3 All equipment including refrigeration and air-conditioning units are individually connected
to a solid-state, over/under-voltage cut-out with a user-definable time delay of up to 10 min. The
ability to individually pre-set the turn-on delay prevents simultaneous activation of all equipment
when the power is restored. This prevents over-loading of the power line. In addition, refrigeration
and air-conditioning equipment requires a minimum turn-on delay of 3 min to allow the refrigerant
to stabilize, thus avoiding damage to the compressor. Visual indication of tripping is provided on
each of the units. The prototype, built by one of us, has been functioning satisfactorily for the past 6
years. All the post-prototype units were assembled in the centre by Mr S Charles, a member of the
staff, who is self-taught electronics enthusiast. The electronic components used are available locally.

4 The mains supply is monitored by the duty electrician and in the event of a black-out or
prolonged brown-out, generators are activated manually. Automatic start-up systems, though
available, were not incorporated since there is no requirement for emergency power.

Anyone interested in detailed information about the above-mentioned devices is welcome to
correspond with us.

Schieffelin Leprosy Research &
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J DEVASUNDARAM & A CARIAPPA

VISUAL AIDS; LABELS AND DIAGRAMS TO AID COMPLIANCE

Sir,

I was interested to read in the December 1986 issue of Leprosy Review (page 373) under 'Leprosy
Control and Field Work', about the use of labels and diagrams to aid compliance, from Dr D J
Morton and colleagues in the University of Zimbabwe, Africa. This reminded me of our experiences
in 1984 in Cape Verde, in which I participated in a study of educational drawings for oral
rehydration and infant diarrhoea. The drawings were produced locally and used in rural areas for
display in shops, health posts and schools. They were in colour and depicted a morning, noon and
night sequence, together with a resumé of 'materials' needed for oral rehydration. After some
months we found that in the 'target group' (mothers of children under the age of 5 years), 9 out of 10
could not recognize the rising and noon-day sun, and a cup in our diagram was recognized correctly
by only 50% of those interviewed.

My impression was that the drawn abstractions we used were even more difficult to understand
than written information. Drawings and diagrams for use with people of limited education demand
considerable experience of trans-cultural factors. We should keep in mind that labels, diagrams and
pictures have not universally reached the immediate 'acceptance level' which is second nature to
people in the so-called developed countries.

Gabinete de Coorenacao de Saude Publica,
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A LORETTI

THE COLOUR INDEX (CI) AND HISTOLOGICAL STAINS

Sir,

The largest single user of dyes is the textile industry, where dyers may classify them according to
their mode of action, i.e. mordant or direct, their general chemical character, i.e. acid or basic, or
their colour. Unfortunately many dyes have one or more synonyms, e.g. brilliant crystal scarlet 6R
is also known as crystal ponceau 6R, naphthalene scarlet 6R and ponceau 6R. For this reason a uniform nomenclature for dyes is particularly important if consistent results are to be obtained.

The response of the industrial dyers to this rich source of error was to set up a Colour Index (CI), whereby each chemical structure is given a unique number and a unique generic name, i.e. not a trade name. This Colour Index, first introduced by the Society of Dyers and Colourists of Bradford, England (1924)\(^1\) is now compiled as a 6-volume reference source in association with the American Association of Textile Chemists and Colorists (1971–75).\(^2\)

The result of this is that dyes which are common to both histology and the textile industry can be accurately identified by their Colour Index number (CI number), a selection of which are detailed in Table 1. However, it can be seen from Table 1 that the CI names appear slightly obscure and it is likely (and even preferable) that the classical names will continue to be used in the histology laboratory for many years to come. Nevertheless, it is useful for the laboratory scientist to know of their existence. It should be borne in mind that dyes that are mixtures of chemicals (such as carbol fuchsin) will not have a CI number.

### Table 1. Colour Index of some common dyes

<table>
<thead>
<tr>
<th>CI number</th>
<th>CI name</th>
<th>Classical name</th>
</tr>
</thead>
<tbody>
<tr>
<td>41000</td>
<td>Basic yellow 2</td>
<td>Auramine</td>
</tr>
<tr>
<td>42510</td>
<td>Basic violet 14</td>
<td>Basic fuchsin</td>
</tr>
<tr>
<td>22120</td>
<td>Direct red 28</td>
<td>Congo red</td>
</tr>
<tr>
<td>45380</td>
<td>Acid red 87</td>
<td>Eosin Y</td>
</tr>
<tr>
<td>75290</td>
<td>Natural black 1</td>
<td>Haematoxylin</td>
</tr>
<tr>
<td>42095</td>
<td>Acid green 5</td>
<td>Light green</td>
</tr>
<tr>
<td>52015</td>
<td>Basic blue 9</td>
<td>Methylene blue</td>
</tr>
<tr>
<td>50040</td>
<td>Basic red 5</td>
<td>Neutral red</td>
</tr>
<tr>
<td>42520</td>
<td>Basic violet 2</td>
<td>New fuchsin</td>
</tr>
<tr>
<td>26125</td>
<td>Solvent red 27</td>
<td>Oil red O</td>
</tr>
<tr>
<td>16230</td>
<td>Acid orange 10</td>
<td>Orange G</td>
</tr>
<tr>
<td>10305</td>
<td>Trinitrophenol</td>
<td>Picric acid</td>
</tr>
<tr>
<td>45170</td>
<td>Basic violet 10</td>
<td>Rhodamine B</td>
</tr>
<tr>
<td>75100</td>
<td>Natural yellow 6</td>
<td>Saffron</td>
</tr>
<tr>
<td>26150</td>
<td>Solvent black 3</td>
<td>Sudan black B</td>
</tr>
<tr>
<td>52040</td>
<td>Basic blue 17</td>
<td>Toluidine blue</td>
</tr>
</tbody>
</table>

A further standardization of dyes used in the histology laboratory has been carried out by the American Biological Stain Commission.\(^3\) In addition, as new histological stains are made they are certified by the Commission and listed in their journal, *Stain Technology*.

Despite the better understanding of chemical composition and the more rational identification of dyes, variation in staining characteristics from one batch to another, and from one manufacturer to another, does occur. For this reason it is recommended that if there is any doubt, new batches of dye should be tested with known control material.

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R L Jones
OCCURRENCE OF LEPROSY IN MANY GENERATIONS OF THE SAME FAMILY

Sir,

It was interesting to read the articles, 'The occurrence of leprosy in an eight-member family—a case report' (Lepr Rev 1984, 55, 47–50) and subsequently 'Occurrence of leprosy in many members of the same family' (Lepr Rev 1985, 56, 80–1).

We had the opportunity to see a family with leprosy in 3 generations. The family was living in the eastern part of Libya. Out of 10 members involved, only 2 were known cases and those were registered with the Leprosy Clinic, Benghazi, Libya. The rest were discovered on examination of family contacts. The index case, the grandmother, aged 75 years, was a known case of lepromatous leprosy. No record existed for her husband who had died many years previously. She had 2 sons and 3 daughters. All the 3 daughters, aged 35, 42 and 45 years respectively, were found to have borderline leprosy. Both sons were unaffected.

The 1st daughter had 11 children. Her husband was free but 2 of her sons had leprosy. The elder son aged 30 years had BT leprosy and was a known case registered with our clinic. The other son aged 15 years had BL leprosy. Her other 4 sons and 4 daughters were unaffected.

The 2nd daughter had 4 sons. They were all free. Her husband was also free of leprosy.

The 3rd daughter had 4 sons and 1 daughter. The daughter aged 16 years was found to have borderline leprosy. Her husband was also found to have active BL leprosy. Incidentally her husband had a 2nd wife who was unaffected and so were her children.

The index case (grandmother) was living with her eldest son who did not have leprosy. He had 6 daughters and 3 sons. Two daughters aged 25 years and 16 years respectively were found to have borderline leprosy.

The family of the 2nd son could not be examined.

All the newly detected cases were registered and put on multiple drug therapy.

In this family, leprosy was thus seen in 3 generations. Surprisingly, none of the newly detected cases complained about the disease. All were detected by active clinical examination. The occurrence of leprosy in this particular family leads one to speculate that heredity plays an important role in leprosy, although close contact must also be recognized as a factor in transmission. Another interesting observation was that only 2 males (if we exclude the husband of the 3rd daughter) were involved as compared to 7 females.

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M SINGH, F H SHAHAT, M S BELHAJ
& E A MANGOUSH

THE CLASSIFICATION OF LEPROSY, A STATE OF CONFUSION

Sir,

In 1960, the 2nd WHO Expert Committee on Leprosy (TRS 189) agreed '... that radical changes in the classification from congress to congress should be avoided since such action would lead to utter confusion, neutralizing all efforts to arrive at the universal use of the same