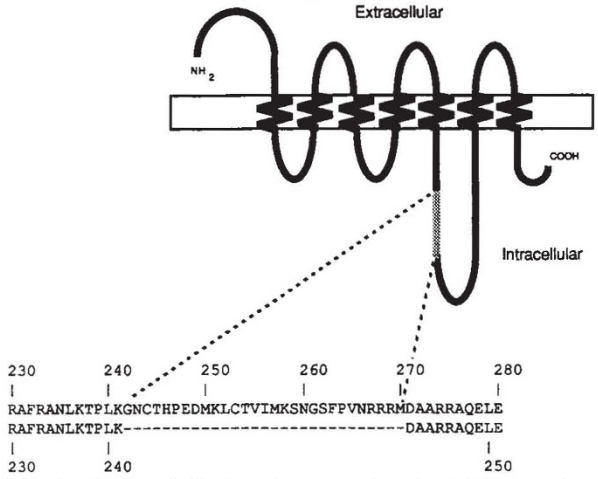
D_2 receptor, a missing exon

SIR—A DNA sequence encoding a dopamine D_2 receptor in rat brain has recently been determined by Bunzow *et al.*[1]. This sequence encodes a protein of 415 amino acids which is found in brain and anterior pituitary and is a member of a family of receptors which are coupled to G proteins. Interest in D_2 receptors stems largely from their involvement in the pathology of neurological and psychiatric disorders such as parkinsonism[2], schizophrenia[2,3] and drug addiction[4].



Schematic diagram of the dopamine D_2 receptor showing the location of an additional exon in the third cytoplasmic loop. The amino-acid sequence of the exon is shown in single letter code above that of the previously published structure[1].

Using a cloning strategy based on the polymerase chain reaction (PCR) and oligonucleotide primers corresponding to consensus sequences of the third and sixth transmembrane segments of this gene family[5], we have obtained several clones from a rat pituitary complementary DNA library encoding the dopamine D_2 receptor. Sequence comparisons showed that these clones were consistent with the published structure except for an additional 29 amino acids in the third cytoplasmic loop inserted after Lysine 241 (see figure). Additional analysis of genomic DNA indicated that this insert is encoded by a separate exon between the flanking sequences reported by Bunzow *et al.*[1]. This region of the receptor is involved in G-protein coupling and varies dramatically between different receptor subtypes[6]. There is also considerable evidence that the dopamine D_2 receptor can couple to either the cyclic AMP or the phosphoinositol second messenger pathways, or to both[2,7]. It is possible that the additional exon confers an alternative G-protein specificity on the D_2 subtype, through differential splicing of messenger RNA. Although receptors of this type are normally encoded by a single exon, the D_2 receptor is encoded by multiple exons[1].

Using oligonucleotide primers corresponding to sequences upstream and downstream from this portion, we found by PCR that the larger form of the dopamine D_2 receptor predominates in both rat pituitary and brain cDNA. In neither tissue did we observe the smaller form. The northern analysis of RNA transcripts in brain and pituitary illustrated by Bunzow *et al.*[1] could therefore represent the larger or both forms of the receptor.

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Exxon Valdez bird toll

SIR—On 24 March 1989, the oil tanker *Exxon Valdez* spilled 260,000 barrels of Alaska North Slope crude oil into Prince William Sound. Oil drifted in a southwesterly direction into the Gulf of Alaska and eventually covered 25,000 km^2 of coastal and offshore waters occupied by more than half a million marine birds. As predicted[1,2], we have witnessed an unprecedented toll of marine birds from oil pollution, which is summarized here.

Dead birds found on beaches and floating in open waters were retrieved by fishermen under contract to Exxon Oil Company, volunteers, and personnel from the US Fish and Wildlife Service (USFWS), Alaska Department of Fish and Game, International Bird Rescue Center and other organizations. Oiled birds were processed and identified (when possible) by USFWS biologists. Data presented here (see Table) include birds retrieved between 25 March and 25 September, 1989.

Preliminary analysis of wildlife surveys conducted before[2,3] and after (USFWS, unpublished data) the spill indicates that about 600,000 marine birds were present in areas contacted by oil. About half that number comprised species with high vulnerability to oil (such as guillemots). More than 35,000 dead birds (89 species) were retrieved from affected areas by 25 September 1989 (see Table). However, the 5,000 deaths — mainly of kittihawks, puffins and shearwaters — in August and September, most are the result of natural causes. Most birds (90 per cent) were killed outside Prince William Sound in the Gulf of Alaska, and the relative composition of oiled birds varied markedly between those areas. In Prince William Sound, proportionally more coastal species were killed. In the Gulf of Alaska, common guillemots were most affected, with few other species comprising more than 2 per cent of the total kill. Species killed in large numbers relative to their local densities included yellow-billed loons, harlequin ducks, pigeon guillemots, marbled murrelets and bald eagles.

Based on the results of corpse-drift experiments conducted elsewhere[4-6] and numbers of birds at risk, we tentatively conclude that the number of birds retrieved represents 10–30 per cent of the actual kill, which was probably between 100,000 and 300,000 birds. The lower estimate is conservative because outside Prince William Sound, logistics, weather and geography prevented a complete and timely search of affected areas. On the other hand, the upper estimate is probably high as it implies an almost total loss of populations at high risk — which we did not observe.

Few, it any, oil spills have had as large an impact on marine bird populations as the *Exxon Valdez* spill. Well-documented[6-8] large oil spills, such as those from the *Torrey Canyon* (7,815 birds retrieved out of an estimated kill of 30,000), *Hamilton Trader* (4,092 out of 10,000), and *Amoco Cadiz* (4,572 out of 20,000), have rarely resulted in retrievals or estimated losses of more than 20,000 birds[9,10]. A few lesser-known incidents of acute oil pollution in the North and Baltic seas have resulted in estimated losses of 30,000–50,000 birds, usually alcids and seaducks[11-13]. On a global scale, the northern Gulf of Alaska harbours enormous populations of marine birds and so the magnitude of losses from the *Exxon Valdez* oil spill was predictable[1,2], and, not surprisingly[1], exceeds any other record of oil-related mortality we can find. If the spill had occurred in summer or autumn, the toll could have