NaOH Etch of Fission Tracks in a Soda-Lime-Silica Glass

It is generally known that fission tracks intersecting the surface of man-made soda–lime–silica glasses can be etched with HF at room temperature[1,2]. In this technique the acid attacks the tracks preferentially so that they become visible in the microscope before the glass itself is destroyed. An NaOH etch at 60° C has been reported for phosphate glasses[3].

I wish to report a glass which I believe to be a soda–lime–silica glass that is etched by NaOH rather than HF. The material consists of numerous glass beads of a recognisable type known as 'trade wind beads' found in archaeological sites of the past millennium in eastern and southern Africa. They are consistently similar in appearance and chemical composition. Their composition is partly reported in Table 1 and abundant further information is available elsewhere[4].

Table 1 Partial Composition of 'Trade Wind Beads'

Element	Concentration	Method	No. of samples
Na	15.0±1.5%	NAA	133
Ca	4.4±1.9%	NAA	133
K	2.3±1.4%	NAA	133
Pb	~0.01–7.0%	XRF	~200
P	~0.1%	ES	2
U	112±55 p.p.m.	NAA	133
(SiO_2	~56%	XRF	1)

NAA, neutron activation analysis; XRF, X-ray fluorescence analysis; ES, emission spectroscopic analysis.

The beads are thought to contain fossil fission tracks useful for dating. In an attempt to etch such tracks, experiments were carried out on artificially produced fission tracks in 'trade wind beads'. HF at various concentrations at room temperature attacked the glass without etching the tracks preferentially. Successful etching was accomplished by refluxing for 7 min in NaOH. The reflux solution was made by dissolving 12 g NaOH in 16 g water.

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1 Fleischer, R. L., Price, P. B., and Walker, R. M., *A. Rev. Nucl. Sci.*, **15**, 1 (1965).
2 Fleischer, R. L., Price, P. B., and Walker, R. M., *Science, N.Y.*, **149**, 383 (1965).
3 Becker, K., *Health Phys.*, **12**, 769 (1966).
4 Davison, Claire C., thesis, Univ. California (Lawrence Berkeley Laboratory Report No. LBL 1240, 1972).

BIOLOGICAL SCIENCES

Proximal Myopathy induced by 5-HT-Imipramine simulates Duchenne Dystrophy

It has recently been observed that patients with Duchenne muscular dystrophy (DMD) have a significant defect in the uptake of serotonin (5-HT) into platelets[1], raising the possibility that a defect in the metabolism of biogenic amines may be important in the pathogenesis of the skeletal muscle lesions of this disorder. If monoamines such as 5-HT or norepinephrine (NE) which are dependent on the intracellular enzyme, monoamine oxidase (MAO), have a diminished ability to reach this site of metabolism, increased tissue levels of these compounds could result and produce skeletal muscle damage. The effect of such a defect would be enhanced in skeletal muscle as catechol-O-methyltransferase, the alternative pathway for metabolism of biogenic amines, is absent from this tissue[2]. Support for this hypothesis of skeletal muscle damage is found in the observation that skeletal muscle from biopsies of Duchenne dystrophy patients has shown the presence of an abnormal intrafibrillar fluorescence (Falck-Hillarp technique) presumed to be a monoamine-like substance[3]. Furthermore, injection of the MAO inhibitor, pargyline, results in a myopathy with evidence for accumulation of monoamines[4]. The pargyline induced myopathy, however, has serious limitations as a model for Duchenne dystrophy, as the lesions are limited to the soleus muscle and, furthermore, MAO activity has been found to be normal in patients with DMD (ref. 1).

Here we report the findings of an experimental myopathy using 5-HT in combination with imipramine, a pharmacological agent blocking uptake of biogenic amines into platelets[5] and thereby producing a defect in biogenic amine metabolism closely simulating that observed in patients with DMD. The results of this study are extremely important in two ways. First, the very typical early and mid-stage histological lesions of DMD were reproduced and, second, this study represents the first demonstration of an experimental animal model with skeletal muscle lesions predominating in the proximal musculature, as is so characteristic of the clinical disease.

Fifty male Osborne-Mendel and Sprague-Dawley rats weighing 135 to 150 g were used. Fresh frozen sections of soleus, gastrocnemius, anterior tibialis, extensor digitorum longus, quadriceps, iliopsoas and deltoid muscles were stained by modified trichrome, haematoxylin and eosin, reduced nicotinamide dinucleotide-tetrazolium reductase and myofibrillar adenosine triphosphatase[6]. At the beginning of the study all rats were injected intraperitoneally with imipramine, 10 mg kg^{-1}, for 3 d. On the fourth day the animals received 5-HT, i.p., 100 mg kg^{-1}. Each week two rats were killed 4 d after the injection of 5-HT, the remaining rats being injected with the same dosage schedule for a total of 11 weeks.

Skeletal muscle lesions were seen in all rats examined and increased in severity as the number of injections in each rat increased. The distribution of the histological lesions in these animals was quite striking as the lesions were very severe in the quadriceps, moderately severe in iliopsoas and occasionally present in the soleus. In the gastrocnemius, anterior tibialis and extensor digitorum longus, only rare skeletal muscle pathology was encountered. The earliest lesions consisted of necrosis and phagocytosis and regeneration of individual muscle fibres or small groups of muscle fibres surrounded by a field of normal muscle (Figs 1 and 2). This is identical to the early lesions of DMD (ref. 7). In animals receiving multiple injections, the lesions showed signs of chronicity manifested by a marked variability in fibre size, internal sarcolemmal nuclei and proliferation of connective tissue surrounding individual muscle fibres (Fig. 3). This is characteristic of the mid-stage histological lesions of DMD (ref. 7). No abnormalities were seen in the intramuscular nerves and the intramuscular arteries showed no signs of occlusion, although, rarely, they were surrounded by a small round cell infiltrate, similar to that described in DMD (ref. 8).

Control animals included those injected i.p. with imipramine 10 mg kg^{-1} as above but without follow-up injections of 5-HT. Another group of animals was injected i.p. daily for 7 d with imiptamine, 25 and 50 mg kg^{-1}. A third group of controls included those injected with single