touch only the fringe of the ideas involved in the extensive investigation of the authors mentioned. Nevertheless, the present brief communication will serve a useful purpose if it directs attention to an important investigation which throws much valuable light on the difficult problem of the quantitative correlation of linked physico-chemical processes in a living system.

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[1] *J. Physiol.*, **100**, 1 (1941).

Role of Potassium in Yeast

IN our previous communication[1] it was described how ammonia could entirely replace potassium in yeast under suitable conditions. It became then a question of interest as to how the 'ammonia yeast' functioned compared with the normal or 'potassium yeast' when both were maintained under strictly similar conditions, except that the ammonium ion replaced potassium. The rate of fermentation of glucose, the reproduction in suitable media and the resting metabolism were examined.

Preparation of the yeast for examination. Two 5-gm. samples of the same baker's pressed yeast were taken, one immersed in Ringer-Barkan fluid (as described in ref. 1) containing $N/5$ NH_4Cl and no potassium, and the other in a similar solution, K being replaced by NH_4 ions. The mixture was bubbled with .3 per cent carbon dioxide and 97 per cent oxygen, the medium being changed every twenty-four hours. After four days there was no measurable amount of potassium left.

The 'potassium yeast' after centrifuging contained 205 m.eq. K/kilo moist yeast and no appreciable amount of ammonia, and the 'ammonia yeast' contained 248 m.eqs./NH_3–N and no potassium.

Fermentation. 1-gm. samples of the centrifuged yeasts were suspended in 10 c.c. of water and allowed to act on an equal volume of 6 per cent glucose. It was found that the mean rate of carbon dioxide production by the 'ammonia yeast' was 40 per cent of that for the 'potassium yeast'—this latter being 0·26 m.eq./hr./c.c. yeast suspension.

Growth and reproduction. Small platinum inoculations were made into 100 c.c. of sterile medium containing 2·5 gm. glucose, 0·5 gm. K (or NH_4) acid phosphate, 0·1 gm. $MgSO_4$, and 20 ml. of boiled 1/5 water extract of 'potassium (or ammonia) yeast'. A curious difference appears between the two yeasts. The 'potassium yeast' grows much faster at first, but reaches an upper limit after twenty-four hours. The 'ammonia yeast' is considerably slower at first, but after two to three days exceeds the 'potassium yeast' and passes on to a far higher level (about four times the number of cells). The early upper level of the 'potassium yeast' is not due to an exhaustion of the glucose by fermentation, as shown by the use of higher glucose concentrations leaving much unfermented sugar after twenty-four hours, but no appreciable change in the number of yeast cells. It appears to be related to an exhaustion of some substance in the yeast extract added.

Resting metabolism. The oxygen uptake at 30° C. was examined in the Warburg apparatus, 3 ml. being taken of a 1/100 suspension in a medium consisting of 0·095 gm. Na_2HPO_4, 0·080 gm. NaH_2PO_4 and 0·60 gm. NaCl made up to 100 ml. (pH = 6·7). The Qo_2 for 'potassium yeast' was found to be −5·35 and that for the 'ammonia yeast' was −6·36. Thus the resting metabolism of the 'ammonia yeast' is *higher* than that of the 'potassium yeast'.

Experiments such as described in the previous letter and in other communications[2,3] show that potassium exists in cells in the ionized form, and it would appear that the main biological reason for the accumulation of potassium in cells (at least those with distensile membranes) is a necessary process for the accumulation of appreciable non-diffusible material or such as the cell can retain[4]. At the same time, the potassium ion even in one-celled organisms, such as yeast, may exert some specific ionic effects, though the above experiments indicate only a difference between the ammonium and potassium ion without it being possible to say which, if any, plays a merely passive role. They show, none the less, that potassium (at least over 0·1–1·0 mgm./100 gm.) is not essential for fermentation, growth or resting metabolism of the living yeast cell.

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Oct. 3.

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Nature of the Disturbed Calcium Metabolism in Thyrotoxicosis and Myxœdema

VARIOUS theories have been put forward to explain the excessive calcium output in thyrotoxicosis including an increased metabolism *per se*[1], neutralization of acid products[2], direct stimulating catabolic action of thyroxin on bone, and a co-existing hyperparathyroidism[3]. For reasons to be given later, all these theories are unsatisfactory. As a result of direct experiments and observations[4] on normal subjects and cases of thyrotoxicosis, myxœdema and parathyroid tetany, a new theory has been formulated. It is believed that in thyrotoxicosis, an excessive secretion of thyroxin acts directly on the kidneys, stimulating them to increase their output of calcium. This may be achieved either as a result of the increased metabolism *per se* or by lowering the renal threshold for calcium.

As Aub *et al.*[1] have shown that an increased metabolism *per se* does not increase the calcium output, the excessive calcium loss would appear to be due to a lowering of the renal threshold for calcium. This leads to a fall in the serum calcium, and as a result there is an increased mobilization of calcium from the bones. In other words, the decalcification in thyrotoxicosis is due to a *vis à fronte*. In myxœdema there is the converse picture, where a diminished thyroxin secretion raises the renal threshold for calcium and causes the calcium output to fall.

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[4] Robertson, J. D., *Lancet*, **1**, 97, 129, 156, 216 (1941).