1967), which has subsequently been shown to reflect a loss of uronic acid from the matrix.

The tumour only exerts its degradative effect when it comes into contact with the cartilage. Often matrix degradation was recorded in the absence of any detectable damage to chondrocytes. Little or no degradation was noted when fibroblasts, constituting the pannus, were interposed between the tumour and the cartilage. Matrix degradation was also found to be associated with the presence of 'haloes' of staining around the chondrocytes, as observed in man by Sylvén (1947), indicating that the matrix is more concentrated around the chondrocytes.

Administration of either vitamin A or papain *in vivo* caused a similar degradation of the matrix, but only with papain treatment were 'haloes' commonly observed. Vitamin A has been shown to degrade cartilage by releasing acid protease and probably other lysosomal enzymes from chondrocytes (Fell & Dingle, 1963); papain has been shown to be capable of degrading the matrix directly, in a reversible manner (Potter, McCluskey, Weissmann & Thomas, 1960). β -Glucuronidase was demonstrated histochemically within the partly degraded matrix close to the edge of the tumour. This suggested the presence of other acid hydrolases. These results together suggest that the degradation of the matrix in the presence of the tumour is mediated mainly by lysosomal enzymes originating not from the chondrocytes but from a remote source such as the tumour.

A purified lysosomal preparation isolated from the tumour by differential centrifugation followed by density-gradient centrifugation with a glycogen-sucrose gradient was checked for purity, biochemically and by electron microscopy. At pH 5.0 *in vitro* this preparation caused degradation of xiphoid cartilage matrix that was enzymic in nature, in addition to that produced by enzymes released from the chondrocytes.

These findings suggest that the ability of the tumour to degrade the matrix may be mediated by the extracellular release and activity of lysosomal enzymes derived from the tumour.

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The Inhibition of Wheat β -Amylase

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β -Amylases were prepared from native-grown wheats, e.g. Atle Quern and Progress varieties, by a modification of existing methods (Ballou & Luck, 1941; Tipples & Tkachuk, 1965). All preparations contained three components, as shown by gel electrophoresis, a feature previously noted in the β -amylases of Canadian hard red spring wheats (Tipples & Tkachuk, 1965). The action of β -amylase on the starch extracted from the same wheat variety was followed. The effect of special thiol reagents, mercuric chloride and phenylmercuric chloride (PMC), on enzyme activity was also studied. The inhibition of β -amylase by PMC was affected by substrate concentration, and a competition between substrate and PMC for the enzyme was observed.

In the routine estimation of barley α -amylase in the presence of β -amylase (Bendelow, 1963) a saturated solution of PMC is usually added to the crude enzyme preparation to inhibit the β -amylase. This mixture is then added to a starch solution for the final enzyme assay. The validity of this sequence of steps will be discussed and an alternative procedure for the selective assay of α -amylase in the presence of β -amylase will be described.

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The Purification and Properties of Some Glycoside Hydrolases

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Enzymes are normally purified by a combination of the traditional techniques such as salt precipitation, ion-exchange and molecular-sieve chromatography. Several attempts have been made to adsorb an enzyme on to its substrate and these have met with limited success. Clarke & Stone (1965) removed a contaminating β -(1 $\rightarrow$ 3)-glucanase from a β -(1 $\rightarrow$ 4)-glucanase by adsorption of the former enzyme on to insoluble laminarin, and Halliwell (1961) adsorbed cellulase on to cellulose.

The chitinase from a *Streptomyces* sp. was found to bind strongly to chitin and recovery of the enzyme was effected by prolonged incubation

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