i.e., S. sudanense, is changed like the paternal parent (Fig. 1).

Xenia was expressed only when *S. sudanense* was used as a female parent. From the observations (Fig. 2), it is clear that the seed characteristics of *S. bicolor* and *S. caffrorum* are dominant over those of *S. sudanense*. Xenia is expressed because of simple dominance of the male parent for the seed characters. But, Xenia was not expressed for all the characteristics in every cross. The only cross in which xenia was expressed for all the characteristics studied was *S. sudanense* × 148. However seed colour appears to have maternal effect.

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ROLE OF pH IN THE FRUIT ROT OF CHILLIES CAUSED BY *COLLETOTRICHUM CAPSICI*

THE pectic enzymes produced by fungal pathogens have sharp peaks of activity at certain pH values, so that the initial pH of a tissue and the change in pH caused by the pathogen may be significant[1]. The significance of the rise in pH during disease development has been emphasised by several workers. Hancock[2] suggested that processes that induce changes in pH during pathogenesis could contribute significantly to disease development if the activities of certain degradative enzymes were favourable.

Extracts obtained from the diseased tissue were tested for their macerating activity on chilli fruit discs over a pH-range of 3·5–9·5. Time taken for the loss of coherence of cells was noticed. Desired pH values were maintained by using the 3 buffers suggested by Tribe[4] for maintaining uniform concentration of the buffering salts.

As shown in Table I the maceration was rapid at pH 9·0 and above. At any pH, red chilli discs were more readily macerated than the green ones. Data at pH 5·5 are critical since these are the approximate pH values of healthy fruits. At pH 5·5 green chilli discs were not macerated while the red ones were. This is what is perhaps happening *in vivo* when the fruits are infected by the fungus. In nature, the disease due to *C. capsici* is common only in red ripe chillies. In the case of green chilli fruits maceration will not be brought about when they are attacked by the fungus. On the other hand, the red ones are macerated by the enzyme produced by the invading fungus.

A change in pH favours the predominating enzyme action[2]. Since the pH of the infected chilli tissue rose to 8·3 during lesion development, it would appear that only the *trans*-eliminative enzymes were operative in bringing about the rotting. Similar report was made in the case of stem rot of squashes[5]. Thus pH appears to be a key factor to explain why green chillies do not get the *Colletotrichum*-rot while the red ripe ones get.

The initial pH of the chilli fruits, which is around 5·0, would be favourable only for exo- or endo-polygalacturonase (PG) activity, if there was any. *C. capsici* produced moderately active exo- and endo-PG *in vitro*, but these were not detectable in the infected fruits.

It should however be mentioned here that in some instances the green fruits turn red at the site of infection apparently due to the ripening processes setting in locally. In such cases the green fruits also become susceptible and the disease spreads progressively but slowly.

A (non-dialyzable and thermostable) 'Killing factor' is also produced by the fungus *C. capsici* in culture[6]. Its activity is rapid below pH 5·5. This

TABLE I

Effect of pH on the time for maceration of green and red chilli fruit discs by diseased tissue extract

Fruit tissue discs	pH of the diseased tissue extract						
	3·5	4·5	5·5	6·5	7·5	8·5	9·0 and above
Green	'No maceration	(No maceration)		12 h	8 h	6 h	5 h
Red		24 h	8 h	7 h	5½ h	4 h	3½ h

The fungus *Colletotrichum capsici* causing fruit-rot disease in chillies (*Capsicum annuum* L.) produces an actively macerating endo-polygalacturonate *trans*-eliminase (PGTE) *in vivo*[3]. Its optimum activity was near pH 9·1. Other pectic enzymes could not be detected.

non-enzymic factor may also be playing some role in rapidly killing the host tissue especially before the pH drifts to alkalinity. In other words, the 'Killing factor might be playing a role in the initial stages (pH is around 5·0) while the endo-PGTE could assume importance both at the initial stages and to an increased extent at subsequent invasion by the fungus (when the pH increases to 8·5).

The authors are grateful to Prof. T. S. Sadasivan and Prof. C. V. Subramanian for encouragement and facilities.

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PHENOLIC ACIDS OF VIABLE AND NON-VIABLE SEEDS OF GROUNDNUT (*ARACHIS HYPOGAEA*, L.)

THE various factors involving the loss of seed viability were discussed by Roberts[1] and various physiological and biochemical changes associated with seed deterioration were reviewed by Abdul Baki and Anderson[2]. Dey *et al.*[3] reported that there was an accumulation of certain phenolic acids like coumarin, ferulic and sinapic acids in the non-viable seeds of rice. The present paper reports investigation on qualitative and quantitative aspects of phenolic acids in viable and non-viable goundnut seeds.

Four months old (viable) and three year old (non-viable) seeds of groundnut variety TMV-3 were used for qualitative and quantitative determination of individual phenolic acids. The viability of seeds was tested with tetrazolium chloride and by the normal germination test.

Phenolic acids were extracted from both viable and non-viable seeds by the method of Bate-Smith[4]. Two dimensional paper chromatography was carried out to detect the individual phenolic acids as per the technique of Ibrahim and Towers[5]. The individual phenolic acids from the paper were eluted with 95% ethanol and co-chromatographed with authentic samples. The phenolic acid content was estimated with Folin's reagent.

Six monohydroxy phenolic acids (*a*-resorcylic, *p*-hydroxybenzoic, *trans-p*-coumaric, *cis-p*-coumaric, phloretic and vanillic acids) and two dihydroxy phenolic acids (protocatechuic and chlorogenic acids) were present in the viable seeds. In non-viable seeds the number of monohydroxy and dihydroxy phenolic acids remains the same as viable seeds except protocatechic acid. However, more amounts of monohydroxy phenolic acids were present in the viable seeds when compared to non-viable seeds. The content of dihydroxy phenolic acids decreased to half of their amounts in the non-viable seeds. Thus, the total disappearance of photocatechuic acid and decreased levels of all the individual phenolic acids in the non-viable seeds may be suggestive of their primary role similar to essential metabolite levels like carbohydrates, fats and proteins which were shown to decrease in non-viable seeds when compared to viable seeds[1]. It has been shown that large amounts of inhibitors like abscisic acid accumulate in non-viable seeds of groundnut[6]. Therefore, it is quite probable that the phenolic inhibitors do not play an active role in the loss of viability of groundnut seeds.

TABLE I

Phenolic acids of viable and non-viable groundnut seeds µg/gm dry seed

		viable	non-viable
1.	Chlorogenic acid	44·55	20·20
2.	Protocatechuc acid	9·27	..
3.	*Trans-p*-coumaric acid	7·69	2·36
4.	*p*-Hydroxy benzoic acid	12·15	3·23
5.	*a*-Resorcylic acid	16·20	4·01
6.	*cis-p*-coumaric acid	10·07	4·81
7.	Phloretic acid	6·68	1·80
8.	Vanillic acid	5·47	4·61
	Total	110·08	41·02

The authors thank Professor V. S. Rama Das, for providing facilities.

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