BRITISH JOURNAL OF PHARMACOLOGY

The *British Journal of Pharmacology* welcomes contributions in all fields of experimental pharmacology including neuroscience, biochemical, cellular and molecular pharmacology. The Board of Editors represents a wide range of expertise and ensures that well-presented work is published as promptly as possible, consistent with maintaining the overall quality of the journal.

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With effect from 1 January 1997

The *British Journal of Pharmacology* welcomes contributions in all fields of experimental pharmacology, including neuroscience, biochemical, cellular and molecular pharmacology, for publication as full papers or as high priority Special Reports.

Papers should normally be based on new results obtained experimentally and should constitute a significant contribution to pharmacological knowledge. Papers which reassess pharmacological concepts based on earlier results will also be considered as will purely theoretical papers. Papers dealing only with descriptions of methods are acceptable if new principles are involved.

Contributions that have already been published, or accepted or are under consideration for publication, with essentially the same content will not be considered. This restriction does not apply to results published as abstracts of communications, letters to editors, or as contributions to symposia, provided that the submission adds significantly to the information available in the previously published contribution.

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The Journal will not consider papers which describe experiments on animals which do not fall clearly within the current laws governing animal experimentation in the United Kingdom. Authors must make it clear that the procedures they use were as humane as possible and the doses (initial and subsequent) of anaesthetics and analgesics should be clearly stated; the method of assessing anaesthesia, particularly after the administration of skeletal muscle relaxants (neuro- muscular blocking drugs), must be well defined. The Society has an Ethics Committee which can be consulted by authors through the Secretaries to the Editorial Board.

When investigations on normal human subjects are reported, evidence of approval by a local Ethics Committee must be given. Papers concerned with clinical trials on investigations of the effects of drugs on patients are not appropriate for this Journal.

Authors are strongly urged to keep their manuscripts as short as they reasonably can. An effective way is to reduce the Discussion and the number of figures to a minimum and to avoid repetition of information that has already been published. Authors should remember that a reader may be influenced by literary style and will appreciate simple but accurate prose.

It is important to note that failure to comply with 'Instructions to Authors' may lead to considerable editorial delays.

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Manuscripts must be typed on one side of A4 paper. Words at the end of lines should not be divided because they may become incorrectly hyphenated. Handwritten characters or symbols (e.g. Greek letters) should be spelled out in full in the margin. Papers in recent issues of the *British Journal of Pharmacology* should be consulted for the general layout of the paper and also for details. The following subsections are used:

1. Title page
2. Summary
3. Introduction
4. Methods
5. Results
6. Discussion and conclusions
7. Acknowledgements
8. List of references
9. Tables
10. Figures and captions

The type must not be smaller than 12 pitch or 10 point. Each section must be typed in **double spacing** with margins of not less than 2.5 cm all round and each page should be numbered. **The original and one copy of the typescript should be supplied.**

Title page

The **title** should normally contain **no more than 150 characters** and should not consist of a sentence (statement or conclusion) or be interrogative. A **short running title** containing not more than 50 characters and spaces **is also required.** The title page should include the names of authors and their appropriate addresses. It should be made clear which address relates to which author. Authors' present addresses differing from those at which the work was carried out should be given as footnotes on the title page and references at the appropriate place in the author list by superscript numbers. A footnote may also be used to indicate the author to whom correspondence should be sent. The use of footnotes for any other reason is not allowed. If the address to which proofs should be sent is not that of the first mentioned author, clear instructions should be given in a covering note and not on the title page. The title page should be paginated as page 1 of the paper.

Summary

The summary will be printed at the beginning of the paper. It should not exceed 5% of the length of the paper and should contain a brief account of the problem, the methods, results and the conclusions. It should be arranged in **numbered and concise paragraphs.** Up to ten **keywords** or phrases of two to three words (including names and terms used in the title) should be displayed at the end of the summary. Keywords will be used to compile the annual index. The quality of the index will thus be determined by the appropriateness of the keywords. These may be selected by reference to the most recent Index of the Journal. Avoid unhelpful or unqualified terms such as 'rat', 'drug' etc.

Introduction

The introduction should give a short and clear account of the background of the problem and the rationale of the investigation. Only previous work that has a direct bearing on the present problem should be cited.

Methods

The methods must be described in sufficient detail to allow the experiments to be interpreted and repeated by the reader. However, detailed repetition of methods which have been adequately described previously should be avoided and references given, although a brief outline is often helpful.

Drugs should be listed in a separate paragraph. Their names should be 'approved names' as published previously in British Approval Names, 1990 (HMSO). If a drug has no 'approved name' its chemical name must be used and the

rules set out in the current *Handbook for Chemical Society Authors* (London, Chemical Society) observed, or its structural formula given. Cumbersome chemical names should be suitably abbreviated for later reference in the paper.

The doses of drugs should be given as unit weight per body weight, e.g. mmol kg^{-1} or mg kg^{-1}; concentrations should be given in terms of molarity, e.g. nM or μM.

Reference should be made to any statistical analyses that have been performed on the results in order, for example, to determine the significance of differences between results obtained under different conditions.

Results

The description of the experimental results should be succinct but, nevertheless, in sufficient detail to allow the experiments to be repeated by others. Typical single experiments may be presented with a clear statement that *n* number of similar experiments had similar results. Where appropriate, however, the mean results with confidence limits or with standard errors of the means and the number of observations should be given. Statistical tests of significance should be performed where appropriate. The results of such tests should be stated as the numerical value of the probability (*P*) that is calculated, with any necessary clarification (e.g. one-tail or two-tail test).

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The purpose of the discussion is to present a brief and pertinent interpretation of the results against the background of existing knowledge. Any assumptions on which conclusions are based must be stated clearly. A mere recapitulation of the results is not acceptable. A review-like treatment, which reduces the impact on the reader, should also be avoided. The main conclusion should be conveyed in a final paragraph.

Acknowledgements

Acknowledgements should be brief but should include reference to sources of support. Sources of drugs not widely available commerically should be acknowledged.

References

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BOLTON, T.B. & KITAMURA, K. (1983). Evidence that ionic channels associated with the muscarinic receptor of smooth muscle may admit calcium. *Br. J. Pharmacol.*, **78**, 405–416.

BRADING, A.F. (1981). Ionic distribution and mechanisms of transmembrane ion movements in smooth muscle. In *Smooth Muscle: An Assessment of Current Knowledge*. ed. Bülbring, E., Brading, A.F., Jones, A.W. & Tomita, T. pp. 65–92. London: Edward Arnold.

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Each table should be given on a separate page, paginated as part of the paper. Tables should be numbered consecutively with arabic numerals and the number should be followed by a brief descriptive caption, occupying not more than two lines, at the head of the table. The proportions of the text area should be borne in mind when designing the layout of tables. For the sake of clarity, tables should not have more than 120 characters to a line, with spaces between columns counted as four characters. The absolute maximum is 180 characters to a line. Each column should have a heading and the units of measurement should be given in parentheses in the heading. Except in special circumstances, tables should be self-explanatory; the necessary descriptions should be at the bottom of the table.

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To avoid unnecessary Figures, particularly those requiring half-tone reproduction, only critical points of the text should be illustrated. If coloured Figures are desired, the Authors should discuss their requirements with the Secretaries, preferably before submission.

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Line width (axes)	Line width (graphs)	Symbol size	Figure will reduce to this percentage of the original size
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   ○ ● □ ■ △ ▲ ▽ ▼ ◇ ◆ + ×.
5. Lettering/labelling should not be too small after reduction.
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Figure 1 illustrates a simple properly-drawn graph in its original form (a) and in its reduced form (b) as it would appear in the Journal (single column width).

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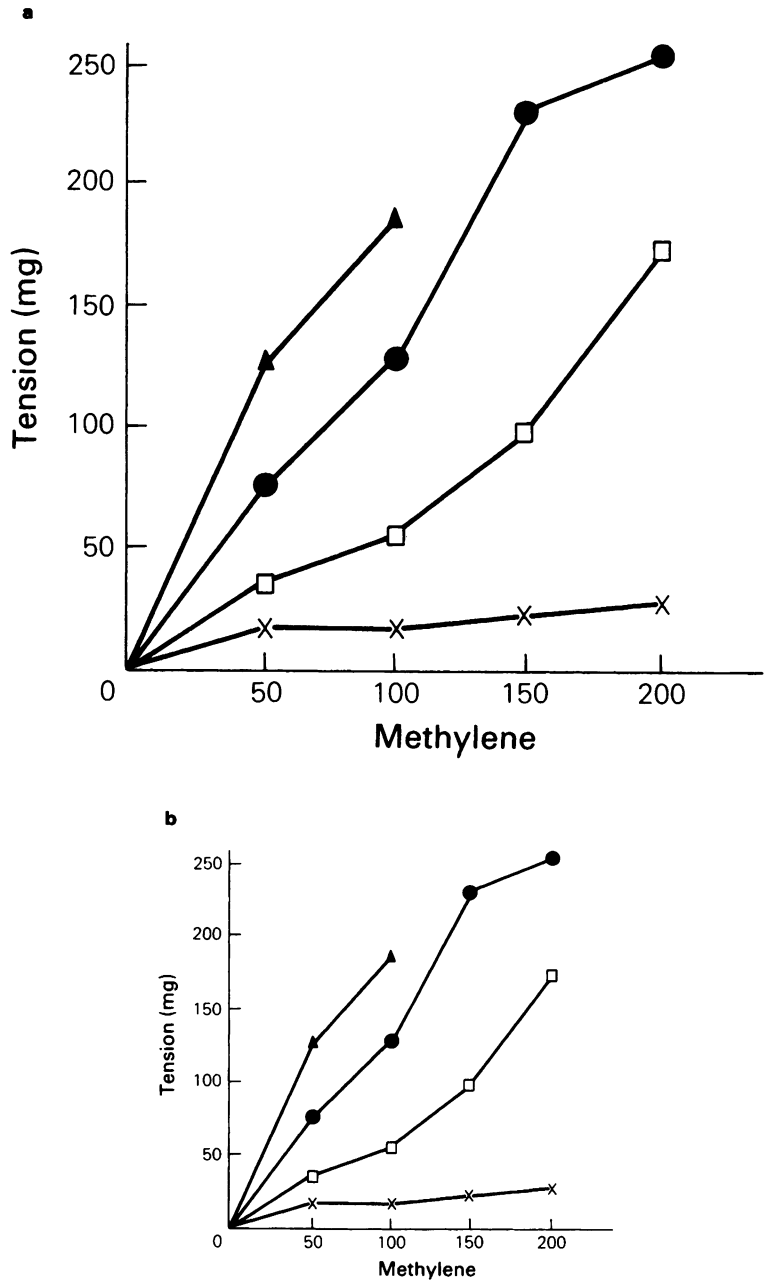

Figure 1 (a) Artwork as drawn. (b) Artwork reduced to 60 per cent of its original size for publication in the Journal to fit in single column width.

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ABBREVIATIONS AND SYMBOLS

Physico-chemical quantities

The *British Journal of Pharmacology* uses the SI symbols for units. The following prefixed for multiples of units should be used:

Multiplier	*Prefix*	*Symbol*
10^{-1}	deci	d
10^{-2}	centi	c
10^{-3}	milli	m
10^{-6}	micro	μ
10^{-9}	nano	n
10^{-12}	pico	p
10^{-15}	femto	f
10^{-18}	atto	a

Multiplier	*Prefix*	*Symbol*
10^{3}	kilo	k
10^{6}	mega	M
10^{9}	giga	G
10^{12}	tera	T

Thus, micron = μm; ångstrom = 0.1 nm. Mixed prefixes are not permissible, thus m μg should be ng. The symbols d (10^{-1}) and c (10^{-2}) should be restricted to those occasions on which there is a strongly felt need for them (e.g. cm).

Use of solidus

The solidus should be avoided as far as possible and the negative index substituted, e.g. mg kg^{-1} rather than mg/kg; pmol mm^{-2} min^{-1} rather than pmol/mm^2/min.

SYMBOLS

Symbols denoting physical quantities are usually printed as italic capitals (indicated by single underline in typescript). A dash over the symbols indicates a mean value; a dot over the symbol indicates a time derivative. Suffixes may be used to indicate 'where' and 'what'. They are printed as inferiors on that line. Multiple suffixes should be avoided if a simpler symbol adequately defined is unambiguous, but if necessary should be separated by commas e.g. $P_{A,\,CO_2}$ denotes partial pressure of CO_2 alveolar air.

CHEMICAL AND BIOLOGICAL ABBREVIATIONS

Authors should also consult *Nomenclature Guidelines for Authors* contained in this tissue of the Journal. The abbreviations listed may be used without definition *except* those for chemicals, drugs and enzymes which must be written in full at first mention in the title, summary and again in the text. At first mention they should be followed by the abbreviation in brackets. Subsequently, the abbreviation alone may be used.

The list of abbreviations for chemical, drug and enzyme names is clearly not comprehensive and includes only a few commonly used examples.

Use abbreviations sparingly as extensive use can make the text hard to follow.

Physico-chemical quantities

Quantity	*Preferred unit*	*Symbol*
Amount (of substance)	mole	mol
Capacitance	farad	F
Concentration	moles per litre	M or mol l^{-1}
Current	ampere	A
Electrical conductance	siemens	S
Electromotive force	volt	V
Flow (blood or other liquid)	litres per second (or min)	1 s^{-1} or 1 min^{-1}
Flow (air or other gas)	liters per second (or min)	1 s^{-1} or 1 min^{-1}
Force	newton	N
Frequency of regular event	hertz	Hz
Length	metre	m
Mass	gram	g
Power	watt	W
Pressure (or partial pressure)	pascal*	Pa
Radioactivity	becquerel or curie	Bq (60 d.p.m.) or Ci (3.7×10^{10} Bq)
Resistance (electrical)	ohm	Ω
Temperature	degree celsius	°C
Time	second (preferred)	s
	minute	min
	hour	h
Volume (blood or other liquid)	litre	l
Volume (air or other gas)	litre	l
Work	joule	J

*mm of mercury (mmHg) are allowed if conventional, and if mercury manometer is used for calibration.

Chemical and biological abbreviations

acetylcholine	ACh
acetylcholinesterase	AChE
adenosine 3′ : 5′-cyclic monophosphate	cyclic AMP
adeonsine 5′-phosphate	AMP
adenosine triphosphatase	ATPase
γ-aminobutyric acid	GABA
analysis of variants	F
adrenaline	Ad
analytical standard of reagent purity	A.R.
anhydrous	anhyd.
approximate(ly)	approx.
approximately equals	≈
aqueous	aq.
arg-vasopressin	AVP
boiling point	b.p.
bovine serum albumin	BSA
cardiovascular system	CVS
catechol-*O*-methyl transferase	COMT
central nervous system	CNS
cerebrospinal fluid	CSF
chi-squared (statistics)	χ^2
clearance	*c*
coenzyme A	CoA
concentrated	conc.
correlation coefficient	*r*
cubic	cu.
degree of freedom (statistics)	d.f.
deoxyribonucleic acid	DNA
deoxyribunuclease	DNase
dextro-(absolute configuration)	D-
dextro-(optical rotation)	(+)-
diameter	diam.
diameter, inside	i.d.
diameter, outside	o.d.
diffusion coefficient	*D*
3,4-dihydroxyphenylalanine	DOPA
3,4-dihydroxyphenylethylamine	dopamine
direct current	d.c.
disintegration per minute	d.p.m.
dissociation constant	K_D
dissociation constant, negative logarithm of	pK
distilled	dist.
dry ice	solid CO_2
edition	edn
editor(s)	ed.
effective concentration	EC_{50}
effective dose, median	ED_{50}
electrocardiogram	ECG
electrocorticogram	ECoG
electroconvulsive therapy	ECT
electroencephalogram	EEG
electromyogram	EMG
electron spin resonance	e.s.r.
endothelial-derived relaxing factor	EDRF
epithelial-derived relaxing factor	EpDRF
equilibrium constant	*K*
equivalent (general use)	equiv.
erythrocyte	r.b.c.
erythrocyte sedimentation rate	ESR
ethylenediaminetetracetic acid	EDTA
excitatory postsynaptic potential	e.p.s.p.
experiment	expt
experimental	exptl
fatty acids, nonesterified	NEFA
figure(s) (with reference number)	Figure(s)
figure (diagram)	figure
gas-liquid chromatography	g.l.c.
glomerular filtration rate	GFR
haemoglobin	Hb
half-life	$t_{\frac{1}{2}}$
high-frequency	h.f.
high performance liquid chromatography	h.p.l.c.
human serum albumin	HSA
hydrogen-ion concentration	$[H^+]$
hydrogen-ion activity, negative logarithm of (hydrogen-ion exponent)	pH
6-hydroxydopamine	6-OHDA
N-[2-Hydroxyethyl]piperazine-N′-[2-ethanesulphonic acid]	HEPES
5-hydroxyindoleacetic acid	5-HIAA
5-hydroxytryptamine	5-HT
immunoglobulins	IgA, IgD, IgE, IgG, IgM
inhibitor contstant	K_i
inhibitory concentration	IC_{50}
inhibitory postsynaptic potential	i.p.s.p.
insoluble	insol.
international unit	iu
intra-arterial	i.a.
intracellular fluid	ICF
intradermal	i.d.
intramuscular	i.m.
intraperitoneal	i.p.
intracerebroventricular	i.c.v.
intravenous	i.v.
isotope (atomic mass) e.g. iodine-131	^{131}I
isotopically substituted compounds e.g.	[^{14}C]-ethanol
laevo-(absolute configuration)	L-
laevo-(optical rotation)	(−)-
lethal dose, median	LD_{50}
leukotriene	LT
logarithm to base e	$\log_e$ or ln
logarithm to base 10	$\log_{10}$
maximum	max.
mean arterial pressure	$\underline{M}AP$
mean value of (statistics)	$\bar{x}$
melting point	m.p.
meta	*m*-
Michaelis constant	K_M
minimum	min.
mobility (electrophoresis)	*m*
monoamine oxidase	MAO
noradrenaline	NA
nuclear magnetic resonance	n.m.r.
number	no. or No.
number of observations (statistics)	*n*
ortho	*o*-
packed cell volume	PCV
page/pages	p./pp.
para-	*p*-
paragraph	para. or ¶

parts per million	p.p.m.
per centplatelet activating factor	%PAF
posterior	post.
probability (significance level in a statistical test)	*P*
radioimmunoassay	RIA
rectus (configuration by the sequence rule)	**R**
red blood corpuscle	RBC
relative band speed to front (chromatography)	R_F
relative molecular mass	M_r
relative retention time (gas chromatography)	t_r
renal plasma flow	RPF
resistance (respiratory)	*R*
respiratory conductance	Sgaw
revolutions per minute	r.p.m.
ribonucleic acid	RNA
section	§
sedimentation coefficient (ultracentrifugation)	*s*
sinister (configuration by the sequence rule)	S
soluble	sol.
solution	soln.
Spearman rank coefficient	r_s
standard deviation (of observed sample)	s.d.
standard error (of estimate mean value)	s.e.mean
standard error (of sampling)	s.e.
standard temperature and pressure	STP
subcutaneous	s.c.
sum (statistical):	
of hypothetical popultaion	Σ
of observed sample	S or Σ
temperature	temp.
thin layer chromatography	t.l.c.
time, clock – 24 h clock used e.g. 18 h 30 min	*t*
time constant	τ
2-amino-2-hydroxymethyl-propan-1,3-diol	Tris
ultraviolet	u.v.
unit	u
vacuum	vac.
valency	e.g. Fe^{2+}; Fe(II) protoporphyrin
volume by volume	v/v
wavelength	λ
weight	wt.
weight by volume	w/v

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NOMENCLATURE GUIDELINES FOR AUTHORS

With effect from 1 January 1997

The Nomenclature Working Party (NWP) of the Editorial Board of the *British Journal of Pharmacology* has consulted many acknowledged experts in an effort to clarify and standardize receptor and other nomenclature systems for use by Editors until a complete set of recommendations from the International Union of Pharmacology Committee on Receptor Nomenclature and Drug Classification (NC-IUPHAR) is published.

NWP is unanimous in its view that, with rare exceptions, the Journal should use spellings, names and abbreviations that have been chosen by international bodies convened for the purpose.

For receptor nomenclature, with few exceptions, the Journal generally follows the guidelines laid down in the current *Trends in Pharmacological Sciences* Receptor and Ion Channel Nomenclature Supplement and the reports of NC-IUPHAR published in *Pharmacological Reviews*.

1 *Definition of receptors and subtypes*

Receptors and their subtypes are defined in terms of the relative potencies of agonists and selectivities of antagonists in functional studies, by the binding of such ligands, by transductional mechanisms and by structural information, where available.

2 *Format of receptor names*

It was agreed that, until NC-IUPHAR provides full recommendations:

(a) Editors will permit with reluctance new nomenclature systems in papers accepted for publication if, and only if, there are compelling reasons to introduce a new terminology (or modify an accepted one). The criteria upon which the new receptor type or subtype are defined must be given, together with adequate explanations of the relationship between the previous nomenclature (fully referenced) and the proposed one.

N.B. The new nomenclature should not appear in the Title, Short Title or Keywords, unless qualified by the adjective putative (e.g. . . . mediated by the putative imidazoline I_2 receptor).

(b) Only well-established and universally accepted subtype names (e.g. muscarinic and nicotinic acetylcholine receptors; α- and β-adrenoceptors) will be acceptable without any reference to the originator of these terms. In cases of controversy concerning further subdivision of the subtype, full referencing must be given.

(c) When receptors are expressed from DNA or RNA that has been introduced into cells and these receptors display a similar pharmacological profile to the native receptors, they should be denoted by use of lower case, e.g. m1 for expressed receptor and M_1 for native receptor. The stoichiometry of the expressed receptor should be indicated, where appropriate, e.g. for an immature muscle nicotinic acetylcholine receptor, it might be $(\alpha 1)_2\beta 1\gamma\delta$.

(d) Receptor subtypes should be designated by means of a subscript numeral or capital letter. Some double subscripts (i.e. numeral plus letter) are acceptable.

(e) Greek letters and Roman numerals should be avoided in any new nomenclature. The name should not include the letter 'R' or 'r' as an abbreviation for receptor.

(f) Mammalian systems are the basis of receptor classifications. Evolutionary changes may be so great that receptors in non-mammalian species are difficult to classify within this nomenclature. Therefore non-mammalian species should be clearly indicated, e.g. torpedo nicotinic acetylcholine receptor, chick β-adrenoceptor, locust GABA receptor.

3 *Types of receptor*

The NWP accepts that there are additional receptors to those described below which can be considered to be well characterised. In many cases, however, their existence has been confirmed only in cloning studies and it is as yet unclear how they relate to similar subdivisions proposed on the grounds of differences in agonist and antagonist potencies in various tissues.

(a) *Acetylcholine receptors* The two principal subfamilies are muscarinic and nicotinic acetylcholine receptors.

Muscarinic acetylcholine receptors The principal subtypes are M_1, M_2, M_3, M_4 and M_5.

Nicotinic acetylcholine receptors Receptors should be described as nicotinic (not n), and where it is known the stoichiometry should be given at first mention.

(b) *Adenosine receptors* Known also as P_1 purinoceptors (see Purinoceptors, 3v).

(c) *Adrenoceptors* The principal subtypes are α_1-, α_2-, β_1-, β_2- and β_3-adrenoceptors. Additional subtypes must be fully referenced. (See Bylund, D.B., *et al.* (1994) *Pharmacol. Rev.*, **46**, 121-136 and Hieble, J.P., *et al.* (1995) *Pharmacol. Rev.*, **47**, 267-270).

(d) *Angiotensin receptors* The principal subtypes are AT_1 and AT_2.

(e) *Bombesin receptors* Proposed subtypes, such as BB_1 and BB_2, may be used but must be fully referenced.

(f) *Bradykinin receptors* The principal subtypes are B_1 and B_2.

(g) *Calcitonin gene-related peptide (CGRP) receptors* Proposed CGRP receptor subtypes must be fully referenced.

(h) *Cannabinoid receptors* The principal subtypes are CB_1 and CB_2.

(i) *Chemokine receptors* The principal subgroups are CC (CK_1, CK_2, CK_3, CK_4) and CXC ($IL8_A$, $IL8_B$). Subtypes must be fully referenced.

(j) *Cholecystokinin (CCK) receptors* The principal subtypes are CCK_A and CCK_B.

(k) *Dopamine receptors* The principal subtypes are D_1, D_2, D_3, D_4 and D_5.

(l) *Endothelin receptors* The principal subtypes are ET_A and ET_B. (See Masaki, T., *et al.* (1994) *Pharmacol. Rev.*, **46**, 137-142).

(m) *Excitatory amino acid receptors* Three ionotropic subtypes are recognised and named: (1) NMDA receptors; (2) AMPA receptors, and (3) kainate receptors. A second class is the metabotropic glutamate (mGlu) receptor family. Further subtypes must be fully referenced.

(n) *γ-Aminobutyric acid, (GABA) receptors* The principal subtypes are $GABA_A$, $GABA_B$ and $GABA_C$. Modulatory sites on the $GABA_A$ receptor should be referenced.

(o) *Histamine receptors* The principal subtypes are H_1, H_2 and H_3.

(p) *5-Hydroxytryptamine (5-HT) receptors* The principal subtypes are $5\text{-}HT_1$, $5\text{-}HT_2$, $5\text{-}HT_3$ and $5\text{-}HT_4$. Further subdivisions require full referencing. (See Hoyer, D., *et al.*, (1994) *Pharmacol. Rev.*, **46**, 157–203).

(q) *Leukotriene (LT) receptors* Receptors should be designated according to the leukotriene that selectively or preferentially binds to them. All leukotriene receptor subtypes should be fully referenced.

(r) *Melatonin receptors* The principal subtypes are ML_{1A}, ML_{1B} and ML_2. All melatonin receptor subtypes should be fully referenced.

(s) *Neuropeptide Y (NPY) receptors* The principal subtypes are NPY_1 and NPY_2. All NPY receptors should be fully referenced.

(t) *Opioid receptors* The principle subtypes are OP_1, (formerly δ), OP_2 (formerly K) and OP_3 (formerly μ). (See Dhawan, B.N., *et al.*, *Pharmacol. Rev.* in press). Other proposed subtypes should be fully referenced.

(u) *Prostanoid receptors* The principal types are DP, EP, FP, IP and TP. When first mentioned, the style prostanoid (XP) receptor should be used, thereafter XP receptor (where X denotes the type). Proposed subtypes should be referred to as XP_n, (e.g. EP_1, EP_2) and referenced. (See Colman, R.A., *et al.*, (1994) *Pharmacol. Rev.*, **46**, 205–229).

(v) *Purinoceptors* The principal subtypes are P_1 and P_2. Subdivision of P_1 into A_1, A_2 and A_3 subtypes and of P_2 into P_{2X} and P_{2Y} are permitted. (See Fredholm, B.B., *et al.* (1994) *Pharmacol. Rev.*, **46**, 143–156). Further subtypes, e.g. P_{2X1} should be fully referenced.

(w) *Somatostatin (SST) receptors* Proposed subtypes should be fully referenced.

(x) *Tachykinin receptors* The term tachykinin is preferred to neurokinin. The principal subtypes are NK_1, NK_2 and NK_3.

(y) *Vasoactive intestinal peptide (VIP) receptors* Proposed subtypes should be fully referenced.

(z) *Vasopressin and oxytocin receptors* The principal subtypes are V_{1A}, V_{1B}, V_2 and OT.

4 *Naming of ion channels*

Ion channels are described by an abbreviation of the ion permeating the channel (e.g. K^+ channel, Na^+ channel, Cl^- channel, Ca^{2+} channel etc.). Ionic currents are referred to by either the full description of the current (e.g. Ca^{2+}-activated K^+ current) or as an abbreviation using the prefix *I* followed by the atomic species carrying the current as a subscript (e.g. I_{Na}, I_{Ca}). Where it is important to specify the activator of the current, this may be added to the atomic species subscript in parenthesis (e.g. $I_{K(Ca)}$ for a calcium-activated potassium current or $I_{K(V)}$ for a voltage-activated potassium current). When channels or current are identified only by application of pharmacological agents (rather than by other physiological mechanisms), the pharmacological blocking/activating agent can be placed in the subscript (e.g. I_{PNS} for a current activated (or blocked) by 'PNS2314' or $I_{K,PNS}$ for a potassium current activated (or blocked) by 'PNS2314').

When a pharmacological agent is used to describe a current, it must be fully defined in the text.

When, for example, a system has two currents carried by the same ionic species and activated by similar means but with (for example) different kinetics, the distinguishing factor may be added to the subscript or as a hyphen after the abbreviation (e.g. '$I_{K(Ca,slow)}$' or '$I_{K(Ca)}$-slow', which would refer to a kinetically slow calcium-activated potassium current).

Examples of some commonly used abbreviations are shown below:

Potassium currents: $I_{K(V)}$, $I_{K(Ca)}$, $I_{K(A)}$, $I_{K(IR)}$
Chloride currents: $I_{Cl(Ca)}$, $I_{Cl(cAMP)}$, $I_{Cl(swell)}$
Calcium currents: $I_{Ca(L)}$, $I_{Ca(T)}$, $I_{Ca(N)}$.

5 *Naming of nerve fibres*

Many nerve fibres are now known to release more than one transmitter, and future work may show that this is in fact the general rule. In that case, the concept of the same transmitter being released either at different developmental stages or under various experimental conditions would no longer hold, and single adjectives that imply this (e.g. cholinergic, noradrenergic) would become inappropriate when applied to nerve fibres, as distinct from transmitter functions. For the present, those nerve fibres that are known to function by releasing more than one identified transmitter may be described accordingly; for example, noradrenergic-purinergic, cholinergic-peptidergic (in alphabetical order, the order implying no priority of function). N.B. The suffix 'ergic' should continue to be applied only to nerve fibres and to the transmission event, in accordance with Dale's intentions. For example, 'cholinergic' indicates that the nerve fibre, or the transmission, functions under particular conditions through the release of a choline-like substance. The suffix should not be used loosely to mean 'pertaining to'. Hence, for example, the expression 'cholinergic receptor' (rather than acetylcholine receptor) is an inappropriate use of the term. Transmission events involving nitric oxide may be referred to as *nitrergic*. However, *nitrergic* may be used to describe axons only when there is sufficient evidence that nitric oxide is released from them as a neurotransmitter.

(a) *Catecholamine releasing nerve fibres* Nerve fibres that are known to function by releasing noradrenaline are to be described as noradrenergic. The term adrenergic should now be reserved for nerve fibres known to release adrenaline. Where the identity of the catecholamine is uncertain, catecholaminergic should be used.

The adjective to be applied to nerve fibres that release dopamine as a transmitter is dopaminergic (not DAergic).

(b) *Some other adjectives describing nerve fibre function* NANC is an acceptable abbreviation of non-adrenergic, non-cholinergic for peripheral efferent nerve fibres when the identity of the transmitter(s) is unknown

other than the fact that neither (nor)-adrenaline nor acetylcholine is involved. It should be defined when introduced. NANCergic, e-NANC (or NANC-e) and i-NANC (or NANC-i) are not acceptable terms.

Glutamatergic, not glutaminergic, should be used to describe nerve fibres releasing glutamate. In referring to peptide-releasing nerve fibres (e.g. those that may release substance P or vasoactive intestinal peptide) the nomenclature to be used is peptidergic (X), e.g. peptidergic (SP), peptidergic (VIP), not SPergic, VIPergic.

The terms 5-hydroxytryptamine (5-HT) and 5-hydroxytryptaminergic (i.e. nerves releasing 5-hydroxytryptamine) are preferred to those of serotonin and serotoninergic. The term 5-HTergic is not acceptable.

Likewise, the terms purinergic (ATP) and purinergic (adenosine) are preferred.

6 *Terms used to describe agonist and antagonist action*

The following terms can be used without full definition. Where appropriate, other terms may be used but must be accompanied by a full definition.

Terms used to describe affinity and potency

(a) EC_{50} The concentration of an agonist that produces 50% of the maximal response for that agonist *in vitro*. The agonist may be stimulatory or inhibitory. When EC_{50} values are determined in the presence of other agonists or antagonists the concentration of the latter should be stated. Related terms, e.g. EC_{25}, are acceptable if accompanied by a full definition.

(b) IC_{50} This term may be used in the following ways: (i) The concentration of antagonist that reduces the response to a sub-maximal concentration of agonist by 50%; the concentration of agonist should be stated. (ii) The concentration of competing agonist or antagonist that inhibits the binding of a radioligand by 50%; the concentration of radioligand should be stated.

(c) ED_{50} This term may be used in the following ways: (i) The dose of an agonist or antagonist that produces 50% of the maximal possible effect of that agonist or antagonist *in vitro*. (ii) The dose of drug that produces the effect under investigation in 50% of the population.

(d) K The dissociation equilibrium constant (mol L^{-1}) for ligand-receptor interactions. The reciprocal is called the affinity constant or association equilibrium constant. When necessary for clarity, subscripts (letters or numerals, or a combination of both) may be added but these must be clearly explained when first used.

(e) n_H The Hill coefficient.

(f) pA_2 The negative logarithm to base 10 of the concentration of an antagonist that makes it necessary to double the concentration of agonist needed to elicit a given submaximal response. Note that the definition is empirical and does not pre-suppose the mechanism of antagonism. The pA_2 value can be determined from a Schild plot with unconstrained slopes, but only provides an estimate of the pK_B if the antagonism has been shown to meet all of the criteria of competition.

(g) pD_2 The negative logarithm to base 10 of the EC_{50}.

(h) pIC_{50} The negative logarithm to base 10 of the IC_{50}.

(i) pK The negative logarithm to base 10 of K (with or without subscripts as appropriate: see 6(d).

Terms used to describe the mode of antagonism

(a) *Competitive antagonism* In competitive antagonism the binding of agonist and antagonist is mutually exclusive. This may be because the agonist and antagonist compete for the same binding site or combine with adjacent sites that overlap. A third possibility is that different sites are involved but they influence the receptor macromolecule in such a way that agonist and antagonist molecules cannot be bound at the same time.

(b) *Irreversible competitive antagonism* Used to describe antagonists that bind irreversibly.

(c) *Non-competitive antagonism* Agonist and antagonist can be bound simultaneously; antagonist binding reduces or prevents the action of the agonist.

(d) *Irreversible non-competitive antagonism* Used to describe non-competitive antagonists that bind irreversibly.

For a more detailed account of the terms used to describe agonist and antagonist action see Jenkinson, D.H., *et al.* (1995) *Pharmacol. Rev.*, **47**, 225–266.

7 *Enzymes*

The International Union of Biochemistry and Molecular Biology Enzyme Commission (EC) number and full name (Enzyme Nomenclature 1992, Academic Press, San Diego and London) must be quoted when first mentioned in text. Subsequently the accepted trivial name is used. Trivial names may be used in the title.

8 *Other nomenclature requirements*

(a) *Racemates* Authors must state unambiguously in the Methods section of papers which isomers were used, e.g. (+)- or (−)-propranolol, and must bring to the attention of the reader the composite character of drugs that are mixtures of stereoisomers. Furthermore, the implications of the composite nature of such drugs studied for the interpretation of the data measured and the conclusions drawn must be made explicit. Note that the terms d- or l- for dextro- and laevo-rotatory are now obsolete, and the prefixes (+)- or (−)- respectively should be used. Capital D and L refer to the absolute configurations and of course remain acceptable when appropriate.

(b) *Purines* This term should not be used as a synonym for purine nucleotides or nucleosides.

(c) *Eicosanoids* The system of nomenclature to be used for eicosanoids is that published in *Methods in Enzymology* (1990) **187**, 1–9. This scheme incorporates recent changes in the style of abbreviation of hydroperoxy-, epoxy- and oxo-unsaturated fatty acids, e.g. 12(S)-hydroperoxyeicosatetraenoic acid which was formerly abbreviated as 12(S)-HPETE now becomes 12(S)-HpETE. In manuscripts, the first use of the full chemical name of any eicosanoid should indicate double bond geometry when this is known.

(d) *Cell lines* Cell type, species and source should be defined.

(e) *Molecular biology* Abbreviations pertaining to molecular biological techniques need to be defined or presented in such a way that they can be recognised by the non-specialist, e.g. the oligonucleotide sequence, TAGC.

(f) *Tension* Tension is force and should be calibrated in newtons (1 newton = 1 kg m s^{-1}) or in kg weight, g weight, or mg weight etc. It should not be calibrated in units of mass (e.g. kg). (See Miller D.J. (1988) *Trends Pharmacol. Sci.*, **9**, 124–5).

(g) *Ions* When referring to ions, the charge should be indicated, e.g. Na^+, Ca^{2+}, $2Na^+/Ca^{2+}$ exchange, etc.

(h) *Inhibitors of nitric oxide synthase* The most commonly used and currently accepted abbreviations for N^G-nitro-L-arginine and N^G-nitro-L-arginine methyl ester are L-NOARG and L-NAME respectively.