Unique Expression of Major Histocompatibility Complex
Class I Proteins in the Absence of Glucose Trimming and Calnexin Association*

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Recent evidence indicates that efficient expression of major histocompatibility complex (MHC) complexes requires their interaction with the resident endoplasmic reticulum (ER) chaperone calnexin, which for certain proteins functions as a lectin specific for monoglycosylated glycans. In the current report, we studied the expression of MHC class I proteins in BW wild type thymoma cells (BW WT) and glucosidase II-deficient BW PHAR2.7 cells. Consistent with a requirement for glucose (Glc) trimming for interaction of class I proteins with calnexin, we found that nascent H-2Kk proteins associated with calnexin in untreated BW WT cells, but not in BW WT cells treated with the glucosidase inhibitor castanospermine (cas), or in untreated glucosidase II-deficient BW PHAR2.7 cells. Suprisingly, we found that H-2Kk expression occurred with similar efficiency in BW PHAR2.7 cells as in BW WT cells and that formation of nascent H-2Kk complexes was perturbed by cas treatment in BW WT cells but not in BW PHAR2.7 cells. Finally, it was noted that expression of the molecular chaperone Bip was markedly increased in BW PHAR2.7 cells relative to BW WT cells, which is suggested to play a role in regulating the expression of H-2Kk complexes in BW PHAR2.7 cells. The current study demonstrates that Glc trimming is required for efficient interaction of nascent H-2Kk proteins with calnexin; that expression of MHC class I proteins can, under certain conditions, proceed effectively in the absence of Glc trimming and calnexin association; and that Bip expression is markedly increased under conditions where diglycosylated glycans persist on nascent glycoproteins within the ER. These data are consistent with the hypothesis that alternative oligomerization pathways exist for class I proteins within the quality control system of the ER that have differential requirements for removal of Glc residues from nascent glycan chains.

Most major histocompatibility complex (MHC) class I molecules expressed at the cell surface consist of heavy chains (HC) noncovalently associated with β2-microglobulin proteins and processed peptides (1, 2). The molecular chaperone calnexin plays an important role in the assembly of nascent HC-β2-peptide complexes within the endoplasmic reticulum (ER) and moderates their transport to the Golgi compartment (3–6). Assembly of nascent murine MHC class I complexes is believed to occur by: (i) association of newly synthesized HC with calnexin to form HC-calnexin protein complexes, (ii) interaction of β2-microglobulin proteins with calnexin-HC proteins to form calnexin-HC-β2 complexes; and finally (iii) addition of peptide to calnexin-associated HC-β2 proteins to form HC-β2-peptide complexes, which rapidly dissociate from calnexin and exit the ER (7–9).

A growing body of evidence signifies that removal of glucose (Glc) residues from nascent oligosaccharide chains is important for association of certain glycoproteins with calnexin, including donotypic αβ chains of the T cell antigen receptor complex (10), viral gene products (11, 12), and many unidentified proteins (10). Removal of Glc residues from immature GlcMan5GlcNAc2 (Glc = glucose, Man = mannose; and Glc-NAc = N-acetylglucosamine) glycan chains is accomplished by the sequential action of ER glucosidase I and glucosidase II enzymes, which remove the outermost and two proximal Glc residues, respectively (13). Since calnexin associates with glycoproteins containing monoglycosylated (Glc5Man5GlcNAc2) glycan chains (12, 14), both glucosidase I and glucosidase II activities are necessary to create glycan substrates for calnexin binding. Oligosaccharide chains are not strictly required for calnexin association, however, as several nonglycosylated molecules interact stably with calnexin, including recombinant multidrug resistance P glycoprotein lacking N-linked addition sites (15) and the CD3ε subunit of the T cell antigen receptor complex (16).

To further our understanding of the role of Glc trimming and calnexin association in the assembly and expression of immune receptor molecules, we studied the expression of MHC class I proteins in BW wild type thymoma cells (BW WT) and the glucosidase II-deficient BW variant cell line, PHAR2.7. We found that expression of H-2Dk proteins was markedly reduced on BW PHAR2.7 cells relative to BW WT cells, and, unexpectedly, that expression of H-2Kk proteins was equivalent on both cell types. Our results show that Glc trimming and calnexin association are required for efficient formation of nascent H-2Kk complexes in BW WT cells but not BW PHAR2.7 cells, indicating that alternative folding pathways exist within the ER quality control system for assembly of MHC class I protein complexes. Lastly, Bip expression was observed to be significantly increased in BW PHAR2.7 cells relative to BW WT cells, which is implicated to play a role in regulating H-2Kk expression in BW PHAR2.7 cells.

EXPERIMENTAL PROCEDURES

Cells and Reagents—BW5147 cells (17) and BW PHAR2.7 cells (18) were maintained by weekly passage in RPMI 1640 medium containing 5% fetal calf serum at 37 °C in 5% CO2. Castanospermine (cas) was...
Because glucosidase II-deficient BW PHAR2.7 cells—
dictated that expression of both H-2Dk and H-2Kk proteins would
lated glycans important in calnexin binding (14, 18), we pre-
purchased from Calbiochem and was used at a 50–100
(15-5-5) and anti-H-2Kk (36-7-5) mAbs, followed by fluorescein isothio-
munoprecipitated material was analyzed on one-dimensional SDS-PAGE
gels under reducing conditions and visualized by chemiluminescence.
expression of MHC class I proteins on BW PHAR2.7 cells relative to BW
WT cells was 0.35 for H-2Dk proteins and 1.1 for H-2Kk proteins. The
data shown are representative of at least five independent experiments; variation
between experiments was less than 10%. B, BW WT and BW PHAR2.7 cells were surface-labeled by biotinylation, solubilized in 1%
Nonidet P-40, and lysates were precipitated with anti-H-2Dk (15-5-5)
and anti-H-2Kk (36-7-5) mAbs preadsorbed to protein A-Sepharose. Im-
munoprecipitated material was analyzed on one-dimensional SDS-PAGE
gels under reducing conditions and visualized by chemiluminescence
The positions of H-2Dk and H-2Kk proteins are indicated.

Cell type: BW WT or BW PHAR2.7

A

B

FIG. 1. Surface expression of H-2Dk and H-2Kk proteins on BW
WT cells and BW PHAR2.7 cells. A, Surface expression of H-2Dk
and H-2Kk complexes was examined by indirect staining with anti-H-2Dk
(15-5-5) and anti-H-2Kk (36-7-5) mAbs, followed by fluorescein isothio-
cyanate (FITC)-conjugated goat anti-mouse IgG2a Ab molecules (GAM).
Control staining was performed with GAM Ab only. The relative
expression of MHC class I proteins on BW PHAR2.7 cells relative to BW
WT cells was 0.35 for H-2Dk proteins and 1.1 for H-2Kk proteins. The
data shown are representative of at least five independent experiments; variation
between experiments was less than 10%. B, BW WT and BW PHAR2.7 cells were surface-labeled by biotinylation, solubilized in 1%
Nonidet P-40, and lysates were precipitated with anti-H-2Dk (15-5-5)
and anti-H-2Kk (36-7-5) mAbs preadsorbed to protein A-Sepharose. Im-
munoprecipitated material was analyzed on one-dimensional SDS-PAGE
gels under reducing conditions and visualized by chemiluminescence
The positions of H-2Dk and H-2Kk proteins are indicated.

Cell type: BW WT or BW PHAR2.7

A

B

FIG. 2. Effect of cas treatment on surface expression of H-2Dk
and H-2Kk proteins on BW WT cells and BW PHAR2.7 cells. Surface
expression of H-2Dk and H-2Kk complexes was examined by cell
surface staining as described in Fig. 1A. Where indicated, cells were
cultured overnight at 37 °C in the presence of the glucosidase inhibitor
castanospermine (Cas) at a concentration of 100 μg/ml. The data shown
are representative of at least three independent experiments; variation
between experiments was less than 10%. Viability was identical in media
containing Cas and cas-treated groups (data not shown).

To study the requirement for glucosidase activity for expression
of MHC class I complexes on BW WT cells, experiments were
performed using the glucosidase inhibitor castanospermine (Cas)
(13). As shown in Fig. 2, surface expression of H-2Dk complexes
on BW WT cells was markedly reduced by overnight culture in cas (Fig. 2). Not suprisingly, surface density of H-2Dk complexes
was similar on untreated and cas-treated BW PHAR2.7 cells
(Fig. 2), as expression of H-2Dk complexes was already decreased
on glucosidase II-deficient BW PHAR2.7 cells. Most importantly,
these data show that cas treatment dramatically reduced expres-
sion of H-2Kk complexes on BW WT cells, but not BW PHAR2.7 cells
(Fig. 2). Reduced expression of MHC class I complexes on
cas-treated BW WT cells was specific in that expression of other
surface molecules (CD45 proteins, thymic shared antigen pro-
Teins, and peanut agglutinin lectin-binding proteins) was unaf-
fected by cas treatment, and no change in class I expression was
observed following culture with the mannosidase inhibitor deoxy-
mannosilirimycin (data not shown). These results show that
impaired glucosidase activity is correlated with decreased expres-
sion of H-2Dk complexes on BW WT cells and that BW WT and BW PHAR2.7 cells exhibit differential requirements for glucosidase activity in the surface expression of H-2Kk complexes.

Intracellular Transport of Nascent H-2Kk Complexes in BW
WT and BW PHAR2.7 Cells—Because of our curious results
regarding H-2Kk expression on BW PHAR2.7 cells, we
performed a series of studies to examine the molecular require-
ments for formation of nascent H-2Kk complexes in BW WT
and BW PHAR2.7 cells. Although the anti-H-2Dk Abs used in
our studies efficiently recognized surface H-2Dk proteins on
BW WT and BW PHAR2.7 cells, they failed to effectively im-
munoprecipitate newly synthesized H-2Dk proteins from either
cell type; therefore, our studies focused on H-2Kk molecules. In
our first set of experiments, the intracellular transport of nas-
cent H-2Kk complexes was examined. For these studies, BW

RESULTS

Surface Expression of MHC Class I Proteins on BW WT and
BW PHAR2.7 Cells—Because glucosidase II-deficient BW
PHAR2.7 cells are impaired in the generation of monoglucosyl-
lated glycans important in calnexin binding (14, 18), we
predicted that expression of both H-2Dk and H-2Kk proteins
would be markedly decreased on BW PHAR2.7 cells relative to BW
WT cells. As shown in Fig. 1A, H-2Dk expression was markedly
reduced on BW PHAR2.7 cells relative to BW WT cells, but,
suprisingly, H-2Kk expression was comparable on both cell

types (Fig. 1A, 3rd column). These findings were confirmed by
immunoprecipitation studies of surface-labeled BW WT and BW
PHAR2.7 cells (Fig. 1B).

Glycosidase Digestion, Gel Electrophoresis, and Immunoblotting—
Digestion with Endo H (Genzyme) was performed as described previ-
ously (10). Digestion with PNGase F (Glyko, Novato, CA) was per-
fomed according to the manufacturer’s instructions. One- and two-
dimensional gel electrophoreses were carried out as described pre-
vously (10). For immunoblotting, samples were probed with a 1:200
dilution of antisera in phosphate-buffered saline containing 5% milk
and 0.02% NaN3, followed by 125I-labeled protein A (10 μCi/ml) (ICN).

Flow Cytometry—Cell surface staining of H-2Dk and H-2Kk proteins
was performed by incubating cells with primary anti-H-2Dk and anti-
H-2Kk Abs and, after washing, with secondary goat anti-mouse IgG
(GAM) Ab conjugated to fluorescein isothiocyanate (Pharmingen). Cells
were analyzed on a FACScan flow cytometer (Beckton Dickinson).
All fluorescence data were collected using logarithmic amplification
on 50,000 viable cells as determined by forward light scatter intensity and
propidium iodide exclusion.
with anti-H-2Kk Abs. Immunoprecipitated material was analyzed on two-dimensional NEPHGE/SDS-PAGE gels. As shown in Fig. 4A, numerous proteins coprecipitated with calnexin molecules in BW WT lysates, including nascent H-2Kk proteins (Fig. 4A, left, top panel, arrow). Identity of H-2Kk proteins in calnexin precipitates was verified by mixing experiments and release/re-capture studies (data not shown). As is evident, markedly fewer proteins were present in anti-calnexin precipitates of BW PHAR2.7 lysates relative to BW WT lysates (Fig. 4A, right, top panel). Most importantly, nascent H-2Kk proteins were not associated with calnexin in BW PHAR2.7 lysates (Fig. 4A, right, top panel, arrow), despite the abundant existence of H-2Kk proteins within the lysate (Fig. 4A, right, bottom panel, arrow). Association of newly synthesized H-2Kk proteins with calnexin molecules in BW WT lysates was inhibited by treatment with the glucosidase inhibitor cas (Fig. 4B, arrow), as was the association of numerous unidentified protein species with calnexin (Fig. 4B). These data demonstrate that Glc trimming is required for effective interaction of calnexin with nascent H-2Kk proteins in BW thymoma cells and provide evidence that efficient formation of H-2Kk complexes in BW PHAR2.7 cells does not result from their association with calnexin.

Requirement for Glc Trimming for Association of Nascent H-2Kk Proteins with Calnexin—As calnexin association is believed to be an important step in the assembly of nascent MHC class I protein complexes (3–6, 14), we next examined the association of newly synthesized H-2Kk molecules with calnexin in BW WT and BW PHAR2.7 cells. For these studies, cells were metabolically labeled, solubilized in 1% digitonin, and lysates immunoprecipitated with anti-H-2Kk mAb (37–6-5), and precipitates were analyzed on two-dimensional NEPHGE/SDS-PAGE gels under reducing conditions. Precipitates were either mock-treated or digested with Endo H and analyzed on SDS-PAGE gels under reducing conditions. The positions of H-2Kk proteins and β2-microglobulin proteins are indicated. K malignant = Kk proteins resistant to digestion with Endo H; K normal = Kk proteins sensitive to digestion with Endo H.

WT and BW PHAR2.7 cells were metabolically pulse-labeled with [35S]methionine, chased for the time period indicated, solubilized in 1% Nonidet P-40 detergent, and lysates immunoprecipitated with anti-H-2Kk mAb, and precipitates were digested with Endoglycosidase H (Endo H), specific for removal of immature N-glycan chains. Nascent H-2Kk glycoproteins synthesized in BW PHAR2.7 cells migrated with slightly decreased mobility compared to H-2Kk proteins made in BW WT cells, reflecting persistence of Glc residues on glycan chains of H-2Kk proteins in BW PHAR2.7 cells (Fig. 3; compare pulse groups). As demonstrated, significant amounts of nascent H-2Kk complexes were formed in both BW WT and BW PHAR2.7 cells during the 30-min pulse period (Fig. 3). H-2Kk complexes efficiently exited the ER and transited through the Golgi compartment in both BW WT and BW PHAR2.7 cells as demonstrated by their acquisition of Endo H resistance during the chase period (Fig. 3). Note that maturation of oligosaccharide chains can occur under conditions of impaired glucosidase activity via the alternative processing pathway for N-linked oligosaccharides, which involves deglucosylation of glycan chains by endomannosidase enzymes (21, 22).

These data show that formation of nascent H-2Kk complexes proceeds with similar efficiency in BW WT and BW PHAR2.7 cells and that H-2Kk complexes are effectively transported from the ER to the Golgi compartment in both cell types, two findings which are in agreement with our results regarding surface expression of H-2Kk proteins on BW WT and BW PHAR2.7 cells (Fig. 1, A and B).

Fig. 3. Intracellular transport of nascent H-2Kk complexes in BW WT and BW PHAR2.7 cells. A, BW WT and BW PHAR2.7 cells were metabolically pulse-labeled with [35S]methionine for 30 min, chased for the time period indicated, solubilized in 1% Nonidet P-40 detergent, and lysates immunoprecipitated with anti-H-2Kk mAb (37–6-5). Precipitates were either mock-treated or digested with Endo H and analyzed on SDS-PAGE gels under reducing conditions. The positions of H-2Kk proteins and β2-microglobulin proteins are indicated. K malignant = Kk proteins resistant to digestion with Endo H; K normal = Kk proteins sensitive to digestion with Endo H.

Because our results in BW PHAR2.7 cells indicated that Glc trimming and calnexin association are not required for effective assembly of nascent H-2Kk complexes, the effect of glucosidase inhibition on formation of nascent H-2Kk complexes in BW WT cells was examined. Significantly fewer H-2Kk complexes existed in anti-H-2Kk precipitates of cas-treated BW WT cells than in untreated BW WT cells (Fig. 5A). In contrast, formation of nascent H-2Kk complexes in BW PHAR2.7 cells was not perturbed by cas treatment (Fig. 5B). Indeed, equivalent amounts of H-2Kk complexes were isolated from lysates of untreated and cas-treated BW PHAR2.7 cells (Fig. 5B). It should be appreciated that nascent H-2Kk proteins synthesized in cas-treated BW PHAR2.7 cells showed a slight shift in molecular weight relative to H-2Kk proteins made in untreated BW PHAR2.7 cells (Fig. 5B, compare pulse groups), demonstrating that cas treatment was effective in these experiments.

Fig. 4. Association of nascent H-2Kk proteins with calnexin in BW WT and BW PHAR2.7 cells. A, digitonin lysates of radiolabeled BW WT and BW PHAR2.7 cells were immunoprecipitated with anti-calnexin Ab or anti-H-2Kk mAb (37–6-5), and precipitates were analyzed on two-dimensional NEPHGE/SDS-PAGE gels under reducing conditions. The position of radiolabeled H-2Kk proteins is indicated. B, radiolabeled lysates of untreated and cas-treated BW WT cells were immunoprecipitated with anti-calnexin Ab, and precipitates were analyzed as in A. The position of H-2Kk proteins is indicated.
We conclude from these data that removal of Glc residues from core glycan chains and calnexin association is required for effective formation of H-2K\(^{k}\) complexes in BW WT and BW PHAR2.7 cells. Impaired formation of nascent H-2K\(^{k}\) complexes in cas-treated BW WT cells provides a molecular basis for reduced surface expression of H-2K\(^{k}\) proteins on BW WT cells cultured overnight in cas (Fig. 2). Moreover, since cas impaired formation of nascent H-2K\(^{k}\) complexes in BW WT cells and other T cell types, including thymocytes and T hybridoma cells,\(^2\) but not BW PHAR2.7 cells, we further conclude that unique assembly mechanisms exist in BW PHAR2.7 cells for assembly of nascent H-2K\(^{k}\) complexes in the absence of Glc trimming and calnexin association.

Increased Expression of Bip Chaperone Proteins in BW PHAR2.7 Cells Relative to BW WT Cells—Besides calnexin, the only molecular chaperone reported to associate with murine class I chains is Bip, albeit weakly (9). As shown in Fig. 6A, expression of Bip proteins was significantly increased in BW PHAR2.7 cells relative to BW WT cells (Fig. 6A). Up-regulation of Bip in BW PHAR2.7 cells was specific in that equivalent amounts of calnexin were expressed in BW WT and BW PHAR2.7 cells (Fig. 6A; lane 2). Similarly, expression of an uncharacterized 44-kDa protein was similar in BW WT and BW PHAR2.7 cells (Fig. 6A, *), demonstrating that this 44-kDa protein is not primarily Bip.

Impaired formation of nascent H-2K\(^{k}\) complexes in cas-treated BW WT cells was associated with reduced Bip expression (Fig. 6B, *). Importantly, small yet detectable amounts of nascent H-2K\(^{k}\) molecules were observed to coprecipitate with Bip proteins in BW PHAR2.7 lysates but not BW WT lysates (Fig. 6B; arrow), indicating that Bip expression is markedly increased in BW PHAR2.7 cells relative to BW WT cells and provide evidence that nascent H-2K\(^{k}\) proteins made in BW PHAR2.7 cells associate with Bip molecules.

\(^2\) J. P. Balow and K. P. Kearse, unpublished observations.
DISCUSSION

The quality control system of the ER ensures that properly folded, fully assembled protein complexes are expressed on the cell surface. The molecular chaperone calnexin is believed to play a key role in maintaining the fidelity of class I complexes expressed on the cell surface by functioning in the assembly of nascent class I HC-β2m-peptide complexes within the ER and regulating their transport to the Golgi compartment. The data in the current study show that: (i) removal of Glc residues from core glycans is required for efficient interaction of nascent H-2Kk proteins with calnexin; (ii) calnexin association is not essential for stable expression of H-2Kk complexes; (iii) expression of Bip chaperones is markedly increased in glucosidase II-deficient BW PHAR2.7 cells; and (iv) H-2Kk proteins were associated with Bip proteins in BW PHAR2.7 cells but not BW WT cells.

Recent evidence indicates that initial association of oligomeric glycoproteins with calnexin requires recognition of monoglucosylated GlcMan2GlcNAc2 species on glycoproteins (12, 14). In agreement with this idea, we found that nascent H-2Kk chains synthesized in BW WT thymoma cells, but not in cas-treated BW WT cells or in glucosidase II-deficient BW PHAR2.7 cells, associated with calnexin. Degradation of murine and human MHC class I proteins under conditions of impaired glucosidase activity has been reported (23, 24). As the Abs used in our studies effectively recognize β2m-assembled H-2Kk chains, but not unassembled H-2Kk chains, it remains to be determined if reduced formation of H-2Kk complexes in cas-treated BW WT cells results from rapid degradation of nascent H-2Kk HC or the failure of H-2Kk HC to assemble effectively with β2m molecules. Regardless, our data clearly show that Glc trimming and calnexin association are not essential for stable expression of H-2Kk complexes as H-2Kk proteins are effectively expressed in glucosidase II-deficient BW PHAR2.7 thymoma cells.

Maintenance of class I expression is important for immune surveillance function within the body. The data in the current study show that alternative folding pathways exist within the ER that sustain expression of class I protein complexes under conditions where Glc trimming and calnexin association are impaired. Unlike H-2Kk proteins, H-DP proteins were not stably expressed on BW PHAR2.7 cells, indicating that assembly mechanisms exist within the ER of BW PHAR2.7 cells that function effectively for the oligomerization of H-2Kk proteins but not H-2D proteins. Regarding this issue, we have considered the possibility that stable expression of H-2Kk proteins in BW PHAR2.7 cells results from mutations introduced during their selection procedure (18) that allow H-2Kk polypeptides made in BW PHAR 2.7 cells to uniquely assemble with β2m proteins and processed peptide in the absence of Glc trimming and calnexin association. While the current studies do not exclude this possibility, preliminary sequencing data suggest that H-2 K k molecules expressed in BW WT and BW PHAR2.7 cells are identical. 3

The molecular basis for proficient expression of H-2Kk complexes in BW PHAR2.7 cells is unclear, but as suggested in this report may involve interaction with Bip proteins. Unlike human class I molecules which stably interact with Bip proteins (9, 25), association with murine class I molecules with Bip is controversial as Bip associates weakly with endogenous mouse class I proteins (9) and fails to interact with mouse gene products expressed in human cell lines (25). Indeed, no association between Bip and H-2Kk proteins in BW WT thymoma cells was observed in our studies (Fig. 5B). It is well documented that Bip expression increases during periods of ER stress and is stimulated by treatment with compounds that interfere with glycan addition or processing of oligosaccharide side chains (26, 27). The current report shows that expression of Bip proteins is markedly elevated in glucosidase II-deficient BW PHAR2.7 cells relative to BW WT cells, suggesting that persistence of diglucosylated glycans on nascent glycoproteins results in the accumulation of numerous malfolded proteins within the ER, which, in turn, results in increased Bip synthesis via feedback regulatory mechanisms (28). Thus, it is conceivable that quantitatively increased levels of Bip proteins in BW PHAR2.7 cells represent the molecular basis for effective expression of H-2Kk complexes in BW PHAR2.7 cells. As we were unable to determine if H-2Kk proteins associated with Bip in BW PHAR2.7 cells represent intermediates in the assembly of H-2Kk-β2m-peptide complexes or end-stage H-2Kk proteins targeted for degradation, the fate of Bip-associated H-2Kk proteins in BW PHAR2.7 cells is unclear. Alternatively, it is reasonable to speculate that stable expression of H-2Kk proteins in BW PHAR2.7 cells results from their association with an unknown chaperone protein(s) that, like Bip, functions independently of glycan processing and is up-regulated during periods of ER stress. We were unsuccessful in our attempts to sustain expression of H-2Kk complexes in BW WT cells by extended culture in parentheses; Drs. Richard Hodes, Jeroen Van Leeuwen, David Segal, and Dinah Singer for critical reading of the manuscript; Drs. Dinah Singer, Hidde Ploegh, and David B. Williams for helpful discussion; Dr. David B. Williams for the gift of anti-calnexin Ab and Dr. Rosalind Kornfeld for the gift of BW WT and BW PHAR2.7 cells.

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REFERENCES

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