In the brain, highly connected and heterogeneous GABAergic cells are crucial in controlling the activity of neuronal networks. They accomplish this task by communicating through remarkably diverse sets of inhibitory processes, the complexity of which is reflected by the variety of interneuron classification schemes proposed in recent years. It is now becoming clear that the subcellular localization and intrinsic properties of heteropentameric GABA$_A$ receptors themselves also constitute major sources of diversity in GABA-mediated signaling. This review summarizes some of the factors underlying this diversity, including GABA$_A$ receptor subunit composition, localization, activation, number and phosphorylation states, variance of GABA concentration in the synaptic cleft, and some of the presynaptic factors regulating GABA release.

The ‘80/20 rule’ originally formulated by the Italian economist Vilfredo Pareto (1848–1923), and mostly known today as Murphy’s law of management, states that 80% of the profits in a given company are produced by 20% of the employees. This principle could equally well apply to some neuroscience research laboratories, and it also happens to describe the rules of connectivity in complex networks including the World Wide Web (WWW), where 80% of links point to only 15–20% of web pages [1]. Whenever and wherever the Pareto principle surfaces, a power law is most likely to be behind it [1]. Do Murphy’s laws of management and the rules of WWW interconnections also apply to the workload and connectivity of various neurons in the CNS? If so, it should be noted that 17–20% of the neurons in the brain are GABAergic [2]. Are these cells the CNS equivalents of Google-like websites, pointed to by >80% of the neuronal connections? Although they receive only ~6000 synapses each, considerably fewer than the 10 000 or so found on single pyramidal cells [2], GABAergic cells might still be better connected than the principal cells they innervate. Spinning this idea one step further, even within the population of GABAergic cells there might be some groups better connected than others [3]. Interneurons that specifically innervate other GABAergic cells [4] usually express calretinin. Perhaps not by chance, calretinin-positive cells represent ~17% of the GABAergic interneurons [5].

Classically, GABA$_A$-receptor-mediated inhibition has been considered the province of fast synaptic (phasic) neurotransmission. However, over the past few years it has become apparent that some types of GABA$_A$ receptor also participate in a distinct form of ‘tonic’ inhibition produced by the continuous activation of extrasynaptic GABA$_A$ receptors [6]. Most recently, this distinction has become blurred by the finding that ‘slow’ types of phasic inhibition can be produced by spillover of transmitter onto ‘perisynaptic’ and extrasynaptic receptors [7,8]. No matter which type of inhibition one considers, diversity is still the rule.

Phasic (synaptic and ‘perisynaptic’) inhibition
Synaptic GABA$_A$ receptors are anchored by specific proteins [9] of lesser renown than those found at synapses harboring glutamate receptors [10]. Nevertheless, just like glutamate receptors, the number of synaptic GABA$_A$ receptors is subject to large alterations during neuronal plasticity and development [11]. Synaptic GABA$_A$ receptors usually contain $\gamma$ subunits, in particular $\gamma_2$ [11], which is a key factor for benzodiazepine sensitivity [12]. Phasic (synaptic) GABA-mediated transmission [13–15] is produced by high concentrations of GABA (0.3–1.0 mM) that are short-lived in the cleft (<1 ms) [16,17]. Depending on the synapse, the GABA concentration transient might or might not saturate the dozens of GABA$_A$ receptors present on the postsynaptic side. The activation of synaptic GABA$_A$ receptors produces an inhibitory postsynaptic current (IPSC) shaped by the properties and number of receptors and by the magnitude and duration of the GABA transient. Moreover, the kinetics of GABA$_A$ receptor openings might even be affected by the anchoring and targeting proteins responsible for clustering receptors at synapses [18].

Some GABA$_A$ receptors can be found at or beyond the perimeter of GABA synapses [7,8]. Comparable to the activation of metabotropic glutamate receptors at excitatory synapses, the localization of which calls for redefinition of the limits of a Sherringtonian synapse [2],
perisynaptic GABA<sub>A</sub> receptors can be activated by transmitter overspill [8]. Accordingly, phasic GABA release should not be considered to activate only synaptic GABA<sub>A</sub> receptors. Less is known at present about the concentration and time course of transmitter release in this situation, or whether spillover often accompanies classical fast synaptic transmission [19] or whether it arises from separate classes of GABAergic synapses [8,20].

**Tonic inhibition**

Many cells display tonic currents activated by the near-micromolar GABA levels [21] always present in the extracellular space. The charge carried by the activation of tonically active GABA<sub>A</sub> receptors can be more than three times larger than that produced by phasic inhibition, even when the frequency of phasic events is large [22–24] (Figure 1). Experimental and theoretical studies indicate that a tonic GABA conductance produces a shunt that affects excitability and gain control [6]. Perhaps the best demonstration that this is crucial in regulating neuronal excitability comes from genetic ablation experiments targeting extrasynaptic receptors. The missing tonic GABA current of cerebellar granule cells in mice devoid of α6 GABA<sub>A</sub> receptor subunits (α6<sup>−/−</sup>) is replaced by a continuously active K<sup>+</sup> conductance of equal magnitude [25].

Cerebellar granule cells of α6<sup>−/−</sup> animals also lack δ-subunit-containing GABA<sub>A</sub> receptors because of a specific partnership between the α6 and δ subunits [26]. This led to the idea that tonic inhibition in granule cells must be mediated by δ-subunit-containing GABA<sub>A</sub> receptors known to be located exclusively extrasynaptically [7]. Indeed, these GABA<sub>A</sub> receptors have a prominent role in mediating tonic inhibition. Their extrasynaptic location [7,8] and unusually high GABA affinity [27] are in line with mediating the tonic GABA conductance in both cerebellar and dentate gyrus granule cells [28].

The diverse effects of tonic inhibition in a network can be appreciated by its different actions on various classes of inhibitory interneurons and principal cells. Selective relief of tonic inhibition in interneurons by relatively low concentrations of picrotoxin leads to a considerable increase in spontaneous IPSC (sIPSC) frequency in CA1 pyramidal neurons [23]. Yet the same picrotoxin concentration has no effect on the frequency or amplitude of sIPSCs recorded in the interneurons themselves, consistent with a different pharmacological profile of the tonic current in GABAergic cells that specifically innervate other interneurons [4].

**Figure 1.** Comparison of phasic and tonic currents in recorded in adult rodent CA1 pyramidal cells at near physiological temperature (34°C). (a) The average phasic current caused by the spontaneous inhibitory postsynaptic currents (sIPSCs) in a mouse CA1 pyramidal cell over 1 s was calculated as the mean current (red line) generated by all events, and was compared with the baseline (black line). Despite the high frequency of sIPSCs (~50 Hz) and their relatively large amplitudes, the mean current was only 14 pA, less than a third of the 43 pA generated by tonic inhibition in the same cell (b). Thus, only 25% of the total time-averaged inhibitory conductance is mediated by phasic (synaptic) inhibition, whereas 75% is generated by tonic inhibition. The magnitude of tonic inhibition (red line) was measured as described in Ref. [22] after the perfusion of a saturating concentration of bicuculline (BMI) during the time indicated by the bar, to subtract the baseline current (black line). Horizontal scale bar: 200 ms in (a) and 20 s in (b). (c) In a rat CA1 pyramidal cell, occurrence of large-amplitude slow spontaneous events, such as the first event on this trace, might significantly add to the mean GABA current experienced by the cell. However, these events could originate from the overspill of transmitter onto extrasynaptic receptors [see text for details], and therefore might be considered as phasic activation of extrasynaptic or perisynaptic receptors of different molecular composition from those found at synapses.

**Causes of diversity in GABA-mediated signaling**

**Subunit composition of synaptic GABA<sub>A</sub> receptors**

GABA<sub>A</sub> receptors are pentameric hetero-oligomers assembled from members of seven different subunit classes, some of which have multiple members: α(1–6), β(1–3), γ(1–3), δ, ε, θ, and π [12]. In theory, a bewildering array of various combinations could assemble from these many subunits and their splice variants. However, GABA<sub>A</sub> receptor subunits do form preferred assemblies, with perhaps dozens of distinct subunit combinations actually present in the brain [12]. Studies in expression systems have revealed numerous physiological and pharmacological differences between the properties of GABA<sub>A</sub> receptors composed of different subunits [29], indicating that subunit composition is one of the major factors underlying diversity. Characteristics imparted by individual subunits that are revealed by studies of recombinant receptors can be used to infer their presence at specific synapses, although it must be recognized that receptor properties depend on the expression system that is used [30].

Complex patterns of subunit distribution in different brain regions and cell types support the view that dozens of GABA<sub>A</sub> receptor combinations are present in the brain. Moreover, even individual neurons can express several different subunit combinations. Because subunit composition strongly influences decay kinetics [29], one might expect to find a variety of kinetically distinct IPSCs in a given cell with multiple GABA<sub>A</sub> receptor subunits. However, it is not always straightforward to relate immunohistochemical information about subunit localization to physiological characteristics of synaptic responses. For example, hippocampal CA1 pyramidal neurons express a large number of GABA<sub>A</sub> receptor subtypes [31], including receptors with α1, α2 and α5.
subunits. However, only two kinetically distinct IPSCs have been described in these cells [32]. Which receptor combinations give rise to these responses? To answer this question, it might be instructive to consider subunit distribution patterns. Most α2-subunit-containing receptors are found at synapses on axon initial segments [33] and at somatic synapses made by parvalbumin-negative basket cells [34]. By contrast, parvalbumin-positive interneurons innervate somatic GABA_A receptors with a large α1:α2 subunit ratio [35]. This clear segregation of α1 and α2 subunits should result in different properties of IPSCs generated at the two kinds of synapse because, in expression systems, α2-subunit-containing GABA_A receptors deactivate more slowly than receptors containing the α1 subunit [36]. Indeed, in mice lacking the α1 subunit, miniature IPSCs (mIPSCs) decay more slowly than those in wild-type mice [37], and in other neurons α1 subunits are responsible for speeding up IPSC decay during neuronal development [38]. However, contrary to expectations, paired recordings between parvalbumin-positive and parvalbumin-negative basket neurons and principal cells have revealed only a single class of fast IPSCs [39]. Those recordings did also reveal a second class of IPSCs with slow kinetics that might arise from the dendrites of CA1 neurons, where α1 and α5 subunits are located [40]. However, as yet there is no direct evidence regarding the subunit composition of receptors that produce slow dendritic IPSCs, or whether these also might arise from a heterogeneous population of presynaptic neurons.

The specificity of α subunits is remarkable when it comes to the effects of benzodiazepines. Point mutations in mice rendering various α subunits insensitive to benzodiazepines have revealed that GABA_A receptors with α1 subunits are responsible for mediating their sedative, anesthetic, anticonvulsant effects, whereas the anxiolytic effects are mediated by α2 subunits [41]. It is not yet known whether these effects result from the subcellular segregation of α subunits at synapses formed by the different interneurons [42] or from the enrichment of specific α subunits in certain brain regions.

**Differential modulation of synaptic and non-synaptic receptors**

Just as subunit composition varies at synapses formed by specific subclasses of interneurons that innervate specific cellular compartments of principal cells [4], there are also differences between the subunit composition of synaptic GABA_A receptors and those located perisynaptically or extrasynaptically. These distinctions translate into explicit differences in the modulation of tonic versus phasic inhibition by endogenous and exogenous compounds. Thus, tonic and phasic inhibitions are modulated distinctly by benzodiazepines, by blocking GABA uptake by the GABA transporter GAT-1, by nicotinic ACh receptor activation, by furosemide, and by competitive and non-competitive GABA_A receptor antagonists [6]. For example, tonic inhibition in CA1 pyramidal neurons, where α5-subunit-containing [43] rather than δ-subunit-containing [28] GABA_A receptors mediate tonic inhibition, is sensitive to benzodiazepines but not to penicillin [6]. Interneurons themselves exhibit a tonic current sensitive to both zolpidem and picrotoxin that is not blocked by low concentrations (500 nM) of SR95531 but is specifically blocked by a low dose of picrotoxin (1 μM) [23].

There is a striking and physiologically relevant difference in the modulation of tonic and phasic inhibitions by neuroactive steroids (neurosteroids). Concentrations of the neurosteroid allotetrahydrodeoxycorticosterone (THDOC) in the physiological range (10 nM) significantly potentiate the tonic conductance in dentate gyrus as well as in cerebellar granule cells without affecting phasic currents, thus identifying a unique site of action for neurosteroids [28]. It will be interesting to characterize the specific effects of ethanol concentrations that are toxic to humans on the diversity of GABA-mediated signaling. For example, δ-subunit-containing GABA_A receptors appear to be highly sensitive to ethanol [44] but the excitatory drive onto certain interneurons is reduced by alcohol [45].

**Desensitization**

A commonly observed use-dependent characteristic of GABA_A receptors is desensitization. When most isolated or expressed receptors are exposed to a steady concentration or repetitive brief pulses of GABA, the amplitude of the response declines. Although such experimental stimuli are very different from the natural stimulus experienced by GABA_A receptors in the brain, several physiologically relevant aspects of inhibition can be derived from this fundamental receptor property. First, it imparts complex kinetic characteristics to synapses: in response to a brief, saturating pulse of agonist designed to mimic the concentration profile of transmitter in the synaptic cleft, an initially rapid decline of the current is followed by a slower decay phase as receptors recover from desensitization and reopen. This mechanism is thought to account for the bi-exponential decay of IPSCs observed at some synapses [46], and it could contribute to changes in response kinetics with repetitive activation [47]. Second, because desensitization reduces the ability of postsynaptic receptors to respond to repetitive activation, it influences the reliability of synaptic transmission at high frequencies. This can differ even for synapses that appear to be otherwise similar physiologically [48]. Third, even low (micromolar) concentrations of neurotransmitter can substantially reduce the availability of synaptic receptors to respond to brief high-concentration transients [49]. This effect persists for many seconds, and leads to homosynaptic and heterosynaptic regulation of synaptic transmission. Network modeling studies support a role for desensitization in regulating coherent oscillations in networks of inhibitory neurons [50].

Although all GABA_A receptors display desensitization, some subunit combinations are affected more than others. For example, δ-subunit-containing GABA_A receptors, which are responsible for generating a tonic GABA conductance in many neurons, are particularly resistant to desensitization [51].

**Number of GABA_A receptors at synapses**

According to both direct (anatomical) and indirect (pharmacological) evidence, the number of GABA_A receptors at...
synapses ranges between tens and hundreds, resulting in a significant variation in their occupancy by GABA released into the cleft [13–15]. There do not seem to be any rules governing the formation of synapses with large numbers of GABA<sub>A</sub> receptors. It might turn out that the number of GABA<sub>A</sub> receptors at a synapse, as well as the specific subunit composition, is determined by the afferent input. Despite several subunit-specific targeting and insertion mechanisms [9], there is no established correlation between subunit composition and GABA<sub>A</sub> receptor number. Even when a single subunit combination (α1β2γ2) is present in a given neuron such as the cerebellar stellate or basket cell, large mIPSCs originate from large synapses with many receptors whereas small mIPSCs are generated at small synapses with many fewer receptors [16,52]. The 20–30-fold difference between the areas of the smallest and largest synapses translates into a similar difference in the volume of the cleft.

The number of synaptic GABA<sub>A</sub> receptors is dynamically regulated not only during development but also during pathological processes such as epilepsy [53]. In tandem with a change in the pharmacology of GABA<sub>A</sub> receptors, probably reflecting a change in their subunit composition, after kindling there is nearly a doubling of synaptic receptor numbers as reflected by the increased number of gold particles and a commensurate increase in IPSC quantal size [53].

Cl<sup>−</sup> reversal potential
The Cl<sup>−</sup> reversal potential determines whether a GABA synapse produces depolarization or hyperpolarization. Young neurons tend to have higher intracellular concentrations of Cl<sup>−</sup>, but distinct developmental regulation of the Cl<sup>−</sup> reversal potential by various Cl<sup>−</sup> extrusion mechanisms changes the Cl<sup>−</sup> flux through GABA<sub>A</sub> receptors [54]. Interestingly, some adult neurons retain high intracellular Cl<sup>−</sup> levels, and thus are excited by GABA [55]. The Cl<sup>−</sup> reversal potential can be dynamically regulated and in some cells this might be activity-dependent. A distinct form of long-term plasticity of inhibitory synapses that results from a change in the driving force for Cl<sup>−</sup> flux was described in CA1 pyramidal neurons in acute slices and in cell culture [56]. Coincident presynaptic and postsynaptic activation of GABAergic synapses produced a local decrease in KCC2-mediated K<sup>+</sup>–Cl<sup>−</sup> cotransport activity, which shifted the reversal potential for Cl<sup>−</sup> flux and effectively reduced the strength of inhibition. Similar to spike-timing-dependent plasticity at glutamatergic synapses, this change occurred only when postsynaptic spiking took place within 20 ms before or after the activation of GABAergic synapses.

Phosphorylation
Intracellular domains of GABA<sub>A</sub> receptor subunits can be phosphorylated by a variety of kinases at serine, threonine and tyrosine residues [57]. Just as for other ion channels, phosphorylation could underlie GABA<sub>A</sub> receptor plasticity [11,15] and might contribute to the diversity of GABA<sub>A</sub> receptor function in a given cell. The β subunits in particular appear to be targeted by serine/threonine kinases in various preparations [57,58], and this affects cell surface trafficking [58]. Protein kinase B (Akt)-dependent phosphorylation of Ser410 of the β2 subunit, a site that is conserved in all β subunits, produces rapid insertion of GABA<sub>A</sub> receptors into the membrane sufficient to enhance the amplitude of sIPSCs [59], and is responsible for rapid recruitment of GABA<sub>A</sub> receptors at synapses by insulin [60]. By contrast, phosphorylation of tyrosine residues on β2 and/or β3 subunits produces a gain in function rather than number, and γ2 subunits can be tyrosine-phosphorylated by Src and seem to be constitutively tyrosine-phosphorylated in the adult brain [57,58].

Local Ca<sup>2+</sup> influx could tap into the Ca<sup>2+</sup>-dependent component of GABA<sub>A</sub> receptor plasticity [15]. For example, when Ca<sup>2+</sup> enters via NMDA receptors, physical and functional interactions between calcineurin-A and GABA<sub>A</sub> receptor γ2S subunits induce long-term depression at inhibitory synapses [61]. Divergent effects of phosphorylation on GABA<sub>A</sub> receptor assemblies composed of mixed [1–β3 subunits could complicate interpretation of experiments that follow a global approach to study the effect of phosphorylation on native GABA<sub>A</sub> receptors [62]. Although recordings from neurons expressing a single type of β subunit might circumvent this problem, it should be noted that preferred cellular pathways and specific anchoring or auxiliary proteins could be involved in the diversity of GABA-mediated signaling resulting from phosphorylation [57,58].

GABA transient in the cleft
At any given synapse, trial-to-trial variability of postsynaptic responses can originate from the probabilistic nature of quantal transmitter release, from the stochastic behavior of the receptors, or from the fluctuation of the transmitter concentration in the cleft. In different cells this results in a large variety of kinetics of the synaptic currents (Figure 2). In a given neuron, the variability of the decay can be explained by the heterogeneous subunit combinations of receptors at different synapses. However, it is less clear why there are pronounced variations in mIPSC kinetics in some cerebellar interneurons [16,52] expressing only the GABA receptor subtype with α1β2γ2 subunit composition [63]. A major contributor to the considerable amplitude variability is the large variation in the postsynaptic receptor number between synapses [52], but at large synapses, where postsynaptic GABA<sub>A</sub> receptors are not fully occupied, some variation also originates from the fluctuation in the peak transmitter concentration. In every cell type studied so far, the variability in the decay of small amplitude synaptic currents is consistently larger than that of large-amplitude IPSCs [16].

Multivesicular release of GABA described at cerebellar interneuronal synapses could well be responsible for changes in cleft GABA concentrations [64]. GABA released from two or more vesicles not only would increase the peak concentration but also might slow its decay. Synaptic vesicles with considerable size variation between synaptic boutons would tend to be filled with varying amounts of transmitter depending on their volume and could be responsible for a large part of the diversity of synapse-specific GABA-mediated signaling.
aptic GABAA receptors but also binds to GABAB receptors only diffuses across the synaptic cleft to activate postsynaptic receptors with respect to regulation of transmitter release is not known.

Whether a more consistent picture would emerge with the number of well-defined and functionally distinct subsets. Pal interneurons might not be segregated into a small number of well-defined and functionally distinct subsets. A similar variability in the decay of mIPSCs can be found in olfactory bulb granule cells (olfactory GC). The average mIPSC \( t_{\text{w}} \) (14.8 ms; red trace) was approximately three times longer than that recorded in the layer 2/3 pyramidal cells. There is nearly a threefold difference between the \( t_{\text{w}} \) of the rapidly (8.3 ms) and slowly (27.0 ms) decaying events. The decay of mIPSCs also shows a remarkable variability in a cerebellar interneuron (cerebellar IN; rapidly decaying mIPSC \( t_{\text{w}} = 1.9 \text{ ms} \), versus slowly decaying mIPSC \( t_{\text{w}} = 4.0 \text{ ms} \), even though these neurons appear to express only a single GABAA receptor subunit combination (\( z \pm 2.1 \text{ ms} \), red trace), a usual characteristic of interneurons. Adapted, with permission, from Ref. [16] (\( \square \) 2001) the Biophysical Society.

**Presynaptic regulation by neuromodulators**

Differences in presynaptic regulation of GABA release are another important source of physiological diversity. Interneurons are subject to regulation by a variety of neurotransmitters, including GABA, glutamate, opioids, 5-hydroxytryptamine, ACh, monoamines and endocannabinoids [42]. In addition to altering cellular excitability, many of these substances also act on presynaptic terminals to regulate transmitter release. A survey of morphological, physiological and pharmacological modulation of CA1 interneuron activity found little correlation between these properties [65], suggesting that hippocampal interneurons might not be segregated into a small number of well-defined and functionally distinct subsets. Whether a more consistent picture would emerge with respect to regulation of transmitter release is not known.

When GABA is released from a nerve terminal, it not only diffuses across the synaptic cleft to activate postsynaptic GABA receptors but also binds to GABA receptors on the presynaptic side. These metabotropic receptors thus function as autoreceptors, and they limit transmitter release during subsequent action potentials by reducing \( \text{Ca}^{2+} \) entry via a direct membrane-delimited pathway. As opposed to desensitization, which within milliseconds can limit the postsynaptic response to repetitive stimuli, it takes tens of milliseconds for GABA receptors to reduce release, but their effect lasts for hundreds of milliseconds [66]. GABA receptors are expressed only in subpopulations of GABAergic interneurons [67] and not all synapses are equally sensitive to GABA receptor-mediated suppression [47].

**Concluding remarks**

Many sources of physiological diversity have been discovered at GABA synapses (Figure 3), and it is likely that the list will grow. Will it be possible to identify the functional consequences of this diversity, and to what end will such information be useful? There is even more complexity because functional diversity per se can translate into higher-level network properties, when population variance as well as mean parameter values determine the behavior of interconnected neurons [68]. In fact, systematic approaches to the question of diversity of GABA signaling have already started to provide new insights into roles of inhibition in the brain. For example, the different roles for specific types of inhibition are beginning to be understood. Tonic inhibition mediated by extrasynaptic receptors modulates neuronal gain and can be altered by circulating hormone levels [6], whereas fast synaptic inhibition dramatically narrows the time window during which coincident inputs evoke action potentials [69]. Recently it has become possible to address the functional consequences of GABA receptor subunit diversity using genetically engineered mice with point mutations in specific GABA receptor subunits [41], and such approaches should help bridge the gap between studies at cellular, circuit and behavioral levels. In light of the specific activation of interneuron subclasses during network oscillations [70,71], it will be interesting to examine the role of specific GABA receptor assemblies at the synapses formed by these cells and the mechanisms responsible for producing and maintaining the specificity of such unique receptor targeting. Understanding of the mechanisms underlying GABA receptor signaling...
plasticity is clearly lagging behind that of equivalent mechanisms in the glutamate receptor field, yet the plastic alterations affecting the GABA system might be extremely important in various devastating neurological and psychiatric disorders. Hopefully, rather than becoming a source of bewilderment and confusion, studies of the diversity of inhibitory signaling will illuminate challenging issues and new approaches to understanding brain function in health and disease.

References
7 Nusser, Z. et al. (1998) Segregation of different GABA A receptors to synaptic and extrasynaptic membranes of cerebellar granule cells. J. Neurosci. 18, 1693–1703
24 Rossi, D.J. et al. (2003) Multiple modes of GABAergic inhibition of rat cerebellar granule cells. J. Physiol. 548, 97–110
30 Merck, K. et al. (2003) Recombinant δ1 β2 γ2GABA A receptors expressed in HER283 and in Q76 cells show different kinetics. Neuroreport 14, 195–198
31 Sperrk, G. et al. (1997) GABA A receptor subunits in the rat hippocampus.1. Immunocytochemical distribution of 13 subunits. Neuroscience 80, 987–1000
38 Vicini, S. et al. (2001) GABA A receptor δ1 subunit deletion prevents developmental changes of inhibitory synaptic currents in cerebellar neurons. J. Neurosci. 21, 3009–3016
60 Wan, Q. et al. (1997) Recruitment of functional GABA<sub>A</sub> receptors to postsynaptic domains by insulin. Nature 388, 686–690
67 Sloviter, R.S. et al. (1999) Localization of GABA<sub>B</sub> (R1) receptors in the rat hippocampus by immunocytochemistry and high resolution autoradiography, with specific reference to its localization in identified hippocampal interneuron subpopulations. Neuropharmacology 38, 1707–1721