2]. In 1990, however, Canham[3] set the field alight by observing that silicon could give out relatively bright, visible, photoluminescent emission when its surface was made porous through a simple hydrofluoric acid etch.

In photoluminescence, the material is excited using short-wavelength light, though the precise mechanism of the porous-silicon photoluminescence is still not entirely clear. Electrically exciting the emission, for example by incorporating the porous silicon in a diode and driving current through it, is also possible, and is generally more useful for devices. In electrically driven porous silicon, the mechanism of the emission may involve very highly excited electrons (or their positive equivalents in semiconductors, holes) forced into the tiny, porous silicon structures, generating photons in various possible ways as they lose energy in a kind of 'avalanche' process[4].

In the past year or so there have been encouraging improvements in the efficiency of such porous-silicon light emission[5]. Early attempts at electrically driven emitters, though encouraging, had several kinds of practical problems, including slow turn-off times (as long as milliseconds), and short useful lifetimes. Part of the lifetime problem may be that the surface of the porous silicon is not very stable chemically, with silicon–hydrogen bonds being easily broken, leading to 'dangling bond' surface states. These states are a common problem in many light-emitting devices because they often lead to undesirable, nonradiative recombination of electrons and holes.

Two separate approaches reported this year, one from a Belorussian–Italian group[4], and the second from Rochester[6], showed significant improvements in both useful lifetime and speed of response. The first group effectively encapsulated the porous silicon in aluminium and aluminium oxide, whereas the Rochester group oxidized the porous silicon surface. It may be that the emission from these oxidized porous silicon structures is from an entirely new mechanism, involving defects in the silicon oxide.

The latter oxidation approach is used by Hirschman and colleagues[1]. Porous sili-

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2. Lu, Z. H., Lockwood, D. J. & Baribeau, J.-M. *Nature* **378**, 258–260 (1995).
3. Canham, L. T. *Appl. Phys. Lett.* **57**, 1046–1048 (1990).
4. Lazarouk, S. *et al. Appl. Phys. Lett.* **68**, 1646–1648 (1996).
5. Loni, A., Simons, A. J., Cox, T. J., Calcott, T. D. J. & Canham, L. T. *Electron. Lett.* **31**, 1288–1289 (1995).
6. Tsybeskov, L., Duttagupta, S. P., Hirschman, K. D. & Fauchet, P. M. *Appl. Phys. Lett.* **68**, 2058–2060 (1996).
7. Krishnamoorthy, A. V. & Miller, D. A. B. *IEEE J. Sel. Top. Quantum Electron.* **2**, 55–76 (1996).

con becomes more chemically stable when oxidized, and the interface between silicon and silicon oxide is known to have very few of the undesirable surface states that can quench light emission. Silicon oxides are also compatible with the many processes used in silicon-circuit manufacture, and can probably withstand the subsequent processing steps needed for functional circuits, allowing the successful integration demonstrated by Hirschman *et al.*

One caution is that fields like these can become like the search for the Holy Grail. The search inspires, but it may not be clear exactly what to do, or what the actual benefit is, if the Grail is found. For some applications, such as densely packed optical interconnection to and from silicon electronics, even the very best light-emitting diodes are doubtful candidates because of the fundamental inefficiency of incoherent light emission. In this application, integrating III–V optoelectronic devices (such as high-performance modulators or lasers) with silicon circuitry may be more promising, and may incidentally allow interconnections within computers to keep up with the advance of silicon information processing[7].

Even after the demonstration from Hirschman *et al.*, there is still some basic technological work to be done — to integrate emitters with the dominant form of silicon electronics (CMOS), for example. But the thrust of the new work towards integration with silicon electronics is well chosen. The benefit of silicon light-emission will probably be in cost-effective, integrated systems, possibly of surprising kinds. Examples could be displays, integrated optoisolators, low-density optical interconnections, or possibly radical applications such as microscopic sensor systems with built-in illuminators.

Certainly, this field is exciting, is progressing fast, and is dispelling doubts that the initially encouraging discoveries might turn to disappointment. There is increasing hope that the light at the end of the silicon tunnel is not an oncoming train. □

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MUSCULAR DYSTROPHY

Utrophin to the rescue

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DUCHENNE muscular dystrophy (DMD) is a fatal genetic disease caused by the absence of dystrophin in muscle. The disease is characterized by the progressive loss of muscle strength, and patients usually die by their early twenties of respiratory or cardiac failure. Although great progress has been made in understanding the molecular genetics of DMD, no effective treatment for this devastating disease has yet been developed. Because dystrophin is a structural muscle protein, therapies for DMD will probably involve the replacement of dystrophin or the upregulation of a functionally related protein, such as utrophin. The paper by Kay Davies and colleagues on page 349 of this issue[1] shows for the first time that utrophin can effectively rescue dystrophin-deficient muscle *in vivo*, providing strong support for a therapeutic strategy to fight DMD involving the upregulation of utrophin.

Dystrophin is tightly associated with a large oligomeric complex of membrane glycoproteins that are collectively referred to as the dystrophin–glycoprotein complex (DGC; refs 2, 3). The DGC spans the sarcolemma of skeletal and cardiac muscle, and biochemical and molecular studies have shown that it provides an important structural link between the actin cytoskeleton and the extracellular matrix[2] (see Fig. 1). The involvement of this cytoskeleton–extracellular matrix connection in muscle physiology is not fully understood; however, it probably stabilizes the sarcolemma, thereby protecting it from stresses that develop during muscle contraction[4,5].

The absence of dystrophin in DMD patients results in several characteristic pathological features, including muscle-cell necrosis and regeneration[6], and elevated serum levels of muscle creatine kinase. At advanced stages of the disease, muscle is eventually replaced by fat and connective tissue. On the molecular level, the lack of dystrophin leads to a dramatic reduction in the levels of all of the other DGC components[2], and loss of the DGC breaks the transmembrane linkage and weakens the muscle-cell membrane. Elegant studies from the laboratory of Jeff Chamberlain[7,8] have shown that in the *mdx* mouse — a dystrophin-deficient animal model for DMD — restoration of the DGC by transgenic expression of dystrophin is necessary to rescue normal muscle physiology. In particular, the actin-binding and β-dystroglycan-binding domains are required[8]. These findings have been corroborated by the adenoviral expression of dystrophin and internally deleted forms of dystrophin (or minigenes) in *mdx* mice.

Utrophin is structurally similar to dystrophin[9], and it is mainly expressed at the neuromuscular junction in adult skeletal muscle (Fig. 1), although it is also found at the sarcolemma in fetal and regenerating muscle and, at very low levels, in DMD muscle[9]. Molecular studies of muscle from *mdx* mice have shown that utrophin associates with sarcolemmal glycoproteins to form a complex that is similar to the DGC (ref. 10), suggesting that utrophin can replace dystrophin at the sarcolemma in dystrophin-deficient muscle.

By expressing high levels of utrophin in *mdx* mice, Tinsley *et al.*[1] now demonstrate that utrophin can functionally replace dystrophin (Fig. 2). They show that overexpression of utrophin leads to the restoration of all of the components of the DGC, including the dystroglycan and sarcoglycan sub-complexes. Furthermore, serum creatine-kinase levels and muscle pathology also seem to be corrected, indicating that the restored complex is functional. Overexpression of utrophin even rescues the deterioration of the diaphragm, which is the most severely affected muscle group in the *mdx* mouse[11].

The results from the Davies group[1] provide the impetus for an exciting new avenue in DMD-therapy research — the identification of small molecules that increase the expression of utrophin in dystrophin-deficient skeletal muscle. These could be used to upregulate utrophin expression in all

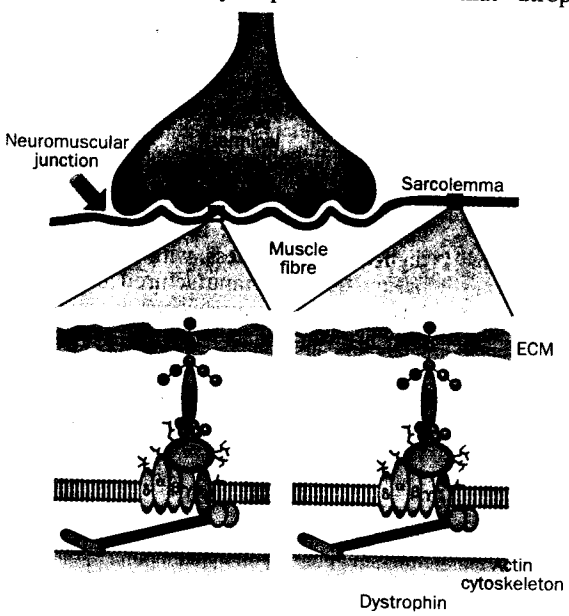

FIG. 1 Localization of dystrophin and utrophin in the muscle cell. The nerve terminal contacts the sarcolemma, creating the neuromuscular junction: expression of utrophin is confined to this region, whereas dystrophin is found throughout the sarcolemma. Both proteins are associated with a glycoprotein complex that binds to the extracellular matrix (ECM).