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Review

Chiral separation principles in capillary electrophoresis

Gerald Gübitz*, Martin G. Schmid

Institute of Pharmaceutical Chemistry, Karl-Franzens University of Graz, Universitätsplatz 1, A-8010 Graz, Austria

Abstract

Capillary electrophoretic techniques for the separation of enantiomers are reviewed. The techniques used for chiral separation include capillary zone electrophoresis, electrokinetic chromatography, isotachophoresis, capillary gel electrophoresis and capillary electrochromatography. The separation principles and the chiral recognition mechanism are discussed and a comprehensive collection of applications to drugs and other compounds of interest is given in tables. © 1997 Elsevier Science B.V.

Keywords: Reviews; Enantiomer separation; Capillary electrophoresis; Electrochromatography; Drugs; Pesticides; Amino acids

Contents

*Corresponding author.

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1. Introduction

The development of methods for the separation of enantiomers has attracted great interest in the past twenty years, since it became evident that the biological or pharmacological activity of compounds of biological or pharmaceutical interest is mostly restricted to one of the enantiomers. There can be qualitative and quantitative differences in the pharmacological activity. The pharmacologically inactive enantiomer can exhibit unwanted side effects, antagonistic activities or even toxic effects. Even if these side effects are not drastic, the unwanted enantiomer has to be metabolized in the organism and represents an unnecessary burden for the organism. Therefore, there is considerable pressure to develop analytical methods for enantiomer separation for enantiomeric purity control, pharmacological studies, pharmacodynamic investigations, clinical studies, etc. Chromatographic techniques such as thin-layer chromatography (TLC), gas chromatography (GC), supercritical fluid chromatography (SFC) and, above all, high-performance liquid chromatography (HPLC), frequently have been used for chiral separations. Capillary electrophoresis (CE) has been found to be a powerful alternative to chromatographic techniques and several chiral separation principles successfully applied in HPLC have been transferred to CE.

The advantages of CE are the small amounts of chiral selector and solvents required. This permits the use of expensive reagents and makes it easy to change the selector and the electrolyte when screening for a suitable selector and conditions. Furthermore, only small sample volumes are required and efficiency is very high.

The most frequently applied CE techniques for chiral separations are capillary zone electrophoresis (CZE), with the addition of a chiral selector to the background electrolyte (BGE), electrokinetic chromatography (EKC), using a chiral pseudostationary phase or a charged chiral selector, and capillary gel electrophoresis (CGE), with incorporation of a chiral selector into a gel. Only a few papers deal with isotachophoresis (ITP) for chiral separation. This technique, however, might gain in interest for sample clean-up and preconcentration when used with ITP–CE coupled techniques.

A recent trend is capillary electrochromatography (CEC), where chiral-coated capillaries or capillaries packed with a chiral stationary phase are used.

The growing interest in using CE for chiral separations is reflected in the dramatically increasing number of publications during the past seven years (Fig. 1).

Since several excellent reviews on chiral separations by CE have appeared in the past few years [1–8], this review will mainly be devoted to recent developments and applications. A comprehensive collection of applications to drugs, pesticides, amino acids and other compounds of interest, however, is given in Tables 1–4. To make it easier for the reader to find a suitable method for a compound of interest, the compounds are listed alphabetically in the tables

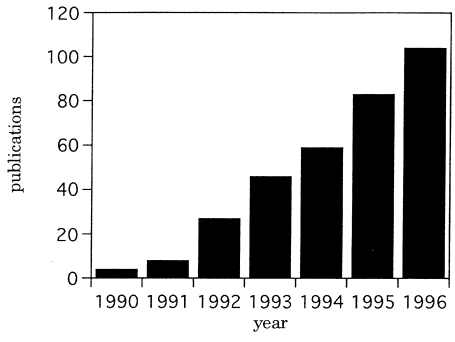

Fig. 1. Number of publications/year dealing with chiral separations by CE.

Table 1
Enantiomer separation of drugs by CE using different selectors

Drug	Selector	BGE	Reference
Acebutolol	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	DDCV (25 m*M*)	25 m*M* borate–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	Transferrin (0.1–0.3 g/ml region)	0.1 *M* MES, pH 6	[174]
	AGP (1 m*M* region)	50 m*M* phosphate, pH 6	[171]
	β-CD-Phos (10 m*M*) or SBE-β-CD (3 m*M*)	50 m*M* phosphate, pH 5	[93]
Acenocoumarol	SBE-β-CD (6 mg/ml)	50 m*M* phosphate, pH 6	[88]
	MeNH-β-CD (5 m*M*) or	50 m*M* phosphoric acid, 50 m*M* acetic acid	[74]
	$(MeNH)_7$-β-CD (5 m*M*)	50 m*M* boric acid and NaOH, pH 7	
N-Acetyl-3-mercaptovaline	DR: OPA–L-Phe or L-Tyr	MEKC	[17]
	DR: NDA–L-Phe or L-Tyr	MEKC	[17]
O-Acetylpseudoephedrine	β-CD (10 m*M*)	5 m*M* sodium acetate, pH 5.48	[26]
	10 m*M* β-alanine		
Aldose reductase inhibitor	β-CD or DM-β-CD or	20 m*M* Tris–10 m*M* phosphate, pH 11	[251]
	HE-β-CD or HP-β-CD (9 m*M*)		
Alimemazine	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
Alprenolol	HP-β-CD (20 m*M*)	50 m*M* phosphate, pH 2.5	[48]
	70 m*M* TMA		
	DM-β-CD (15 m*M*)	0.1 *M* TEA–phosphate, pH 3, 30% MeOH	[44]
	β-CD, DM-β-CD or HP-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	Cellobiohydrolase I (40 ng/ml)	0.4 *M* phosphate, pH 5.1, 25% 2-propanol	[168]
	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	Succ-β-CD (3.3%)	50 m*M* borate, pH 7.3	[83]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	DDCV (25 m*M*)	25 m*M* borate-10 m*M* TEA, pH 8.8, 25% ACN	[210]
	AGP–CSP (packed)	4 m*M* phosphate, pH 6.8, 4% 2-propanol	[176]
	HP-β-CD (120 m*M*)	0.1 *M* citric acid–phosphate, pH 2.5	[86]
Ambucetamide	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
3-Aminoalkyl-6-carboxamido-1,2,3,4-tetrahydrocarbazole	β-CD (20 m*M*), STDC (50 m*M*)	30 m*M* phosphate–10 m*M* boric acid, pH 7.02	[300]
Aminoglutethimide	α-CD or γ-CD (10 or 5 m*M*)	phosphate–borate, pH 7	[253]
	CM-β-CD (5 m*M*) and β-CD (1 m*M*)	phosphate–borate, pH 9, 50% MeOH	[253]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	DDCV (80 m*M*)	25 m*M* phosphate–borate, pH 9.25	[206]
	18C6TCA (10 m*M*)	20 m*M* Tris–phosphate, pH 2.06	[147]
	SBE-γ-CD (1 m*M*) or γ-CD (20 m*M*)	40 m*M* phosphate, pH 3	[92]
	β-CD-Phos (3 m*M*)	50 m*M* phosphate, pH 5	[93]
	SBE-β-CD (3 m*M*), CM-γ-CD (1 m*M*)	50 m*M* phosphate, pH 5	[93]
Aminoglutethimide analogues	γ-CD or	50 m*M* phosphate, pH 2.5	[105]
	DM-β-CD (15 m*M*)		
Aminopromazine	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
Amphetamine	DM-β-CD (5 m*M*)	98.8% 25 m*M* Tris–phosphate	[91]
		pH 2.45, 1.2% MeOH	
	β-CD polymer (0.2 g/l)	50 m*M* phosphate, pH 2.5	[52]
	SBE-β-CD (1 m*M*)	98.8% 25 m*M* Tris–phosphate,	[91]
		pH 2.4, 1.2% MeOH	
	SBE-β-CD (4.6 m*M*)	20 m*M* citric acid–phosphate, pH 2.5	[86]
	Rifamycin B (25 m*M*)	0.1 *M* phosphate, pH 7, 30% 2-propanol	[128]
	DR: OPA–*N*-acetyl-L-cysteine	MEKC	[17]
	Various methylated CDs (7.5–30 m*M*)	30 m*M* Tris, pH 2.4	[41]
	Poly-β-CD (0.1 g/l)	50 m*M* phosphate, pH 6	[55]

(*continued on p. 182*)

Table 1. Continued

Drug	Selector	BGE	Reference
Amphetamine and analogues (methamphetamine, 4-hydroxy-amphetamine, 4-methoxy-amphetamine,	β-CD or DM-β-CD or TM-β-CD or HP-β-CD (10 m*M*)	50 m*M* phosphate, pH 2.5	[10,58]
3,4-methylene-methamphetamine, 3,4-methylenedioxy-ethamphetamine, 2,5-dimethoxy-amphetamine, 2,5-dimethoxy-metamphetamine, 4-bromo-2,5-dimethoxy-amphetamine)	β-CD (10 m*M*)	50 m*M* TEA–phosphate, pH 2	[60]
	DR: Marfey's reagent	MEKC	[10]
	DR: GITC	10 m*M* phosphate–borate, pH 9, 0.1 *M* SDS	[12]
Amphetamine and analogues (methyldimethoxyethylamphetamine, methyldimethoxymethylamphetamine, methylamphetamine, ephedrine, epinephrine, pseudoephedrine)	HP-β-CD (120 m*M*)	0.1 *M* citric acid–20 m*M* phosphate, pH 2.5	[86]
Amphetamine analogues (methyldimethoxymethylamphetamine, methylamphetamine, methyldimethoxyethylamphetamine)	HP-β-CD (60 or 120 m*M*)	50 m*M* borate, pH 9.5, 5% 1-propanol, [116] 50 m*M* STDC	
Anisodamine	HP-β-CD (30 m*M*)	0.1 *M* Tris–phosphate, pH 2.3	[255]
Arabasine	Heparin (2%)	10 m*M* phosphate, pH 5	[187]
Arotinolol	Ovomucoid (0.5 m*M*)	50 m*M* phosphate, pH 5, 6% 2-propanol	[160]
	AGP (0.5 m*M* region)	50 m*M* phosphate, pH 6, 10% 2-propanol	[171]
Arterenol	CM-β-CD (2%)	20 m*M* citric acid, pH 2.5	[87]
Atenolol	M-β-CD (24 m*M*) or DM-β-CD (28 m*M*)	20 m*M* Tris–phosphate, pH 2.4	[251]
	M-β-CD (37 m*M*)	40 m*M* lithium phosphate, pH 3	[32]
	DM-β-CD (40 m*M*)	50 m*M* lithium phosphate, pH 2.5	[33]
	β-CD (12 m*M*)	40 m*M* borate, pH 9.3, 32 m*M* SDS	[107]
	DM-β-CD (30 m*M*)	0.1 *M* TEA–phosphate, pH 3	[44]
	DM-β-CD, HP-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	β-CD polymer (0.1 g/ml)	50 m*M* phosphate, pH 2.5	[52]
	SBE-β-CD (2 or 4.6 m*M*)	20 m*M* citric acid–phosphate, pH 2.5	[86]
	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	Sulfated β-CD (2%)	10 m*M* phosphate buffer, pH 3.8	[97]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	STDC (50 m*M*)	16 m*M* NaCl and 50 m*M* polyoxyethyleneether	[224]
	CSA (30 m*M*)	1 *M* acetic acid, 0.2 m*M* Tween 20, ACN	[249]
	SBE-β-CD (2 m*M*)	50 m*M* phosphate, pH 5	[93]
Atropine	HP-β-CD (15–100 m*M*)	0.1 *M* TAPS, pH 8–10	[256]
	AGP (0.75 m*M* region)	50 m*M* phosphate, pH 6, 10% 2-propanol	[171]
Azelastine	β-CD-Phos (1 m*M*)	50 m*M* phosphate, pH 7	[93]
	CM-γ-CD (1 m*M*)	50 m*M* phosphate, pH 5	[93]
Baclofen (CBI)	β-CD (20 m*M*) 50 m*M* STDC	30 m*M* phosphate, 10 m*M* boric acid, pH 7,	[115]
	18C6TCA (10 m*M*)	20 m*M* Tris–phosphate, pH 2.06	[147]
Bambuterol	DM-β-CD or TM-β-CD (15 or 5 m*M*)	50 m*M* phosphate, pH 2.5	[257]
	HP-β-CD (60 m*M*)	phosphate, pH 3, 20 m*M* TBA	[50]
Bamethan	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
Bendroflumethiazide	Vancomycin (2 m*M*)	50 m*M* phosphate, pH 7, 25 m*M* SDS	[136]
Benzetimide	HP-β-CD (10 m*M*)	citrate–NaOH–HCl, pH 4	[258]
Bifonazole	SBE-β-CD (0.1 m*M*)	50 m*M* phosphate, pH 3, 20% MeOH	[259]

Table 1. Continued

Drug	Selector	BGE	Reference
Bisoprolol	CSA (30 m*M*)	1 *M* acetic acid, 0.2 m*M* Tween 20, ACN	[249]
	γ-CD-Phos (5 m*M*)	50 m*M* phosphate, pH 5	[93]
Bromacil	Vancomycin (2 m*M*)	50 m*M* phosphate, pH 7, 25 m*M* SDS, 5% MeOH	[136]
Bromodiphenhydramine	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
Brompheniramine	Heparin (2%)	10 m*M* phosphate, pH 5	[187]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
Bunitrolol	Ovomucoid (0.5 m*M*)	50 m*M* phosphate–10 m*M* CHAPS, pH 5	[160]
	CSA (30 m*M*)	1 *M* acetic acid, 0.2 m*M* Tween 20, ACN	[249]
	β-CD-Phos (5 m*M*)	50 m*M* phosphate, pH 5	[93]
	SBE-β-CD (3 m*M*)	50 m*M* phosphate, pH 5	[93]
	γ-CD-Phos (10 m*M*)		
Buphenine	Transferrin (0.1–0.3 g/ml, region)	0.1 *M* MES, pH 6	[174]
Bupivacaine	DM-β-CD (10 m*M*)	18 m*M* Tris–phosphate, pH 2.9, 0.1% MHEC,	[260]
	0.03 m*M* HTAB		
	DM-β-CD (30 m*M*)	0.1 *M* TEA–phosphate, pH 3	[44]
	β-CD polymer (0.2 g/ml)	50 m*M* phosphate, pH 2.5	[52]
	0.1 *M* SDS, 2 *M* urea, 15% MeOH		
	SBE-β-CD (10 mg/ml)	50 m*M* phosphate, pH 6	[88]
	Heparin (2%)	10 m*M* phosphate, pH 5	[187]
	Sulfated β-CD (2%)	10 m*M* phosphate buffer, pH 3.8	[97]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	STDC (15 m*M*)	phosphate, pH 3.13 (4 m*M* Brij-35)	[222]
	Transferrin (0.1–0.2 g/ml, region)	0.1 *M* MES, pH 6	[173]
	AGP (0.5 m*M* region)	50 m*M* phosphate, pH 5, 10% 1-propanol	[171]
	β-CD-Phos (10 m*M*), γ-CD-Phos (5 m*M*)	50 m*M* phosphate, pH 5	[93]
	SBE-β-CD (3 m*M*), CM-γ-CD (10 m*M*)	50 m*M* phosphate, pH 5	[93]
	DM-β-CD (10 m*M*)	0.1 m*M* TEA–phosphoric acid, pH 3	[262]
Bupropion	Sulfated β-CD (2%)	10 m*M* phosphate buffer, pH 3.8	[97]
Butabarbital	β-CD (30 m*M*)	20 m*M* phosphate–borate, pH 7	[105]
Butaclamol	SBE-γ-CD (0.1 m*M*) or γ-CD (20 m*M*)	40 m*M* phosphate, pH 3	[92]
Butetamate	β-CD (1.8%), CSA (40 m*M*)	0.1 *M* phosphate, pH 2	[110]
Canadine	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Carbinoxamine	β-CD (30 m*M*)	0.1 *M* phosphate, pH 2.5, 1.5 *M* urea	[263]
	Heparin (2%)	10 m*M* phosphate, pH 5	[187]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Carprofen	β-CD (coated Chirasil-Dex) CEC	20 m*M* borate–phosphate, pH 7	[264]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[131]
	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 7	[124]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6, (ACN)	[127,132]
	TM-β-CD (10 m*M*)	75 m*M* formate, pH 4	[266]
	β-CD-NH_2 (20 m*M*)+TM-β-CD (10 m*M*)	34 m*M* phosphate, pH 2.3	[76]
Carnithine	DR: FLEC	50 m*M* phosphate, pH 2.6, 20 m*M* TBAB	[23]
Carvedilol	DM-β-CD (10 m*M*)	18 m*M* Tris–phosphate, pH 2.9,	[260]
	0.1% MHEC, 0.03 m*M* HTAB		
	β-CD (16.3 m*M*)	50 m*M* phosphate, pH 3.3	[252]
Cathinone	DM-β-CD (5 m*M*)	90% (25 m*M* Tris–phosphate, pH 2.45)	[91]
		10% MeOH	
	SBE-β-CD (1 m*M*)	98.8% 25 m*M* Tris–phosphate, pH 2.4,	[91]
		1.2% MeOH	
Chloramphenicol	DM-β-CD (10 m*M*)	20 m*M* Tris–citric acid, pH 3.5, 0.1% HEC	[28]
Chlorcyclizine	γ-CD or M-β-CD (0.1 *M*)	FA, 0.1 *M* Tris 0.15 *M* citric acid, pH 5.1	[248]
Chlormezanone	SBE-β-CD (5 m*M*)	50 m*M* phosphate, pH 5	[93]

(*continued on p. 184*)

Table 1. Continued

Drug	Selector	BGE	Reference
Chlorodiltiazem	Chondroitin sulfate (3%)	20 m*M* phosphate–borate, pH 2.4	[189]
	Heparin (3%)	20 m*M* phosphate–borate, pH 6	[189]
	Dextrin (3%)	20 m*M* phosphate, pH 2.5	[185]
p-Chlorophenprocoumon	Glucidex 2 (3%)	10 m*M* Tris–phosphate, pH 7	[182]
Chloroquine	Heparin (2%)	10 m*M* phosphate, pH 5	[187,188]
	Sulfated β-CD (2%)	10 m*M* phosphate buffer, pH 3.8	[97]
	Dextransulfate (2%)	50 m*M* phosphate, pH 6	[188]
p-Chlorowarfarin	Glucidex 2 (3%)	10 m*M* Tris–phosphate, pH 7	[182]
	Dextrin (6%)	20 m*M* phosphate, pH 2.5	[185]
Chlorphenamine	Transferrin (0.1–0.3 g/ml region)	0.1 *M* MES, pH 6	[174]
Chlorpheniramine	β-CD (0.1 *M*)	pH 3, 5 *M* urea, 50 m*M* SDS	[106]
	β-CD (30 m*M*)	0.1 *M* phosphate, pH 2.5, 10% MeOH, 1.5 *M* urea	[263]
	HP-β-CD or β-CD (30 or 15 m*M*)	0.1 *M* TEA–phosphate, pH 3	[44]
	β-CD, DM-β-CD, TM-β-CD, HP-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	Heparin (2%)	10 m*M* phosphate, pH 5	[187,188]
	Heparin (3%)	20 m*M* phosphate–borate, pH 6	[189]
	Ovomucoid (0.25 m*M*)	10 m*M* phosphate, pH 5, 9% 2-propanol	[167]
	Ovomucoid (0.5 m*M*)	50 m*M* phosphate, pH 5, 8% 1-propanol	[160]
	MeNH-β-CD (5 m*M*)	0.1 *M* phosphoric acid–TMA, pH 2.5	[74]
	Sulfated β-CD (2%)	10 m*M* phosphate buffer, pH 3.8	[97]
	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
	Dextransulfate (2%)	50 m*M* phosphate, pH 5	[188]
	SBE-β-CD (0.2 m*M*), CM-γ-CD (1 m*M*) γ-CD-Phos (1 m*M*)	50 m*M* phosphate, pH 5	[93]
Chlorprenaline	AGP (0.5 m*M*)	50 m*M* phosphate, pH 4	[160]
	AGP (0.3 m*M* region)	50 m*M* phosphate, pH 6, 10% EtOH	[171]
	β-CD-Phos (0.2 m*M*)	50 m*M* phosphate, pH 5	[93]
	SBE-β-CD (0.2 m*M*), CM-γ-CD (2 m*M*) γ-CD-Phos (0.5 m*M*)	50 m*M* phosphate, pH 5	[93]
Chlortalidone	β-CD-NH_2 (5 m*M*)	39.1 m*M* phosphate–18 m*M* ammediol, pH 2.3	[75]
	Succ-β-CD (3.3%)	50 m*M* borate, pH 7.3	[83]
	HP-β-CD–CSP (packed)	CEC: 5 m*M* phosphate, pH 6.5 ACN	[246]
	HP-β-CD/ODS	CEC: 1 m*M* phosphate, pH 6.5 ACN	[246]
Cicletanine	γ-CD (25 m*M*)	0.1 *M* borate, pH 8.6, 0.11 *M* SDS, 10% ACN	[219]
Cicloprofen	β-CD (coated Chirasil-Dex) CEC	20 m*M* borate–phosphate, pH 7	[264]
Cimaterol	SBE-β-CD (2 m*M*)	20 m*M* citric acid–phosphate, pH 2.5	[86]
	HP-β-CD (120 m*M*)	0.1 *M* citric acid–phosphate, pH 2.5	[86]
	HP-β-CD (60 or 120 m*M*)	50 m*M* borate, pH 9.5, 5% 1-propanol, 50 m*M* STC	[116]
Cinchonine/cinchonidine[a]	Lambda-carrageenan (0.28%)	25 m*M* citric acid–29 m*M* Tris, pH 7.2	[191]
Clenbuterol	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	β-CD (16 m*M*)	0.2 *M* phosphate–0.1 *M* citric acid, pH 4	[307]
	HP-β-CD (30 m*M*)	50 m*M* borate–phosphate, pH 2.2	[268]
	β-CD (6–16 m*M*)	phosphate, pH 2.5, 4, 5.5, 0–8% MeOH	[269]
	HP-β-CD (60 m*M*)	phosphate, pH 3, 20 m*M* TBA	[50]
	β-CD polymer (0.1 g/l)	50 m*M* phosphate, pH 2.5	[52]
	SBE-β-CD (2 or 4.6 m*M*)	20 m*M* citric acid–phosphate, pH 2.5	[86]
	SBE-β-CD (1 m*M*)	50 m*M* phosphate, pH 3.1	[90]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	Dextrinsulfopropylether (4%)	80 m*M* Tris–phosphate, pH 3	[190]
	Various methylated CDs (7.5–30 m*M*)	30 m*M* Tris, pH 2.4	[41]
	Poly-β-CD (0.1 g/l)	50 m*M* phosphate, pH 6	[55]

Table 1. Continued

Drug	Selector	BGE	Reference
	HP-β-CD (120 m*M*)	0.1 *M* citric acid–phosphate, pH 2.5	[86]
	HP-β-CD (60 or 120 m*M*)	50 m*M* borate, pH 9.5, 5% 1-propanol, 50 m*M* STC	[116]
Clentiazem	Dextrin (3%)	20 m*M* phosphate, pH 2.5	[185]
Clentiazem intermediate	DEAE–dextran (3%)	20 m*M* phosphate, pH 8	[138]
	Dextrin (3%)	20 m*M* phosphate, pH 2.5	[185]
Clidiniumbromide	γ-CD (15 m*M*)	0.1 m*M* phosphate, pH 2.5	[63]
	α-CD (15 m*M*)	0.1 *M* phosphate, pH 2.5	[61]
Clofedanol	β-CD (100 m*M*)	FA, 0.1 *M* Tris 0.15 *M* citric acid, pH 5.1	[248]
	Transferrin (0.1–0.3 g/ml region)	0.1 *M* MES, pH 6	[174]
Cocaine	DM-β-CD (5 m*M*)	98.8% (25 m*M* Tris–phosphate, pH 2.45) 1.2% MeOH	[91]
Coumachlor	Vancomycin (2 m*M*)	50 m*M* phosphate, pH 7, 25 m*M* SDS, 10% ACN	[136]
	poly-(L-SUVal) (0.5%)	25 m*M* phosphate, pH 5.9	[203]
5-Cyclobutyl-5-phenyl-hydantoin	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Cyclodrine	TMA-β-CD (16 m*M*)	50 m*M* phosphate, pH 2.5	[79]
	β-CD (3%), CSA (40 m*M*)	0.15 *M* phosphate, pH 2, 5% urea	[109]
Cyclohexylglutarimide	DDCV (80 m*M*)	25 m*M* phosphate–borate, pH 9.25	[206]
Cyclopentolate	TMA-β-CD (16 m*M*)	50 m*M* phosphate, pH 2.5	[79]
	β-CD (1.8%), CSA (40 m*M*)	0.1 *M* phosphate, pH 2	[110]
Cyclophosphamide	AGP–CSP (packed)	2 m*M* phosphate, pH 5.5, 3% 2-propanol	[176]
Denopamine	DM-β-CD (20 m*M*)	25 m*M* phosphate, pH 2.7, 2 *M* urea	[53,270,279]
	AGP (0.5 m*M* region)	50 m*M* phosphate, pH 6, 10% methanol	[171]
	SBE-β-CD (0.5 m*M*), γ-CD-Phos (1 m*M*)	50 m*M* phosphate, pH 5	[93]
Deprenyl and its metabolites (amphetamine, methamphetamine, propargylamphetamine)	DM-β-CD (6 m*M*)	20 m*M* Tris–phosphate, 0.1% or 0.5% HPMC	[265]
Diclofensine	HP-β-CD (60 or 120 m*M*)	50 m*M* borate, pH 9.5, 5% 1-propanol, 50 m*M* STC	[116]
Dihydropyridine derivative	HP-β-CD (0.3%)	10 m*M* citric acid–Tris, pH 6	[254]
	HP-γ-CD (0.5%)	20 m*M* phosphate, pH 6	[254]
Disopyramide	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	AGP–CSP (packed)	4 m*M* phosphate, pH 6.8, 15% 2-propanol	[176]
Diltiazem	Chondroitin sulfate (3%)	20 m*M* phosphate–borate, pH 2.4	[189]
	Dextran sulfate (3%)	20 m*M* phosphate–borate, pH 5.5	[189]
	Dextrin (3%)	20 m*M* phosphate, pH 2.5	[185]
Diltiazem and analogues	STDC (50 m*M*)	20 m*M* phosphate–borate, pH 7	[220]
Diltiazem intermediate	DEAE–dextran (3%)	20 m*M* phosphate, pH 8	[138]
	Dextrin (3%)	20 m*M* phosphate, pH 2.5	[185]
Dimethindene	HP-β-CD (30 μg/ml)	50 m*M* phosphate, pH 3.3	[252]
	HP-β-CD (30 m*M*)	0.1 *M* phosphate–TEA, pH 3	[44]
	CM-β-CD (2%)	20 m*M* phosphate, pH 5.8	[87]
	CM-β-CD (2%)	20 m*M* citric acid, pH 2.5	[254]
	CM-β-CD (2%)	20 m*M* citric acid, pH 2.5	[271]
	SBE-β-CD (0.08 m*M*)	50 m*M* phosphate, pH 3.1	[90]
	Heparin (2%)	10 m*M* phosphate, pH 5	[187]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Diniconazole	γ-CD or DM-β-CD (50 m*M*)	0.1 *M* borate, pH 9, 0.1 *M* SDS, 2 *M* urea	[272,273]

(*continued on p. 186*)

Table 1. Continued

Drug	Selector	BGE	Reference
Doxylamine	CM-β-CD (2%)	20 m*M* phosphate, pH 5.8	[87]
	CM-β-CD (2%)	20 m*M* citric acid, pH 2.5	[254]
	CM-β-CD (2%)	20 m*M* citric acid, pH 2.5	[271]
	Heparin (2%)	10 m*M* phosphate, pH 5	[187]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Econazole	SBE-β-CD (0.1 m*M*)	50 m*M* phosphate, pH 3, 20% MeOH	[259]
Enilconazole	β-CD (20 m*M*) or γ-CD (50 m*M*)	50 m*M* phosphate, pH 3, 10% MeOH	[259]
	HP-β-CD (20 m*M*)	50 m*M* phosphate, pH 3, 10% MeOH	[259]
Enpiroline	Heparin (2%)	10 m*M* phosphate, pH 5	[187]
Eperisone	Ovomucoid (0.05 m*M*)	50 m*M* phosphate–0.5 *M* PEA, pH 5	[167]
	AGP (0.5 m*M* region)	50 m*M* phosphate, pH 6, 10% 1-propanol	[171]
	SBE-β-CD (5 m*M*), CM-γ-CD (1 m*M*)	50 m*M* phosphate, pH 5	[93]
	γ-CD-Phos (5 m*M*)		
Ephedrine	DM-β-CD (20 m*M*)	10 m*M* Tris–phosphate, pH 2.4	[30]
	DM-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	β-CD (20 m*M*)	0.15 *M* TBA–phosphate, pH 2.5	[47]
	DM-β-CD (50 m*M*)	50 m*M* Li phosphate, pH 2.5	[33]
	DM-β-CD (40 g/l)	30 m*M* Tris–phosphate, pH 2.5	[274]
	DM-β-CD (18 m*M*)	20 m*M* phosphate, pH 2.5, 10 m*M* TBAB, 0.1% HPC	[84]
	DM-β-CD or HP-β-CD (30 m*M*)	0.1 *M* phosphate–TEA, pH 3	[44]
	DM-β-CD (15 m*M*)	50 m*M* phosphate, pH 2.5	[257]
	β-CD, DM-β-CD or HP-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	DM-β-CD (20 m*M*)	20 m*M* phosphate, pH 2.5	[275]
	CM-β-CD (2%)	20 m*M* phosphate, pH 5.8	[87]
	CM-β-CD (2%)	20 m*M* citric acid, pH 2.5	[254,271]
	SBE-β-CD (1 m*M*)	98.8% 25 m*M* Tris–phosphate, pH 2.4, 1.2% MeOH	[91]
	SBE-β-CD (40 m*M*)	20 m*M* borate, pH 10	[84]
	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	SBE-β-CD (1.5 m*M*)	20 m*M* phosphate, pH 2.5	[85]
	Succ-β-CD (3.3%)	50 m*M* borate, pH 7.3	[83]
	DDCV (50 m*M*)	25 m*M* phosphate–borate, pH 8	[208]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	DR: GITC	10 m*M* phosphate–borate, pH 9, 0.1 *M* SDS	[12]
	DM-β-CD (18 m*M*)	45 m*M* Tris, pH 2.4	[51]
Epinastine	BSA (0.75 m*M*)	50 m*M* phosphate, pH 6	[160]
	AGP (0.1 m*M* region)	50 m*M* phosphate, pH 6, 10% 1-propanol	[171]
	SBE-β-CD (0.2 m*M*), CM-γ-CD (0.1 m*M*)	50 m*M* phosphate, pH 5	[93]
	γ-CD-Phos (0.1 m*M*)	50 m*M* phosphate, pH 5	[93]
Epinephrine	DM-β-CD (20 m*M*)	10 m*M* Tris–phosphate, pH 2.4	[30]
	DMβ-CD (20 m*M*)	0.1 *M* phosphate, pH 2.5	[276]
	DM-β-CD (20 m*M*)	50 m*M* phosphate, pH 2.5	[277]
	DM-β-CD (30 m*M*)	0.1 *M* phosphate, pH 2.5, 10% MeOH	[263]
	DM-β-CD or M-β-CD (9 m*M*)	20 m*M* Tris–phosphate, pH 2.3	[251,278]
	β-CD (20 m*M*)	0.15 *M* phosphate–TBA, pH 2.5	[47]
	β-CD, DM-β-CD or HP-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	β-CD polymer (0.1 g/ml)	50 m*M* phosphate, pH 2.5	[52]
	CEC: γ-CD (coated acrylamide)	20 m*M* borate–phosphate, pH 7, 10% MeOH	[245]
	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	SBE-β-CD (2.5 m*M*)	0.2 *M* phosphate buffer, pH 2.5	[85]
	Sulfated β-CD (9 m*M*)	10 m*M* phosphate, pH 3.2	[96]
	γ-CD/PAA (coated)	CEC: 20 m*M* borate–phosphate, pH 7, 10% 2-propanol	[245]
	Various methylated CDs (7.5–30 m*M*)	30 m*M* Tris, pH 2.4	[41]
	DM-β-CD (18 m*M*)	45 m*M* Tris, pH 2.4	[51]
	HP-β-CD (120 m*M*)	0.1 *M* citric acid–phosphate, pH 2.5	[86]

Table 1. Continued

Drug	Selector	BGE	Reference
Etilefrin	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	DM-β-CD	25 m*M* phosphate, pH 3, 2 *M* urea	[279]
	SBE-β-CD (1 m*M*)	50 m*M* phosphate, pH 3.1	[90]
	Succ-β-CD (3.3%)	50 m*M* borate, pH 7.3	[83]
	AGP (0.5 m*M* region)	50 m*M* phosphate, pH 6	[171]
	SBE-β-CD (1 m*M*) or γ-CD-Phos (5 m*M*)	50 m*M* phosphate, pH 5	[93]
Etodolac	M-β-CD or γ-CD (10 m*M*)	20 m*M* borate–phosphate, pH 7	[264]
	β-CD (coated Chirasil-Dex) CEC	20 m*M* borate–phosphate, pH 7	[264]
Etopropazine	HP-β-CD (100 m*M*)	FA, 0.1 *M* Tris–0.15 *M* citric acid, pH 5.1	[248]
	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
Fadrozole	β-CD or DM-β-CD (30 m*M*)	20 m*M* phosphate–borate, pH 7,	[105]
	0.1 *M* SDS, 2 *M* urea, 15% MeOH		
Fenfluramine	DM-β-CD (15 m*M*)	0.1 *M* phosphate–TEA, pH 3, 30% MeOH	[44]
meta-Fenfluramine	TM-β-CD (40 m*M*)	0.1 *M* phosphate–TEA, pH 2.5	[258]
ortho-Fenfluramine	γ-CD (30 m*M*)	0.1 *M* phosphate–TEA, pH 2.5	[258]
Fenoldopam and trimethyl derivative	β-CD (20 m*M*)-STDC (50 m*M*)	30 m*M* phosphate–10 m*M* boric acid, pH 7.2	[113]
Fenoprofen	β-CD (15 m*M*)	0.2 *M* MES, pH 4.5, 0.2% HEC	[34]
	HP-β-CD (20 m*M*)	0.2 *M* MES, pH 4.41, 0.2% HEC	[35]
	β-CD (15 m*M*)	0.6 *M* MES, pH 4.65, 0.2% HEC	[280]
	TM-β-CD (30 m*M*)	0.1 *M* MES, pH 5	[40]
	Dextrin (10%)	0.1 *M* phosphate–pyrophosphate, pH 6 or 7	[181]
	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,124]
	Ristocetin (2 m*M*)		[127,131]
	Teicoplanin (2 m*M*)		[127,132]
	MeNH-β-CD (5 m*M*) or	50 m*M* phosphoric acid, 50 m*M* acetic acid	[74]
	$(MeNH)_7$-β-CD (5 m*M*)	50 m*M* boric acid and NaOH, pH 7	
	β-CD (10 m*M*)	0.2 *M* MES, pH 4.6	[68]
Fenoterol	AGP (0.5 m*M* region)	50 m*M* phosphate, pH 6, 10% MeOH	[171]
	SBE-β-CD (5 m*M*)	50 m*M* phosphate, pH 5	[93]
	γ-CD-Phos (5 m*M*)		
Fluoxetine	TM-β-CD (10 m*M*)	18 m*M* Tris, pH 2.7, 0.1% MHEC, CTAB	[260]
	SBE-β-CD (7.5 m*M*)	1% Tris–acetic acid, pH 5.5 and 10% ACN	[281]
	Dextrin 10 (20%)	25 m*M* Tris–phosphate, pH 3.4	[180]
	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
Flurbiprofen	TM-β-CD (30 m*M*)	0.1 *M* MES, pH 5	[40]
	β-CD (coated Chirasil-Dex)	CEC: 20 m*M* borate–phosphate, pH 7	[264]
	Avidin (0.025 m*M*)	50 m*M* phosphate, pH 6, 10% ethanol	[166]
	Maltooligosaccharides (2.5–10%)	10 m*M* phosphate, pH 7	[179]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[131]
	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 7 (SDS)	[124,129]
	Dextrin (6%)	20 m*M* phosphate, pH 2.5	[185]
	β-CD-NH_2 (20 m*M*)+TM-β-CD (10 m*M*)	34 m*M* phosphate, pH 2.3	[76]
Folinic acid[a]	Avidin (0.025 m*M*)	50 m*M* phosphate, pH 6, 10% ethanol	[166]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[131]
	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 7	[124]
Formoterol	Dextrinsulfopropylether (4%)	80 m*M* Tris–phosphate, pH 3	[190]
Gallopamil	HP-β-CD (20 m*M*)	0.1 *M* phosphate–TEA, pH 3	[44]
Glutethimide	DM-β-CD (15 m*M*)	50 m*M* phosphate, pH 2.5	[105]
	DDCP (25 m*M*)	25 m*M* phosphate–borate, pH 8.8	[209]
	PMPC (wallcoated)	CEC: 40 m*M* phosphate, pH 7, 20% ACN	[192]
	Rifamycin SV (25 m*M*)	0.1 *M* phosphate, pH 7, 30% 2-propanol	[128]

(*continued on p. 188*)

Table 1. Continued

Drug	Selector	BGE	Reference
Glycopyrronium	HP-β-CD (12 m*M*)	0.1 *M* Tris–phosphate, pH 2.3	[255]
Heptaminol	DR: OPA–*N*-acetyl-L-cysteine	MEKC	[17]
Hexobarbital	α-CD or β-CD,	50 m*M* phosphate–10 m*M* borate	[264]
	DM-β-CD (10 m*M*)	pH 9	
	β-CD or DM-β-CD,	100 m*M* Tris–boric acid, pH 8.3,	[254]
	HP-β-CD (0.5–1.3%)	2 m*M* EDTA	
	β-CD or DM-β-CD (30 m*M*)	20 m*M* phosphate–borate, pH 7,	[105]
	0.1 *M* SDS, 2 *M* urea, 15% MeOH		
	β-CD (packed)	CEC: 4 m*M* phosphate, pH 6.8, 5% ACN	[177]
	CEC: 5 m*M* TEAA, 15% MeOH,, pH 4.71		[177]
	CE-β-CD (2%)	20 m*M* phosphate buffer, pH 5.8	[87]
	AGP–CSP (packed)	2 m*M* phosphate, pH 5.5, 2% 2-propanol	[176]
	Rifamycin SV (25 m*M*)	0.1 *M* phosphate, pH 7, 30% 2-propanol	[128]
	Chirasil-Dex (coated)	borate–phosphate, pH 7	[243]
Homatropin	β-CD (15 m*M*)	35 m*M* phosphate, pH 6.25, 0.2% HEC	[282]
	Succ-β-CD (3.3%)	50 m*M* borate, pH 7.3	[83]
	AGP (0.05 m*M* region)	50 m*M* phosphate, pH 5, 10% 1-propanol	[171]
	HP-β-CD (10 m*M*)	25 m*M* phosphate, pH 2.5	[67]
Homochlorcyclizine	BSA (0.5 m*M*)	50 m*M* phosphate, pH 6	[160]
Hydantoin derivatives	Sulfated β-CD (3%)	10 m*M* phosphate, pH 8, 10% MeOH	[95]
Hydroxychloroquine	Heparin (2%)	10 m*M* phosphate, pH 5	[187]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Hydroxyzine	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
5-(4-Hydroxyphenyl)-5-phenyl-hydantoin	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Ibuprofen	β-CD (15 m*M*)	200 m*M* MES, pH 4.5, 0.2% HEC	[34,283]
	TM-β-CD (30 m*M*)	100 m*M* MES, pH 5	[40]
	β-CD or γ-CD (coated Chirasil-Dex)	20 m*M* borate–phosphate, pH 7	[264]
	Avidin (0.025 m*M*)	50 m*M* phosphate, pH 6, 10% ethanol	[166]
	BSA (1 mg/ml)	10 m*M* phosphate, pH 7.12, 5% dextran	[157]
	Dextrin 10 (15 m*M*)	20 m*M* TAPS–6.5 m*M* Tris, pH 7.7, 4% ethanol	[180]
	Maltooligosaccharides (2.5–10%)	10 m*M* phosphate, pH 7	[179]
	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 7	[124]
	Dextrin (6%)	20 m*M* phosphate, pH 2.5	[185]
	β-CD (15 m*M*)	0.2 *M* MES, pH 4.6	[67]
Idazoxan	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Imafen	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	SBE-β-CD (1 m*M*)	50 m*M* phosphate, pH 3.1	[90]
Imazalil	β-CD-Phos (0.2 m*M*)	50 m*M* phosphate, pH 5	[93]
	SBE-β-CD (5 m*M*) or CM-γ-CD (0.5 m*M*)	50 m*M* phosphate, pH 5	[93]
Indapamide	Sulfated β-CD (2%)	10 m*M* phosphate, pH 8	[95]
Indoprofen	TM-β-CD (30 m*M*)	0.1 *M* MES, pH 5	[40]
	Ristocetin (2 m*M*)	0.1 *M* phosphate buffer, pH 6	[131]
	Vancomycin (2 m*M*)	0.1 *M* phosphate buffer, pH 7 (SDS)	[124,129]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate buffer, pH 6	[127,132]
	TM-β-CD (10 m*M*)	35 m*M* formate, pH 4	[266]
Isolysergic acid	γ-CD (30 m*M*)	0.1 *M* phosphate, pH 2.5	[284]
Isoprenaline	DM-β-CD or HP-β-CD (15 or 30 m*M*)	0.1 *M* phosphate–TEA, pH 3	[44]
	SBE-β-CD (1 m*M*)	50 m*M* phosphate, pH 3.1	[90]
Isoproterenol	DM-β-CD (18 m*M*)	10 m*M* Tris–phosphate, pH 2.4	[30]
	DM-β-CD (20 m*M*)	50 m*M* phosphate, pH 2.5	[277]
	β-CD (20 m*M*)	0.15 *M* phosphate–TBA, pH 2.5	[47]
	DM-β-CD (20 m*M*)	50 m*M* phosphate, pH 2.5, 70 m*M* TMA	[48]
	DM-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]

Table 1. Continued

Drug	Selector	BGE	Reference
	β-CD or DM-β-CD, TM-β-CD or HP-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	Sulfated β-CD (9 m*M*)	10 m*M* phosphate, pH 3.2	[96]
	MeNH-β-CD (5 m*M*)	0.1 *M* phosphoric acid–TMA, pH 2.5	[74]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	DM-β-CD (18 m*M*)	45 m*M* Tris, pH 2.4	[51]
	DM-β-CD (30 m*M*)	25 m*M* phosphate, pH 2.5	[285]
Isothipendyl	γ-CD (15 m*M*)	0.1 m*M* phosphate, pH 2.5	[63]
Isoxuprine	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Ketamine	DM-β-CD (20 m*M*)	50 m*M* phosphate, pH 2.5	[277]
	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	β-CD polymer (0.2 g/ml)	50 m*M* phosphate, pH 2.5	[52]
	MeNH-β-CD (5 m*M*)	0.1 *M* phosphoric acid–TMA, pH 2.5	[74]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	AGP (0.2 m*M* region)	50 m*M* phosphate, pH 6	[171]
	β-CD-Phos (0.7 m*M*)	50 m*M* phosphate, pH 5	[93]
	α-CD (15 m*M*)	0.1 *M* phosphate, pH 2.5	[61]
Ketoconazole	SBE-β-CD (0.1 m*M*)	50 m*M* phosphate, pH 3, 40% MeOH	[259]
	β-CD (20 m*M*) or γ-CD (50 m*M*)	50 m*M* phosphate, pH 3, 10% MeOH	[259]
Ketoprofen	TM-β-CD (30 m*M*)	0.1 *M* MES, pH 5	[40]
	Avidin (0.025 m*M*)	50 m*M* phosphate, pH 6, 10% EtOH	[166]
	Dextrin (10%)	0.1 *M* phosphate–pyrophosphate, pH 6 or 7	[181]
	Dextrin 10 (15 m*M*)	20 m*M* TAPS–6.5 m*M* Tris, pH 7.7, 4% EtOH	[180]
	Maltooligosaccharides (2.5–10%)	10 m*M* sodium phosphate, pH 7	[179]
	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 6 (SDS)	[124,127,129]
	Ristocetin (2 m*M*)		[131,127]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6, (ACN)	[127,132]
	β-CD-NH_2 (20 m*M*)+TM-β-CD (10 m*M*)	34 m*M* phosphate, pH 2.3	[76]
Ketotifen and its intermediates	β-CD or DM-β-CD (1 m*M*)	5 m*M* acetate, pH 5.5, 0.2% HEC, 10 m*M* β-alanine	[27]
	β-CD (10 m*M*)	20 m*M* Tris–citric acid, pH 3.5, 0.005% HEC	[28]
Kynunerine	HSA (1 mg/ml)	10 m*M* borate, pH 9.5	[163]
Labetalol[a]	Transferrin (0.1–0.2 g/ml region)	0.1 *M* MES, pH 6	[173]
Lansoprazole	BSA (40 μ*M*)	30 m*M* phosphate, pH 7.4, 5% 1-propanol	[175]
Laudanosine	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	STDC (50 m*M*)	20 m*M* phosphate–borate, pH 7	[219]
	Lambda-carrageenan (0.28%)	25 m*M* citric acid–29 m*M* Tris, pH 7.2	[191]
	γ-CD (10 m*M*), poly-(D-SUVal) (0.5%)	25 m*M* borate, pH 9	[118]
	poly-(L-SUVal) (0.25%)	25 m*M* phosphate, pH 5.6	[203]
Laudanosoline	Lambda-carrageenan (0.28%)	25 m*M* citric acid–29 m*M* Tris, pH 7.2	[191]
	Dextran 70 (15%)	20 m*M* phosphate, pH 2.5	[185]
	poly-(L-SUVal) (0.25%)	25 m*M* phosphate, pH 5.6	[203]
Leucovorin[a]	BSA (1 mg/ml)	10 m*M* phosphate, pH 7.12, 5% dextran	[157]
	BSA (1 mg/ml)	20 m*M* phosphate, pH 7 or 7.2	[156]
	BSA (1 mg/ml)	20 m*M* phosphate, pH 7,02	[158]
Lisuride	γ-CD (30 m*M*)	0.1 *M* phosphate, pH 2.5	[284]
Lobelin	DM-β-CD (6 m*M*)	0.1 *M* Tris–phosphate, pH 2.3	[267]
Lofexidine	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	SBE-β-CD (0.1 m*M*)	50 m*M* phosphate, pH 3	[259]

(*continued on p. 190*)

Table 1. Continued

Drug	Selector	BGE	Reference
	SBE-β-CD (1 m*M*)	50 m*M* phosphate, pH 3.1	[90]
	β-CD (20 m*M*) or γ-CD (50 m*M*)	50 m*M* phosphate, pH 3, 10% MeOH	[259]
	HP-β-CD (20 m*M*)	50 m*M* phosphate, pH 3, 10% MeOH	[259]
Loxiglumide	Vancomycin (3m*M*)	50 m*M* phosphate, pH 6	[134]
Meclizine	SBE-β-CD (5 m*M*), CM-γ-CD (3 m*M*)	50 m*M* phosphate, pH 5	[93]
Meclozine	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
Mefenorex	Various methylated CDs (7.5–30 m*M*)	30 m*M* Tris, pH 2.4	[41]
Mefloquine	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	SBE-β-CD (0.08 m*M*)	50 m*M* phosphate, pH 3.1	[90]
	Heparin (2%)	10 m*M* phosphate, pH 5	[187]
	DM-β-CD (2.5 m*M*)	0.1 *M* phosphate, pH 2.5	[286]
Meluol	γ-CD (30 m*M*)	0.1 *M* phosphate, pH 2.5	[284]
Mepenzolate	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Mephenytoin and	β-CD (20 m*M*)/STDC (50 m*M*)	30 m*M* phosphate–10 m*M* borate, pH 7.2	[113]
4-hydroxymephenytoin	β-CD (50 m*M*)	10 m*M* phosphate–6 m*M* borate, pH 9.l, 0.1 *M* SDS, 10% 2-propanol	[287]
Mephobarbital	β-CD (30 m*M*)	20 m*M* phosphate–borate, pH 7, 0.1 *M* SDS, 2 *M* urea, 15% MeOH	[105]
	Wall-immobilized derivatised β-CD, CEC	50 m*M* phosphate, pH 7.8	[244]
Mepivacaine	DM-β-CD (10 m*M*)	18 m*M* Tris–phosphate, pH 2.9, 0.1% MHEC, 0.03 m*M* HTAB	[260]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	STDC (12, 40 m*M*)	phosphate, pH 3.13 (1 m*M* Brij-35)	[222]
	DM-β-CD (10 m*M*)	0.1 m*M* TEA–phosphoric acid, pH 3	[262]
N-(2-Mercaptopropionic)-acid	DR: OPA–L-Phe or L-Tyr	MEKC	[17]
	DR: NDA–L-Phe or L-Tyr	MEKC	[17]
N-(2-Mercaptopropionyl)-glycine	DR: OPA–L-Phe or L-Tyr	MEKC	[17]
	DR: NDA–L-Phe or L-Tyr	MEKC	[17]
Merucathinone	DM-β-CD (5 m*M*)	90% (25 m*M* Tris–phosphate, pH 2.45), 10% MeOH	[91]
Metamphetamine	SBE-β-CD (1 m*M*)	98.8% 25 m*M* Tris–phosphate, pH 2.4, 2% MeOH	[91]
	DM-β-CD (5 m*M*)	98.8% (25 m*M* Tris–phosphate, pH 2.45), 1.2% MeOH	[91]
	SBE-β-CD (4.6 m*M*)	20 m*M* citric acid–phosphate, pH 2.5	[186]
	Poly-β-CD (0.1 g/l)	50 m*M* phosphate, pH 2.5	[56]
Metanephrine	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	Sulfated β-CD (9 m*M*)	10 m*M* phosphate, pH 3.2	[96]
	AGP (1 m*M* region)	50 m*M* phosphate, pH 6	[171]
	β-CD-Phos (0.5 m*M*) or γ-CD-Phos (5 m*M*)	50 m*M* phosphate, pH 5	[93]
Metaproterenol	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	DM-β-CD (10 m*M*)	25 m*M* phosphate, pH 2.5	[67,285]
Methotrexate	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 6	[127]
	Ristocetin (2 m*M*)		
	Teicoplanin (2 m*M*)		
Methoxamine	18C6TCA (30 m*M*)	pH 2.07	[142]
	Succ-β-CD (3.3%)	50 m*M* borate, pH 7.3	[83]
	DM-β-CD (12 m*M*)	0.1 *M* Tris–phosphate, pH 2.3	[56]
Methoxyphenamine	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	HP-β-CD (0.1 *M*)	25 m*M* phosphate, pH 2.5	[67]
5-(4-Methylphenyl)-5-phenyl-hydantoin	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Methcathinone	SBE-β-CD (1 m*M*)	98.8% 25 m*M* Tris–phosphate, pH 2.4, 1.2% MeOH	[91]

Table 1. Continued

Drug	Selector	BGE	Reference
N-Methylephedrine	DM-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	DM-β-CD (40 g/l)	30 m*M* Tris–phosphate, pH 2.5	[274]
	DM-β-CD	pH 2.5, HPC, TBA	[84]
	DM-β-CD (18 m*M*)	20 m*M* phosphate, pH 2.5,10 m*M* TBAB	[84]
	SBE-β-CD (40 m*M*)	20 m*M* borate, pH 10	[84]
	Sulfated β-CD (9 m*M*)	10 m*M* phosphate, pH 3.2	[96]
N-Methylpseudoephedrine	DM-β-CD (18 m*M*)	20 m*M* phosphate, pH 2.5, 10 m*M* TBAB, HTC, TBA	[84]
	DM-β-CD (18 m*M*)	20 m*M* phosphate, pH 2.5, 10 m*M* TBAB	[84]
	SBE-β-CD (40 m*M*)	20 m*M* borate, pH 10	[84]
	DDCV (10 m*M*)	50 m*M* phosphate, pH 8	[211]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
2-Methyltaurine (DNS derivative)	β-CD (60 m*M*)+γ-CD (10 m*M*)	0.1 *M* borate, 0.1 *M* SDS, 20% MeOH	[111]
Metomidate	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	SBE-β-CD (0.1 m*M*)	50 m*M* phosphate, pH 3	[259]
	SBE-β-CD (1 m*M*)	98.8% 25 m*M* Tris–phosphate pH 2.4, 1.2% MeOH	[91]
	SBE-β-CD (1 m*M*)	50 m*M* phosphate, pH 3.1	[90]
	β-CD (20 m*M*) or γ-CD (50 m*M*)	50 m*M* phosphate, pH 3, 10% MeOH	[259]
	HP-β-CD (20 m*M*)	50 m*M* phosphate, pH 3, 10% MeOH	[259]
Metoprolol	DM-β-CD (37 m*M*)	40 m*M* Li phosphate, pH 3	[32]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	CSA (30 m*M*)	1 *M* acetic acid, 0.2 m*M* Tween 20, ACN	[249]
	AGP–CSP (packed)	2 m*M* phosphate, pH 6.8, 2% 2-propanol	[176]
	AGP (0.5 m*M* region)	50 m*M* phosphate, pH 6, 10% MeOH	[171]
	DM-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[288]
	Cellobiohydrolase I (40 ng/ml)	0.4 *M* phosphate, pH 5.1, 25% 2-propanol	[168]
	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	Cellulase–BSA gel	50 m*M* phosphate, pH 6.8, 1% 2-propanol	[162]
	CEC: Imprinted polymer	4 *M* acetate, pH 3, 80% ACN	[154]
	γ-CD-Phos (10 m*M*)	50 m*M* phosphate, pH 5	[93]
	HP-β-CD (60 or 120 m*M*)	50 m*M* borate, pH 9.5, 5% 1-propanol, 50 m*M* STC	[116]
Mexilethine	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	18C6TCA (10 m*M*)	20 m*M* Tris–phosphoric acid, pH 2.06	[147]
	AGP (0.5 m*M* region)	50 m*M* phosphate, pH 6, 8% 2-propanol	[171]
	SBE-β-CD (2 m*M*) γ-CD-Phos (2 m*M*)	50 m*M* phosphate, pH 5	[93]
Mianserine	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	SBE-β-CD (0.05 m*M*)	50 m*M* phosphate, pH 3.1	[90]
	β-CD, γ-CD (0.1 *M*)	FA, 0.1 *M* Tris–0.15 *M* citric acid, pH 5.1	[248]
	Dextrinsulfopropylether (4%)	80 m*M* Tris–phosphate, pH 3	[190]
	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
	SBE-γ-CD (0.04 m*M*) or γ-CD (20 m*M*)	40 m*M* phosphate, pH 3	[92]
Mianserine analogues	CM-β-CD (5 m*M*)	50 m*M* phosphate, pH 3	[89]
	SBE-β-CD (0.1 m*M*)	50 m*M* phosphate, pH 3	[89]
Miconazole	SBE-β-CD (0.1 m*M*) HP-β-CD, M-β-CD	50 m*M* phosphate, pH 3, 20% MeOH	[259]
	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
Midodrine	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
MPPEA	Various methylated CDs (7.5–30 m*M*)	30 m*M* Tris, pH 2.4	[41]
Nadolol	HP-β-CD (20 m*M*)	50 m*M* phosphate, pH 2.5, 70 m*M* TMA	[48]
	HP-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	HP-β-CD (60 or 120 m*M*)	50 m*M* borate, pH 9.5, 5% 1-propanol, 50 m*M* STC	[116]

(*continued on p. 192*)

Table 1. Continued

Drug	Selector	BGE	Reference
Naproxen	HP-β-CD (10 m*M*)	0.2 *M* MES–TBA, pH 5, 0.4% polymeric additive	[289]
	HP-β-CD (5 m*M*)	0.2 *M* MES, pH 4.86, 0.2% HEC	[35]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[131]
	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 7	[124]
	Dextrin (6%)	20 m*M* phosphate, pH 2.5	[185]
	HP-β-CD (10 m*M*)	25 m*M* acetate, pH 4.6	[67]
	β-CD-NH_2 (20 m*M*)+TM-β-CD (10 m*M*)	34 m*M* phosphate, pH 2.3	[76]
Nebracetam	γ-CD-Phos (4 m*M*)	50 m*M* phosphate, pH 5	[93]
Nefopam	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	β-CD or γ-CD (100 m*M*)	FA, 0.1 *M* Tris, 0.15 *M* citric acid, pH 5.1	[248]
	HP-β-CD or M-β-CD		
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Nicartipine	AGP (0.2 m*M* region)	50 m*M* phosphate, pH 5, 10% 1-propanol	[171]
	β-CD-Phos (5 m*M*)	50 m*M* phosphate, pH 5	[93]
	SBE-β-CD (0.5 m*M*), CM-γ-CD (1 m*M*)	50 m*M* phosphate, pH 5	[93]
Nicergoline	γ-CD (30 m*M*)	0.1 *M* phosphate, pH 2.5	[284]
Nomifensine	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
Norephedrine	DM-β-CD (20 m*M*)	10 m*M* Tris–phosphate, pH 2.4	[30]
	DM-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	DM-β-CD (40 g/l)	30 m*M* Tris–phosphate, pH 2.5	[274]
	DM-β-CD (18 m*M*)	20 m*M* phosphate, pH 2.5, 10 m*M* TBAB	[84]
	DM-β-CD (5 m*M*)	90% (25 m*M* Tris–phosphate, pH 2.45), 10% MeOH	[91]
	DM-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	SBE-β-CD (40 m*M*)	20 m*M* borate, pH 10	[84]
	Sulfated β-CD (2–4%)	10 m*M* phosphate	[95]
	18C6TCA (30 m*M*)	10 m*M* Tris–citric acid, pH 2.2	[141]
	18C6TCA (30 m*M*)	pH 2.07	[142]
	18C6TCA (10 m*M*)	FA	[148]
	Succ-β-CD (3.3%)	50 m*M* borate, pH 7.3	[83]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	18C6TCA (10 m*M*)	20 m*M* Tris–phosphoric acid, pH 2.06	[147]
	DR: OPA–*N*-acetyl-L-cysteine	MEKC	[17]
	DM-β-CD (18 m*M*)	45 m*M* Tris, pH 2.4	[51]
	β-CD-Phos (5 m*M*)	50 m*M* phosphate, pH 5	[93]
Norepinephrine	DM-β-CD (18 m*M*)	10 m*M* Tris–phosphate, pH 2.4	[30]
	DM-β-CD (20 m*M*)	50 m*M* phosphate, pH 2.5	[277]
	β-CD (20 m*M*)	0.15 *M* phosphate–TBA, pH 2.5	[47]
	HP-β-CD (20 m*M*)+18C6TCA (5 m*M*)	30 m*M* Tris–citric acid, pH 2.5	[140,149]
	DM-β-CD, HP-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	β-CD polymer (0.1 g/l)	50 m*M* phosphate, pH 2.5	[52]
	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	18C6TCA (30 m*M*)	10 m*M* Tris–citric acid, pH 2.2	[141]
	18C6TCA (30 m*M*)	pH 2.07	[142]
	18C6TCA (10 m*M*)	FA, 2.5 m*M* TBAP	[148]
	SBE-β-CD (2.5 m*M*)	0.2 *M* phosphate, pH 2.5	[85]
	Sulfated β-CD (9 m*M*)	10 m*M* phosphate, pH 3.2	[96]
	18C6TCA (10 m*M*)	20 m*M* Tris–phosphoric acid, pH 2.06	[147]
	18C6TCA (5 m*M*)+HP-β-CD (20 m*M*)	30 m*M* Tris–citric acid, pH 2.5	[149]
	DM-β-CD (18 m*M*)	45 m*M* Tris, pH 2.4	[51]

Table 1. Continued

Drug	Selector	BGE	Reference
Norfenefrine	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	Sulfated β-CD (9 m*M*)	10 m*M* phosphate, pH 3.2	[96]
Normetanephrine	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	18C6TCA (30 m*M*)	10 m*M* Tris–citric acid, pH 2.2	[141]
	Sulfated β-CD (9 m*M*)	10 m*M* phosphate, pH 3.2	[96]
Nornicotine	Heparin (2%)	10 m*M* phosphate, pH 5	[187]
Norphenylephrine	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
Norpseudoephedrine	β-CD (10 m*M*)	5 m*M* sodium acetate, pH 5.48, 10 m*M* β-alanine	[26]
	DM-β-CD (5 m*M*)	90% (25 m*M* Tris–phosphate, pH 2.45), 10% MeOH	[91]
	SBE-β-CD (1 m*M*)	98.8% 25 m*M* Tris–phosphate, pH 2.4, 1.2% MeOH	[91]
	TMA-β-CD (16 m*M*)	50 m*M* phosphate, pH 2.5	[79]
Norverapamil	TM-β-CD (25 m*M*)	0.1 *M* phosphate–TEA, pH 3	[44]
	TM-β-CD (60 m*M*)	60 m*M* phosphate, pH 2.5	[290]
	Dextrin 10 (20%)	25 m*M* Tris–phosphate, pH 3.4	[180]
Octopamine	DM-β-CD (20 m*M*)	50 m*M* phosphate, pH 2.5	[277]
	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	18C6TCA (30 m*M*)	pH 2.07	[142]
	Sulfated β-CD (9 m*M*)	10 m*M* phosphate, pH 3.2	[96]
	Succ-β-CD (3.3%)	50 m*M* borate, pH 7.3	[83]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	18C6TCA (10 m*M*)	20 m*M* Tris–phosphoric acid, pH 2.06	[147]
Ofloxacin	BSA (0.4%)	0.1 *M* phosphate buffer, pH 8	[159]
Ofloxacin and analogues	γ-CD (20 m*M*)+10 m*M* Zn(II)sulfate +10 m*M* D-Phe	10 m*M* acetate, pH 6.5	[237]
Omeprazole	BSA (100 μ*M*)	30 m*M* phosphate, pH 7.4, 5% 1-propanol	[175]
Orphenadrine	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
	γ-CD (15 m*M*)	0.1 *M* phosphate, pH 2.5	[63]
	α-CD (15 m*M*)	0.1 *M* phosphate, pH 2.5	[61]
Oxaminiquine	β-CD (25 m*M*)	50 m*M* phosphate, pH 12	[291]
	Heparin (3 m*M*)	50 m*M* phosphate, pH 3	[291]
Oxazepam	HSA (packed)	CEC: 4 m*M* phosphate, pH 7	[165]
Oxomemazine	HP-β-CD (15 m*M*)	0.1 *M* phosphate–TEA, pH 3	[44]
	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
	γ-CD (15 m*M*)	0.1 *M* phosphate, pH 2.5	[63]
	α-CD (15 m*M*)	0.1 *M* phosphate, pH 2.5	[61]
Oxprenolol	DM-β-CD (20 m*M*)	50 m*M* phosphate, pH 2.5, 70 m*M* TMA	[48]
	DM-β-CD (37 m*M*)	50 m*M* Li phosphate, pH 3	[32]
	DM-β-CD or HP-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	β-CD polymer (0.1 g/ml)	50 m*M* phosphate, pH 2.5	[52]
	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	DDCV (25 m*M*)	25 m*M* borate–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	AGP–CSP (packed)	4 m*M* phosphate, pH 6.8, 4% 2-propanol	[176]
	HP-β-CD (120 m*M*)	0.1 *M* citric acid–phosphate, pH 2.5	[86]
	HP-β-CD (60 or 120 m*M*)	50 m*M* borate, pH 9.5, 5% 1-propanol, 50 m*M* STC	[116]
Oxyphencyclimine	BSA (0.5 m*M*)	50 m*M* phosphate, pH 6	[160]
	Ovomucoid (0.5 m*M*)	50 m*M* phosphate, pH 5, 8% 1-propanol	[160]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
	AGP (0.2 m*M* region)	50 m*M* phosphate, pH 6, 10% 2-propanol	[171]
	SBE-β-CD (5 m*M*)	50 m*M* phosphate, pH 5	[93]

(*continued on p. 194*)

Table 1. Continued

Drug	Selector	BGE	Reference
Pentobarbital	TM-β-CD (10 m*M*)	50 m*M* phosphate–10 m*M* borate, pH 9	[292]
	TM-α-CD (10 m*M*)		
	γ-CD (30 m*M*)	20 m*M* phosphate–borate, pH 9, 50 m*M* SDS	[108]
	60 m*M* L-MEN or 40 m*M* CSA		
	AGP–CSP (packed)	2 m*M* phosphate, pH 5.5, 2% 2-propanol	[176]
p-Hydroxynorpseudoephedrine	β-CD (15 m*M*)	5 m*M* sodium acetate, 0.2% HEC, pH 5.48; 10 m*M* β-alanine	[26]
Phendimetrazine	TMA-β-CD (16 m*M*)	50 m*M* phosphate, pH 2.5	[79]
Phenglutarimide	DDCV (80 m*M*)	25 m*M* phosphate–borate, pH 9.25	[206]
Pheniramine	Heparin (2%)	10 m*M* phosphate, pH 5	[187]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Phenprocoumon	Glucidex 2 (3%)	10 m*M* Tris–phosphate, pH 7	[182]
Phensuximide	Sulfated β-CD (3%)	10 m*M* phosphate, pH 7	[95]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Phenylephrine	AGP (1 m*M* region)	50 m*M* phosphate, pH 6	[171]
	β-CD-Phos (5 m*M*) or SBE-β-CD (1 m*M*)	50 m*M* phosphate, pH 5	[93]
Pholedrine	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	TMA-β-CD (16 m*M*)	50 m*M* phosphate, pH 2.5	[79]
Picumeterol	β-CD (16 m*M*)	0.2 *M* phosphate–0.1 *M* citric acid, pH 4	[307]
	DM-β-CD (30 m*M*)	25 m*M* borate–phosphoric acid, pH 2.3	[293,294]
Pinacidil	HP-β-CD (9 m*M*)	0.1 *M* Tris–phosphate, pH 2.3, 0.5% HPC	[295]
Pindolol	DM-β-CD (20 m*M*)	50 m*M* phosphate, pH 2.5, 70 m*M* TMA	[48]
	DM-β-CD (10 m*M*)	18 m*M* Tris–phosphate, pH 2.9, 0.1% MHEC, 0.03 m*M* HTAB	[260]
	DM-β-CD (15 m*M*)	0.1 *M* phosphate–TEA, pH 3	[44]
	β-CD or DM-β-CD, HP-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	CM-β-CD (2%)	20 m*M* citric acid, pH 2.5	[271]
	Cellobiohydrolase I (40 ng/ml)	0.4 *M* phosphate, pH 5.1, 25% 2-propanol	[168]
	Fungal cellulase (20μM)	50 m*M* phosphate, pH 7.4	[155]
	Ovomucoid (0.5 m*M*)	50 m*M* phosphate, pH 5, 8% ethanol	[160]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	CSA (30 m*M*)	1 *M* acetic acid, 0.2 m*M* Tween 20, ACN	[249]
	Rifamycin B (25 m*M*)	0.1 *M* phosphate, pH 7, 30% 2-propanol	[128]
	Lambda-carrageenan (0.28%)	25 m*M* citric acid–29 m*M* Tris, pH 4	[191]
	Cellulase–BSA gel	50 m*M* phosphate, pH 6.8, 1% 2-propanol	[162]
	AGP (0.1 m*M* region)	50 m*M* phosphate, pH 6, 10% EtOH	[171]
	CM-γ-CD (5 m*M*)	50 m*M* phosphate, pH 5	[93]
	HP-β-CD (120 m*M*)	0.1 *M* citric acid–phosphate, pH 2.5	[86]
	HP-β-CD (60 or 120 m*M*)	50 m*M* borate, pH 9.5, 5% 1-propanol, 50 m*M* STC	[116]
Piperoxam	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Pranoprofene	TM-β-CD (10 m*M*)	35 m*M* formate, pH 4	[266]
Pantoprazole	BSA (85 μ*M*)	30 m*M* phosphate, pH 7.4, 5% 1-propanol	[175]
Practolol	DM-β-CD (40 m*M*)	50 m*M* phosphate, pH 2.5	[33]
Prilocaine	STDC (12, 40 m*M*)	phosphate, pH 3.13 (1 m*M* Brij-35)	[222]
	DM-β-CD (10 m*M*)	0.1 m*M* TEA–phosphoric acid, pH 3	[262]
Primaquine	DM-β-CD (20–40 m*M*)	25 m*M* phosphate, 2 *M* urea,, pH 2.5	[279]
	TM-β-CD (20 m*M*)	25 m*M* phosphate, pH 2.7, 2 *M* urea	[270]
	γ-CD (20 m*M*)	25 m*M* phosphate–borate, pH 2.7	[53]
	Heparin (2%)	10 m*M* phosphate, pH 5	[187]
	Ovomucoid (0.5 m*M*)	50 m*M* phosphate, pH 5, 8% 1-propanol	[160]

Table 1. Continued

Drug	Selector	BGE	Reference
	M-β-CD (100 m*M*)	FA, 0.1 *M* Tris 0.15 *M* citric acid, pH 5.1	[248]
	Sulfated β-CD (2%)	10 m*M* phosphate buffer, pH 3.8	[97]
	18C6TCA (10 m*M*)	20 m*M* Tris–phosphoric acid, pH 2.06	[147]
	Dextrin (9%)	20 m*M* phosphate, pH 2.5	[185]
	AGP (0.5 m*M* region)	50 m*M* phosphate, pH 6, 10% 1-propanol	[171]
	β-CD-Phos (5 m*M*) or γ-CD-Phos (0.2 m*M*)	50 m*M* phosphate, pH 5	[93]
	SBE-β-CD (5 m*M*) or CM-γ-CD (0.1 m*M*)	50 m*M* phosphate, pH 5	[93]
Promethazine	β-CD (3 m*M*)	10 m*M* sodium acetate, pH 5.55 10 m*M* β-Alanine	[296]
	γ-CD	pH 3	[279]
	SBE-β-CD (6 mg/ml)	50 m*M* phosphate, pH 6	[88]
	BSA (0.05 m*M*)	50 m*M* phosphate, pH 7.4, 3% *n*-propanol	[155]
	HSA (35 μ*M*)	50 m*M* phosphate, pH 7	[164]
	Orosomucoid (21 μ*M*)	50 m*M* phosphate, pH 6.8	[155]
	Transferrin (0.1–0.2 g/ml region)	0.1 *M* MES, pH 6	[173]
	AGP (0.5 m*M* region)	50 m*M* phosphate, pH 5, 10% 1-propanol	[171]
	γ-CD (15 m*M*)	0.1 m*M* phosphate, pH 2.5	[63]
	β-CD-Phos (5 m*M*) or γ-CD-Phos (5 m*M*)	50 m*M* phosphate, pH 5	[93]
	SBE-β-CD (5 m*M*) or CM-γ-CD (0.1 m*M*)	50 m*M* phosphate, pH 5	[93]
Propiomazine	β-CD (0.1 *M*)	0.15 *M* citric acid–0.1 *M* Tris, FA	[248]
Propranolol	β-CD (40 m*M*)	0.1 *M* phosphate, pH 2.5, urea, MeOH	[42]
	HE-β-CD or HP-β-CD (28 m*M*)	20 m*M* Tris–phosphate, pH 2.4	[251]
	DM-β-CD (37 m*M*)	40 m*M* Li phosphate, pH 3, MeOH or ACN	[32,297]
	β-CD, DM-β-CD, TM-β-CD (20 m*M*)	50 or 100 m*M* phosphate, pH 2.5 TBA or TMA	[47,48]
	β-CD (12 m*M*)	40 m*M* borate, pH 9.3, 32 m*M* SDS	[107]
	TM-β-CD (15 m*M*)	0.1 *M* phosphate–TEA, pH 3	[44]
	HP-β-CD (10 m*M*)	0.2 *M* TAPSO–TBA hydroxide, pH 7, 0.4% Polymer additive	[45]
	HP-β-CD (15 m*M*)	0.1 *M* TAPS, pH 7.6	[256]
	β-CD or DM-β-CD. TM-β-CD or HP-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	β-CD polymer (20 m*M*)	50 m*M* phosphate, pH 2.5	[52]
	CM-β-CD (2%)	20 m*M* phosphate, pH 5.8	[87]
	CM-β-CD (2%)	20 m*M* citric acid, pH 2.5	[271]
	BSA (0.5 m*M*)	50 m*M* phosphate, pH 6	[160]
	Cellobiohydrolase I (40 ng/ml)	0.4 *M* phosphate, pH 5.1, 25% 2-propanol	[168]
	CM-β-CD (10 m*M*)	0.1 *M* phosphate–TEA, pH 3	[81]
	MeNH-β-CD (5 m*M*)	0.1 *M* phosphoric acid–TMA, pH 2.5	[74]
	Succ-β-CD (3.3%)	50 m*M* borate, pH 7.3	[83]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	DDCV (25 m*M*)	25 m*M* borate–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	CSA (30 m*M*)	1 *M* acetic acid, 0.2 m*M* Tween 20, ACN	[249]
	Rifamycin B (25 m*M*)	0.1 *M* phosphate, pH 7, 30% 2-propanol	[128]
	Lambda-carrageenan (0.28%)	25 m*M* citric acid–29 m*M* Tris, pH 4	[191]
	Cellulase–BSA gel	50 m*M* phosphate, pH 6.8, 1% 2-propanol	[162]
	Transferrin (0.1–0.2 g/ml region)	0.1 *M* MES, pH 6	[173]
	CEC: Imprinted polymer	4 *M* acetate, pH 3, 80% ACN	[154]
	HE-β-CD or DM-β-CD or Ac-β-CD (25 m*M*)	50 m*M* phosphate, pH 3, MeOH	[275]
Propriomazine	HSA (35 μ*M*)	50 m*M* phosphate, pH 7	[164]
	β-CD (0.1 *M*)	FA, 0.1 *M* Tris–0.15 *M* citric acid, pH 5.1	[248]

(*continued on p. 196*)

Table 1. Continued

Drug	Selector	BGE	Reference
Pseudoephedrine	DM-β-CD (10 m*M*)	5 m*M* sodium acetate, pH 5.48, 0.2% HEC; 10 m*M* β-alanine	[26]
	β-CD (20 m*M*)	0.15 *M* TBA–phosphate, pH 2.5	[47]
	DM-β-CD (18 m*M*)	20 m*M* phosphate, pH 2.5, 10 m*M* TBAB	[84]
	DM-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	SBE-β-CD (1 m*M*)	98.8% 25 m*M* Tris–phosphate, pH 2.4, 1.2% MeOH	[91]
	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	SBE-β-CD (1.5 m*M*)	20 m*M* phosphate, pH 2.5	[85]
	Sulfated β-CD (9 m*M*)	10 m*M* phosphate, pH 3.2	[96]
	DDCV (50 m*M*)	25 m*M* phosphate–borate, pH 8	[208]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	DR: GITC	10 m*M* phosphate–borate, pH 9, 0.1 *M* SDS	[12]
	DM-β-CD (18 m*M*)	45 m*M* Tris, pH 2.4	[51]
Pseudomerucathine	DM-β-CD (5 m*M*)	90% (25 m*M* Tris–phosphate, pH 2.45), 10% MeOH	[91]
Pyridogluthedimide	DDCV (80 m*M*)	25 m*M* phosphate–borate, pH 9.25	[206]
Quinacrine	Heparin (2%)	10 m*M* phosphate, pH 5	[187]
Quinagolide	β-CD (30 m*M*)	50 m*M* phosphate, pH 2.5	[141]
	α-CD (60 m*M*)/18C6H4 (20 m*M*)	10 m*M* Tris–citric acid, pH 2.2	[149]
Remoxipride	HP-β-CD (60 m*M*)	phosphate, pH 3, 20 m*M* TBA	[50]
Ropivacaine	DM-β-CD (50 m*M*)	1 m*M* formate, pH 2.85	[298]
	DM-β-CD (10 m*M*)	0.1 m*M* TEA–phosphoric acid, pH 3	[262]
Salbutamol	SBE-β-CD (2 m*M*)	20 m*M* citric acid–phosphate, pH 2.5	[86]
	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	CSA (30 m*M*)	1 *M* acetic acid, 0.2 m*M* Tween 20, ACN	[249]
	β-CD-Phos (0.4 m*M*)	50 m*M* phosphate, pH 5	[93]
	SBE-β-CD (3 m*M*)	50 m*M* phosphate, pH 5	[93]
	HP-β-CD (120 m*M*)	0.1 *M* citric acid–phosphate, pH 2.5	[86]
	DM-β-CD (112 m*M*)	50 m*M* phosphate–0.1 *M* citric acid, pH 2.5	[299]
Secobarbital	α-CD+TM-β-CD (10 m*M*)	50 m*M* phosphate–10 m*M* borate, pH 9	[292]
	γ-CD (30 m*M*)	20 m*M* phosphate–borate, pH 7, 0.1 *M* SDS, 2 *M* urea, 15% MeOH	[105]
	γ-CD (35 m*M*)	30 m*M* phosphate, pH 7, 0.1 *M* SDS, 15% MeOH	[92]
Selegilin	Poly-β-CD (0.1 g/l)	50 m*M* phosphate, pH 6	[55]
Simendan	Dextrin 10 (15 m*M*)	20 m*M* TAPS–6.5 m*M* Tris, pH 7.7, 4% ethanol	[180]
Sotalol	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	DM-β-CD (15 m*M*)	0.1 *M* phosphate–TEA, pH 3	[44]
Sulconazole	Dextrin (3%)	20 m*M* phosphate, pH 2.5	[185]
	SBE-β-CD (5 m*M*) or CM-γ-CD (10 m*M*)	50 m*M* phosphate, pH 5	[93]
Sulpiride	AGP (1 m*M* region)	50 m*M* phosphate, pH 6	[171]
	β-CD-Phos (10 m*M*) or SBE-β-CD (1 m*M*)	50 m*M* phosphate, pH 5	[93]
Suprofen	TM-β-CD (30 m*M*)	0.1 *M* MES, pH 5	[40]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[131]
	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 7	[124]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,132]
	TM-β-CD (10 m*M*)	75 m*M* formate, pH 4	[266]
	β-CD-NH_2 (20 m*M*)+TM-β-CD (10 m*M*)	34 m*M* phosphate, pH 2.3	[76]
Synephrine	DM-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	Sulfated β-CD (9 m*M*)	10 m*M* phosphate, pH 3.2	[96]
	Succ-β-CD (3.3%)	50 m*M* borate, pH 7.3	[83]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
Temazepam	HSA (packed)	CEC: 4 m*M* phosphate, pH 7	[165]

Table 1. Continued

Drug	Selector	BGE	Reference
Terbutaline	β-CD or DM-β-CD (15 or 5 m*M*)	0.1 *M* phosphate, pH 2.5	[42]
	HP-β-CD or β-CD (15 m*M*)	0.1 *M* phosphate–TEA, pH 3	[44]
	HP-β-CD (60 m*M*)	phosphate, pH 3, 20 m*M* TBA	[50]
	β-CD polymer (10 mg/ml)	50 m*M* phosphate, pH 2.5	[52]
	β-CD, DM-β-CD or HP-β-CD (5–25 m*M*)	50 m*M* phosphate, pH 2.5, 10.6 and 11.6	[257]
	SBE-β-CD (2 m*M*)	20 m*M* citric acid–phosphate, pH 2.5	[86]
	SBE-β-CD (4.6 m*M*)	20 m*M* citric acid–phosphate, pH 2.5	[86]
	Rifamycin B (25 m*M*)	60% 0.1 *M* phosphate, 40% 2-propanol	[126]
	TMA-β-CD (16 m*M*)	50 m*M* phosphate, pH 2.5	[79]
	MeNH-β-CD (5 m*M*) or	0.1 *M* phosphoric acid–TMA, pH 2.5	[74]
	sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	AGP (1 m*M* region)	50 m*M* phosphate, pH 6	[171]
	DM-β-CD (50 m*M*)	25 m*M* phosphate, pH 2.5	[68]
	SBE-β-CD (0.2 m*M*)	50 m*M* phosphate, pH 5	[93]
	HP-β-CD (120 m*M*)	0.1 *M* citric acid–phosphate, pH 2.5	[86]
Terguride	γ-CD (30 m*M*)	0.1 *M* phosphate, pH 2.5	[284]
Tetrahydropapaveroline	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	STDC (50 m*M*)	20 m*M* phosphate–borate, pH 7	[220]
Tetranisole	Heparin (2%)	10 m*M* phosphate, pH 5	[187]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
Tetryzoline	β-CD (15 m*M*)	0.1 *M* phosphate, pH 2.5	[62]
	γ-CD (15 m*M*)	0.1 *M* phosphate, pH 2.5	[63]
	α-CD (15 m*M*)	0.1 *M* phosphate, pH 2.5	[61]
Thalidomide	SBE-β-CD (2 or 4.6 m*M*)	20 m*M* citric acid–phosphate, pH 2.5	[86]
	DDCV (80 m*M*)	25 m*M* phosphate–borate, pH 9.25	[206]
Thalidomide and metabolites	CM-β-CD (15 m*M*)	50 m*M* phosphate, pH 6	[82]
Thiazinamium	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
Thiopental	γ-CD (30 m*M*)	20 m*M* borate–phosphate, pH 9,	[108]
		50 m*M* SDS, 60 m*M* L-MEN or 40 m*M* CSA	
Thioridazine	γ-CD (5 m*M*)	10 m*M* acetate, pH 5.47, 0.08% HEC,	[28]
		10 m*M* β-alanine	
	γ-CD (5 m*M*)	10 m*M* NaOH, MES, 0.084% HEC,	[296]
		10 m*M* aminocaproic acid,, pH 5.57	
	γ-CD (5 m*M*)	20 m*M* Tris–phosphate, pH 2.5	[28]
	HSA (35 μM)	50 m*M* phosphate, pH 7	[164]
	β-CD or γ-CD or M-β-CD (100 m*M*)	FA, 0.1 *M* Tris–0.15 *M* citric acid, pH 5.1	[248]
	β-CD-Phos (5 m*M*)	50 m*M* phosphate, pH 5	[93]
	CM-γ-CD (5 m*M*), γ-CD-Phos (5 m*M*)	50 m*M* phosphate, pH 5	[93]
Tiaprofenicacid	MeNH-β-CD (5 m*M*) or	50 m*M* phosphoric acid, 50 m*M* acetic acid	[74]
	$(MeNH)_7$-β-CD	50 m*M* boric acid and NaOH ad, pH 7	
Timepidium	γ-CD (20 m*M*)	25 m*M* phosphate–borate, pH 2.7	[53]
	γ-CD (20 m*M*)	25 m*M* phosphate–borate, pH 9, 50 m*M* DTAC	[53]
	Dextrin (9%)	20 m*M* phosphate, pH 2.5	[185]
Timolol	HP-β-CD (60 or 120 m*M*)	50 m*M* borate, pH 9.5, 5% 1-propanol,	[116]
		50 m*M* STC	
Tocainide analogues	γ-CD (50 m*M*)	40 m*M* phosphate, pH 3, 0.05% PVA	[46]
Tolperisone	Ovomucoid (0.5 m*M*)	50 m*M* phosphate, pH 5, 10% 2-propanol	[167]
	Sulfated β-CD (2%)	10 m*M* phosphate buffer, pH 3.8	[97]
	AGP (0.5 m*M* region)	50 m*M* phosphate, pH 6, 10% 2-propanol	[171]
	CM-γ-CD (1 m*M*) or γ-CD-Phos (3 m*M*)	50 m*M* phosphate, pH 5	[93]
Tranylcypromine	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	Various methylated CDs (7.5–30 m*M*)	30 m*M* Tris, pH 2.4	[41]
Trihexyphenidyl	M-β-CD (100 m*M*)	FA, 0.1 *M* Tris 0.15 *M* citric acid, pH 5.1	[248]
	Succ-β-CD (3.3%)	50 m*M* borate, pH 7.3	[83]

(*continued on p. 198*)

Table 1. Continued

Drug	Selector	BGE	Reference
	Sulfated β-CD (2%)	10 m*M* phosphate buffer, pH 3.8	[97]
	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
	AGP (0.5 m*M* region)	50 m*M* phosphate, pH 6, 10% 1-propanol	[171]
	SBE-β-CD (2 m*M*)	50 m*M* phosphate, pH 5	[93]
Trimebutine	BSA (0.5 m*M*)	50 m*M* phosphate, pH 6	[160]
	Ovomucoid (0.5 m*M*)	50 m*M* phosphate, pH 5, 8% 1-propanol	[160]
	AGP (0.1 m*M* region)	50 m*M* phosphate, pH 6, 10% 1-propanol	[171]
	β-CD-Phos (0.4 m*M*)	50 m*M* phosphate, pH 5	[93]
	SBE-β-CD (2 m*M*) or CM-γ-CD (5 m*M*)	50 m*M* phosphate, pH 5	[93]
Trimepazine	β-CD or HP-β-CD (100 m*M*)	FA, 0.1 *M* Tris 0.15 *M* citric acid, pH 5.1	[248]
Trimetoquinol	DM-β-CD (10–40 m*M*)	25 m*M* phosphate, 2 *M* urea, pH 2.5	[279]
	DM-β-CD (5%)	25 m*M* phosphate, pH 2.7	[53]
	Chondroitin sulfate (3%)	20 m*M* phosphate–borate, pH 2.4	[189]
	Conalbumin (0.5 m*M*)	50 m*M* phosphate, pH 7	[160]
	Dextran sulfate (3%)	20 m*M* phosphate–borate, pH 5.5	[189]
	STDC (50 m*M*)	20 m*M* phosphate–borate, pH 7	[220]
	Dextran 70 (15%)	20 m*M* phosphate, pH 2.5	[185]
	SBE-β-CD (0.2 m*M*) or CM-γ-CD (10 m*M*) γ-CD-Phos (2 m*M*)	50 m*M* phosphate, pH 5	[93]
Trimetoquinol, analogues	β-CD polymer (1%)	25 m*M* phosphate, pH 2.7, 2 *M* urea	[270]
	β-CD (40 m*M*) or β-CD polymer (3%)	25 m*M* phosphate, pH 6.5 (2 *M* urea)	[53]
	Chondroitin sulfate (3%)	20 m*M* phosphate–borate, pH 2.4	[189]
Trimipramine	HP-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	β-CD or γ-CD or HP-β-CD (100 m*M*)	FA, 0.1 *M* Tris–0.15 *M* citric acid, pH 5.1	[248]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	AGP (0.12 m*M* region)	50 m*M* phosphate, pH 5, 10% 1-propanol	[171]
	SBE-β-CD (5 m*M*) or CM-γ-CD (5 m*M*) γ-CD-Phos (5 m*M*)	50 m*M* phosphate, pH 5	[93]
Tropicamide	TMA-β-CD (16 m*M*)	50 m*M* phosphate, pH 2.5	[79]
	β-CD (15 m*M*)	0.1 *M* phosphate, pH 2.5	[62]
	γ-CD (15 m*M*)	0.1 *M* phosphate, pH 2.5	[63]
	α-CD (15 m*M*)	0.1 *M* phosphate, pH 2.5	[61]
Uniconazole	γ-CD (50 m*M*)	0.1 *M* borate, pH 9, 0.1 *M* SDS, 2 *M* urea, Isobutanol (10%)	[272]
Verapamil	TM-β-CD (12 m*M*)	20 m*M* Tris–phosphate, pH 2.7, 0.1% MHEC, 0.05 m*M* HTAB, 2% ethylene glycol	[260]
	TM-β-CD or β-CD (25 or 15 m*M*)	0.1 *M* phosphoric acid–diethanolamine, pH 3	[44]
	TM-β-CD (60 m*M*)	60 m*M* phosphate, pH 2.5	[290]
	TM-β-CD or HP-β-CD (20 m*M*)	50 m*M* TMA–phosphate, pH 2.5	[49]
	Dextrin 10 (20%)	25 m*M* Tris–phosphate, pH 3.4	[180]
	Ovomucoid (0.5 m*M*)	50 m*M* phosphate, pH 5, 10% 2-propanol	[167]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	Dextrin (15%)	20 m*M* phosphate, pH 2.5	[185]
	Dextrinsulfopropylether (4%)	80 m*M* Tris–phosphate, pH 3	[190]
	Maltooligosaccharides (10%)	50 m*M* Tris– or TEA–phosphate, pH 3.25	[183]
	AGP (0.5 m*M* region)	50 m*M* phosphate, pH 6, 10% 1-propanol	[171]
	β-CD-Phos (0.4 m*M*)	50 m*M* phosphate, pH 5	[93]
	SBE-β-CD (3 m*M*) or CM-γ-CD (1 m*M*)	50 m*M* phosphate, pH 5	[93]
	γ-CD-Phos (1 m*M*)	50 m*M* phosphate, pH 5	[93]
Verapamil and analogues	STDC (50 m*M*)	16 m*M* NaCl and 50 m*M* polyoxyethyleneether	[223]
Warfarin	DM-β-CD (4 m*M*)	0.1 *M* phosphate, pH 8.35, 2% MeOH	[301]
	SBE-β-CD (6 mg/ml)	50 m*M* phosphate, pH 6	[88]
	Avidin (0.025 m*M*)	50 m*M* phosphate, pH 6, 10% THF	[166]
	BSA (0.05 m*M*)	50 m*M* phosphate, pH 6.8	[155]

Table 1. Continued

Drug	Selector	BGE	Reference
	Dextrin (10%)	0.1 *M* phosphate–pyrophosphate, pH 6 or 7	[181]
	Glucidex 2 (3%)	10 m*M* Tris–phosphate, pH 7	[182]
	Dextrin 10 (15 m*M*)	20 m*M* TAPS–6.5 m*M* Tris, pH 7.7, 4% ethanol	[180]
	MeNH-β-CD (5 m*M*) or	50 m*M* phosphoric acid, 50 m*M* acetic acid	[74]
	$(MeNH)_7$-β-CD (5 m*M*)	50 m*M* boric acid and NaOH, pH 7	
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	Vancomycin (2 m*M*)	50 m*M* phosphate, pH 7, 25 m*M* SDS	[136]
	Dextrin (6%)	20 m*M* phosphate, pH 2.5	[185]
	poly-(L-SUVal) (0.5%)	25 m*M* phosphate, pH 5.9	[203]
Zoplicone	HP-β-CD (30 m*M*)	50 m*M* phosphate, pH 3.3	[252]
	β-CD (15 m*M*)	0.1 *M* phosphate, pH 2.5	[62]

[a]Diastereoisomers only.

without dividing the tables according to separation principles.

2. Indirect separation by derivatization to diastereomers

Indirect separation of enantiomers involves the formation of diastereomeric derivatives using chiral derivatization reagents. The diastereomeric pairs formed can be separated in an achiral environment. Since this approach is more complicated and requires an additional reaction step, direct separation is usually preferred, if possible. A suitable derivatization reagent has to be available in approximately 100% enantiomeric purity and corresponding functional groups have to be present in the analyte. Furthermore, care must be taken that no racemization takes place during the derivatization process. Therefore, only a limited number of indirect methods for chiral separation have been reported in CE. In the case of small molecules and aliphatic compounds, which often are not able to exhibit suitable stereoselective interactions with the chiral selector necessary for chiral recognition, the indirect method is the only practicable way.

Several reagents, familiar from HPLC, have also been used for indirect separation in CE. Most of the separations were carried out using the principle of micellar electrokinetic chromatography (MEKC). 1-Fluoro-2,4-dinitro-5-L-alanine (Marfey's reagent) was used for the derivatization of amino acids, dipeptides [9] and amphetamine analogues [10]. 2,3,4,6-Tetra-*O*-acetyl-D-glucopyranosyl isothio-

Table 2
Chiral separation of pesticides and herbicides

Compound	Selector	BGE	Reference
Ethofamesate	SBE-β-CD (40 mg/ml)	pH 9	[261]
Napropamide	SBE-β-CD (40 mg/ml)	pH 9	[261]
Phenoxy acid derivatives	TM-β-CD (25 m*M*)	25 m*M* acetate, pH 4.5	[303]
Phenoxy acid derivatives (as ANDSA derivatives)	β-CD and TM-β-CD		[304]
Phenoxy acids	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 6	[127]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,131]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6 (ACN)	[127,132]
	OM (60 m*M*)	0.1 *M* phosphate–acetate, pH 5 (pH 7)	[214]
	OG (10–150 m*M*)	0.2 *M* phosphate, pH 6.5	[213]
Polychlorinated biphenyls	γ-CD (50 m*M*)	0.1 *M* CHES, pH 10, 2 *M* urea, 0.11 *M* SDS	[305]
Silvex	Deoxy Big CHAP (50 m*M*)	0.4 *M* borate, pH 10	[216]

Table 3
Chiral separation of amino acids and derivatives by CE

Compound	Selector	BGE	Reference
N-Acetyl amino acids	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,132]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,131]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6 (ACN)	[127,132]
Amino acids	18C6TCA (30–50 m*M*)	pH 2.2	[141]
	18C6TCA (30 m*M*)	6 m*M* BTA–5 m*M* Tris–citric acid, pH 2.2	[139]
	18C6TCA (30 m*M*)	0.1 m Tris, pH 2.5	[302]
	18C6TCA (25–50 m*M*)	FA 2.5 m*M* TBAP	[148]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.2	[96]
	L-Hydroxyproline (50 m*M*)	ammonia, pH 4.0 (SDS, urea)	[234]
	Cu(II) (25 m*M*)		
	DR: Marfey' reagent	MEKC	[9]
	DR: GITC	MEKC	[11]
	DR: OPA–*N*-acetyl-L-cysteine	MEKC	[13]
	DR: OPA–TATG	MEKC	[15,16]
	DR: (+)-O,O′-Diacetyl-L-tartaric anhydride	MEKC	[18–20]
	DR: FLEC	40 m*M* borate, pH 9.2, 15 m*M* SDS, 15% 2-propanol	[21]
	DR: SAMBI or SNEIT	MEKC	[24]
Amino acid carbamates	NG (80 m*M*)	50 m*M* bicarbonate–0.1 *M* NaOH, pH 10.5	[212]
FMOC-amino acids	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 6	[127]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,131]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6 (ACN)	[127,132]
AQC-amino acids	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 6	[127]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,131]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6 (ACN)	[127,132]
	DDCV (100 m*M*)	25 m*M* phosphate–borate, pH 9	[206]
AQC sulfur- and selenium-	Vancomycin (0.7 m*M*)	20 m*M* MOPS–Tris, pH 7	[130]
containing amino acids	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,131]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6 (ACN)	[127,132]
N-Benzoyl amino acids	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 6	[127]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,131]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6 (ACN)	[127,132]
	SDVal (25 m*M*)	25 m*M* borate–50 m*M* phosphate, pH 7	[197]
N-3,5-DNB-amino acids	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6 (ACN)	[127,132]
	Sulfated β-CD (9 m*M*)	10 m*M* phosphate, pH 3.2	[96]
	SDVal (25 m*M*)	25 m*M* borate–50 m*M* phosphate, pH 7	[197]
	Quinine (2.4 m*M*)	13 m*M* acetate in MeOH	[250]
	NEC-β-CD (9.7 m*M*)	50 m*M* phosphate, pH 6.52	[66]
N-3,5,DNPyr-amino acids	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6 (ACN)	[127,132]
Carbobenzyloxy amino acids	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6 (ACN)	[127,132]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,131]
Di- and tripeptides	18C6TCA (10–25 m*M*)	10 m*M* Tris–citric acid, pH 2, (methanol)	[144]
	18C6TCA (5–15 m*M*)	10 m*M* Tris–citric acid, pH 2.5	[143]
	DR: Marfey' reagent	MEKC	[9]
	DR: FLEC	40 m*M* borate, pH 9.2, 15 m*M* SDS, 15% 2-propanol	[22]
DNS-amino acids	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 6	[127]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,131]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6 (ACN)	[127,132]
	β-CD (100 m*M*)	NMF, 10 m*M* NaCl	[247]

Table 3. Continued

Compound	Selector	BGE	Reference
	Deoxy Big CHAP (50 m*M*)	0.3 *M* borate, pH 10	[216]
	STDC (50 m*M*)	50 m*M* phosphate, pH 3 (SDS)	[218]
	Glycyrrhizic acid (30 m*M*)	20 m*M* borate–10 m*M* phosphate, pH 7, 10 m*M* SDS	[226]
	L-Histidine (5 m*M*), Cu(II) (2.5 m*M*)	10 m*M* acetate, pH 7–8	[230]
	Aspartame (5 m*M*), Cu(II) (2.5 m*M*)	10 m*M* acetate, pH 7.4	[231]
	N,*N*-Didecyl-L-alanine (5 m*M*)	10 m*M* acetate, pH 7.4, 20 m*M* SDS	[232]
	Cu(II) (25 m*M*)	10% glycerol	
	N,*N*-Didecyl-L-alanine (4 m*M*)	20 m*M* acetate, pH 7, 50 m*M* SDS	[233]
	Cu(II) (2 m*M*)	5% glycerol	
	Rifamycin SV (25 m*M*)	0.1 *M* phosphate, pH 7, 30% 2-propanol	[128]
2,4-DNP-amino acids	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 6, (SDS)	[127,129]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,131]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6, (ACN)	[127,132]
	BSA (0.3 m*M*) or AGP (0.3 m*M*) or	20 m*M* phosphate, pH 7.1	[169]
	Ovomucoid (0.3 m*M*) or		
	Casein (25 mg/ml)		
FMOC di- and tripeptides	Vancomycin (1 m*M*)	50 m*M* phosphate, pH 7.6	[133]
PTH amino acids	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,132]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,132]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6, (ACN)	[127,132]
	Digitonin (25 m*M*)	50 m*M* phosphate, 50 m*M* SDS, pH 3	[195]
	β-Escin (25 m*M*)	50 m*M* phosphate, 50 m*M* SDS, pH 3	[226]
	SDVal (50 m*M*)	30 m*M* SDS, 0.5 *M* urea, pH 9, 10% MeOH	[198]

cyanate (GITC) was applied to the indirect chiral separation of amino acids [11], some sympathomimetics and amphetamine analogues [12]. Kang and Buck [13] derivatized amino acids with *o*-phthalaldehyde (OPA) and *N*-acetyl-L-cysteine or *N-tert*.-butyloxycarbonyl-L-cysteine to diastereomeric isoindole derivatives which were resolved by MEKC. Dette et al. [14] reversed this process and utilized an amino acid, L-valine, as a reagent in combination with OPA for determination of the optical purity of *N*-acetyl-L-cysteine. OPA in combination with 2,3,4,6-tetra-*O*-acetyl-1-thio-β-D-glucopyranose (OPA/TATG) was used to resolve 36 amino acids by Tivesten and Folestad [15]. More recently, the same group presented an on-column chiral derivatization procedure using OPA/TATG. The chiral reagent, dissolved in acetonitrile, is injected onto the capillary as a plug just after the sample plug. As the reagent components are uncharged, they move with the same velocity as the electroosmotic flow (EOF). The negatively charged amino acids have a lower net mobility, therefore, the reagent passes the sample zone, thereby forming the diastereomeric derivatives in-situ [16].

Leroy et al. [17] used OPA or naphthalene-2,3-dicarboxaldehyde (NDA) in the presence of either chiral thiols or amines for the resolution of drugs with primary amino groups or thiol drugs, respectively.

(+)-*O*,*O′*-Diacetyl-L-tartaric anhydride was employed for the chiral separation of tryptophan and amino acid derivatives using a polyacrylamide-coated capillary and polyvinylpyrrolidone as the additive to the BGE [18–20].

(−)-[1-(9-Fluorenyl)-ethyl]chloroformate (FLEC) was used for the indirect separation of amino acids [21], di- and tripeptides [22] and carnitine [23]. Other chiral derivatization reagents for amino acids are (*S*)-1-phenylethyl isothiocyanate and (*S*)-1-(1-naphthyl)ethyl isothiocyanate [24], which have been used successfully in HPLC.

Recently, Noe and Freissmuth [25] have reported the chiral separation of aldohexoses and aldotrioses after derivatization with *S*(−)-1-phenylethylamine.

Table 4
Enantiomer separation of miscellaneous compounds by CE

Compound	Selector	BGE	Reference
Aldoses	DR: *S*-(−)-1-phenylethylamine	50 m*M* borate, pH 10.3	[25]
Aldoses as 5′-amino-2-naphthalenesulfonates	β-CD (12 m*M*)	0.5 *M* borate, pH 8–10	[120]
amino alcohols	18C6TCA (30 m*M*)	10 m*M* Tris–citric acid, pH 2.2	[141]
	18C6TCA (30 m*M*), pH 2.07		[142]
	18C6TCA (10 m*M*)	FA 40 m*M* TBAP	[148]
	Sulfated β-CD (9 m*M*)	10 m*M* phosphate, pH 3.2	[96]
Aminotetraline derivatives	18C6TCA (30 m*M*)	50 m*M* phosphate, pH 2.2	[145]
	18C6TCA (10–15 m*M*)	10 m*M* Tris–citric acid, pH 2.2	[146]
1′-(9-Anthryl)-2,2,2-trifluoroethanol	PMPC (coated)	CEC: 40 m*M* phosphate, pH 7, 40% ACN	[192]
Benzoin	β-CD-NH_2 (5 m*M*)	39.1 m*M* phosphate, 18 m*M* ammediol, pH 2.3	[75]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	DDCAS (25 m*M*)	25 m*M* acetate–phosphate, pH 4–5.5	[211]
	DDCV (25 m*M*)	0.1 *M* CHES–10 m*M* TEA, pH 8.8, 25% ACN	[210]
	AGP–CSP (packed)	2 m*M* phosphate, pH 5.5, 5% 1-propanol	[176]
	HSA (packed)	CEC: 4 m*M* phosphate, pH 7	[165]
1,1′-Binaphthyl-2,2′-diamine	Deoxy Big CHAP (15 m*M*)	0.15 *M* borate, pH 10	[216]
	18C6TCA (10 m*M*)	20 m*M* Tris–phosphoric acid, pH 2.06	[147]
	Dextrin (6%)	20 m*M* phosphate, pH 2.5	[185]
	Dextran 40 (3%)	20 m*M* phosphate, pH 2.5	[185]
	poly-(L-SUVal) (0.5%)	25 m*M* phosphate, pH 5.9	[203]
1,1′-Binaphthyl-2,2′-dicarboxylic acid	Maltose or maltotriose or maltoheptaose (0.4 *M*)	40 m*M* carbonate, pH 9	[184]
	Cannamycin (3%) or fradiomycin (3%)	20 m*M* phosphate–borate, pH 7.5–9, 30% MeOH	[138]
	Dextran 40 (3%)	20 m*M* phosphate, pH 2.5	[185]
1,1′-Binaphthyl-2,2′-diol	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	Big CHAP (20 m*M*)	0.15 *M* borate, pH 10	[216]
	STDC (50 m*M*)	20 m*M* phosphate–borate, pH 7	[220]
	STDC (50 m*M*)	16 m*M* NaCl and 50 m*M* polyoxyethyleneether	[223]
	β-CD-Phos (5 m*M*)+α-CD (10 m*M*) +TM-β-CD (10 m*M*)	25 m*M* phosphate, pH 10.5, (55 m*M* SDC)	[65]
	poly-(L-SUVal) (0.5%)	25 m*M* phosphate, pH 5.9	[203]
1,1′-Binaphthyl-2,2′-diyl hydrogen phosphate	*N*-*p*-*tert*.-butyl calix[4]arene tetra-carboxylyl-L-alanine *tert*.-butyl ester	40 m*M* phosphate, pH 11	[151]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	Deoxy Big CHAP (15 m*M*)	0.1 *M* borate, pH 10	[216]
	Quinine (2.4 m*M*)	13 m*M* acetate in MeOH	[250]
	Streptomycin (3%) or fradiomycin (3%)	20 m*M* phosphate–borate, pH 7.5–9, 30% MeOH	[138]
	Dextrin (3%)	20 m*M* phosphate, pH 2.5	[185]
	Dextrinsulfopropylether (2%)	40 m*M* phosphate, pH 6	[190]
	SBE-β-CD (0.5 m*M*)	50 m*M* phosphate, pH 6	[77]
Diols	β-CD (1.8%)–borate	50 m*M* borate, pH 9.3, (MeOH)	[121,122]
Hydrobenzoin	β-CD-NH_2 (5 m*M*)	39.1 m*M* phosphate–18 m*M* ammediol, pH 2.3	[75]
	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	β-CD (1.8%) or Succ-β-CD (3%)	50 m*M* borate, pH 9.3, (MeOH)	[122]
Hydroxy acids	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 6	[127]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,131]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6, (ACN)	[127,132]

Table 4. Continued

Compound	Selector	BGE	Reference
	Sulfated β-CD (9 m*M*)	10 m*M* phosphate, pH 3.2	[96]
	TMA-β-CD (16 m*M*)	50 m*M* acetate, pH 5	[79]
	β-CD-NH_2 (5 m*M*)	46 m*M* caproate–18 m*M* ammediol, pH 4.75	[75]
	L-Proline or L-hydroxyproline (12 m*M*), Cu(II) (6 m*M*)	20 m*M* phosphate, pH 4.4	[235]
	or aspartame (16 m*M*), Cu(II) (8 m*M*)	20 m*M* acetate, pH 4	[235]
	Allyl-TER (15 mg/ml)	0.1 *M* β-alanine-acidic acid, pH 4.2	[193]
	Poly-β-CD (0.1 g/l)	50 m*M* phosphate, pH 6	[306]
	γ-CD (15 m*M*), quinine (40 m*M*)	0.1 *M* acetate, pH 5	[110]
Naphtylethylamine	18C6TCA (30 m*M*)	10 m*M* Tris–citric acid, pH 2.2	[141]
	18C6TCA (10 m*M*)	FA, 0.1 *M* TBAP	[148]
	18C6TCA (10 m*M*)	20 m*M* Tris–phosphoric acid, pH 2.06	[147]
N-[1-(1-Naphthyl)ethyl]phthalamic acid	Quinine (2.4 m*M*)	13 m*M* acetate in MeOH	[250]
Phenylethylamine	18C6TCA (10 m*M*)	FA, 0.1 *M* TBAP	[148]
Terpenes	α-CD (1 m*M*)+ sulfated β-CD (6.5 m*M*)	10 m*M* phosphate, pH 3.3	[98]
Troeger's base	Sulfated β-CD (2%)	10 m*M* phosphate, pH 3.8	[97]
	Dextrinsulfopropylether (4%)	80 m*M* Tris–phosphate, pH 3	[190]
	poly-(L-SUVal) (0.5%)	25 m*M* phosphate, pH 5.9	[203]
Tryptophan esters	Transferrin (0.1 g/ml region)	0.1 *M* MES, pH 6	[172]
Various organic acids	Vancomycin (2 m*M*)	0.1 *M* phosphate, pH 6	[127]
	Ristocetin (2 m*M*)	0.1 *M* phosphate, pH 6	[127,131]
	Teicoplanin (2 m*M*)	0.1 *M* phosphate, pH 6, (ACN)	[127,132]
	β-CD-NH_2 (5 m*M*)	46 m*M* caproate, 18 m*M* ammediol, pH 4.75	[75]

Since sugars are not easily accessible to direct resolution methods, this technique represents a useful contribution to chiral sugar analysis.

3. Inclusion complexation

3.1. Cyclodextrins

3.1.1. Use of neutral cyclodextrins

Inclusion into the chiral cavity of cyclodextrins (CDs) represents the most frequently used approach for chiral separation by CE. CDs are cyclic oligosaccharides consisting of six (α-CD), seven (β-CD) or eight (γ-CD) glucopyranose units. CDs possess a truncated cone with a hydrophobic cavity and a hydrophilic surface. The depth of the cavity and the solubility can be increased by derivatization. The hydroxy groups in positions 2, 3 and 6 are available for derivatization. It has been shown that the enantioselectivity can drastically vary among the various derivatives. The most frequently used derivatives are heptakis-*O*-methyl cyclodextrin (M-CD), heptakis (2,6-di-*O*-methyl) cyclodextrin (DM-CD), heptakis (2,3,6-tri-*O*-methyl) cyclodextrin (TM-CD), hydroxyethyl cyclodextrin (HE-CD) and hydroxypropyl cyclodextrin (HP-CD). Not only neutral CD derivatives, but also charged CDs have been developed. The chiral recognition mechanism is based on inclusion of a bulky hydrophobic part of the molecules, preferably aromatic moieties, in the hydrophobic cavity of the CD. An additional requirement is that secondary interactions have to take effect; these include dipole–dipole interactions or hydrogen bonds between the hydroxyl groups at position 2 or 3 at the mouth of the CD and polar substituents close to the chiral centre of the analyte. Since uncharged CDs migrate at the same velocity as the EOF, they allow the separation of charged analytes only. The chiral resolution of uncharged analytes can be performed by MEKC or by using charged cyclodextrin derivatives.

The first application of CDs for chiral separations by CE was reported by Smolkova's group, using the principle of ITP [26]. This approach was shown to be applicable to the chiral resolution of drugs such as

ephedrine analogues [27], chloramphenicol, ketotifen and thioridazine [28]. The use of CDs incorporated in gels was reported by Guttman et al. [29], while Fanali [30] published the first paper dealing with the application of CDs in CZE and investigating the chiral separation of several sympathomimetic drugs. Subsequently, more than 700 compounds were resolved using CDs as chiral selectors; these include β-blockers, sympathomimetics, antipsychotics, antidepressants, hypnotics, barbiturates, local anaesthetics, bronchodilators, non-steroidal anti-inflammatories, anti-asthmatics, anticoagulants, anti-epileptics, antihypertensives, calcium-channel blockers, antimalaria drugs, antibacterials, antivirals, antifungals, etc.

Since another contribution in this issue is focused on chiral separations by CE using CDs, the applications are not discussed here in detail. A collection of applications to drugs and other compounds, however, is given in Tables 1–4.

Several parameters, such as the concentration of the chiral selector, pH, the nature and ionic strength of the BGE and the addition of organic modifiers or other additives, have been found to have an important influence on the resolution.

Wren and Rowe [31–33] postulated a separation model and showed that an optimum selector concentration exists. Modified models were discussed by Rawjee et al. [34–36] and Surapaneni et al. [37]. The degree of substitution in derivatized CDs has been found to be of great importance for chiral recognition [38–40]. Recently, Weseloh et al. [41] investigated methylated β-CDs with different substitution patterns and compared these selectors by means of the chiral separation of tranylcypromine, clenbuterol, adrenaline, amphetamine and mefenorox.

Low pH is usually used to separate cationic drugs, while high pH is used for anionic compounds. The addition of organic modifiers can drastically influence both the efficiency and the resolution. By adding 30% methanol to the BGE containing 40 m*M* β-CD in phosphate buffer, Fanali [42] observed a significant improvement in the resolution of propranolol. There are several other examples where the addition of organic modifiers improved the resolution [43–45]. Resolution can also be optimized through manipulation of the EOF. Depending on requirements, the EOF can be enhanced, suppressed or even reversed. The EOF can be suppressed not only by decreasing the pH but also by coating the capillary wall with polyacrylamide or by adding alkylhydroxyalkylcellulose [28] or polyvinylalcohol [46], to create a dynamical coating. The addition of tetraalkyl ammonium ions can also suppress or reverse the EOF [47–50]. Quang and Khaledi [49] observed a reversal of the EOF by adding tetrabutyl ammonium hydroxide to a BGE containing phosphate buffer, pH 2.5, and DM-β-CD, TM-β-CD or HP-β-CD as chiral selectors. Since the migration of cationic analytes is opposite to that of the EOF, an increase in the mobility difference between the analyte and the selector–analyte complex is obtained. This system allowed the resolution of trimipramine [49], β-blockers [48] and sympathomimetics [47]. A similar approach was employed by Stalberg et al. [50] to improve the resolution of local anaesthetic drugs. An interesting new technique for reversal of the EOF has been developed by Hong and Lee [51]. By applying positive radial potential gradients across the capillary wall, the surface charge and the ξ-potential at the capillary–solution interface became less negative. The EOF is thereby reversed and is opposite to the electrophoretic migration of the cationic analytes. The chiral separation of some sympathomimetic drugs using this procedure illustrates the improvement in resolution and efficiency.

Uncharged β-CD polymers were used to resolve trimetoquinol analogues [52,53], β-blockers, sympathomimetics, anaesthetics [52,54], amphetamine analogues [55,56] and 2-hydroxy acids [57].

The chiral resolution of amphetamine analogues was also studied by Varesio and Veuthey [58], Wang et al. [59] and Cladrowa-Runge et al. [10], who compared different CD derivatives. An investigation of the optical resolution of designer drugs on the basis of methylenedioxyamphetamines using CDs has recently been reported by Gaus et al. [60]. The systematic screening of a broad spectrum of drugs and comparing α-CD [61], β-CD [62] and γ-CD [63] as chiral selectors was undertaken by Koppenhoefer et al. This work demonstrated that the ring size of the CD has to be adapted to the size and structure of the analyte. Lindner et al. [43] investigated the chiral separation of several types of amino acid derivatives, such as carboxybenzyl (CBZ)-, 3,5-dinitrobenzyl (DNB)-, 2,4-dinitrophenyl (DNP)-, 9-fluorenylmethoxycarbonyl (FMOC)-, 5-dimethylaminonaphthylsulfonyl (DNS)- and 6-amino-

quinolyl-*N*-hydrosuccinimidylcarbamoyl (AQC)-amino acids, using native CDs and CD derivatives. The authors showed that suitable selectors and conditions have to be chosen for the different types of AA derivatives. Modified CDs were found to exhibit better enantioselectivity in most cases. The addition of organic modifiers and decreasing the temperature to 5°C enhanced chiral resolution. The resolution of underivatized di- and tripeptides using β- or γ-CD was recently described by Wan and Blomberg [22]. Yoshinaga and Tanaka [64] studied the influence of urea and urea derivatives on the chiral recognition of CDs by means of DNS-AAs. Urea has been used by several groups to increase the solubility of β-CD. These authors found that urea and urea derivatives greatly influence the enantioselectivity. Interestingly, enantioselectivity was found to be enhanced with unmodified CDs and reduced with CD derivatives. In a recent study, Nishi [65] compared the enantioselectivity of native CDs, HP-α (or β and γ)-CD, DM-β-CD, TM-β-CD, 6-*O*-α-D-glucosyl-α (or β)-CD (Glu-β-CD), 6-*O*-α-D-maltosyl-β-cyclodextrin (Mal-β-CD), heptakis(2,3-di-*O*-acetyl)-β-cyclodextrin (Ac-β-CD) and α-(or β and γ)-CD phosphate using three binaphthyl derivatives as test solutes. The author has shown that, in many cases, the use of a mixture of different CDs improved the resolution. Gahm and Stalcup [66] have recently introduced naphthylethylcarbamoylated β-CDs for the chiral separation of 3,5-DNP-AAs by CE and HPLC. Besides stereoselective inclusion, $\pi-\pi$ interactions between the nitro groups of the analyte and the naphthyl moiety are discussed to be effective.

A useful guide for a rational procedure in chiral separation problems using CDs was given by Guttman et al. [67–69]. A ''CD-array chiral analysis'' scheme is presented, starting with unmodified CDs and continuing with several CD derivatives at different concentrations. For basic analytes, low pH is to be applied and for acidic compounds, a high pH should be used. Several starting buffers are recommended. Further means for optimizing the resolution are changing the temperature and the electric field strength, EOF manipulation and reversing the polarity. The system was explicated with a series of drugs.

An ultrafast chiral separation of basic drugs using CDs has recently been reported by Aumatell and Guttman [70] using DM-β-CD as a selector. Chiral resolution of metaproterenol and isoproterenol was achieved within 40–50 s. The peak efficiency was said to be more than one million theoretical plates/m.

3.1.2. Use of charged cyclodextrins

Charged CDs have been employed for both charged and uncharged analytes. In the ionized state, they migrate with their own electrophoretic mobility, therefore, this approach can also be regarded as a kind of EKC [71].

Further advantages of charged CD derivatives are better solubility and the ability to display additional electrostatic interactions. Ion-pairing effects might also play an important role.

3.1.3. Use of cationic CDs

Terabe et al. [71,72] used mono(6-β-aminoethylamino-6-deoxy)-β-CD for the chiral separation of DNS-AAs. 6^{A}-Methylamino-β-CD, 6^{A},6^{D}-dimethylamino-β-CD and heptamethylamino-β-CD were investigated to determine if they were suitable for the resolution of hydroxy acid derivatives [73] and several anionic and cationic drugs [74]. A reversal of the enantiomer migration order was observed for phenylacetic acid with the change from a coated to an uncoated capillary [74]. Lelièvre et al. [75] have recently reported the use of mono(6-amino-6-deoxy)-β-CD (β-CD-NH_2) for the chiral separation of benzoin and its derivatives, hydroxy acids, several organic acids and chlorthalidone. Continuing these studies, the same group demonstrated that a dual CD system can greatly enhance stereoselectivity. While β-CD-NH_2 alone showed no enantioselectivity for non-steroidal anti-inflammatories, a mixture of β-CD-NH_2 and TM-β-CD allowed baseline resolution of these compounds [76]. Quaternary ammonium salts of β-CD were used for the resolution of mandelic acid analogues and binaphthyl derivatives [77]. Rousell and Favrou [78] investigated 2-hydroxy-3-trimethylammoniopropyl-β-CD as a new chiral selector for the separation of phenylthiohydantoin (PTH)- and methylthiohydantoin (MTH) AAs and some basic and acidic drugs and compared the results with those obtained with β-CD and carboxymethyl (CM)-β-CD. The same chiral selector was used by Bunke and Jira [79] for the enantiomer separation of basic and acidic

analytes. As with EOF modifiers such as tetraalkylammonium ions, a reversal of the EOF was observed with 2-hydroxy-3-trimethylammoniopropyl (TMA)-β-CD.

3.1.4. Use of anionic CDs

Anionic CDs such as CM-β-CD, carboxyethyl-β-cyclodextrin (CE-β-CD), succinyl-β-cyclodextrin (Succ-β-CD), sulfobutyl-β-cyclodextrin (SBE-β-CD), sulfoethyl-β-cyclodextrin (SEE-β-CD), CD phosphates and sulfated CDs have been applied to chirally resolve cationic, neutral and even anionic compounds.

Schmitt and Engelhardt [87] studied the behaviour of anionic CDs such as CM-β-CD, CE-β-CD and Succ-β-CD for the chiral resolution of cationic as well as uncharged compounds using coated capillaries at different pH values. At pH values below 4, the carboxylic group is not charged and acts as a pseudo-stationary phase, while at pH>5, the charged CD has its own mobility and moves towards the anode, thereby allowing the chiral separation of uncharged analytes to take place. Cationic analytes migrate towards the cathode at low pH values, since the CD derivative is not charged at low pH. Changing to pH>5, the carboxyl functional CD will be negatively charged, forming an ion-pair with the analyte and, thus, transporting the analyte to the anode. This reversed the migration order of the enantiomers. The possibility of changing the migration order of enantiomers is advantageous if the enantiomeric excess of a drug is to be determined. The minor enantiomer should always be the first-migrating component. Similar observations on the reversal of the elution order of the enantiomers of cationic drugs were made by Aturki and Fanali [80] using a carboxyl-functional CD polymer [80].

Reversal of the enantiomer migration order can also be achieved by reversing the EOF using quaternary ammonium bases, either as buffer additives or applied to the capillary wall as a coating.

Recently, an interesting study on the possibility of reversing the enantiomeric elution order was published by Chankvetadze et al. [77]. The studies were carried out with 1,1′-binaphthyl-2,2′-diyl hydrogenphosphate as an anionic model analyte using uncoated capillaries and neutral, positively and negatively charged CDs were compared. One way of reversing the elution order of enantiomers is to change from an uncharged CD to a strongly negatively charged CD (SBE-β-CD) having a higher mobility than that of the analyte. A second option is to change from low to high pH, thus changing the mobilities of both the selector (CM-β-CD) and the analyte. The third approach involves changing the pH and reversing the polarity. For all CD derivatives, except CM-β-CD, a reversal of the enantiomer elution order was observed in the latter case.

Of course, the simplest way to reverse the enantiomer elution order would be to use a chiral selector with the opposite conformation, which is not practicable in the case of CDs. The possibility of reversing the enantiomer elution order by changing from CD–CZE mode to CD–MEKC is discussed in Section 3.1.3.

CM-β-CD was used for the chiral separation of various cationic and uncharged drugs, among them being ephedrine, hexobarbital, dimetinden, doxylamine, pindolol [87], propranolol [81] and thalidomide and its metabolites [82]. Succ-β-CD has recently been shown to exhibit excellent enantioselectivity for several β-blockers, sympathomimetics and various other drugs [83]. Fig. 2 shows the chiral separation of chlortalidone using Succ-β-CD in borate buffer, pH 7.3.

SBE-β-CD proved to be a very effective selector for a broad spectrum of drugs, including sym-

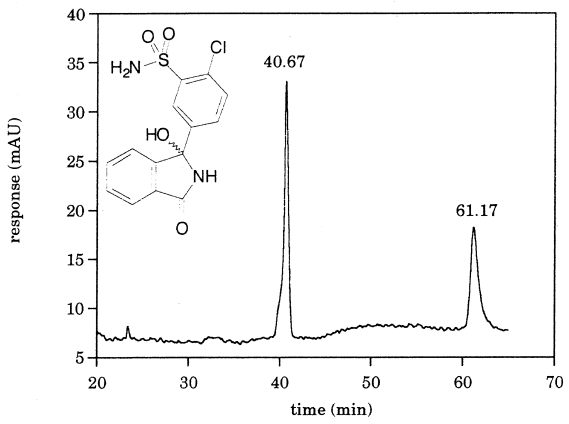

Fig. 2. Electropherogram of the chiral separation of DL-chlortalidone. Electrolyte: 3.3% Succ-β-CD, 50 m*M* borate, pH 7.3. Capillary: fused-silica, untreated, 60 cm×75 μm I.D. (effective length, 51 cm). Voltage: 15 kV; detection: 208 nm (from Ref. [83]).

pathomimetics [84,85], β-blockers [86], anticoagulants [88], mianserine analogues [89] and imidazole derivatives [90]. When chirally separating several drugs of forensic interest, Lurie et al. [91] found that mixtures of SBE-β-CD and neutral CDs show a synergistic effect. Besides SBE-β-CD, SBE-γ-CD has also been tested recently for enantiomer separation and compared with native γ-CD by means of cationic and anionic analytes including DNS-AAs, binaphthyl derivatives, aminoglutethimide, mianserine, butaclamol, etc. A reversal of the enantiomer elution order was observed on changing from γ-CD to SBE-γ-CD [92]. In a recent study, Tanaka et al. [93] compared the enantioselectivity of CM-β-(and γ)-CD, β (and γ)-CD phosphate and SBE-β-CD for a spectrum of about 40 basic racemates.

Sulfated CDs also showed enantioselectivity for a great variety of neutral, basic as well as acidic compounds, including antihistamines, anaesthetics, β-blockers, antimalarials, antidepressants, antiarrhythmics, anticholinergics, etc. [90,94–97].

Gahm et al. [98] explored the synergistic effect of sulfated-β-CD on the optical resolution of monoterpenes with native α-CD. Although sulfated CDs are not able to resolve monoterpenes, they act as a carrier for the analyte to the anode, thus supporting the interactions with α-CD.

Chankvetadze et al. [90] presented an interesting approach, using the chiral selector in a countercurrent flow. SBE-β-CD was applied at the detector end of the capillary and migrated as a zone counter current to the zone of the analyte to the anode. This technique is of interest for avoiding a detector response by the selector when UV-absorbing selectors are used.

An excellent review on the use of charged CDs has recently been published by Chankvetadze et al. [99]. A more detailed discussion of charged CDs is given in another article in this issue.

3.1.5. CD-mediated micellar electrokinetic chromatography

This principle is based on the use of uncharged CDs in combination with an achiral surfactant such as sodium dodecyl sulfate (SDS). Uncharged CDs migrate with an identical velocity to that of the bulk solution. Partition of a hydrophobic analyte takes place between the bulk solution, the CD and the negatively charged SDS micelle, which migrates in the opposite direction to the EOF, thus retaining the analyte. This principle was applied to the resolution of DNS-AAs [100], CBI-AAs [101], FMOC-AAs [102], 2-(9-anthryl)ethylcarbonyl (AEOC)-AAs [103] and several drugs, among them being barbiturates, glutethimide analogues [104,105], chlorpheniramine [106] and β-blockers [107]. The addition of L-menthoxy acetic acid or (+)-*S*-camphor-10-sulfonate to a solution of γ-CD and SDS was found to improve the resolution of some barbiturates [108]. The mechanism of action of these reagents is not known. As barbiturates are acidic compounds, an ion-pairing mechanism, as proposed for basic drugs [109,110], is out of the question. The use of a mixture of β- and γ-CDs in combination with SDS was shown to improve the resolution of DNS-AAs [100], with 2-methyltaurine as the DNS derivative [111]. Changing from CD–CZE mode to CD–MEKC can be a way of reversing the EOF. Katayama et al. [112] reversed the enantiomermigration order of (*R*,*S*)-1-benzyl-4-[(5,6-dimethoxy-1-indanon)-2-yl]methylpiperidine by changing from CD–CZE with DM-β-CD on a coated capillary to CD–MEKC using DM-β-CD and SDS on an uncoated capillary.

In place of SDS, chiral surfactants were also added to CDs for CD–MEKC. Taurodeoxy cholate (STDC) was used in combination with CDs for the chiral separation of DNS-AAs [113,114], CBI-AAs [115] and some drugs, including sympathomimetics, β-blockers, amphetamine derivatives [116] and mephenytoin analogues [113].

Smith et al. [117] described the combination of γ-CD and decanoyl-*N*-methylglucamide as a borate complex for the chiral separation of DNS-AAs. Recently, a polymer surfactant, poly(sodium *N*-undecenyl-D-valinate (polySUVal) was employed, together with γ-CD, by Wang and Warner [118] for the resolution of 1,1′-binaphthol, laudanosine and verapamil. Compared to the use of polySUVal alone [119], enantioselectivity was seen to be improved.

3.1.6. Use of cyclodextrins in combination with ion-pairing reagents

This principle was first investigated by Bunke et al. [109]. The addition of (+)-*S*-camphor-10-sulfonic acid (CSA) to β-CD resulted in the resolution of

cationic drugs such as cyclodrin, while with β-CD or CSA alone, there was no resolution. Tropicamide, a neutral drug, which is not able to form an ion-pair, was not resolved. In the same vein, this group recently observed that the chiral recognition supporting effect did not depend on whether the counter ion was chiral or not [110]. The authors assumed that the analyte is included in the cavity of the CD as an ion-pair, thereby enabling the chiral C-atom of the analyte to reach a better position for its chiral recognition.

The combination of CD and quinine as an ion-pairing reagent was found to be effective for the chiral separation of acidic analytes.

3.1.7. Mixed CD–borate–diol complexation

Cis-1,2-diols are known to form borate complexes. This phenomenon has been widely used in CE for the achiral separation of carbohydrates. This basic principle has been found to be adaptable to the chiral separation of compounds with a cis-1,2-diol structure. Stefanson and Novotny [120] described the chiral separation of fluorescently labelled sugars as borate complexes using β-CD or linear dextrins. Jira et al. [121] showed that various diol compounds, including some quinazoline derivatives of potential pharmaceutical interest and containing diol groups in the side chain, can be resolved with CDs as borate complexes. Schmid et al. [122] studied different CD derivatives using borate buffer, pH 9.3, to determine their suitability for resolving cis-1,2-diols. A dual chiral recognition mechanism is proposed based on inclusion of the aromatic substituent of the diol–borate complex into the cavity of the CD and interaction of the borate with the hydroxyl groups at C-2 and C-3 at the mouth of the CD. These interactions can simply be hydrogen bonds; however, the formation of mixed borate complexes is also possible. Although these hydroxyl groups are in the *trans* position, they are diequatorially positioned and might participate in mixed borate complexation. This hypothesis is supported by the fact that no separation was observed when borate was replaced by other electrolytes. Furthermore, the availability of free hydroxyl groups at C-2 and C-3 was a requirement for obtaining resolution. Good resolution was obtained using β-CD or Succ-β-CD. M-β-CD, HE-β-CD and HP-β-CD resulted in partial resolution, depending on the availability of hydroxyl groups at C-2 and C-3 in limited amounts due to the varying substitution pattern. Since chiral separation failed to occur with 2,6-DM-β-CD and 2,3,6-TM-β-CD, the authors conclude that these hydroxyl groups are essential for chiral recognition.

Fig. 3 illustrates the resolution power of this technique by means of the chiral separation of hydrobenzoin.

The formation of a ternary CD–borate–diol complex has also been taken into account by Noroski et al. [123] in connection with the enantiomer separation of a new cholesterol-lowering drug (BMS-180431-09) having a diol structure, using β-CD or HP-β-CD, borate and SDS as the BGE. Negative results were also obtained in this case with 2,6-DM-β-CD and triacetyl-β-CD.

3.2. Use of macrocyclic antibiotics

Macrocyclic antibiotics have been introduced by Armstrong et al. [124–126] as a new class of chiral selectors, which show enantioselectivity for a broad spectrum of compounds. Macrocyclic antibiotics have multiple chiral atoms and several functional groups that are capable of multiple interactions to enable chiral recognition. There are hydrophobic pockets that can interact with hydrophobic groups,

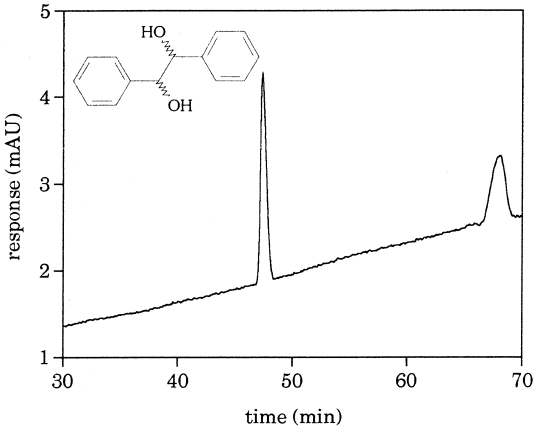

Fig. 3. Electropherogram of the chiral resolution of *RR*,*SS*-hydrobenzoin. Conditions: 1.8% β-CD, 50 m*M* borate, pH 9.3, 20% MeOH. Capillary: untreated fused-silica, 60 cm×75 μm I.D. (effective length, 51 cm); voltage: 18 kV, detection: 214 nm (from Ref. [122]).

and pendent polar arms that can form hydrogen bonds. The primary interactions of macrocyclic antibiotics are assumed to be charge–charge or ionic interactions. The secondary interactions proposed are hydrogen bonding, steric repulsion, hydrophobic-, dipole–dipole- and $\pi-\pi$ interactions [124–127].

Two classes of macrocyclic antibiotics have been investigated as chiral selectors in HPLC and CE:

The ansamycins, rifamycin B [126,128] and rifamycin SV [128], and the glycopeptides, vancomycin [124,127,129], ristocetin A [127,131] and teicoplanin [127,132].

Rifomycin B is negatively charged and is suitable for resolving cationic compounds like β-blockers, sympathomimetics and several barbiturates [126,128], while rifamycin SV was shown to posses higher resolution power towards rather negatively charged compounds containing two rings [128].

Macrocyclic glycopeptide antibiotics have an aglycone portion consisting of fused macrocyclic rings that form a ''basket'' shape and have several pendent sugar moieties. They are positively charged under the conditions applied and have been used in the chiral separation of a broad spectrum of anionic solutes. About 350 compounds have been resolved successfully with glycopeptide antibiotics, including non-steroidal anti-inflammatory drugs, antineoplastics, hydroxy acids, some herbicides and *N*-blocked amino acids [124,127–136]. Teicoplanin is surface active and forms micelles, due to its appended hydrocarbon chain. Self-aggregation seems to decrease enantioselectivity compared to that of vancomycin and ristocetin A [132]. A comparison of the enantioselectivities of vancomycin, teicoplanin and ristocetin A, including modelling studies, is given by Gasper et al. [127]. The most versatile selector among the macrocyclic glycopeptide antibiotics was found to be ristocetin A. About 120 compounds, including *N*-blocked amino acids, non-steroidal anti-inflammatory drugs, hydroxy acids, etc., were resolved into their enantiomers [127,131]. The addition of SDS to vancomycin was found to enhance the efficiency by one order of magnitude and to reverse the enantiomer migration order [129]. When vancomycin binds to SDS, its mobility changes from positive to negative. Partition of acidic analytes between the bulk phase, the free vancomycin and the mixed micelle takes place. These studies have recently been extended to neutral compounds [136]. Vancomycin has also been used for the chiral separation of di- and tripeptides (as their FMOC derivatives) [133] and AQC derivatives of AAs containing sulfur or selenium [130,137].

Recently, Ward et al. [135] created a counter current process by using a coated capillary to suppress EOF, whereby the chiral selector migrates away from the detection zone, thus improving sensitivity. Non-steroidal anti-inflammatories and DNS-AAs were resolved by this approach using vancomycin as the chiral selector.

Limiting factors in the use of macrocyclic antibiotics as chiral selectors in CE are the UV absorption up to 250 nm and the limited stability. Recently, aminoglucoside antibiotics, such as streptomycin, fradiomycin and kanamycin, were also found to be applicable as chiral selectors [138]. Since they are relatively small molecules, a different chiral recognition mechanism is to be expected (see also Section 4.2).

Further information is given in a special contribution in this issue that is devoted to the use of macrocyclic antibiotics as chiral selectors.

3.3. Use of chiral crown ethers

Crown ethers are macrocyclic polyethers that are known to form host–guest complexes with alkali- and earth metal ions as well as with primary ammonium cations. With the use of chiral crown ethers, the inclusion of primary amines is stereoselective.

The main interactions are assumed to be hydrogen bonds between the three hydrogens attached to the nitrogen of the analyte and the dipoles of the oxygens of the macrocyclic ether. The carboxylic groups of the crown ether are perpendicular to the plane of the macrocyclic ring, forming a chiral barrier, which divides the space available for the substituents at the chiral centre of the analyte into two domains. Thus, two different diastereomeric inclusion complexes are formed. In addition, electrostatic interactions of the carboxylic acid groups with polar groups of the analyte are to be taken into account [139,140].

This principle, successfully applied in HPLC, was introduced to CE by Kuhn et al. [141] using (+)-18-

crown-6-tetracarboxylic acid (18C6TCA) for the chiral separation of amino acids. Separations are carried out at low pH (pH 2–2.5) to protonate the amines. At this pH, the EOF is very low. The crown ether was either used without any additional electrolyte or in the presence of Tris–citric acid or Tris–phosphoric acid buffers. Indirect detection by the addition of benzyltrimethylammonium chloride to the BGE was employed to separate aliphatic amino acids [139].

Besides amino acids, 18C6TCA also showed excellent enantioselectivity for amino alcohols, among them being several sympathomimetic drugs [139,142].

They have also been used for the chiral resolution of racemic dipeptides [143,144] and some tripeptides [143]. Fig. 4 shows the resolution of DL-Leu–DL-Leu into its four possible stereoisomers [144]. Although the chiral center is in the δ-position to the amino group, dipeptides were excellently resolved. In this case, the main interactions responsible for chiral recognition are postulated to be hydrogen bonds between the polar groups of the dipeptide and the carboxylic group of the selector. 18C6TCA could also resolve aminotetraline derivatives [145,146].

More recently, Nishi et al. [147] reported the chiral separation of drugs containing primary amino groups, among them being aminoglutethimide, baclofen, mexiletine and primaquine, using 10 m*M* 18C6TCA, 20 m*M* Tris–phosphoric acid, pH 1.9.

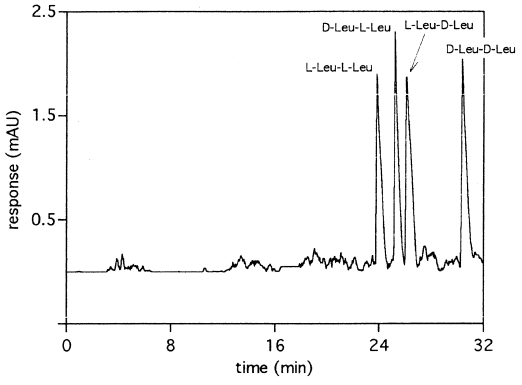

Fig. 4. Electropherogram of the chiral resolution of DL-leucyl–DL-leucine. Conditions: 15 m*M* 18C6TCA, 10 m*M* Tris–citrate, 20% methanol; capillary: untreated fused-silica, 80 cm×75 μm I.D. (effective length, 71 cm); voltage: 30 kV; detection: UV, 206 nm (from Ref. [144]).

A new approach involves the use of 18C6TCA in a non-aqueous medium [148]. Using 18C6TCA in formamide and tetra-*n*-butylammonium perchlorate, several amino compounds were separated into their enantiomers, among them being 1-phenylethylamine, which was not resolved in aqueous medium.

Cyclodextrins were found to exhibit a synergistic effect on the resolution when used in combination with a crown ether [140,149,150]. Noradrenaline was baseline resolved with a combination of 5 m*M* 18C6TCA and 20 m*M* HP-β-CD, while no or poor resolution was obtained with HP-β-CD or 18C6TCA alone [140,149]. A similar observation was made in the chiral separation of *o*, *m* and *p*-fluoro-DL-phenylalanines using a mixture of β-CD and 18C6TCA by gel electrophoresis [150].

Of course, this efficient technique of crown ether inclusion is restricted to compounds containing primary amino groups.

3.4. Use of chiral calixarenes

A new approach is the use in HPLC and CE of calixarenes, which are also able to form inclusion complexes. The first attempt to use a chiral calixarene for chiral CE separations has recently been reported by Pena et al. [151]. The authors synthesized a water-soluble *N*-acylcalix(4)arene amino acid derivative and showed preliminary results for the chiral resolution of racemic 1,1′-binaphthyl-2,2′-diyl hydrogenphosphate by CE using 40 m*M* phosphate buffer, pH 11.

It is to be expected that calixarenes will become a new class of efficient chiral selectors.

3.5. Use of chiral imprinted polymers

The use of molecularly imprinted polymers represents an innovation in chiral CE. This principle, successfully applied for chiral separations in HPLC, has recently been introduced to CE by Lin et al. [152,153]. D-Phenylalanine or D-phenylalanine anilide was used as a print molecule, loosely bound to a methacrylate polymer. After the print molecule is removed, the polymer contains imprints of the print molecule, which shows high enantioselectivity for the same or closely related molecules. The imprinted polymer was either filled into the capillary,

incorporated in an acrylamide gel [152], or bound to the capillary wall [153]. By means of CEC, phenylalanine, tyrosine and phenylglycine were resolved into their enantiomers.

Schweitz et al. [154] prepared chiral stationary phases for CEC by molecularly imprinting propranolol and metoprolol [154]. The authors described the in-situ preparation of imprinted polymers by filling the capillary with a mixture of the print molecule and the polymerization components, with polymerization taking place inside the capillary. The polymers were covalently bound to the inner wall of fused-silica capillaries by [(methacryloxy)propyl]trimethoxysilane. The imprint molecule is removed by flushing the capillary with acetonitrile and the electrolyte.

Generally, this approach shows high enantioselectivity, however, the efficiency seems to be somewhat low and the technique is, of course, limited to very closely related compounds.

4. Chiral affinity EKC

4.1. Use of proteins

After their successful use as chiral selectors in HPLC, proteins also found their way into CE.

Depending on the pH selected, proteins can be positively or negatively charged. Their charge gives them electrophoretic mobility and they can be used for the separation of neutral and charged analytes.

Several proteins, such as bovine serum albumin (BSA) [155–162], human serum albumin (HSA) [163–165], α_1-acid glycoprotein (AGP) [160], avidin [166], conalbumin [160], cellulase [155,162], ovomucoid [160,167], cellobiohydrolase I [168] and casein [169], have been used for chiral separation.

The first application of proteins as chiral selectors was reported by Birnbaum and Nilsson [161]. These authors filled a capillary with a gel consisting of BSA crosslinked with glutaraldehyde and used it to separate tryptophan enantiomers. Later, the same group used a gel consisting of a copolymerisate of BSA or cellulase crosslinked with glutaraldehyde for the resolution of β-blockers [162].

Most of the separations were done with the proteins as additives to the BGE, following the principle of affinity EKC.

The chiral separation of leucovorin stereoisomers was investigated by Barker et al. [156] using BSA. A reversal of the migration order was observed when there was a change from a bare capillary to a polyethylene glycol (PEG)-coated capillary; this was due to the difference in the EOF velocities. The synergistic effect of dextran and BSA was studied by either adding dextran to the buffer [157] or by using an immobilized BSA–dextran polymer network [158]. In a study by Busch et al. [155], the proteins BSA, AGP, ovomucoid (OVM) and fungal cellulase were compared for the chiral separation of benzoin, warfarin and promethazine [155]. Chiral CE using HSA was also found to be useful for studies of enantioselective drug–protein binding and displacement interactions [164,170].

To avoid detection problems caused by the protein passing the detector cell, Valtcheva et al. [168] introduced a 2–3-cm long plug of agarose at the injection end of the capillary, thus preventing any hydrodynamic flow. Using cellobiohydrolase I (CBH I) as the selector, they resolved several β-blockers. For the same purpose, Tanaka and Terabe [160] employed a ''partial separation zone technique''. In this approach, the capillary was partially filled with a solution of a protein, which did not reach the detector. The feasibility of this method was demonstrated with a variety of drugs using different proteins (BSA, OVM, AGP, conalbumin) [160,171]. Recently, Kilár and Fanali [172] introduced iron-free human transferrin as a chiral selector in CE and used it as a stationary zone by keeping the pH at the isoelectric point of the protein, thus preventing migration of the protein to the detector. This new approach was first applied to the chiral separation of tryptophan esters [172] and has been extended more recently to a broad spectrum of drugs [173,174].

Wistuba et al. [169] have explored, for the first time, the use of casein from goat's milk as a chiral selector and compared the results with those obtained with BSA, AGP or OVM using DNP-AAs as test solutes.

Eberle et al. [175] recently studied the chiral separation of the proton pump inhibitors pontoprazole, omiprazole and lansoprazole by using BSA as an additive in the BGE. The addition of 1-propanol was shown to improve the resolution. The use of packed capillaries with AGP or HSA as immobilized

chiral selectors for electrochromatography [165,176] is discussed in Section 8.

4.2. Use of polysaccharides

Besides CDs, linear saccharides such as maltodextrins and maltooligosaccharides were also found to be useful as chiral selectors. They are composed of α-(1,4)-linked D-glucose units. The helical structure of dextrins might be responsible for chiral recognition [178]. Hydrogen bonds and dipole–dipole interactions are assumed to be the main interactions. Such selectors were successfully applied to the chiral resolution of non-steroidal anti-inflammatory drugs and anticoagulants [179–183]. Even small oligosaccharides were found to be able to exhibit chiral recognition. Kano et al. [184] showed that maltose, maltotriose and maltoheptaose can be used for the chiral separation of binaphthyl derivatives. Nishi et al. [185] compared the enantioselectivity of dextrin and dextran for a broad spectrum of drugs. Anionic compounds including ibuprofen, naproxen, warfarin, diltiazem synthetic intermediate and 1,1′-binaphthyl-2-2′-diyl hydrogenphosphate were resolved under neutral conditions, while for cationic drugs, such as timepidium, primaquine, sulconazole, trimetoquinol, diltiazem and clentiazem, an acidic medium was required.

Charged polysaccharides were used to resolve neutral cationic drugs by means of EKC. Using dextran sulfate, Nishi et al. [186] resolved the enantiomers of trimetoquinol and dilitiazem analogues. Mucopolysaccharides, such as heparin and chondroitin sulfate, proved to be very effective chiral selectors. Ionic and hydrophobic interactions as well as hydrogen bonds are assumed to be responsible for chiral recognition. Heparin was used for the chiral resolution of antimalarials, antihistamines [187,188] dilitiazem analogues and trimetoquinol derivatives [189]. Compared to heparin and dextran sulfate, chondroitin sulfate possesses less ionic residues, therefore, ionic interactions are weaker, resulting in the wider applicability of chondroitin sulfate. Nishi et al. [189] demonstrated the high resolving power of chondroitins by means of trimetoquinol derivatives, diltiazem and analogues, primaquine, propranolol and some other drugs. Chondroitin C was found to be superior to chondroitin A. A new anionic polysaccharide, dextrin sulfopropyl ether (DSPE), has recently been developed by Jung et al. [190] and applied to a broad spectrum of drugs and other compounds. Different concentrations of DSPE in borate, phosphate, acetate and Tris–phosphate buffers of different ionic strengths and pH values were investigated, testing mianserine, verapamil, clenbuterol, formoterol, Troeger's base and binaphthyl derivatives as model analytes. As follows from a comparison of the results of DSPE with those of dextrin 10, DSPE exhibited increased the separation power for cationic analytes.

Beck and Neau [191] have recently introduced lambda-carrageenan as a new polysaccharide based selector. Lambda-carrageenan is a high molecular mass sulfated polysaccharide that has shown enantioselectivity to propranolol, pindolol, tryptophan derivatives, laudanosine, laudanosoline and the diastereomeric pairs, cinchonine and cinchonidine.

Nishi et al. [138] showed that cationic selectors on carbohydrate bases, diethylaminoethyl (DEAE)–dextran and aminoglycoside antibiotics, such as streptomycin sulfate, kanamycin sulfate and fradiomycin sulfate, can be used as chiral selectors for acidic compounds [138]. Synthetic intermediates of diltiazem were resolved with DEAE–dextran. Although aminoglycoside antibiotics are relatively small molecules containing only three or four sugar units, they were shown to exhibit chiral recognition. This was demonstrated by the enantioseparation of acidic binaphthyl derivatives. A new approach, the use of 3,5-dimethylphenylcarbamoyl cellulose and *p*-methylbenzoyl cellulose-coated capillaries, recently introduced by Francotte and Jung [192] for chiral separations by electrochromatography, is discussed in Section 8.

4.3. Ergot alkaloids

The use of ergot alkaloids as novel chiral selectors in CE was reported by Ingelse et al. [193]. A BGE containing 15 mg/ml of (+)-(5*R*,8*S*,10*R*)-1-allylterguride (allyl-TER), 100 m*M* β-alanine–acetic acid, pH 4.2, chirally resolved hydroxy acids. A drawback of this promising selector, however, is its limited solubility.

5. Micellar electrokinetic chromatography using chiral surfactants

This technique was introduced by Terabe et al. [194]. Generally, charged surfactants that form micelles which move via their own electrophoretic mobility, or uncharged surfactants together with SDS, are used. Charged as well as neutral analytes can be resolved by this technique, whereby partition of the analyte takes place between the aqueous BGE and the micelles. Analytes are adsorbed on the hydrophobic surface of the micelle and interact with the polar groups of the surfactant.

Long alkyl-chain amino acid derivatives, such as sodium *N*-dodecanoyl-L-valinate (SDVal) [195–200], sodium *N*-dodecanoyl-L-alaninate (SDAla) [197], sodium *N*-dodecyl-L-glutamate (SDGlu) [200], sodium *N*-dodecyl-L-serine (SDSer) [201] and sodium *N*-dodecyl-L-threonine (SDThr) [202] have been used for the chiral separation of amino acid derivatives, benzoin and warfarin. Recently, the use of a polymer surfactant, poly(sodium *N*-undecenyl-L-valinate) (poly SUVal) was described for the chiral separation of binaphthyl, laudanosine [119] and 3,5-dinitrobenzoyl amino acid [202] anticoagulants and binaphthyl derivatives [203].

New surfactants based on long chain amino acids, (*R*)- and (*S*)-dodecoxycarbonyl valine (DDCV) and (*R*)-dodecoxycarbonyl proline (DDCP), which were shown to exhibit improved enantioselectivity for a wide variety of compounds, have recently been introduced [204–210]. Among their applications, these chiral selectors were successfully used for the chiral separation of some β-blockers [210], sympathomimetics [208,210], piperidinediones [209] and AQC-AAs [206]. Hydrophobic interactions, dipole–dipole interactions and hydrogen bonds are the main interactions. In varying the structure of this class of reagents, Mazzeo et al. [211] synthesized (*S*)-2-[(dodecoxycarbonyl) amino]-3(*S*)-methyl-1-sulfoxypentane (DDCAS) and checked its enantioselectivity by using benzoin as a model.

A new group of surfactants containing a sugar moiety, such as heptyl-, octyl-, nonyl- and decyl-β-D-glucopyranosides, has recently been introduced and used for the chiral resolution of amino acids, as carbamates [212]. Mechref and El Rassi [213] used *n*-octyl-, *n*-nonyl-β-D-glucopyranosides [213] and octyl-β-D-maltopyranoside [214] for the optical resolution of phenoxy acid herbicides. The use of ionic alkyl-β-D-glucopyranosides, dodecyl-β-D-glucopyranoside monophosphate and -monosulfate was reported by Tickle et al. [215] and applied to the chiral resolution of DNS-AAs, binaphthyl compounds and several drugs. In-situ charged micelles were created by Mechref and El Rassi [216] by combining *N*,*N*-bis(3-D-gluconamidopropyl)cholamide (Big CHAP), -deoxycholamide (deoxy Big CHAP) (Fig. 5) and borate. The surface charge density of the micelles can be adjusted by varying the borate concentration. This makes it possible to manipulate the migration window of the Big CHAP– and deoxy Big CHAP–borate micellar system. Enantiomer resolution was optimized by varying the pH and the borate concentration. The feasibility of the system was demonstrated by using DNS-AAs, binaphthyl compounds and silvex herbicide as test solutes [216]. Fig. 6 shows the influence of borate concentration on the chiral resolution of BNDA.

Phospholipids represent a new class of chiral surfactants. Nimura et al. [217] demonstrated the applicability of L-α-palmitoyllysophosphatylcholine, a zwitterionic chiral surfactant, to the enantioseparation of DNS-AAs.

Bile acid salts, such as sodium cholate (SC), sodium deoxycholate (SDC), sodium taurocholate (STC) and sodium deoxytaurocholate (STDC), have been introduced as chiral selectors for CE by Terabe et al. [218]. Bile salts have a steroidal skeleton and form helical micelles with a reversed micelle conformation [219,220]. Bile salts have been used successfully for the chiral separation of DNS-AAs [218], carboline compounds [221], bi-naphthyl derivatives, diltiazem analogues [220], trimetoquinol analogues [219] and several other compounds. Amini et al. [222] resolved local anaesthetics with STDC

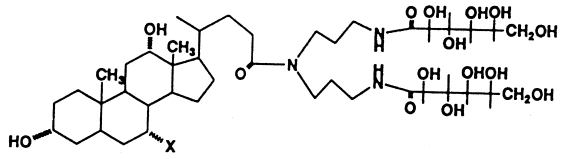

Fig. 5. Structure of Big CHAP (*X*=OH) and Deoxy Big CHAP (*X*=H) surfactants (Reprinted with permission from Ref. [216]).

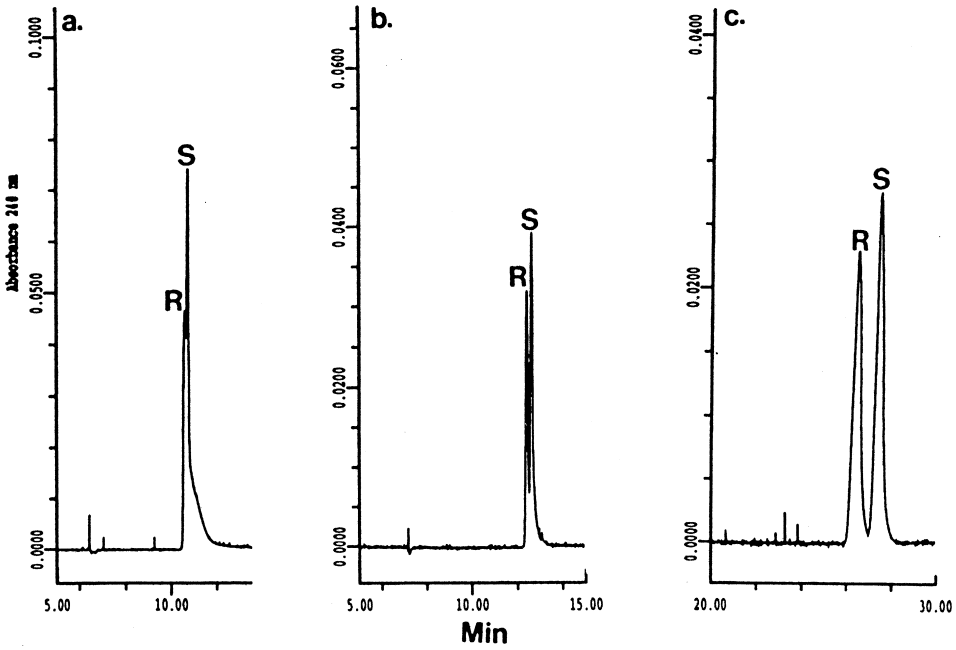

Fig. 6. Electropherogram of BNDA enantiomers obtained with Deoxy Big CHAP with various concentrations of borate in the running electrolytes. Capillary: untreated fused-silica, 50 cm (to detector), 57 cm (total length)×50 μm I.D.; running electrolytes: 25.0 m*M* (a), 50.0 m*M* (b) or 150.0 m*M* (c) sodium borate containing 15.0 m*M* Deoxy Big CHAP (pH 10.0) and 10% (v/v) methanol; capillary temperature: 15.0°C; voltage: 20 kV (reprinted with permission from Ref. [216]).

and observed an improvement in resolution and a reversal of the migration order of the enantiomers when Brij 35 [polyoxyethylene(23)-dodecanol], for forming mixed micelles, was added [222]. Similarly, a mixture of bile salts and polyoxyethylene ethers was used for the chiral separation of verapamil analogues, atenolol and bi-naphthol [223,224].

Bile salts were also found to exhibit a synergistic effect in chiral recognition in combination with CDs [113,115,116,225].

Saponins, such as digitonin [195], glycyrrhizic acid and escin [226], were also employed as micelle-forming chiral selectors. These selectors were used as mixed micelles with SDS, STDC [218] or octyl-β-glucoside [226] for the resolution of DNS-AAs and PTH-AAs.

More recently, new surfactants based on tartaric acid amides of long chain amines and taurine [227,228] have been synthesized and used to resolve several synthetic aromatic amides and urea derivatives. These new chiral selectors may have potential in the chiral resolution of a broad spectrum of compounds.

6. Use of microemulsions

Only one paper deals with the use of a microemulsion system for chiral CE separations [229]. This microemulsion system consisted of a lipophilic chiral selector, (2*R*,3*R*)-dibutyl tartrate, SDS as a surfactant and 1-butanol as a co-surfactant in the BGE. Ephedrine was used to illustrate the applicability of this system for chiral separation. No further developments on this approach, however, have been reported to date.

7. Ligand exchange capillary electrophoresis (LE-CE)

This chiral separation principle has been used

widely in HPLC by either adding a chelate complex to the mobile phase or by applying LE chiral stationary phases (CSPs). The first application of the principle of LE in CE was reported by Gassman et al. [230]. The authors used an L-histidine–Cu(II) complex as an additive to the BGE for the chiral separation of DNS-AAs. Chiral resolution was lost when Cu(II) was replaced by Co(II). Enantioselectivity was improved when aspartame was used instead of histidine [231]. The addition of tetradecyl sulfate, as a micelle-forming surfactant, was shown to be advantageous in resolving monobasic DNS-AAs. Mixed micelles consisting of *N*,*N*-didecyl-L-alanine–Cu(II) and SDS were used by Cohen et al. [232] and later by Sundin et al. [233] to resolve DNS-AAs.

The first direct separation of underivatized amino acids was reported by Schmid and Gübitz [234] using L-proline– and L-hydroxyproline–Cu(II) complexes as chiral selectors. Eleven amino acids containing aromatic groups were successfully resolved into their enantiomers. The addition of SDS significantly improved the resolution and resulted in reversal of the migration order of the analytes according to their hydrophobicity and, moreover, in a reversal of the enantiomeric migration order. Hydrophobic interactions between the micelle and the hydrophobic substituent of the amino acid are assumed to be responsible for the stronger retention of the more hydrophobic amino acids. In the mixed complex of the chiral selector with a D-enantiomer, the hydrophobic substituent of the amino acid might be more easily accessible for hydrophobic interactions with the pseudo-stationary micelle phase than in the complex with the L-enantiomer, resulting in reversal of the enantiomeric migration order compared to the experiments carried out in the absence of SDS. To illustrate this, Fig. 7 shows the chiral separation of some amino acids with and without SDS.

Using Cu(II) complexes of L-proline, L-hydroxyproline or aspartame, Desiderio et al. [235] chirally separated 2-hydroxy acids. An interesting approach has recently been presented by Krasensky et al. [236] for improving the detection sensitivity. A capillary containing Cu(II)acetate and L-proline or aspartame was coupled to a capillary without a chiral selector at the detector side by a bifurcated block. This circumvented interferences in detection by the chiral selector. In the chiral separation of hydroxy acids, this system achieved a detection limit of 10^{-18} *M*.

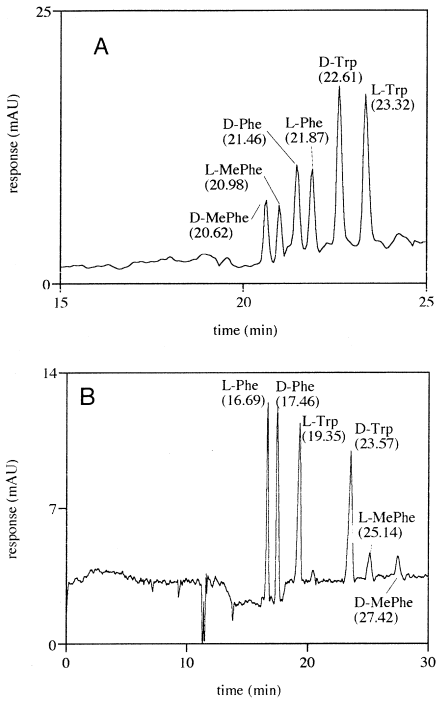

Fig. 7. Electropherograms of the enantiomer separation of DL-Phe, DL- Trp and DL-MePhe with and without SDS. Conditions: (A) 80 m*M* L-4-hydroxyproline, 40 m*M* Cu(II)sulfate, adjusted with ammonia to pH 4.0; (B) 50 m*M* L-4-hydroxyproline, 25 m*M* Cu(II)sulfate, 15 m*M* SDS, 3 *M* urea, adjusted with ammonia to pH 4.0. Capillary: untreated fused-silica, 75 cm×75 μm I.D. (effective length, 66 cm). U=27 kV, λ=208 nm, ambient temperature (modified from Ref. [234]).

Recently, the combination of ligand-exchange and host–guest interactions has been investigated as a new tool for chiral separations [237]. Horimai et al. [237] mixed γ-CD with a solution of Zn(II)-D-phenylalanine and used this dual selector system to successfully separate ofloxacin and analogues into their enantiomers. Instead of using chiral chelate complexes as selectors, LE–CE was also shown to be applicable to the resolution of racemic metal complexes. Fanali et al. [238] reported the resolution of racemic Co(III) complexes with ethylenediamine, *o*-phenanthroline, etc., using L-(+)-tartrate as the chiral selector.

The limitation of the very effective technique of LE–CE is the prerequisite for chelate complex-forming groups in the analyte.

8. Capillary electrochromatography (CEC)

This challenging approach, first regarded with some scepticism by several CE experts, has come to be of increasing interest in recent years. The efficiency obtained is rather higher than expected. Several applications to chiral separations have already been reported. There are different ways of performing chiral separations by CEC. One is to immobilize the chiral selector on the wall by coating or chemically binding it. The mobile phase is transported through the capillary by the EOF. This approach has been introduced by the group of Schurig [239–243]. Permethylated β- or γ-CD was attached via an octamethylene spacer to dimethylpolysiloxane (Chirasil-Dex) and coated onto the wall of the capillary. The high efficiency (250 000 theoretical plates/m) was demonstrated by means of 1,1′-dinaphthyl-2.2′-diyl hydrogenphosphate [240].

Hexobarbital [241,243], edolac [240] and several non-steroidal antiinflammatory drugs [240–242] were resolved on Chirasil-Dex using CEC. Schurig et al. [243] demonstrated the concept of unified enantioselective chromatography through the chiral separation of hexobarbital by GC, HPLC, SFC and CEC using the same capillary coated with Chirasil-Dex (Fig. 8). A dual chiral recognition system, consisting of Chirasil-Dex and a CD derivative added to the BGE, was investigated with hexobarbital as a model by the same group [241].

Armstrong et al. [244] synthesized a similar selector using a permethylated allyl-substituted β-CD, which was coupled to an organohydrosiloxane polymer. Capillaries coated with this selector were used in CEC, GC and SFC. In the CEC mode, racemic mephobarbital was resolved.

An alternative, used by Szeman and Ganzler [245], is to coat the capillary wall with an acrylamide polymer to which a CD derivative is bound. This approach was tested with the chiral separation of ephedrine, hexobarbital, epinephrine and a benzodioxane derivative.

CEC can also be performed with packed capillaries. Frits are prepared by sintering a zone of the packing material in front of the detection window. Capillaries packed with a β-CD–CSP, for HPLC, 5 μm, were used by Li and Lloyd [177] for the resolution of some amino acid derivatives, benzoin

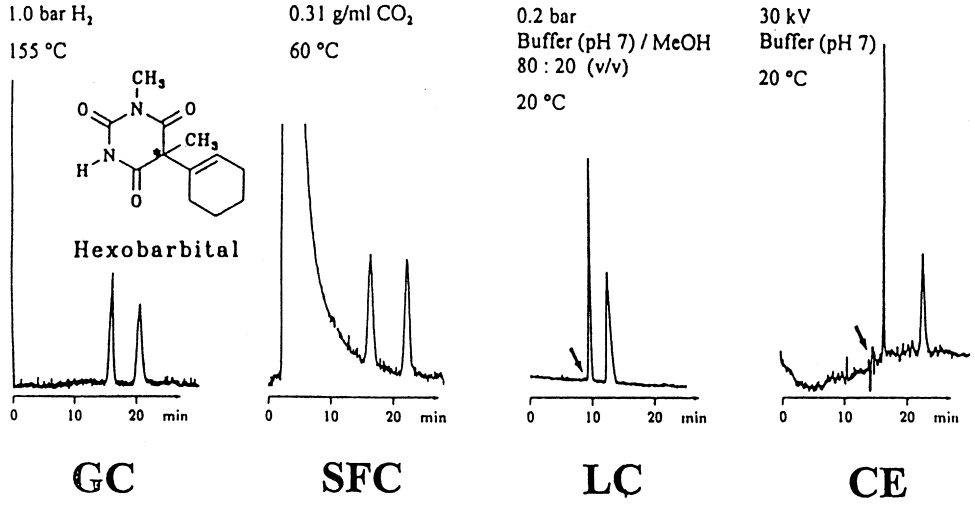

Fig. 8. Enantiomer separation of hexobarbital on a 1 m×50 μm I.D. fused-silica column coated with Chirasil-DEX (film thickness, 250 nm) by GC, SFC, LC and CEC in ca. 20 min. Effective column length in LC and CEC: 85 cm. Buffer: borate–phosphate (pH 7) (the arrow indicates the dead volume) (reprinted with permission from Ref. [243]).

and hexobarbital. For the resolution of anionic analytes, the EOF was reversed by the addition of TEAA. Recently, Lelièvre et al. [246] compared a HP-β-CD–CSP (5 μm) and HP-β-CD as an additive in the mobile phase using an achiral phase (3 μm ODS) to resolve chlortalidone by CEC. In the latter approach, the CD derivative is assumed to be adsorbed on the hydrophobic stationary phase. Resolution was found to be superior on the CSP, however, efficiency was lower. With increasing amounts of acetonitrile, the peak shape improved and the migration time decreased; this was, however, at the expense of resolution. Li and Lloyd [176] packed a capillary with a 5 μm AGP–CSP and resolved some β-blockers, barbiturates, ifosfamide and disopyramide on this phase. As observed in HPLC, the addition of 2-propanol improved the resolution. Lloyd et al. [165] compared the efficiency of CEC using immobilized HSA (7 μm) in packed capillaries, and free solution CE using HSA in the BGE with benzoin, temazepam and oxazepam as test analytes. The resolution of the benzodiazepines on the packed capillary was good, the efficiency, however, was rather poor and marked band-broadening was observed. An improvement is to be expected from the use of smaller particles (<3μm).

Encouraged by the success of HPLC–CSPs based on cellulose derivatives, Francotte and Jung [192] coated capillaries with 3,5-dimethylphenylcarbamoylcellulose and *p*-methylbenzoylcellulose and used these capillaries for both CEC and open tubular LC (OTLC). Several parameters influencing resolution, such as thickness of coating, concentration of organic modifiers and temperature, were investigated. The efficiency and resolution were compared for both OTLC and CEC using glutethimide as a model compound and were found to be higher in the CEC mode (Fig. 9).

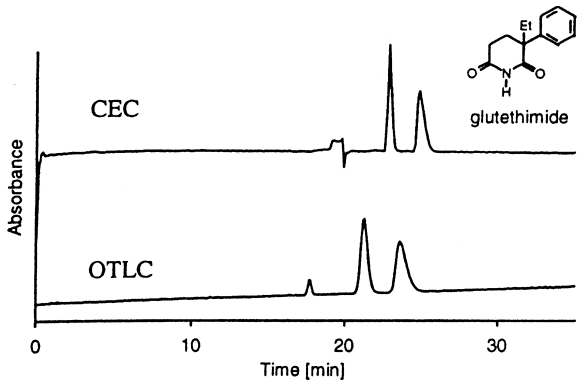

Fig. 9. Enantiomer separation of racemic glutethimide by CEC and OTLC in the same capillary coated with 0.025 mm PMBC. Column: 57 cm (50 cm injector to detector)×50 μm I.D. fused silica; voltage: 30 kV (CEC); inlet pressure: 35 mbar (OTLC); mobile phase: 40 m*M* phosphate buffer, pH 7, with 20% (v/v) acetonitrile (reprinted with permission from Ref. [192]).

Another innovative approach, the use of imprinted polymers as CSPs in CEC [152–154], is discussed in Section 3.5.

9. Chiral separation by CE in non-aqueous medium

A recent promising trend in CE is the use of non-aqueous BGE. Non-aqueous solvents are advantageous in some cases for solubility reasons, to reduce interactions with the capillary wall and to avoid interferences in the interactions of the analyte with the chiral selector by water. Valkó et al. [247] employed β-CD in *N*-methylformamide (NMF) or formamide (FA) for the resolution of DNS-AAs. With NMF, a fast EOF, high electrophoretic mobilities and good efficiency were observed; however, chiral resolution was found to be better with FA.

Wang and Khaledi [248] investigated the resolution of basic drugs by non-aqueous CE comparing β-CD, γ-CD, M-β-CD and HP-β-CD. The results obtained in three organic solvents, FA, NMF and dimethylformamide (DMF) were compared to those achieved in water and in 6 *M* urea in water. Binding constants were determined for trimipramine, mianserine and thioridazine and were found to be significantly lower in the organic solvents. For that reason, higher selector concentrations were required in non-aqueous medium. Compared to aqueous systems, a significant improvement in the resolution was obtained, especially for tricyclic systems. The best separations were obtained with β-CD and no or only partial resolution was observed with γ-CD. Since tricyclic compounds are too large for β-CD and would fit better into γ-CD, the authors concluded that the primary mechanism in non-aqueous solvents is not inclusion complexation but rather polar interactions to the hydroxyl at the mouth of the CD.

Mori et al. [148] demonstrated the applicability of (+)-18-crown-6-tetracarboxylic acid in formamide and ammonium perchlorate for the chiral resolution of compounds with primary amino groups (see also Section 3.3).

The use of chiral ion-pairing reagents (+)-*S*-camphor-10-sulfonic acid [249] and quinine [250] in non-aqueous medium is discussed in Section 10.

The use of non-aqueous solvents will certainly extend the potential of CE for chiral separations.

10. Use of chiral ion-pairing reagents

The use of ion-pairing reagents as synergistic components with CDs is discussed in Section 3.1.4.

No successful application of chiral ion-pairing reagents in CE using aqueous BGEs has been reported to date.

Recently, (+)-*S*-camphor-10-sulfonic acid (CSA) was employed as an ion-pairing reagent for the chiral resolution of cationic drugs in non-aqueous medium in the presence of Tween 20, using acetonitrile as the solvent [249]. Among several basic drugs tested, only compounds with a β-amino alcohol structure were resolved. A two-point interaction is given by ionic attraction between the sulfonate group of the counter-ion and the basic group of the analyte and the formation of hydrogen bonds between the oxo group of the CSA and the hydroxy group in the amino alcohol. In aqueous medium, the formation of hydrogen bonds is largely suppressed. Tween 20 seems to act as a hydrophobic pseudo-stationary phase. Fig. 10 shows the influence of Tween 20 concentration on the resolution of atenolol. This approach was successfully applied to the enantiomer separation of several β-blockers and sympathomimetics with an amino alcohol structure. The enantioselectivity was more pronounced for compounds containing an isopropylamino- or *tert.*-butylamino group than for compounds containing methylamino groups.

Stalcup and Gahm [250] showed that quinine can act as a chiral ion-pairing reagent in non-aqueous solvents for acidic compounds. The power of this approach was demonstrated by the enantiomer separation of DNP-AAs, 1,1′-binaphthyl-2,2′-diyl hydrogenphosphate and *N*-[1-(1-naphthyl)ethyl]phthalamic acid using ammonacetate-methanol as the BGE. Similar to observations in HPLC, the addition of acetic acid improved the resolution. In addition to ionic interactions, $\pi-\pi$ interactions between the quinoline ring and the nitro groups in the AA derivatives are to be taken into account.

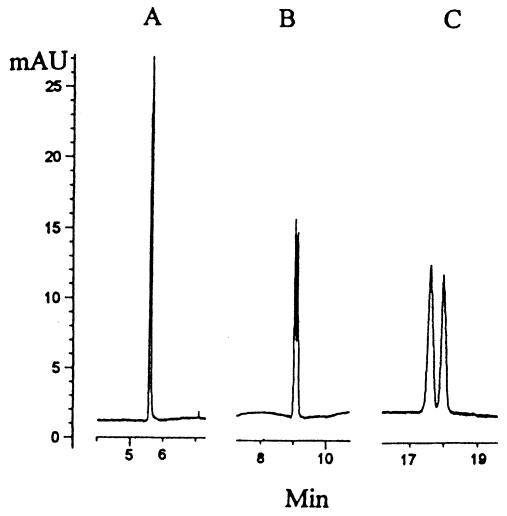

Fig. 10. Electropherogram of 0.2 mg/ml atenolol with increasing concentrations of Tween 20 added to the electrophoresis medium (1 *M* acetic acid in acetonitrile. (A) (+)-*S*-Camphorsulfonate (30 m*M*); (B) 30 m*M* (+)-*S*-camphorsulfonate, 0.1 m*M* Tween 20 and (C) 30 m*M* (+)-*S*-camphorsulfonate, 0.2 m*M* Tween 20) (reprinted with permission from Ref. [249]).

It is to be expected that this promising technique will find broad application in future.

11. Selection of the chiral separation principle

The most versatile group of selectors was found to be CDs, followed by macrocyclic antibiotics, proteins and polysaccharides (Fig. 11). These selectors showed enantioselectivity for a broad spectrum of compounds of very different structure, including a large variety of drugs (Tables 1–4).

Finding a suitable chiral selector is still, in many cases, based on trial and error, both in HPLC and CE. However, a few predictions can be made if typical structural elements are present. After choosing a selector, several parameters are to be varied, i.e., the nature, ionic strength and pH of the buffer,

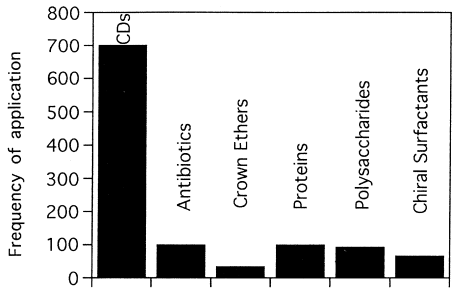

Fig. 11. Frequency of the application of the different chiral selectors.

the addition of organic modifiers, the addition of the resolution supporting reagents, the addition of micelle-forming compounds, manipulation of EOF (enhancing, suppressing, reversing), the temperature, etc. A guide for proceeding in chiral separations with CDs was given by Guttman et al. [67–69].

Table 5 gives some suggestions for choosing a chiral selector.

12. Conclusions

Capillary electrophoresis has proved to be a powerful technique for chiral separations. CE is advantageous over HPLC with respect to speed, flexibility regarding choice of the chiral selector and conditions and economy of operation. Besides separation principles transferred from HPLC to CE, some new approaches have been developed for chiral CE separation. An important advantage is the simple possibility of changing the enantiomer migration order by adding EOF modifiers, changing the pH, or changing from uncharged to charged chiral selectors. It should be emphasized, however, that CE should not be regarded as a substitute for HPLC. In many cases, CE and HPLC are complementary with respect to enantioselectivity. Several separation problems have been solved by CE and not by HPLC and vice versa.

It is to be expected that some promising recent approaches, such as CEC, CE in non-aqueous

Table 5
Selection of the separation principle

Primary amines	Crown ethers
Metal complexing compounds	LE
Diols containing aromatic substituents	Mixed borate–CD complexation
Bulky hydrophobic compounds, aromates containing polar groups in the side chain	CDs, macrocyclic antibiotics
Cationic compounds	Negatively charged selectors: anionic chiral surfactants, negatively charged CDs, negatively charged antibiotics, negatively charged polysaccharides, proteins, CD–MEKC, anionic ion-pairing reagents in non-aqueous medium
Anionic compounds	Positively charged selectors: cationic CDs, cationic antibiotics, cationic ion-pairing reagents in non-aqueous medium
Neutral compounds	MEKC with charged selectors: charged CDs, CD–MEKC, charged polysaccharides, charged antibiotics, charged chiral surfactants
Compounds having no groups for stereoselective interactions but functional groups that are suitable for derivatization	Indirect methods using chiral derivatization reagents

medium and the use of chiral ion-pairing reagents, will certainly extend the application spectrum of chiral CE.

13. Abbreviations

ACN	Acetonitrile
AEOC-AAs	2-(9-Anthryl)ethylcarbonyl amino acids
AGP	α_1-Acid glycoprotein
Allyl-TER	(+)-(5*R*,8*S*,10*R*)-1-Allyl-terguride
Ammediol	2-Amino-2-methyl-1,3-propanediol
ANDSA	7-Aminonaphthalene-1,3-disulfonic acid
AQC-AAs	6-Aminoquinolyl-*N*-hydroxysuccinimidylcarbamoyl amino acids
BGE	Background electrolyte
Big CHAP	*N*,*N*-Bis(3-D-gluconamidopropyl)cholamide
BN	1,1′-Binaphthol
BNC	1,1′-Binaphthyl-2,2′-dicarboxylic acid
BNDA	1,1′-Binaphthyl-2,2′-diamine
BNP	1,1′-Binaphthyl-2,2′-diyl hydrogenphosphate
BSA	Bovine serum albumin
BTA	Benzyltrimethylammonium chloride
18C6TCA	(+)-18-Crown-6-tetracarboxylic acid
CBHI	Cellobiohydrolase I
CBI-AAS	1-Cyano-benz[*f*]isoindole amino acids
CBZ-AAs	Carboxybenzyl amino acids
CD	Cyclodextrin
CDen	Mono-(6-β-aminoethylamino-6-deoxy)-β-cyclodextrin
CD–MEKC	Cyclodextrin-modified micellar electrokinetic chromatography
β-CD-NH_2	Mono(6-amino-6-deoxy)-β-cyclodextrin
CE	Capillary electrophoresis
CEC	Capillary electrochromatography
CE-β-CD	Carboxyethyl-β-cyclodextrin
CHAPS	[3-(3-Cholamidopropyl)dimethylammonio]-1-propanesulfonate
CHES	2-[*N*-Cyclohexylamino]ethanesulfonic acid
CGE	Capillary gel electrophoresis
CM-β-CD	Carboxymethyl-β-cyclodextrin
CPC	Cetyltrimethylammonium chloride
CSA	(+)-*S*-Camphor-10-sulfonic acid
CSP	Chiral stationary phase
CTAB	Cetyltrimethylammonium bromide
DDCAS	(*S*)-2-[(Dodecoxycarbonyl)amino]-3(*S*)-methyl-1-sulfoxypentane
DDCP	*N*-Dodecoxycarbonyl proline
DDCV	*N*-Dodecoxycarbonyl valine
DEAE-	Diethylaminoethyl-
Deoxy Big CHAP	*N*,*N*-bis(3-D-gluconamidopropyl)deoxycholamide
DM-β-CD	Heptakis(2,6-di-*O*-methyl)-β-cyclodextrin
DMF	Dimethylformamide
DNB-AAs	3,5-Dinitrobenzoyl amino acids
DNP-AAs	2,4-Dinitrophenyl amino acids
DNPyr-AAs	*N*-3,5-Dinitropyridyl amino acids
DNS-AAs	5-Dimethylaminonaphthylsulfonyl amino acids
DR	Derivatization reagent
DSPE	Dextrin sulfopropyl ether
DTAC	Hydroxypropylmethylcellulose
EKC	Electrokinetic chromatography
EOF	Electroosmotic flow
FA	Formamide
FLEC	(−)-[1-(9-Fluorenyl)-ethyl]chloroformate
FMOC	9-Fluorenylmethoxycarbonyl amino acids
GITC	2,3,4,6-Tetra-*O*-acetyl-D-glucopyranosyl isothiocyanate
HEC	Hydroxyethylcellulose
HE-β-CD	Hydroxyethyl-β-cyclodextrin
HPC	Hydroxypropylcellulose
HP-β-CD	Hydroxypropyl-β-cyclodextrin
HPMC	Hydroxypropylmethylcellulose
HSA	Human serum albumin
ITP	Isotachophoresis
L-MEN	L-Menthoxyacetic acid
M-β-CD	Permethylated-β-cyclodextrin
MeNH-β-CD	6^A-Methylamino-β-cyclodextrin
$(MeNH)_7$-β-CD	Heptamethylamino-β-cyclodextrin
MEKC	Micellar electrokinetic chromatography
MES	2-(*N*-Morpholino)ethanesulfonic acid
MHEC	Methylhydroxyethylcellulose
MOPS	3-(*N*-Morpholino)propanesulfonic acid
MTH-AAS	Methylthiohydantoin amino acids
NDA	Naphthalene-2,3-dicarboxaldehyde
NG	Nonyl-β-D-glucopyranoside
NMF	*N*-Methylformamide
OG	Octyl-β-D-glucopyranoside
OM	Octyl-β-D-maltopyranoside
OVM	Ovomucoid
PAA	Polyacrylamide
PEA	Phosphorylethanolamine
PMPC	*para*-Methylbenzoyl cellulose
poly-β-CD	β-Cyclodextrin polymer
poly(SUVal)	Poly(sodium *N*-undecylenyl-L-valinate)
PTH-AAs	Phenylthiohydantoin amino acids
PVA	Polyvinyl alcohol
SAMBI	(*S*)-1-Phenylethyl isothiocyanate

SC	Sodium cholate
SDAla	Sodium *N*-dodecanoyl-L-alaninate
SDC	Sodium deoxycholate
SDGlu	Sodium *N*-dodecanoyl-L-glutamate
SDS	Sodium dodecyl sulfate
SDThr	Sodium *N*-dodecanoyl-L-threoninate
SDVal	Sodium *N*-dodecanoyl-L-valinate
SNEIT	(*S*)-1-(1-Naphthyl)ethyl isothiocyanate
STC	Sodium taurocholate
STDC	Sodium taurodeoxycholate
Succ-β-CD	Succinyl-β-cyclodextrin
SUVal	Sodium *N*-undecylenyl-L-valinate
TAA	Tetraalkylammonium
TAPS	3-[*N*-Tris(hydroxymethyl)methylamino]-propanesulfonic acid
TAPSO	3-[*N*-Tris(hydroxymethyl)methyl-amino]-2-hydroxy-propanesulfonic acid
TATG	2,3,4,6-Tetra-*O*-acetyl-1-thio-β-D-glucopyranose
TBA	Tetrabutylammonium
TEA	Triethanolamine
TEAA	Triethylammonium acetate
TMA	Tetramethylammonium
TMA-β-CD	2-Hydroxy-3-trimethylammoniopropyl-β-cyclodextrin
TM-β-CD	Heptakis(2,3,6-tri-*O*-methyl)-β-CD
Tris	Tris(hydroxymethyl)aminomethane

References

[1] J. Snopek, I. Jelinek, E. Smolkova-Keulemansova, J. Chromatogr. 609 (1992) 1.
[2] R. Kuhn, S. Hofstetter-Kuhn, Chromatographia 34 (1992) 505.
[3] T.J. Ward, Anal. Chem. 66 (1994) 633A.
[4] M.M. Rogan, K.D. Altria, D.M. Goodall, Chirality 6 (1994) 25.
[5] S. Fanali, M. Cristalli, R. Vespalec and P. Bocek, in A. Chrambach, M.J. Dunn and B.J. Radola (Editors), Advances in Electrophoresis, VCH, Weinheim, 1994, p. 3.
[6] H. Nishi, S. Terabe, J. Chromatogr. 496 (1995) 245.
[7] S. Fanali, J. Chromatogr. A 735 (1996) 77.
[8] H. Nishi, J. Chromatogr. A 735 (1996) 57.
[9] A.D. Tran, T. Blanc, E.J. Leopold, J. Chromatogr. 516 (1990) 241.
[10] S. Cladrowa-Runge, R. Hirz, E. Kenndler, A. Rizzi, J. Chromatogr. A 710 (1995) 339.
[11] H. Nishi, T. Fukuyama, M. Matsuo, J. Microcol. Sep. 2 (1990) 234.
[12] I.S. Lurie, J. Chromatogr. 605 (1992) 269.
[13] L. Kang, R.H. Buck, Amino Acids 2 (1992) 103–109.
[14] C. Dette, H. Wätzig, A. Aigner, Pharmazie 49 (1994) 245.
[15] A. Tivesten, S. Folestad, J. Chromatogr. A 708 (1995) 323.
[16] A. Tivesten, E. Örnskov, S. Folestad, J. High Resolut. Chromatogr. 19 (1996) 229.
[17] P. Leroy, L. Bellucci, A. Nicolas, Chirality 7 (1995) 235.
[18] W. Schützner, S. Fanali, A. Rizzi, E. Kenndler, J. Chromatogr. 639 (1993) 375.
[19] W. Schützner, G. Caponecchi, S. Fanali, A. Rizzi, E. Kenndler, Electrophoresis 15 (1994) 769.
[20] W. Schützner, S. Fanali, A. Rizzi, E. Kenndler, J. Chromatogr. A 719 (1996) 411.
[21] H. Wan, P.E. Andersson, A. Engstrom, L.G. Blomberg, J. Chromatogr. A 704 (1995) 179.
[22] H. Wan, L.G. Blomberg, J. Chromatogr. A 758 (1997) 303.
[23] C. Vogt, A. Georgi, G. Werner, Chromatographia 40 (1995) 287.
[24] R. Bonfichi, C. Dallanoce, S. Lociuro, A. Spada, J. Chromatogr. A 707 (1995) 355.
[25] C.R. Noe, J. Freissmuth, J. Chromatogr. A 704 (1995) 503.
[26] J. Snopek, I. Jelinek, E. Smolkova-Keulemansova, J. Chromatogr. 438 (1988) 211.
[27] I. Jelinek, J. Snopek, E. Smolkova-Keulemansova, J. Chromatogr. 439 (1988) 386.
[28] J. Snopek, H. Soini, M. Novotny, E. Smolkova-Keulemansova, J. Chromatogr. 559 (1991) 215.
[29] A. Guttman, A. Paulus, A.S. Cohen, N. Grinberg, B.L. Karger, J. Chromatogr. 448 (1988) 41.
[30] S. Fanali, J. Chromatogr. 474 (1989) 441.
[31] S.A.C. Wren, R.C. Rowe, J. Chromatogr. 603 (1992) 235.
[32] S.A.C. Wren, R.C. Rowe, J. Chromatogr. 635 (1993) 113.
[33] S.A.C. Wren, J. Chromatogr. 636 (1993) 57.
[34] Y.Y. Rawjee, D.U. Staerk, G. Vigh, J. Chromatogr. 635 (1993) 291.
[35] Y.Y. Rawjee, G. Vigh, Anal. Chem. 66 (1994) 619.
[36] Y.Y. Rawjee, R.L. Williams, L.A. Buckingham, G. Vigh, J. Chromatogr. A 688 (1994) 273.
[37] S. Surapaneni, K. Ruterbories, T. Lindstrom, J. Chromatogr. A 761 (1997) 249.
[38] I.E. Valko, H.A.H. Billiet, J. Frank, K.C.A.M. Luyben, J. Chromatogr. A 678 (1994) 139.
[39] M. Yoshinaga, M. Tanaka, J. Chromatogr. A 679 (1994) 359.
[40] S. Fanali, Z. Aturki, J. Chromatogr. A 694 (1995) 297.
[41] G. Weseloh, H. Bartsch, W.A. König, J. Microcol. Sep. 7(4) (1995) 355.
[42] S. Fanali, J. Chromatogr. 545 (1991) 437.
[43] W. Lindner, B. Böhs, V. Seidel, J. Chromatogr. A 697 (1995) 549.
[44] I. Bechet, P. Paques, M. Fillet, P. Hubert, J. Crommen, Electrophoresis 15 (1994) 818.
[45] A. Guttman, N. Cooke, J. Chromatogr. A 680 (1994) 157.
[46] D. Belder, G. Schomburg, J. High Resolut. Chromatogr. 15 (1992) 686.
[47] C.Y. Quang, M.G. Khaledi, Anal. Chem. 65 (1993) 3354.
[48] C.Y. Quang, M.G. Khaledi, J. High Resolut. Chromatogr. 17 (1994) 99.
[49] C.Y. Quang, M.G. Khaledi, J. Chromatogr. A 692 (1995) 253.

[50] O. Stalberg, H. Brotell, D. Westerlund, Chromatographia 40 (1995) 697.
[51] S. Hong, C.S. Lee, Electrophoresis 16 (1995) 2132.
[52] B.A. Ingelse, F.M. Everaerts, J. Sevcik, Z. Stransky, S. Fanali, J. High Resolut. Chromatogr. 18 (1995) 348.
[53] H. Nishi, K. Nakamura, H. Nakai, S. Terabe, Chromatographia 40 (1995) 638.
[54] B.A. Ingelse, F.M. Everaets, C. Desiderio, S. Fanali, J. Chromatogr. A 709 (1995) 89.
[55] B.A. Ingelse, F.M. Everaerts, J. Sevcik, Z. Stransky, S. Fanali, J. High Resolut. Chromatogr. 18 (1995) 348.
[56] J. Sevcik, Z. Stransky, B.A. Ingelse, K. Lemr, J. Pharm. Biomed. Anal. 14 (1996) 1089.
[57] S. Fanali, Z. Aturki, Electrophoresis 16 (1995) 1505.
[58] E. Varesio, J.-L. Veuthey, J. Chromatogr. A 717 (1995) 219.
[59] Z. Wang, Y.L. Sun, Z.P. Sun, J. Chromatogr. A 735 (1996) 295.
[60] H.J. Gaus, Z.Z. Gogus, K. Schmeer, B. Behnke, K.A. Kovar, E. Bayer, J. Chromatogr. A 735 (1996) 221.
[61] B.C. Lin, Y.B. Ji, Y.Y. Chen, U. Epperlein, B. Koppenhoefer, Chromatographia 42 (1996) 106.
[62] B. Koppenhoefer, U. Epperlein, B. Christian, B. Lin, Y. Ji, Y. Chen, J. Chromatogr. A 735 (1996) 333.
[63] B. Koppenhoefer, U. Epperlein, B. Christian, J. Yibing, C. Yuying, L. Bingcheng, J. Chromatogr. A 717 (1995) 181.
[64] M. Yoshinaga, M. Tanaka, J. Chromatogr. A 710 (1995) 331.
[65] H. Nishi, J. High Resolut. Chromatogr. 18 (1995) 659.
[66] K.-H. Gahm, A.M. Stalcup, Anal. Chem. 67 (1995) 19.
[67] A. Guttman, Electrophoresis 16 (1995) 1900.
[68] A. Guttman, S. Brunet, C. Jurado, N. Cooke, Chirality 7 (1995) 409.
[69] A. Guttman, S. Brunet, N. Cooke, LC·GC Int. 9 (1996) 88.
[70] A. Aumatell, A. Guttman, J. Chromatogr. A 717 (1995) 229.
[71] S. Terabe, Trends Anal. Chem. 8 (1989) 129.
[72] S. Terabe, H. Otsuka, T. Ando, J. Chromatogr. 332 (1985) 211.
[73] A. Nardi, A. Eliseev, P. Bocek, S. Fanali, J. Chromatogr. 638 (1993) 247.
[74] S. Fanali, E. Camera, Chromatographia 43 (1996) 247.
[75] F. Lelièvre, P. Gareil, A. Jardy, Anal. Chem. 69 (1997) 385.
[76] F. Lelièvre, P. Gareil, Anal. Chem. 69 (1997) 393.
[77] B. Chankvetadze, G. Schulte, G. Blaschke, J. Chromatogr. A 732 (1996) 183.
[78] C. Roussel, A. Favrou, J. Chromatogr. A 704 (1995) 67.
[79] A. Bunke, T. Jira, Pharmazie 51 (1996) 672–673.
[80] Z. Aturki, S. Fanali, J. Chromatogr. A 680 (1994) 137.
[81] M. Fillet, I. Bechet, P. Chiap, P. Hubert, J. Crommen, J. Chromatogr. A 717 (1995) 203.
[82] C. Weinz, G. Blaschke, J. Chromatogr. B 674 (1995) 287.
[83] M.G. Schmid, K. Wirnsberger, G. Gübitz, Pharmazie 51 (1996) 852–854.
[84] C. Dette, S. Ebel, S. Terabe, Electrophoresis 15 (1994) 799.
[85] R.J. Tait, D.O. Thompson, V.J. Stella, J.F. Stobaugh, Anal. Chem. 66 (1994) 4013.
[86] A. Aumatell, R.J. Wells, D.K.Y. Wong, J. Chromatogr. A 686 (1994) 293.
[87] T. Schmitt, H. Engelhardt, Chromatographia 37 (1993) 475.
[88] C. Desiderio, S. Fanali, J. Chromatogr. A 716 (1995) 183.
[89] B. Chankvetadze, G. Endresz, D. Bergenthal, G. Blaschke, J. Chromatogr. A 717 (1995) 245–253.
[90] B. Chankvetadze, G. Endresz, G. Blaschke, Electrophoresis 15 (1994) 804.
[91] I.S. Lurie, R.F.X. Klein, T.A. Dalcason, M.J. LeBelle, R. Brenneisen, R.E. Weinberger, Anal. Chem. 66 (1994) 4019.
[92] M. Jung, E. Francotte, J. Chromatogr. A 755 (1996) 81.
[93] Y. Tanaka, M. Yanagawa, S. Terabe, J. High Resolut. Chromatogr. 19 (1996) 421.
[94] B. Chankvetadze, G. Endresz, G. Blaschke, J. Cap. Elec. 2 (1995) 235.
[95] W.H. Wu, A.M. Stalcup, J. Liq. Chromatogr. 18 (1995) 1289.
[96] K.-H. Gahm, A.M. Stalcup, Chirality 8 (1996) 316–324.
[97] A.M. Stalcup, K.-H. Gahm, Anal. Chem. 68 (1996) 1360–1368.
[98] K.-H. Gahm, L.W. Chang, D.W. Armstrong, J. Chromatogr. A 759 (1997) 149.
[99] B. Chankvetadze, G. Endresz, G. Blaschke, Chem. Soc. Rev. 25 (1996) 141.
[100] S. Terabe, Y. Miyashita, Y. Ishihama, O. Shibata, J. Chromatogr. 636 (1993) 47.
[101] T. Ueda, R. Mitchell, F. Kitamura, T. Metcalf, T. Kuwana, A. Nakamoto, J. Chromatogr. 593 (1992) 265.
[102] H. Wan, P.E. Andersson, A. Engstrom, L.G. Blomberg, J. Chromatogr. A 704 (1995) 179.
[103] H. Wan, A. Engstrom, L.G. Blomberg, J. Chromatogr. A 731 (1996) 283.
[104] I. Jelinek, J. Snopek, J. Dian, E. Smolkova-Keulemansova, J. Chromatogr. 470 (1989) 113.
[105] E. Francotte, S. Cherkaoul, M. Faupel, Chirality 5 (1993) 516.
[106] K. Otsuka, S. Terabe, J. Liq. Chromatogr. 16 (1993) 945.
[107] H. Siren, J.H. Jumppanen, K. Manninen, M.L. Riekkola, Electrophoresis 15 (1994) 779.
[108] H. Nishi, T. Fukuyama, S. Terabe, J. Chromatogr. 553 (1991) 503.
[109] A. Bunke, T. Jira, G. Gübitz, Pharmazie 50 (1995) 570.
[110] A. Bunke, T. Jira, Pharmazie 51 (1996) 479.
[111] S. Anselmi, D. Braghiroli, M. Di Bella, M.G. Schmid, R. Wintersteiger, G. Gübitz, J. Chromatogr. B 681 (1996) 83.
[112] H. Katayama, Y. Ishihama, N. Asakawa, J. Chromatogr. A 764 (1997) 151.
[113] G.N. Okafo, C. Bintz, S.E. Clarke and P. Camilleri, J. Chem. Soc., Chem. Commun., (1992) 1189.
[114] M. Lin, N. Wu, G.E. Barker, P. Sun, C.W. Huie, R.A. Hartwick, J. Liq. Chromatogr. 16 (1993) 3667.
[115] G.N. Okafo, P. Camillieri, J. Microcol. Sep. 5 (1993) 149.
[116] A. Aumatell, R.J. Wells, J. Chromatogr. A 688 (1994) 329.
[117] J.T. Smith, W. Nashabeh, Z.E. Rassi, Anal. Chem. 66 (1994) 1119.
[118] J. Wang, I.M. Warner, J. Chromatogr. A 711 (1995) 297.
[119] J.A. Wang, I.M. Warner, Anal. Chem. 66 (1994) 3773.
[120] M. Stefansson, M. Novotny, J. Am. Chem. Soc. 115 (1993) 11573.
[121] T. Jira, A. Bunke, M.G. Schmid, G. Gübitz, J. Chromatogr. A 761 (1997) 269.

[122] M.G. Schmid, K. Wirnsberger, T. Jira, A. Bunke, G. Gübitz, Chirality 9 (1997) 153.
[123] J.E. Noroski, D.J. Mayo, M. Moran, J. Pharm. Biomed. Anal. 13 (1995) 45.
[124] D.W. Armstrong, K.L. Rundlett, J.R. Chen, Chirality 6 (1994) 496.
[125] D.W. Armstrong, Y.B. Tang, S.S. Chen, Y.W. Zhou, C. Bagwill, J.R. Chen, Anal. Chem. 66 (1994) 1473.
[126] D.W. Armstrong, K. Rundlett, G.L. Reid, Anal. Chem. 66 (1994) 1690.
[127] M.P. Gasper, A. Berthod, U.B. Nair, D.W. Armstrong, Anal. Chem. 68 (1996) 2501.
[128] T.J. Ward, C. Dann III, A. Blaylock, J. Chromatogr. A 715 (1995) 337.
[129] K.L. Rundlett, D.W. Armstrong, Anal. Chem. 67 (1995) 2088.
[130] R. Vespalec, H.A.H. Billiet, J. Frank, P. Bocek, Electrophoresis 17 (1996) 1214.
[131] D.W. Armstrong, M.P. Gasper, K.L. Rundlett, J. Chromatogr. A 689 (1995) 285.
[132] K.L. Rundlett, M.P. Gasper, E.Y. Zhou, D.W. Armstrong, Chirality 8 (1996) 88.
[133] H. Wan, L.G. Blomberg, J. Microcol. Sep. 8(5) (1996) 339.
[134] S. Fanali, C. Desiderio, J. High Resolut. Chromatogr. 19 (1996) 322.
[135] T.J. Ward, C. Dann III, A.P. Brown, Chirality 8 (1996) 77.
[136] D.W. Armstrong, K.L. Rundlett, J. Liq. Chromatogr. 18 (1995) 3659.
[137] R. Vespalec, H.A.H. Billiet, J. Frank, K.C.A.M. Luyben, J. High Resolut. Chromatogr. 19 (1996) 137.
[138] H. Nishi, K. Nakamura, H. Nakai, T. Sato, Chromatographia 43 (1996) 426.
[139] R. Kuhn, F. Erni, T. Bereuter, J. Häusler, Anal. Chem. 64 (1992) 2815.
[140] R. Kuhn, C. Steinmetz, T. Bereuter, P. Haas, F. Erni, J. Chromatogr. A 666 (1994) 367.
[141] R. Kuhn, F. Stoecklin, F. Erni, Chromatographia 33 (1992) 32.
[142] E. Höhne, G.-J. Krauss, G. Gübitz, J. High Resolut. Chromatogr. 15 (1992) 698.
[143] R. Kuhn, D. Riester, B. Fleckenstein, K.-H. Wiesmüller, J. Chromatogr. A 716 (1995) 371.
[144] M.G. Schmid, G. Gübitz, J. Chromatogr. A 709 (1995) 81.
[145] Y. Walbroehl, J. Wagner, J. Chromatogr. A 680 (1994) 253.
[146] P. Castelnovo, C. Albanesi, J. Chromatogr. A 715 (1995) 143.
[147] H. Nishi, K. Nakamura, H. Nakai, T. Sato, J. Chromatogr. A 757 (1997) 225.
[148] Y. Mori, K. Ueno, T. Umeda, J. Chromatogr. A 757 (1997) 328.
[149] R. Kuhn, J. Wagner, Y. Walbroehl, T. Bereuter, Electrophoresis 15 (1994) 828.
[150] J.-M. Lin, T. Nakagama, T. Hobo, Chromatographia 42 (1996) 559.
[151] M.S. Pena, Y. Zhang, S. Thibodeaux, M.L. McLaughlin, A.M. de la Pena, I.M. Warner, Tetrahedron Lett. 37(33) (1996) 5841.
[152] J.-M. Lin, T. Nakagama, K. Uchiyama, T. Hobo, Chromatographia 43 (1996) 585.
[153] J.-M. Lin, T. Nakagama, X.Z. Wu, K. Uchiyama, T. Hobo, Fresenius' J. Anal. Chem. 357 (1997) 130.
[154] L. Schweitz, L.I. Andersson, S. Nilsson, Anal. Chem. 69 (1997) 1179.
[155] S. Busch, J.C. Kraak, H. Poppe, J. Chromatogr. 635 (1993) 119.
[156] G.E. Barker, P. Russo, R.A. Hartwick, Anal. Chem. 64 (1992) 3024.
[157] P. Sun, N. Wu, G. Barker, R.A. Hartwick, J. Chromatogr. 648 (1993) 475.
[158] P. Sun, G. Barker, R.A. Hartwick, N. Grinberg, R. Kaliszan, J. Chromatogr. 652 (1993) 247.
[159] T. Arai, M. Ichinose, H. Kuroda, N. Nimura, T. Kinoshita, Anal. Biochem. 217 (1994) 7.
[160] Y. Tanaka, S. Terabe, J. Chromatogr. A 694 (1995) 277.
[161] S. Birnbaum, S. Nilsson, Anal. Chem. 64 (1992) 2872.
[162] H. Ljungberg, S. Nilsson, J. Liq. Chromatogr. 18 (1995) 3685.
[163] R. Vespalec, V. Sustacek, P. Bocek, J. Chromatogr. 638 (1993) 255.
[164] D.K. Lloyd, S. Li, P. Ryan, Chirality 6 (1994) 230.
[165] D.K. Lloyd, S. Li, P. Ryan, J. Chromatogr. A 694 (1995) 285.
[166] Y. Tanaka, N. Matsubara, S. Terabe, Electrophoresis 15 (1994) 848.
[167] Y. Ishihama, Y. Oda, N. Asakawa, Y. Yoshida, T. Sato, J. Chromatogr. A 666 (1994) 193.
[168] L. Valtcheva, J. Mohammed, G. Pettersson, S. Hjertén, J. Chromatogr. 638 (1993) 263.
[169] D. Wistuba, H. Diebold, V. Schurig, J. Microcol. Sep. 7 (1995) 17.
[170] T. Arai, N. Nimura, T. Kinoshita, Biomed. Chromatogr. 9 (1995) 68.
[171] Y. Tanaka, S. Terabe, Chromatographia 44 (1997) 119.
[172] F. Kilár, S. Fanali, Electrophoresis 16 (1995) 1510.
[173] F. Kilár, Electrophoresis 17(12) (1996) 1950.
[174] M.G. Schmid, G. Gübitz and F. Kilár, Electrophoresis, in press.
[175] D. Eberle, R.P. Hummel, R. Kuhn, J. Chromatogr. A 759 (1997) 185.
[176] S. Li, D.K. Lloyd, Anal. Chem. 65 (1993) 3684.
[177] S. Li, D.K. Lloyd, J. Chromatogr. A 666 (1994) 321.
[178] Y.J. Yamashita, Polym. Sci. Part A 3 (1965) 3251.
[179] A. D'Hulst, N. Verbeke, J. Chromatogr. 608 (1992) 275.
[180] H. Soini, M. Stefansson, M.L. Riekkola, M. Novotny, Anal. Chem. 66 (1994) 3477.
[181] C. Quang, M.G. Khaledi, J. High Resolut. Chromatogr. 17 (1994) 609.
[182] A. D'Hulst, N. Verbeke, Chirality 6 (1994) 225.
[183] A. D'Hulst, N. Verbeke, J. Chromatogr. A 735 (1996) 283.
[184] K. Kano, K. Minami, K. Horiguchi, T. Ishimura, M. Kodera, J. Chromatogr. A 694 (1995) 307.
[185] H. Nishi, S. Izumoto, K. Nakamura, H. Nakai, T. Sato, Chromatographia 42 (1996) 617.
[186] H. Nishi, K. Nakamura, H. Nakai, T. Sato, S. Terabe, Electrophoresis 15 (1994) 1335.

[187] A.M. Stalcup, N.M. Agyei, Anal. Chem. 66 (1994) 3054.
[188] N.M. Agyei, K.H. Gahm, A.M. Stalcup, Anal. Chim. Acta 307 (1995) 185.
[189] H. Nishi, K. Nakamura, H. Nakai, T. Sato, Anal. Chem. 67 (1995) 2334.
[190] M. Jung, K.O. Börnsen, E. Francotte, Electrophoresis 17 (1996) 130.
[191] G.M. Beck, S.H. Neau, Chirality 8 (1996) 503.
[192] E. Francotte, M. Jung, Chromatographia 42 (1996) 521.
[193] B.A. Ingelse, J.C. Reijenga, H.A. Claessens, F. Everaerts, J. High Resolut. Chromatogr. 19 (1996) 225.
[194] S. Terabe, K. Otsuka, K. Ichikawa, A. Tsuchiya, T. Ando, Anal. Chem. 56 (1984) 111.
[195] K. Otsuka, S. Terabe, J. Chromatogr. 515 (1990) 221.
[196] A. Dobashi, T. Ono, S. Hara, J. Yamaguchi, Anal. Chem. 61 (1989) 1984.
[197] A. Dobashi, T. Ono, S. Hara, J. Yamaguchi, J. Chromatogr. 480 (1989) 413.
[198] K. Otsuka, J. Kawahara, K. Tatekawa, S. Terabe, J. Chromatogr. 559 (1991) 209.
[199] K. Otsuka, S. Terabe, Electrophoresis 11 (1990) 982.
[200] K. Otsuka, M. Kashihara, Y. Kawaguchi, R. Koike, T. Hisamitu, S. Terabe, J. Chromatogr. A 652 (1993) 253.
[201] K. Otsuka, K. Karuhara, M. Higashimori, S. Terabe, J. Chromatogr. A 680 (1994) 317.
[202] A. Dobashi, M. Masaki, Y. Dobashi, Anal. Chem. 67 (1995) 3011.
[203] K.-A. Kimberly, M.S. Pena, S.A. Shamsi, I.M. Warner, Anal. Chem. 69 (1997) 958.
[204] J.R. Mazzeo, E.R. Grover, M.E. Swartz, J.S. Petersen, J. Chromatogr. A 680 (1994) 125.
[205] S. Honda, M. Kotani, A. Miyoshi, A. Taga, E.R. Grover, Chromatography 16 (1995) 88.
[206] M.E. Swartz, J.R. Mazzeo, E.R. Grover, P.R. Brown, Anal. Biochem. 231 (1995) 65.
[207] J.R. Mazzeo, M.E. Swartz, E.R. Grover, Anal. Chem. 67 (1995) 2966.
[208] M.E. Swartz, J.R. Mazzeo, E.R. Grover, P.R. Brown, J. Chromatogr. A 735 (1996) 303.
[209] M.E. Swartz, J.R. Mazzeo, E.R. Grover, P.R. Brown, H.Y. Aboulenein, J. Chromatogr. A 724 (1996) 307.
[210] A.G. Peterson, E.S. Ahuja, J.P. Foley, J. Chromatogr. B 683 (1996) 15.
[211] J.R. Mazzeo, M.E. Swartz, E.R. Grover, Anal. Chem. 67 (1995) 2966–2973.
[212] P.L. Desbène, C.E. Fulchic, J. Chromatogr. A 749 (1996) 257.
[213] Y. Mechref, Z. El Rassi, J. Chromatogr. A 757 (1997) 263.
[214] Y. Mechref, Z. El Rassi, Chirality 8 (1996) 518.
[215] D.C. Tickle, G.N. Okafo, P. Camilleri, R.F.D. Jones, A.J. Kirby, Anal. Chem. 66 (1994) 4121.
[216] Y. Mechref, Z. El Rassi, J. Chromatogr. A 724 (1996) 285.
[217] N. Nimura, H. Itoh, C. Mitsuno and T. Kinoshita, in Proceedings of Separation Sciences '94, Tokyo, 1994, p. 283.
[218] S. Terabe, H. Shibata, Y. Miyashita, J. Chromatogr. 480 (1989) 403.
[219] H. Nishi, T. Fukuyama, M. Matsuo, S. Terabe, Anal. Chim. Acta 236 (1990) 281.
[220] H. Nishi, T. Fukuyama, M. Matsuo, S. Terabe, J. Chromatogr. 515 (1990) 233.
[221] H. Nishi, T. Fukuyama, M. Matsuo, S. Terabe, J. Microcol. Sep. 1 (1989) 234.
[222] A. Amini, I. Beijersten, C. Pettersson, D. Westerlund, J. Chromatogr. A 737 (1996) 301.
[223] J.G. Clothier, S.A. Tomellini, J. Chromatogr. A 723 (1996) 179.
[224] J.G. Clothier, L.M. Daley, S.A. Tomellini, J. Chromatogr. B 683 (1996) 37.
[225] H. Nishi, J. High Resolut. Chromatogr. 18 (1995) 659.
[226] Y. Ishihama, S. Terabe, J. Liq. Chromatogr. 16 (1993) 933–944.
[227] D.D. Dalton, D.R. Taylor, J. Microcol. Sep. 7 (1995) 513.
[228] D.D. Dalton, D.R. Taylor, D.G. Waters, J. Chromatogr. A 712 (1995) 365.
[229] J.H. Aiken, C.W. Huie, Chromatographia 35 (1993) 448.
[230] E. Gassmann, J.E. Kuo, R.N. Zare, Science 230 (1985) 813.
[231] P. Gozel, E. Gassman, H. Michelsen, R.N. Zare, Anal. Chem. 59 (1987) 44–49.
[232] A.S. Cohen, A. Paulus, B.L. Karger, Chromatographia 24 (1987) 15–24.
[233] N.G. Sundin, T.M. Dowling, N. Grindberg, G. Bicker, J. Microcol. Sep. 8 (1996) 323.
[234] M.G. Schmid, G. Gübitz, Enantiomer 1 (1996) 23.
[235] C. Desiderio, Z. Aturki, S. Fanali, Electrophoresis 15 (1994) 864.
[236] S. Krasensky, S. Fanali, L. Krivankova, P. Bocek, Electrophoresis 16 (1995) 968.
[237] T. Horimai, M. Ohara, M. Ichinose, J. Chromatogr. A 760 (1997) 235.
[238] S. Fanali, L. Ossicini, F. Forest, P. Bocek, J. Microcol. Sep. 1 (1989) 190.
[239] S. Mayer, V. Schurig, J. High Resolut. Chromatogr. 15 (1992) 129.
[240] S. Mayer, V. Schurig, J. Liq. Chromatogr. 16 (1993) 915.
[241] S. Mayer, M. Schleimer, V. Schurig, J. Microcol. Sep. 6 (1994) 43.
[242] S. Mayer, V. Schurig, Electrophoresis 15 (1994) 835.
[243] V. Schurig, M. Jung, S. Mayer, M. Fluck, S. Negura, H. Jakubetz, J. Chromatogr. A 694 (1995) 119.
[244] D.W. Armstrong, Y.B. Tang, T. Ward, M. Nichols, Anal. Chem. 65 (1993) 1114.
[245] J. Szeman, K. Ganzler, J. Chromatogr. A 668 (1994) 509.
[246] F. Lelièvre, C. Yan, R.N. Zare, P. Gareil, J. Chromatogr. A 723 (1996) 145.
[247] I.E. Valkó, H. Sirén, M.-L. Riekkola, Chromatographia 43(5) (1996) 242.
[248] F. Wang, M.G. Khaledi, Anal. Chem. 68 (1996) 3460.
[249] I. Bjornsdottir, S.H. Hansen, S. Terabe, J. Chromatogr. A 745 (1996) 37–44.
[250] A.M. Stalcup, K.H. Gahm, J. Microcol. Sep. 8 (1996) 145.
[251] T.E. Peterson, J. Chromatogr. 630 (1993) 353.
[252] M. Heuermann, G. Blaschke, J. Chromatogr. 648 (1993) 267.

[253] V.C. Anigbogu, C.L. Copper, M.J. Sepaniak, J. Chromatogr. A 705 (1995) 343.
[254] T. Schmitt, H. Engelhardt, J. High Resolut. Chromatogr. 16 (1993) 525.
[255] Z. Wang, A. Huang, Y. Sun, J. High Resolut. Chromatogr. 19 (1996) 697.
[256] Y.Y. Rawjee, R.L. Williams, L.A. Buckingham, G. Vigh, J. Chromatogr. A 688 (1994) 273.
[257] S. Palmarsdottir, L.E. Edholm, J. Chromatogr. A 666 (1994) 337.
[258] R. Porrá, M.G. Quaglia, S. Fanali, Chromatographia 41 (1995) 383.
[259] B. Chankvetadze, G. Endresz, G. Blaschke, J. Chromatogr. A 700 (1995) 43.
[260] H. Soini, M.L. Riekkola, M. Novotny, J. Chromatogr. A 608 (1992) 265.
[261] C. Desiderio, C. Palcaro, S. Fanali, Electrophoresis 18 (1997) 227.
[262] C.E. Sänger-van de Griend, K. Gröningsson, D. Westerlund, Chromatographia 42 (1996) 263.
[263] G.M. McLaughlin, J.A. Nolan, J.L. Lindahl, R.H. Palmieri, K.W. Anderson, S.C. Morris, J.A. Morrison, T.J. Bronzert, J. Liq. Chromatogr. 15 (1992) 961.
[264] S. Mayer, V. Schurig, Electrophoresis 15 (1994) 835.
[265] E. Szökö, K. Magyar, J. Chromatogr. A 709 (1995) 157.
[266] F. Lelièvre, P. Gareil, J. Chromatogr. A 735 (1996) 311.
[267] Z. Wang, A. Huang, Y. Sun, J. High Resolut. Chromatogr. 19 (1996) 478.
[268] K.D. Altria, R.C. Harden, M. Hart, J. Hevizi, P.A. Hailey, J.V. Makwana, M.J. Portsmouth, J. Chromatogr. 641 (1993) 147.
[269] M.M. Rogan, K.D. Altria, D.M. Goodall, Chromatographia 38 (1994) 723.
[270] H. Nishi, K. Nakamura, H. Nakai, T. Sato, J. Chromatogr. A 678 (1994) 333.
[271] T. Schmitt, H. Engelhardt, J. Chromatogr. A 697 (1995) 561.
[272] R. Furuta, T. Doi, J. Chromatogr. A 676 (1994) 431.
[273] R. Furuta, T. Doi, Electrophoresis 15 (1994) 1322.
[274] M.W.F. Nielen, Anal. Chem. 65 (1993) 885.
[275] M.W. Matchett, S.K. Branch, T.M. Jefferies, J. Chromatogr. A 705 (1995) 351.
[276] S. Fanali, P. Bocek, Electrophoresis 11 (1990) 757.
[277] W. Schützner, S. Fanali, Electrophoresis 13 (1992) 687.
[278] T.E. Peterson, D. Trowbridge, J. Chromatogr. 603 (1992) 298.
[279] H. Nishi, Y. Kokusenya, T. Miyamoto, T. Sato, J. Chromatogr. A 659 (1994) 449.
[280] Y.Y. Rawjee, R.L. Williams, G. Vigh, Anal. Chem. 66 (1994) 3777.
[281] S. Piperaki, S.G. Penn, D.M. Goodall, J. Chromatogr. A 700 (1995) 59.
[282] Y.Y. Rawjee, R.L. Williams, G. Vigh, J. Chromatogr. A 652 (1993) 233.
[283] Y.Y. Rawjee, R.L. Williams, G. Vigh, J. Chromatogr. A 680 (1994) 599.
[284] S. Fanali, M. Flieger, N. Steinerova, A. Nardi, Electrophoresis 13 (1992) 39.
[285] A. Aumatell, A. Guttman, J. Chromatogr. A 717 (1995) 229.
[286] S. Fanali, E. Camera, J. Chromatogr. A 745 (1996) 17.
[287] C. Desiderio, S. Fanali, A. Kupfer, W. Thormann, Electrophoresis 15 (1994) 87.
[288] H. Nishi, K. Nakamura, H. Nakai, T. Sato, J. Chromatogr. A 678 (1994) 333.
[289] A. Guttman, N. Cooke, J. Chromatogr. A 685 (1994) 155.
[290] J.M. Dethy, S. Debroux, M. Lesne, J. Longstreth, P. Gilbert, J. Chromatogr. B 654 (1994) 121.
[291] A.M. Abushoffa, B.J. Clark, J. Chromatogr. A 700 (1995) 51.
[292] M. Tanaka, S. Asano, M. Yoshinago, Y. Kawaguchi, T. Tetsumi, T. Shono, Fresenius' J. Anal. Chem. 339 (1991) 63.
[293] K.D. Altria, Chromatographia 35 (1993) 177.
[294] K.D. Altria, J. Chromatogr. 646 (1993) 245.
[295] Z. Wang, A. Huang, Y. Sun, Z. Sun, J. Chromatogr. A 749 (1996) 300.
[296] I. Jelinek, J. Dohnal, J. Snopek, E. Smolkova-Keulemansova, J. Chromatogr. 464 (1989) 139.
[297] S.A.C. Wren, R.C. Rowe, J. Chromatogr. 609 (1992) 363.
[298] M.H. Lamoree, A.F.H. Sprang, U.R. Tjaden, J. van der Greef, J. Chromatogr. A 742 (1996) 235.
[299] M.M. Rogan, K.D. Altria, D.M. Goodall, Electrophoresis 15 (1994) 808.
[300] G.N. Okafo, K.K. Rana, P. Camillieri, Chromatographia 39 (1994) 627.
[301] P. Gareil, J.P. Gramond, F. Guyon, J. Chromatogr. 615 (1993) 317.
[302] Y. Walbroehl, J. Wagner, J. Chromatogr. A 685 (1994) 321.
[303] A.W. Garrison, P. Schmitt, A. Kettrup, J. Chromatogr. A 688 (1994) 317.
[304] Y. Mechref, Z. El Rassi, Anal. Chem. 68 (1996) 1771.
[305] M.L. Marina, I. Benito, J.C. Diez-Maser, M.J. Gonzalez, Chromatographia 42 (1996) 269.
[306] S. Fanali, Z. Aturki, Electrophoresis 16 (1995) 1505.
[307] K.D. Altria, D.M. Goodall, M.M. Rogan, Chromatographia 34 (1992) 19.