Polyploid Wheats and Fraction 1 Protein

With respect to the maternally inherited large subunit of fraction 1 protein, Chen *et al.* (*1*) found that *Triticum boeoticum* and *T. urartu* had identical polypeptide patterns, and *Aegilops speltoides* had a different one identical with that of the tetraploid wheats. From this evidence they inferred that *Ae. speltoides*, but neither *T. boeoticum* nor *T. urartu*, could have been the maternal parent of the tetraploids. The evidence was obtained from a single accession (the progeny of a single plant) per species, and rests on the tacit assumption that there is no variation within species with reference to the large subunit pattern. In support of that assumption Chen *et al.* refer to data (*2*) showing no variation in the large subunit pattern among six accessions of *Nicotiana glauca* and nine of *N. tabacum*. Nevertheless, the basic assumption is questionable. It requires that large subunit mutations presumed to distinguish one species from another within a genus (in this case *Ae. squarrosa* from *Ae. speltoides*) cannot occur within a given species. One accession of *Ae. speltoides* suffices to show that it could have been the maternal parent of the tetraploids, but one accession of *T. boeoticum* is inadequate to show that it could not have been the maternal parent.

Our evidence from 742 reciprocal crosses (*3*) shows that if *T. boeoticum* and *T. urartu* are, in fact, the parents of the tetraploids (*4*), then *T. boeoticum* must be the maternal parent. *Triticum urartu* carries a cytoplasmic lethal factor which results in nonviable seed when it is used as the maternal parent in crosses with *T. boeoticum* or the tetraploid species.

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We share Johnson's concern over the possibility of intraspecies variation in the polypeptide pattern of the large subunit of fraction 1 protein, with respect both to wheat species and to species in the genus *Nicotiana*. However, our analyses of fraction 1 proteins from *Nicotiana* species have completely failed to uncover any evidence of such variation. Since the previous report on *Nicotiana* species (*1*), we have examined a further 14 cultivars of *N. tabacum*, obtained from breeding programs around the world, and ten individual plants from collections of *N. suaveolens*, perhaps the most variable morphologically of all the *Nicotiana* species, and these analyses show the presence of a single type of large subunit pattern for each species.

It would certainly be desirable to analyze the fraction 1 protein from more accessions of *Triticum boeoticum*, as well as the other wheat species, but on the basis of our experience with *Nicotiana* species it seems unlikely that individuals of *T. boeoticum* would be found to have large subunit polypeptides different from those reported (*2*).

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Overlooked Avian Oncornavirus in Cultured Muscle—Functionally Significant?

Among investigators using tissue-cultured "normal" chick embryo (NCE) muscle, live-and-let-live appears to be the attitude in regard to possible or proved contamination of many if not all of their cultures by avian oncornavirus genus C [the currently preferred generic term to include the avian leucosis and sarcoma viruses (*1*)]. There is immunologic evidence that that group of viruses is widespread in chicken flocks (*2*). In a study of cultured NCE muscle, we have found electron microscopic evidence of C-particles and have identified avian oncornavirus by a complement-fixation test for "avian leucosis virus" (the COFAL test). We suggest that viral contamination of cultured NCE muscle is widepread. Yet various authors have been charitable toward the presence or possible presence of that group of viruses in their muscle fibers cultured from chick embryos.

A recently published electron micrograph of cultured NCE muscle (*3*) illustrates C-particles, presumably of avian oncornavirus, in their characteristic appearance [which is as membrane-circumscribed particles, the membrane being a dilated t-tubule (*4*)]; however, that virus and its possible role in the growth and differentiation phenomena examined in the study are not mentioned, nor is the possible virus-enhancing role of the cytochalasin B used in the culture medium. In a tissue culture atlas (*5*), similar C-particles are shown in NCE muscle with-

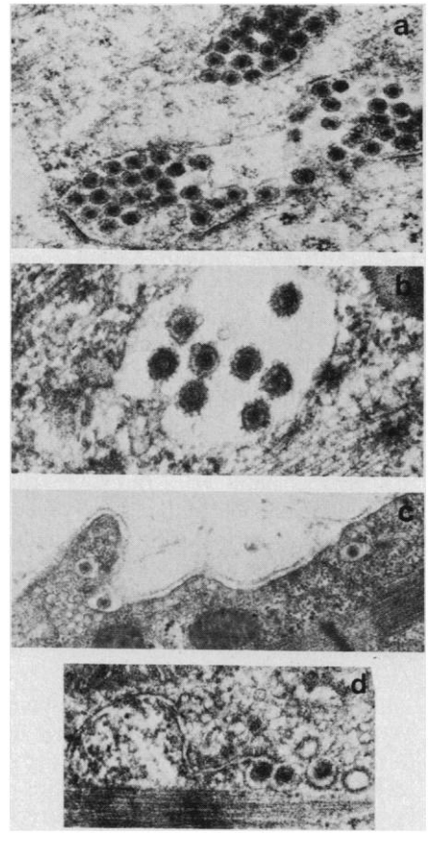

Fig. 1. Numerous C-particles in membrane-bound collections (× 31,000). (b) Two of the C-particles in a sac are connected by "stalks" to the limiting membrane (× 45,000). (c) C-particle free under the basement membrane (center) and possibly in transversely sectioned mouth of t-tubule (left and right) (× 18,000). (d) The limiting membranes are dilations of t-tubules, evident from this less involved region (× 26,000).

out comment. In numerous studies of growth, differentiation, metabolism, physiology, response to cytotoxic agents such as 5-bromodeoxyuridine (BrdU) (*6*), and even viral "infectivity" (*7*) (actually superinfectivity) of cultured NCE muscle [see (*4*) for additional references], including our own previous studies, the possible influence of avian oncornavirus contamination has not heretofore been mentioned (*8*). Even cultured embryonic quail muscle, preferred by some investigators, might be infected, because sensitive tests reveal avian oncornavirus antigen in that species (*9*). In cultured NCE muscle we have found that dinitrophenol greatly enhances the amount of avian oncornavirus shown by electron microscopy as C-particles (Fig. 1) and by the COFAL test, and that chloroquin enhances the amount shown by electron microscopy (*4*). Others have found that cytochalasin B (*10*), BrdU (*11*), and chloroquin (*12*) can enhance expression of several viruses in various other cultured cells.

All investigators using cultured NCE muscle should consider the possible influence on their experiments of avian oncornavirus contamination of their test cells. It would be surprising if viral infestation is without effect, although the effect would not necessarily be adverse. One of the avian oncornavirus group, Rous sarcoma virus, in its malignant transformation of cells, can effect many metabolic changes—for example, enhancing the capacity for anaerobic glycolysis—and it can induce nonmalignant proliferation of otherwise stagnant NCE neuroretinal cells (*13*). We wonder whether the ready growth and development of NCE muscle cells in culture is at least partially the result of a submalignant transformation of them by a small amount of an avian oncornavirus, a possible ameliorative effect of that virus group akin to an effect hypothesized in the "normal" biology of chick embryo cells (*2*). Do we, our cells and ourselves, need the virus as much as it needs us? Is that why we are charitable?

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