Interaction of Porcine Vasoactive Intestinal Peptide with Dispersed Pancreatic Acinar Cells from the Guinea Pig

BINDING OF RADIOIODINATED PEPTIDE

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We have used 125I-labeled vasoactive intestinal peptide (VIP) to study the kinetics, stoichiometry, and chemical specificity with which the labeled peptide binds to dispersed acinar cells prepared from guinea pig pancreas. Binding of 125I-VIP to pancreatic acinar cells was moderately rapid, reversible, specific, saturable, and dependent on incubation temperature. Deterioration of 125I-VIP incubated with pancreatic acinar cells at 37°C was reflected in a decrease in acid-precipitable radioactivity and in the amount of tracer which could bind to fresh acinar cells. On the other hand, 125I-VIP bound to pancreatic acinar cells appeared to be protected from deterioration.

VIP and secretin but not glucagon or COOH-terminal octapeptide of cholecystokinin inhibited binding of 125I-VIP to pancreatic acinar cells. The dose-response curve for inhibition of 125I-VIP binding by VIP or secretin was biphasic and suggested that pancreatic acinar cells have two classes of binding sites: (a) a relatively small number of sites with a high affinity for VIP and a low affinity for secretin, and (b) a relatively large number of sites with a low affinity for VIP and a high affinity for secretin. The difference between the relative affinities of VIP and secretin for the high affinity VIP binding sites appears to be primarily attributable to the NH₂-terminal portions of these molecules since synthetic COOH-terminal fragments VIP₁₋₁₇, VIP₁₋₁₆, and secretin₁₋₁₇ were equipotent in inhibiting 125I-VIP binding. On the other hand, secretin₁₋₁₇, [6-tyrosine] secretin and native secretin were equipotent in inhibiting binding of 125I-VIP to its high affinity site, and these three peptides were 5 times more potent than secretin₁₋₁₇ but 10,000 times less potent than native VIP.

VIP is an octacosapeptide isolated from hog upper small intestine (1) which is similar in chemical structure (2-4) and in spectrum of biologic activities to secretin and glucagon (5). Previous studies have shown that VIP and secretin bind to a common membrane site in liver and fat (6-9) and that this binding correlates with the ability of these peptides to activate adenylate cyclase. Glucagon, which also activates adenylate cyclase in liver and fat (6-9), interacts with a site which is functionally distinct from that for VIP and secretin.

Both VIP and secretin stimulate pancreatic exocrine secretion (1, 10, 11), increase pancreatic tissue concentrations of cyclic AMP in vivo (12) and in vitro (13, 14) and stimulate adenylate cyclase activity in pancreatic membranes (15). In general the potency of VIP has been significantly less (approximately 10 times) than that for secretin, although maximal concentrations of secretin plus VIP give the same response as a high concentration of secretin alone. To explore further the interaction of secretin and VIP with pancreatic tissue in vitro we have prepared 125I-VIP and have studied the kinetics, stoichiometry and chemical specificity with which the labeled peptide interacts with dispersed acinar cells prepared from guinea pig pancreas. The present paper deals with functional characteristics of binding of 125I-VIP to pancreatic acinar cells. The accompanying paper focuses on the stoichiometry and chemical specificity required for VIP and secretin to increase cyclic AMP in pancreatic acinar cells.

MATERIALS AND METHODS

Male Hartley albino guinea pigs (350 to 400 g) were obtained from the Small Animals Section, Veterinary Resources Branch, National Institutes of Health, Bethesda, Maryland. Crude collagenase from Clostridium histolyticum (EC 3.4.4.19) and crude hyaluronidase from bovine testis (EC 3.2.1.35) were purchased from Sigma Chemical Co., St. Louis, Mo. Carrier-free Na¹²⁵I (500-600 μCi/ml) in 0.1 M NaOH was purchased from the Radiochemical Centre, Amersham, England.
Chloramine T was purchased from Eastman Kodak Co., Rochester, N. Y., sodium metabisulfite from Aldrich Chemical Co., St. Louis, Mo.; bacitracin from Calbiochem, La Jolla, Calif.; bovine serum albumin from Miles Laboratories, Inc., Kankakee, Ill., purified soybean trypsin inhibitor from Worthington Biochemical Corp., Freehold, N. J., and trasylof from Bayer, Leverkusen, Germany. All other chemicals used were of the highest grade commercially available.

Highly purified natural porcine vasoactive intestinal peptide and natural porcine secretin were generous gifts from Dr. Viktor Mutt (GHI Research Unit, Karolinska Institute, Stockholm, Sweden). Two preparations of synthetic porcine secretin were used. One was a gift from Dr. E. Wunsch (The Max Planck Institute fur Eiweis- und Lederfororschung, Munich, Germany) and the other was purchased from Schwarz/Mann (Orangeburg, N. Y.). [6-Tyrosine] synthetic porcine secretin was purchased from Schwarz/Mann. The following synthetic fragments of porcine peptides were gifts from Dr. M. A. Ondetti: secretin, (SQ 18,484), secretin, (SQ 19,300), secretin, (SQ 19,301), COOH-terminal octapeptide of cholecystokinin (CCK-OP, SQ 19,844), and secretin, (SQ 19,297), secretin, (SQ 18,484), secretin, (SQ 19,300), and secretin, (SQ 19,301).

**RESULTS**

At 37°C specific binding of 125I-VIP to dispersed guinea pig pancreatic acinar cells was moderately rapid (50% of maximum binding occurred within 5 min), became maximal by 10 to 20 min, and then decreased progressively from 40 to 120 min (Fig. 1). Stepwise reduction in incubation temperature from 37°C to 4°C produced a progressive decrease both in the rate of the binding reaction and in the maximum amount of 125I-VIP which could be bound. At pH 5.3 binding of 125I-VIP was barely detectable. As the pH was increased, binding increased, became maximal in the range 7.0 to 7.5 and then decreased. In acinar cells preincubated with 125I-VIP for 10 min, washed to remove free radioactivity and then resuspended in fresh buffer containing no radioactivity, tracer dissociated at a rate of approximately 1% per min. Adding nonradioactive VIP (5 x 10⁻⁸ M) to the incubation medium did not alter the rate of dissociation. Stepwise reduction of the incubation temperature produced a progressive decrease in the rate of dissociation (Fig. 2). The results in Fig. 2 indicate that, at most, 1% of the bound radioactivity would be lost during the washing-resuspension procedure routinely used to determine 125I-VIP binding to isolated pancreatic acinar cells.

The 125I-VIP used for the present studies was 94 to 97% precipitable by 5% trichloroacetic acid. When placed in the usual incubation medium (final 125I-VIP concentration of 2.6 x 10⁻¹⁰ M) and incubated at 37°C without acinar cells the amount of radioactivity which was trichloroacetic acid-precipitable decreased by 15% in 30 min. When acinar cells (17 x 10⁶ cells/ml) were added the rate of loss of trichloroacetic acid-precipitable radioactivity was doubled (see legend to Fig. 2).
acid-precipitability increased 2- to 3-fold and nonradioactive VIP (5 × 10^{-11} M), bacitracin (0.25 mg/ml) or trasylo (500 Kallikrein inactivating units/ml) offered no apparent "protection." On the other hand, increasing the concentration of bovine serum albumin in the incubation medium from 0.1% (w/v) to 4% increased the amount of 125I-VIP which was trichloroacetic acid-precipitable after 30 min from 69 to 83% of the added counts.

To characterize further the functional alteration of 125I-VIP which occurs in the presence of acinar cells, 125I-VIP was incubated with acinar cells for 20 min at 15° and 37° (Fig. 3). At 37° specific binding of 125I-VIP was rapid and became maximal after 5 to 10 min of incubation. The amount of the free 125I-VIP which was trichloroacetic acid-precipitable decreased progressively so that after 20 min only 50% of the tracer was precipitable. The amount of free 125I-VIP which could bind to fresh cells also decreased progressively, and this decrease was more rapid and more extensive than that observed for trichloroacetic acid precipitation. Reducing the incubation temperature to 15° slowed the rate of binding of 125I-VIP but also reduced deterioration of the tracer in the sense that a greater percentage of the free 125I-VIP was trichloroacetic acid-precipitable and was able to bind the fresh cells.

To test for potential alteration of 125I-VIP bound to the cell, tracer was incubated with acinar cells at 37° and at various times bound 125I-VIP was extracted at 20° for 5 min with 150 mM NaCl containing 1 mM hydrochloric acid (Fig. 4). Approximately 20% of the bound 125I-VIP was removed by this procedure. Compared to free 125I-VIP (Fig. 3) the bound radioactivity which was extracted showed significantly greater biological activity in terms of its ability to bind fresh acinar cells.

The ability of various peptides to inhibit binding of 125I-VIP to pancreatic acinar cells was tested using a 10-min incubation at 37°. These incubation conditions were chosen in an effort to maximize binding of 125I-VIP while at the same time to minimize degradation of the tracer. Inhibition of binding of 125I-VIP by native VIP could be detected at 1 × 10^{-10} M VIP, was half-maximal at 10^{-8} M, and was abolished at 10^{-4} M (Fig. 5A). There appeared to be two components to the curve describing binding of 125I-VIP as a function of the concentration of native VIP. 85% of tracer binding could be inhibited at 10^{-5} M; however, 10^{-4} M VIP was required to abolish tracer binding. After correcting for the degradation of 125I-VIP the results in Fig. 5 were plotted in the form suggested by Scatchard (23). The resulting plot was curvilinear with an upward concavity and the curve could be described by two straight lines. One possible explanation for these results is that there are two functionally independent classes of binding sites. Based on this assumption we have calculated the apparent affinity and capacity for each class of sites using results from l:l separate experiments (Table I). These calculations indicate that there are approximately 9000 high affinity sites per cell and that half of these sites will be occupied at a VIP concentration of 7 × 10^{-10} M. There are approximately 135,000 low affinity sites per cell and these sites are 50% occupied at a VIP concentration of 8 × 10^{-6} M. The portion of the curve reflecting the low affinity binding sites accounts for only 10 to 15% of 125I-VIP binding and has a shallow slope, thereby reducing the precision with which one is able to calculate the.

![Fig. 2. Effect of temperature on dissociation of specifically bound 125I-VIP to pancreatic acinar cells. Pancreatic acinar cells were preincubated with 125I-VIP (2.6 × 10^{-11} M) for 10 min at 37°. The cells were then placed at the indicated temperature for 5 min and the dissociation reaction was initiated by adding nonradioactive VIP (5 × 10^{-11} M). Results are expressed as the percentage of 125I-VIP specifically bound at the time nonradioactive VIP was added.](http://www.jbc.org/)

![Fig. 3. Deterioration of free 125I-VIP in the presence of pancreatic acinar cells. Cells were incubated with 125I-VIP (2.6 × 10^{-11} M) at 15° or 37° and specific binding determined at the times indicated (A). Free 125I-VIP was separated by centrifuging (10,000 × g for 15 s) 300 μl of incubation mixture. Duplicate 20-μl aliquots of supernatant were added to 1% bovine serum albumin in 5% trichloroacetic acid (TCA), mixed, placed at 4° and centrifuged (1500 × g) for 10 min. Radioactivity was determined on supernatant and precipitate and the results are expressed as the percentage of total radioactivity which was precipitated (B). Duplicate 100-μl aliquots of supernatant were incubated for 8 min at 37° with fresh pancreatic acinar cells and the amount of specifically bound 125I-VIP was determined (C).](http://www.jbc.org/)
parameters for this portion of the curve. We should also point out that the values derived from the Scatchard plot should be viewed only as approximations since our conditions do not meet all of the criteria required for this type of analysis (23-25) and our correction for deterioration of free 125I-VIP was based on loss of trichloroacetic acid precipitability which certainly underestimates the extent to which the biologic activity of free 125I-VIP is altered (see Fig. 3).

Secretin, which has a chemical structure similar to that of VIP, also inhibited binding of 125I-VIP to pancreatic acinar cells (Fig. 5). Relatively low concentrations of secretin (i.e., 10^-9 M) produced a small, but reproducible, decrease in binding. At concentrations of secretin above 5 x 10^-7 M inhibition of 125I-VIP binding was more pronounced. These results indicate that the class of sites with a low affinity for VIP has a high affinity for secretin, and that the high affinity VIP binding sites have a low affinity for secretin. From experiments similar to those illustrated in Fig. 5 one can estimate that 50% of the low affinity VIP binding sites will be occupied by 5 x 10^-10 M secretin while approximately 8 x 10^-8 M secretin is required for it to occupy 50% of the sites that have a high affinity for VIP (Table I). Neither glucagon, which is chemically similar to VIP and secretin, nor the COOH-terminal octapeptide of cholecystokinin at concentrations as high as 10^-8 M, inhibited binding of 125I-VIP to pancreatic acinar cells (Fig. 5A).

To explore the relative importance of segments of the VIP molecule in interacting with 125I-VIP binding sites, we tested various synthetic fragments of VIP for inhibition of binding of 125I-VIP to pancreatic acinar cells (Fig. 5B). Neither the NH2-terminal fragment VIP1-8, nor the COOH-terminal fragment VIP16-28, at concentrations as high as 10^-8 M affected binding of 125I-VIP. The COOH-terminal fragments VIP14-28 and VIP15-28 were each capable of inhibiting binding of 125I-VIP; however, each peptide had a relatively low affinity since at concentrations of 10^-8 M, tracer binding was reduced by only 15 to 20% (Table II). Similar tests were carried out with fragments of secretin (Fig. 5C). The NH2-terminal hexapeptide, secretin1-6, at concentrations as high as 3 x 10^-6 M, was without effect. A larger NH2-terminal fragment, secretin1-14, inhibited tracer binding; however, its affinity was low since at a concentration of 3 x 10^-6 M binding of 125I-VIP was reduced by only 10%. The COOH-terminal fragments, secretin14-27 and secretin17-27 each inhibited binding of 125I-VIP and the larger fragment was more potent. Significant inhibition of 125I-VIP binding by the 14-27 fragment could be detected at concentrations of 5 x 10^-6 M and binding was inhibited by 40% when the concentration of secretin14-27 was 3 x 10^-6 M. An effect of secretin15-27 could be detected at 10^-6 M and 125I-VIP binding

![FIG. 4](https://example.com/fig4.png)  
**FIG. 4.** Deterioration of 125I-VIP bound to pancreatic acinar cells. Acinar cells were incubated with 125I-VIP (2.6 x 10^-11 M) at 37°C. At 16-min intervals duplicate 200-μl portions of incubation mixture were taken and the cells were washed 3 times by centrifugation (10,000 x g for 15 s) and resuspension. Bound 125I-VIP was extracted by incubating the washed cells with 200 μl of 150 mM sodium chloride/1 mM hydrochloric acid for 5 min at 20°C and then centrifuging. The radioactivity present in the supernatant (left panel) is expressed as the percentage of I-125I-VIP specifically bound to unextracted cells. Aliquots of the supernatant were neutralized and incubated for 8 min at 37°C with fresh pancreatic acinar cells to determine the percentage of extracted 125I-VIP which could bind specifically.

![FIG. 5](https://example.com/fig5.png)  
**FIG. 5.** Ability of various peptides to inhibit binding of 125I-VIP to dispersed pancreatic acinar cells. Cells were incubated for 10 min at 37°C with 125I-VIP (2.6 x 10^-11 M) plus different concentrations of the indicated peptides. Binding of 125I-VIP is expressed as the percentage of radioactivity specifically bound in the absence of added nonradioactive peptide.
was inhibited by 60% when the fragment was present at a concentration of $3 \times 10^{-8}$ M. [6-Tyrosine] secretin gave results which were identical to those obtained with secretin$_{1-8}$ (data not shown). Our results are not sufficiently precise to enable us to determine whether the 14–27 and 5–27 fragments of secretin can interact with the low affinity VIP binding site; however, both peptides clearly inhibit binding of $^{125}$I-VIP to the high affinity binding sites. If we assume that neither peptide can interact with those binding sites having a low affinity for VIP concentrations of secretin$_{1-8}$ and secretin$_{1-14}$ re- quired to occupy 50% of the high affinity VIP binding sites are in the range of $7 \times 10^{-8}$ M and $1 \times 10^{-8}$ M, respectively (Table II).

### Discussion

The present results illustrate that binding of $^{125}$I-VIP to pancreatic acinar cells, like that of other peptides to their respective target tissues (Fig. 6), is rapid, specific, reversible and dependent on incubation temperature. As has also been seen with other iodinated peptides (26, 27) significant deterioration of free $^{125}$I-VIP occurred during incubation at 37°C. This deterioration was temperature-sensitive and was more extensive when measured by the ability of incubated $^{125}$I-VIP to bind to fresh acinar cells than when measured as the decrease in acid precipitability. Bacitracin, which inhibits inactivation of VIP and glucagon by liver membranes (7, 28), trasyloil or nonradioactive VIP did not alter deterioration of $^{125}$I-VIP, while 4% (w/v) bovine serum albumin reduced but did not abolish deterioration of the tracer. Bound $^{125}$I-VIP appeared to be protected from whatever factors are responsible for inactivation of the tracer. These findings are similar to those reported

Throughout this paper we have referred to binding of $^{125}$I-VIP to acinar cells for two reasons. On microscopic examination of our cell preparation the frequency of acinar cells was 98% and of duct cells plus islet cells was 1%. In cell suspensions prepared with discontinuous albumin density gradient centrifugation and containing approximately 90% duct cells and 10% acinar cells, $^{125}$I-VIP binding was the same as it was in our usual preparation.

<table>
<thead>
<tr>
<th>Compound</th>
<th>$S_{50}$</th>
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<tbody>
<tr>
<td>VIP</td>
<td>$4 \times 10^{-7}$</td>
</tr>
<tr>
<td>Secretin</td>
<td>$3 \times 10^{-7}$</td>
</tr>
<tr>
<td>Glucagon</td>
<td>$2 \times 10^{-7}$</td>
</tr>
</tbody>
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### Table II

Apparent affinities of various synthetic fragments of secretin and VIP for high affinity $^{125}$I VIP binding sites in dispersed guinea pig pancreatic acinar cells

$S_{50}$ is the concentration required for the peptide to occupy 50% of the high affinity $^{125}$I-VIP binding sites. The values for VIP fragments represent the means ±1 S.D. from four separate experiments similar to those illustrated in Fig. 5. The values for secretin fragments represent the means ±1 S.D. from five separate experiments similar to those illustrated in Fig. 5. The values for native VIP and for native secretin are those given in Table I.

Fig. 6. Amino acid sequence for porcine secretin, VIP and glucagon. *Hatched circles* indicate amino acids which are identical to those in the corresponding position in VIP. *Stippled circles* indicate amino acids which are similar in chemical atmosphere (i.e. side chain is hydrophobic, similar in size, similar in charge, etc.).

for glucagon degradation by liver membranes (29, 30) and suggest that the site to which $^{125}$I-VIP binds is functionally distinct from the site(s) at which it is inactivated.

Inhibition of $^{125}$I-VIP binding by native VIP was biphasic. A Scatchard plot of $^{125}$I-VIP binding was curvilinear and could be fitted by two straight lines. Although there are several different phenomena which could give rise to the observed relation between $^{125}$I-VIP binding of VIP concentration (24, 25) and the values derived from the Scatchard plot are strictly estimates, we have interpreted our results as reflecting the presence of two functionally distinct classes of VIP binding sites. The most compelling observation favoring this conclusion is the close agreement between the dose-response curve for VIP inhibition of $^{125}$I-VIP binding and that for VIP stimulation of cellular cyclic AMP given in the accompanying paper (31). Our curvilinear Scatchard plot does not appear to reflect "negative cooperativity" (32) since the rate of dissociation of bound $^{125}$I-VIP was not altered by adding native VIP and it cannot be explained by native VIP reducing inactivation of the tracer and thereby enhancing $^{125}$I-VIP binding. Since we do not know if both classes of VIP-binding sites are present on all cells, the values in Table I give the relative abundance of high and low affinity binding sites in our preparation of pancreatic acinar cells.

VIP, glucagon and secretin are similar in terms of chemical structure (Fig. 6) and spectrum of biologic activities (4–15). Previous studies with iodinated peptides showed that plasma membranes from rat liver (6–9) or rat adipocytes (6–8) possess a single class of VIP binding sites having a high affinity for VIP and a low affinity for secretin. In each of these tissues the concentration of VIP or secretin which produced half-maximal inhibition of $^{125}$I-VIP binding agreed closely with the peptide concentration giving half-maximal stimulation of adenylate cyclase. Glucagon, which also activates adenylate cyclase in liver and fat, does not interact with the receptor which recognizes VIP and secretin. Pancreatic acinar cells possess two functionally distinct classes of binding sites, neither of which interacts with glucagon. One class is similar to that seen in membranes prepared from liver and from adipocytes in that it has a high affinity for VIP and a low affinity for secretin; the other class has a low affinity for VIP and a high affinity for secretin.
Our results with synthetic VIP fragments indicate that a sufficiently large COOH-terminal fragment (VIP₁₄₋₂₈ and VIP₂₁₋₅₆, but not VIP₂₈₋₅₆) can interact with VIP binding sites but with an apparent affinity which is much lower than that for native VIP and agrees with the studies of Bodanszky et al. (3) who found these fragments to be less than 2% as potent as native VIP. Our studies with synthetic fragments of secretin showed that an NH₂-terminal fragment (secretin₁₋₁₄) as well as a COOH-terminal fragment (secretin₁₄₋₂₈) could inhibit binding of [³²P]VIP. The NH₂-terminal fragment was 5 times less potent than secretin₁₋₁₄ which, in turn, was 5 times less potent than the larger COOH-terminal fragment secretin₅₋₂₈. The Sc₅₀ for secretin₁₄₋₂₈ was the same as that for native secretin acting at the high affinity VIP binding site. Unlike native secretin, however, the 3₋₇ fragment did not produce clear biphasic inhibition of [³²P]VIP binding. This suggests that either secretin₁₋₁₄ does not interact with the low affinity VIP binding sites or its affinity for this class of sites is the same as its affinity for the high affinity VIP binding sites. These results with secretin₁₋₁₄ do indicate that the NH₂-terminal four amino acids are important for secretin binding to the low but not to the high affinity VIP binding sites.

The difference between the affinities of secretin and VIP for the high affinity VIP binding sites appears to be primarily attributable to their NH₂-terminal structures. Secretin₁₋₁₄, VIP₁₋₁₄, and VIP₁₋₇₈ were equipotent in inhibiting [³²P]VIP binding. Secretin₁₋₁₄ was equipotent with native secretin and with [6-tyrosine] secretin and each of these peptides was 5 times more potent than secretin₁₋₁₄ but 10,000 times less potent than VIP. Glucagon which is similar in chemical structure to VIP and secretin (Fig. 6) did not inhibit binding of [³²P]VIP to pancreatic acinar cells; however, the basis for this difference between the affinities of secretin and VIP for the high affinity VIP binding sites appears to be primarily attributable to their NH₂-terminal structures. Secretin₁₋₁₄, VIP₁₋₁₄, and VIP₁₋₇₈ were equipotent in inhibiting [³²P]VIP binding. Secretin₁₋₁₄ was equipotent with native secretin and with [6-tyrosine] secretin and each of these peptides was 5 times more potent than secretin₁₋₁₄ but 10,000 times less potent than VIP. Glucagon which is similar in chemical structure to VIP and secretin (Fig. 6) did not inhibit binding of [³²P]VIP to pancreatic acinar cells; however, the basis for this lack of interaction is not readily apparent from comparing the structures of these three peptides.

Of the 9 amino acids which occur in identical positions in secretin and VIP, 8 are in the NH₂-terminal half and 1 is in the COOH-terminal half (Fig. 6). It was initially surprising that secretin₁₋₁₄, which has only 2 amino acid identities with VIP, was approximately 5 times more potent in inhibiting [³²P]VIP binding than was secretin₁₋₁₄, which has 8 amino acid identities with VIP (Fig. 6). There are 10 amino acid similarities between secretin and VIP (Fig. 6). Of these 10 similarities, 3 are in the NH₂-terminal half, while 7 are in the COOH-terminal half. Thus, both secretin and VIP have a common COOH-terminal chemical atmosphere attributable primarily to amino acids having side chains which are hydrophobic and this similarity may account for the ability of secretin₁₋₁₄ to interact with the high affinity VIP binding sites with an affinity which is greater than that for secretin₁₋₁₄ and which is equal to that for VIP₁₋₁₄ and VIP₁₋₇₈.

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REFERENCES
10. Dockray, G. J. (1973) Experientia 29, 1510-1511
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J P Christophe, T P Conlon and J D Gardner


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