Abstract
Satralizumab (Enspryng®), a humanized anti-interleukin-6 (IL-6) receptor monoclonal recycling antibody, has been developed by Chugai Pharmaceutical and Roche for the treatment of neuromyelitis optica spectrum disorder (NMOSD). In June 2020, based on positive results from two pivotal phase III trials, subcutaneous satralizumab received its first global approval in Canada for the treatment of NMOSD in adults and children aged ≥ 12 years who are aquaporin 4 water channel autoantibody (AQP4-IgG) seropositive. Satralizumab was subsequently approved in Japan, Switzerland and the USA. Satralizumab is under regulatory review in the EU, and is undergoing clinical development in several countries worldwide. This article summarizes the milestones in the development of satralizumab leading to this first approval for the treatment of NMOSD.

1 Introduction
Neuromyelitis optica spectrum disorder (NMOSD) is a rare autoimmune disease of the central nervous system (CNS) that is characterized by inflammatory and demyelinating lesions in optic nerve, spinal cord brainstem and cerebrum, which can lead to progressive impairment of vision and motor functions [1–4]. The primary goal of NMOSD treatment is to reduce the risk of irreversible neurological impairment by preventing relapse and reducing the severity of attacks [5, 6]. Recent research suggested that interleukin-6 (IL-6) has an important role in the immunopathogenesis of NMOSD [1, 2]. IL-6 signalling triggers an inflammatory cascade that is thought to lead to differentiation of T cells into proinflammatory TH17 cells, differentiation of B cells into plasmablasts that produce aquaporin 4 water channel autoantibodies (AQP4-IgG), a diagnostic serum marker that is found in ≈ 80% of patients with NMOSD, and an increase in blood–brain barrier (BBB) permeability, allowing penetration of antibodies and proinflammatory cells into the CNS [2, 7–10].

Satralizumab (Enspryng®), a humanized anti-IL-6 receptor monoclonal antibody designed using recycling antibody technology™, has been developed by Chugai Pharmaceutical and Roche for the treatment of NMOSD [11]. Based on positive results from two pivotal phase III trials, satralizumab received its first global approval under priority review in Canada on 1 June 2020 for the treatment of NMOSD as monotherapy or as combination therapy with immunosuppressant in adults and children aged ≥ 12 years who are AQP4-IgG seropositive [11]. On 29 June 2020, satralizumab was approved in Japan, Switzerland and the USA. Satralizumab is under regulatory review in the EU, and is undergoing clinical development in several countries worldwide. This article summarizes the milestones in the development of satralizumab leading to this first approval for the treatment of NMOSD.
subsequently approved in Japan for the prevention of relapses of NMOSD, including neuromyelitis optica (NMO), in adults and children who are AQP4-IgG seropositive [12]. On 13 July 2020, satralizumab was approved in Switzerland for the treatment of NMOSD as monotherapy or as combination therapy with immunosuppressant in adults and adolescents who are AQP4-IgG seropositive [13]. On 17 August 2020, satralizumab was approved in the USA for the treatment of NMOSD in adult patients who are AQP4 antibody positive [14, 15].

Satralizumab is available as a single-use, prefilled syringe containing 120 mg/mL satralizumab for subcutaneous injection and the recommended dosage is 120 mg at week 0, 2 and 4 as loading doses, followed by a maintenance dose of 120 mg every 4 weeks [13, 15–17]. The first subcutaneous injection of satralizumab should be administered under supervision of a health practitioner. During satralizumab treatment, liver enzyme levels should be monitored every 4 weeks for the first 3 months, followed by every 3 months for 1 year; neutrophil counts should be monitored 4–8 weeks after treatment initiation and as clinically indicated thereafter [13, 15–17].

Subcutaneous satralizumab is under regulatory review in the EU [18, 19], and is undergoing clinical development in several countries worldwide. In Japan, phase I clinical development was conducted for the treatment of rheumatoid arthritis; however, no recent development reports have been identified.

### 1.1 Company Agreements

In June 2016, Chugai Pharmaceutical and Roche entered into an exclusive worldwide license agreement under which the latter was granted the worldwide rights for the development and marketing of satralizumab, with the exception of Japan and Taiwan [20, 21]. Under the terms of the agreement, Chugai is entitled to receive an upfront fee, milestone and royalty payments from Roche, while being responsible for continued product manufacturing and supply of satralizumab [20].

### 2 Scientific Summary

#### 2.1 Pharmacodynamics

Satralizumab is a humanized immunoglobulin G2 monoclonal antibody produced in Chinese hamster ovary cells using recombinant DNA technology [16]. The exact mechanisms of action of satralizumab in treating NMOSD is currently unknown, but is thought to target multiple aspects that contribute to the disease in patients with NMOSD by binding to membrane-bound and soluble IL-6 receptors, thereby blocking IL-6 signalling pathways [6, 10, 16]. Through inhibition of downstream IL-6 signalling pathways, satralizumab is thought to reduce inflammation and IL-6 mediated autoimmune T- and B-cell activation, preventing differentiation of B cells into AQP4-IgG-secreting plasmablasts [2, 10]. Satralizumab utilizes a novel recycling antibody technology which allows satralizumab to dissociate from IL-6 receptor in a pH-dependent manner, thereby extending the duration of circulation in the body [2]. After uptake by the cell, the
antibody-antigen complex is transported to the endosome [2, 22, 23]. In the acidic environment of the late endosomal compartment (pH 5.5–6.0), satralizumab dissociates from the IL-6 receptor and is transported back to the plasma membrane via the recycling endosomal pathway by binding to the neonatal Fc receptor (FcRn), leading to release of free satralizumab back into plasma, ready to bind another IL-6 receptor [2, 22, 23]. The drug’s binding affinity to IL-6 receptor is maintained in plasma (pH 7.4) [2, 22]. Satralizumab has ≈ 4-fold higher affinity to IL-6 receptor than tocilizumab [6].

In vitro, satralizumab reduced NMO-induced BBB dysfunction [24]. Moreover, subcutaneous satralizumab significantly inhibited IL-6 receptor signalling for four weeks, with marked, sustained increases in soluble IL-6 receptor levels observed in Japanese and Caucasian healthy volunteers (n = 72) and patients with rheumatoid arthritis (n = 33) or with NMO (n = 104) [25]. In NMOSD patients who received subcutaneous satralizumab 120 mg at week 0, 2, 4 (loading regimen) and once every 4 weeks thereafter (maintenance regimen), the predicted median IL-6 receptor occupancy was maintained at > 95% over the satralizumab 4-week dosing interval [25–27]. Decreases in C-reactive protein, fibrinogen and complement (C3, C4 and CH50) were also observed with satralizumab treatment [16].

2.2 Pharmacokinetics

In healthy volunteers, subcutaneous satralizumab exhibits non-linear pharmacokinetics across the single dose range of 30–240 mg [28].

Following subcutaneous administration of satralizumab at the recommended dosage, the absorption half-life and bioavailability is ≈ 3 days and 78.5%, respectively, with steady state reached 8 weeks after the first administration, according to a population pharmacokinetic analysis in 154 patients with NMO or NMOSD [16]. Satralizumab is distributed in a biphasic manner where the estimated central and peripheral volume of distributions for a typical 60 kg patient is 3.46 L and 2.07 L, respectively. Although the metabolism of satralizumab has not been characterized, the drug is expected to mainly undergo catabolism in the same manner as endogenous IgG. Satralizumab exhibits concentration-dependent, linear and target-mediated (Michaelis-Menten) elimination characteristics, with the estimated linear clearance and associated elimination half-life being 0.0679 L/day and ≈ 30 days, respectively [16].

The pharmacokinetics of satralizumab are not affected by age, gender and race [16]. No formal studies of the effect of hepatic or renal impairment on the pharmacokinetics of satralizumab have been conducted. Higher bodyweight and the presence of anti-drug antibodies (ADAs) appeared to have a significant impact on satralizumab exposure; however, based on the results of exposure-response relationship analyses, no dosage adjustment is required [16].

There are no formal drug interaction studies of satralizumab [16]. Since the expression of CYP450 enzymes is suppressed by overproduction of IL-6 in vitro and in vivo, caution should be exercised when satralizumab is initiated or discontinued in patients concomitantly receiving CYP3A4, CYP1A2, CYP2C9 or CYP2C19 substrates, particularly those with a narrow therapeutic index (e.g. warfarin, carbamazepine, phenytoin and theophylline) and dosage can be adjusted if needed [16].

### Features and properties of satralizumab

**Alternative names**
ENSPRYNG; RG-6168; SA-237

**Class**
Anti-inflammatories; Antirheumatics; Eye disorder therapies; Monoclonal antibodies

**Mechanism of Action**
Interleukin 6 receptor antagonists

**Route of Administration**
Subcutaneous

**Pharmacodynamics**
Inhibits interleukin-6 signalling pathways

**Pharmacokinetics**
Non-linear pharmacokinetics; steady state reached 8 weeks after first administration; estimated mean linear clearance 0.0679 L/day and the associated elimination half-life ≈ 30 days

**Most frequent adverse events**
Headache, arthralgia and injection related reactions

**ATC codes**
WHO ATC code
L04A-C19 (Satralizumab); M01A (Antinflammatory and Antirheumatic Products, Non-steroids)

EphMRA ATC code
L4C (Interleukin Inhibitors); M1A (Anti-Rheumatics, Non-Steroidal)

**Chemical Name**
Immunoglobulin G2, anti-(human interleukin 6 receptor) (human-mus musculus monoclonal SA237 heavy chain), disulfide with human-mus musculus monoclonal SA237 light chain, dimer

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2.3 Therapeutic Trials

The efficacy and safety of satralizumab was evaluated in two pivotal, multinational, randomized, double-blind, placebo-controlled phase III trials, SAkuraStar (NCT02073279) and SAkuraSky (NCT02028884), in patients with NMO or NMOSD, including both AQP4-IgG seropositive and seronegative patients (capped at 30%) [26, 27]. In SAkuraStar (n = 95) and SAkuraSky (n = 83), patients were randomized to receive subcutaneous satralizumab 120 mg or matching placebo at week 0, 2 and 4, then once every 4 weeks; satralizumab was administered as a monotherapy (SAkuraStar [26]) or as an add-on therapy to baseline immunosuppressant therapy (SAkuraSky [27]). In SAkuraSky, baseline immunosuppressant therapy included azathioprine (AZA), mycophenolate mofetil (MMF) or oral corticosteroids at stable doses, with adolescent patients permitted to receive a combination of AZA and oral corticosteroids, or MMF and oral corticosteroids [27]. Patients experiencing a protocol-defined relapse (PDR) or those completing the double-blind period of the study were eligible to enter an open-label extension period, during which all patients received unblinded long-term treatment with satralizumab [26, 27].

During the double-blind period, satralizumab was effective in reducing the risk of an adjudicated NMOSD relapse whether it was administered as a monotherapy or as an add-on therapy to baseline immunosuppressant therapy [26, 27]. In SAkuraStar (median treatment duration: 92.3 weeks with satralizumab vs 54.6 weeks with placebo), the proportion of patients experiencing a PDR was significantly (p = 0.018) lower in the satralizumab monotherapy group than in the placebo group (30% vs 50%), with satralizumab reducing the risk of relapse by 55% [hazard ratio (HR) 0.45, 95% CI 0.23–0.89; primary endpoint in the intention-to-treat population, encompassing both AQP4-IgG seropositive and seronegative subgroups] [26]. The proportion of relapse-free patients in the satralizumab and placebo groups was 89% versus 66% at week 48, 78% versus 59% at week 96 and 74% versus 49% at week 144, respectively [27]. There was no significant difference between satralizumab and placebo groups in the predefined key secondary outcomes, change in Visual Analogue Scale pain score and the Functional Assessment of Chronic Illness Therapy (FACIT) fatigue score from baseline to week 24 [26, 27].

In a prespecified subgroup analysis, relative to placebo, satralizumab was associated with a significantly lower risk of relapse (based on 95% CIs) in patients who were AQP4-IgG seropositive (SAkuraStar: HR 0.26, 95% CI 0.11–0.63; SAkuraSky: HR 0.21, 95% CI 0.06–0.75); there was insufficient evidence (based on 95% CIs) to indicate that satralizumab significantly lowered the risk of NMOSD relapse compared with placebo in patients who were AQP4-IgG seronegative [26, 27]. In the AQP4-IgG seropositive subgroup, the proportion of relapse-free patients receiving satralizumab or placebo was 83% versus 55% at week 48, 77% versus 41% at week 96 and 77% versus 41% at week 144 in SAkuraStar [26] and it was 92% versus 60% at week 48, 92% versus 53% at week 96 and 85% versus 53% at week 144 in SAkuraSky [27]. Furthermore, the treatment benefit of satralizumab over placebo was consistently seen regardless of age (adolescents aged 13–17 years [29]) or geographical region (Asian region [30]).

The results of a pooled analysis of data from SAkuraStar and SAkuraSky were consistent to those observed in the individual trials [31, 32]. In the pooled analysis, satralizumab reduced the risk of PDR by 58% compared with placebo (HR 0.42, 95% CI 0.25–0.71) and the HR for this endpoint in the AQP4-IgG seropositive group was 0.25 (95% CI 0.12–0.50) [31, 32].

### Key clinical trials of satralizumab

| Drug(s) | Indication | Phase | Status | Location(s) | Identifier | Sponsors |
|---------|------------|-------|--------|-------------|------------|----------|
| Satralizumab, placebo | NMO/NMOSD | Phase III | Active, no longer recruiting | Multinational | NCT02073279, BN40900, SA309JG, SAkuraStar | Chugai Pharmaceutical, Roche |
| Satralizumab, placebo | NMO/NMOSD | Phase III | Active, no longer recruiting | Multinational | NCT02028884, BN40898, SA307JG, SAkuraSky | Chugai Pharmaceutical, Roche |
2.4 Adverse Events

Satralizumab was generally well tolerated when administered as a monotherapy or as an add-on therapy to immunosuppressant therapy in patients with NMOSD, based on results from the phase III SAkuraStar and SAkuraSky trials [26, 27, 33]. The most commonly reported adverse events (AEs) with satralizumab included headache, arthralgia and injection related reactions (IRRs) [16]. In 104 satralizumab recipients in SAkuraStar and SAkuraSky, serious treatment-related AEs, including infections, occurred in 2.9% of patients and AEs resulted in dose interruptions in 22.1% of patients and treatment discontinuation in 3.8% patients; these rates were comparable to those seen in placebo recipients [16]. There were no deaths or anaphylactic reactions reported in SAkuraStar and SAkuraSky [26, 27].

In the pooled analysis of data from SAkuraStar and SAkuraSky, the rate of infection in patients receiving satralizumab (n = 104) or placebo (n = 74) was 113.04 and 154.85 events/100 patient-years (PY), respectively; satralizumab was not associated with an increased risk of opportunistic infections [33]. Satralizumab treatment should not be initiated in patients with active infections and if a patient develops an active infection during the treatment, satralizumab should be interrupted until the infection is controlled [13, 15–17].

Although the rate of IRRs was higher with satralizumab than with placebo (18.58 vs 8.99 events/100 PY), reported IRRs were mostly of mild to moderate severity and none required treatment discontinuation [16, 33]; IRRs mostly occurred within 24 h after injections [16]. Laboratory abnormalities (e.g. decreases in neutrophil and platelet counts, increases in liver enzymes and total cholesterol) have been reported with satralizumab; however, the majority of the cases were transient and/or resolved without dose interruption [16]. Neutrophil counts and liver enzyme levels should be monitored during satralizumab treatment [13, 15–17].

Longer term, the nature and frequency of AEs with satralizumab during the open-label extension periods in SAkuraStar and SAkuraSky were consistent to those observed during the double-blind periods in these two trials; no new safety signals were identified [34].

As with all therapeutic proteins, satralizumab has a potential for immunogenicity. In SAkuraStar and SAkuraSky, ADAs were detected in 71% and 41% of satralizumab recipients, respectively [16]. The presence of ADAs was not associated with changes in pharmacodynamics, efficacy and safety of satralizumab [16].

2.5 Ongoing Clinical Trials

The open-label extension periods of phase III SAkuraStar (NCT02073279) and SAkuraSky (NCT02028884) trials are currently ongoing.

3 Current Status

Satralizumab received its first approval on 1 June 2020 in Canada for the treatment of NMOSD in adults and children aged ≥ 12 years who are AQP4-IgG seropositive [11]. Subsequently, satralizumab was approved in Japan on 29 June 2020, for the prevention of relapses of NMOSD, including NMO, in adults and children who are AQP4-IgG seropositive [12]. On 13 July 2020, satralizumab received approval in Switzerland for the treatment of NMOSD as monotherapy or as combination therapy with immunosuppressant in adults and adolescents who are AQP4-IgG seropositive [13]. On 17 August 2020, satralizumab was approved in the USA for the treatment of NMOSD in adult patients who are AQP4 antibody positive [14, 15].

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Declarations

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Conflict of interest Young-A Heo is a salaried employee of Adis International Ltd/Springer Nature, is responsible for the article content and declares no relevant conflicts of interest.

Ethics approval, Consent to participate and consent for publication, Availability of data and material, Code availability Not applicable.

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